

## Bridget Cornell

---

**From:** Marja Ambler  
**Sent:** Wednesday, April 6, 2022 12:50 PM  
**To:** Georgina Balkwell; Bridget Cornell  
**Cc:** Joanne Marchetta; John Marshall; Katherine Hangeland  
**Subject:** FW: TRPA Hearings Officer Meeting — Agenda Item No V.B — April 7 2022  
**Attachments:** NATURE--Radiofrequency EMF irradiation effects on pre-B lymphocytes.pdf; NATURE--Whole-body exposures to radiofrequency-electromagnetic energy can cause DNA damage via an oxidative mechanism.pdf; Expert-report-Christopher-J-Portier-Murray-v-Motorola-3-1-2021-1.pdf; TPC-CELL TOWER SAFETY\_Disinformation Flyer.pdf; TPC-CELL TOWER SAFETY\_Disinformation Flyer2.pdf; Tahoe Prosperity Center Lies.pdf; Rhetoric and frame analysis of ExxonMobil's climate change communications.pdf

Bridget & Georgina,  
Please add to the public record and respond to Mr. Barney receipt of his comments.  
Thank you

Marja Ambler  
Senior Executive Assistant  
775-589-5287



---

**From:** Gerald O. Barney <gerald.barney@groupoffice.ch>  
**Sent:** Tuesday, April 05, 2022 9:27 PM  
**To:** Joanne Marchetta <jMarchetta@trpa.gov>; John Marshall <jmarshall@trpa.gov>; Marja Ambler <mambler@trpa.gov>; Katherine Hangeland <khangeland@trpa.gov>  
**Subject:** TRPA Hearings Officer Meeting — Agenda Item No V.B — April 7 2022

Dear TRPA Hearing's Officer,

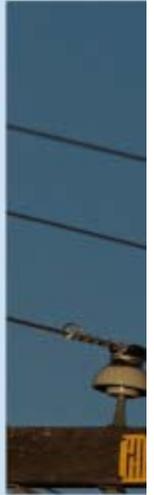
It's official,

**Microwave radiation causes cancer.** The [latest scientific study](#) (*also attached*) published in the most prestigious journal NATURE all but ends any serious debate over whether cell phone radiation frequencies cause non-thermal DNA damage leading to cancer. For the last several decades, the wireless industry used the playbook of the Tobacco and [Fossil Fuels industries](#) to cloud the overwhelming science about the dangers of their technology ([Exxon understood the science about global warming 40 years ago](#), and spent millions to promote misinformation). In a similar vein, the [Tahoe Prosperity Center has spent thousands of dollars of City grant money in promoting disinformation](#) on behalf of Verizon and AT&T locally (their flyers blatantly mischaracterize [findings of the NIH](#), EPA, and the [scientific community](#)). We must stop this dangerous technology

implementation now—just like global warming, we will not be able to go back in time to fix it. This time, we now know what we wish we knew 40 years ago: [their sociopathic game](#).

[Please sign the petition](#).

Click on the booklet below to access 3,300 pages of damning science on the City Council record:



# The Current Science on & Environmental Effects Caused by Cell Tower Radiation

See another collection [here](#).

Please protect us from this known **existential threat**, and [Save Lake Tahoe](#). Don't forget to sign the [petition](#)!

Gerald Barney



OPEN

# Radiofrequency EMF irradiation effects on pre-B lymphocytes undergoing somatic recombination

Elena Ioniță<sup>1,2</sup>, Aurelian Marcu<sup>3</sup>, Mihaela Temelie<sup>1</sup>, Diana Savu<sup>1</sup>, Mihai Șerbănescu<sup>3</sup> & Mihai Ciubotaru<sup>1,2</sup>✉

Intense electromagnetic fields (EMFs) induce DNA double stranded breaks (DSBs) in exposed lymphocytes. We study developing pre-B lymphocytes following V(D)J recombination at their Immunoglobulin light chain loci (*IgL*). Recombination physiologically induces DNA DSBs, and we tested if low doses of EMF irradiation affect this developmental stage. Recombining pre-B cells, were exposed for 48 h to low intensity EMFs (maximal radiative power density flux  $S$  of  $9.5 \mu\text{W}/\text{cm}^2$  and electric field intensity 3 V/m) from waves of frequencies ranging from 720 to 1224 MHz. Irradiated pre-B cells show decreased levels of recombination, reduction which is dependent upon the power dose and most remarkably upon the frequency of the applied EMF. Although 50% recombination reduction cannot be obtained even for an  $S$  of  $9.5 \mu\text{W}/\text{cm}^2$  in cells irradiated at 720 MHz, such an effect is reached in cells exposed to only  $0.45 \mu\text{W}/\text{cm}^2$  power with 950 and 1000 MHz waves. A maximal four-fold recombination reduction was measured in cells exposed to 1000 MHz waves with  $S$  from 0.2 to  $4.5 \mu\text{W}/\text{cm}^2$  displaying normal levels of  $\gamma\text{H2AX}$  phosphorylated histone. Our findings show that developing B cells exposure to low intensity EMFs can affect the levels of production and diversity of their antibodies repertoire.

Somatic or V(D)J recombination is the process that assembles in all jawed vertebrates the gene segments encoding the variable regions of the specific antigen immune receptors (T cell and Immunoglobulin IG) of the lymphoid T and B cells<sup>1</sup>. This process occurs in lymphocyte precursors, is mediated by RAG (recombination activating gene proteins) recombinase a heterotetrameric complex made of a dimer of RAG1 and two monomers of RAG2<sup>2,3</sup>. RAG1 a member of the DDE transposase/Integrase family is the key catalytic component of RAG. RAG binds specifically to recombination signal sequences (RSS) flanking germinal coding V, (D), J gene segments in the variable region at the IG and T cell receptor loci and catalyzes their rearrangement<sup>4</sup>. RAG recombination generates two DNA hairpins at the coding ends and two blunt double stranded DNA cuts at the signal ends. RAG maintains the paired cleaved ends in proximity and allows the ubiquitous set of non-homologous end-joining (NHEJ) DNA repair enzymes (Artemis, ATM, DNAPk, XRCC4, DNA Ligase IV) to resolve the hairpins and join the cleaved ends. For B and T lymphocytes recombination occurs at two stages during their differentiation<sup>5</sup>. We will discuss only the B lineage development in the bone marrow. First two rounds, D to J (in pre-pro stage) followed by V to DJ recombination (in late-pro stage) occur in pro-B cells at their Ig Heavy chain locus (*IgH*). Once *IgH* locus is rearranged, expressed *Ig $\mu$*  together with a surrogate light chain comprising  $\lambda 5$  Vpre B proteins and two  $\text{Ig}\alpha$ ,  $\beta$  signaling subunits assemble the pre-B cell receptor (pre-BCR)<sup>6</sup>, which marks the large pre-B cell stage. Stromal bone marrow cells secreted interleukin IL-7 binds to their receptor (IL-7R), a signal which is transduced as pro-survival and proliferative<sup>7</sup>. First, IL-7R signals through Janus Kinase 3-(JAK-3)<sup>8</sup> phosphorylating and recruiting the signal transducer and activator of transcription 5A and B (STAT5A and B)<sup>9,10</sup> which stimulate transcription of *Ccnd3* encoding Cyclin D3<sup>11</sup> and of the B cell lymphoma 2(*bcl2*) gene<sup>12</sup>. Both Cyclin D3 and the anti-apoptotic BCL2 help pre-B cells through cell cycle G1 checkpoint allowing the replication of their DNA. Secondly, IL-7R signals in large pre-B cells through phosphoinositide 3-kinase (PI3K)<sup>13</sup> and protein Kinase B (AKT) phosphorylating the forkhead box O 1, 3 (FOXO1,3) transcription factors, modification which exports them from nuclei and targets the proteins for degradation<sup>14–16</sup>. FOXO1, 3 activate *e-rag* enhancer and *rag1*, 2 genes transcription<sup>14,17</sup>. In large pre-B cells IL-7R also signals via the nuclear factor kappa light chain

<sup>1</sup>Department of Physics of Life and Environmental Sciences, Horia Hulubei National Institute for R&D in Physics and Nuclear Engineering, 077125 Măgurele, Ilfov, Romania. <sup>2</sup>Department of Immunology, Internal Medicine, Colentina Clinical Hospital, 72202 Bucharest, Romania. <sup>3</sup>Center for Advanced Laser Technologies, National Institute for Laser Plasma and Radiation Physics, 077125 Măgurele, Ilfov, Romania. ✉email: mihai.ciubotaru@nipne.ro

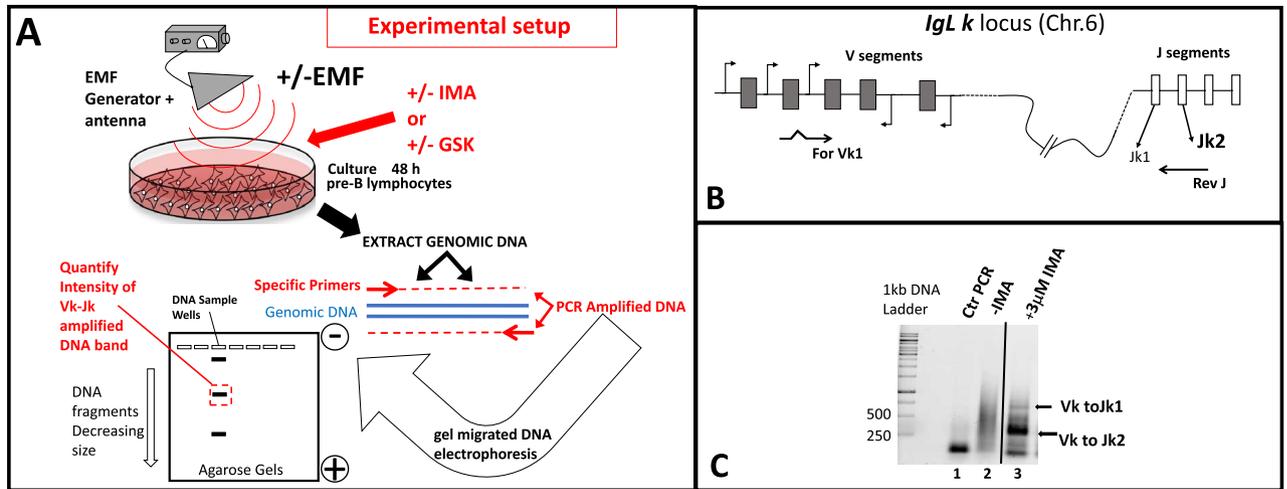
enhancer of activated B cells (NF- $\kappa$ B) stimulated by AKT phosphorylation of IKK $\alpha$  serine 23<sup>18</sup>. NF- $\kappa$ B activates Cyclin D4 kinase which targets FOXO1 for phosphorylation and repression<sup>19</sup>. By inhibiting FOXO1, or phosphorylating STAT-5, IL-7R signals are transiently downregulating RAG proteins in large pre-B cells. After four to five rounds of replication the large pre-B lymphocytes get under the influence of cell surface pre-BCR receptor aggregation and stimulation (in absence of a bonified ligand), a signal which antagonizes that of IL-7R, induces cell cycle arrest and transitions cells towards small pre-B stage<sup>20</sup>. Stimulation of pre-BCR cascades through RAS and extracellular signal- regulated kinase (ERK) upregulating the E2A transcription factor expression. E2A binds both Igk intronic and Igk 3' enhancers making the *Igk* light chain locus accessible for recombination<sup>21</sup>. Another effect of pre-BCR stimulation signals through spleen tyrosine kinase(SYK) and B cell-linker protein(BLNK) which together repress PI3K and AKT but stimulate mitogen activated p38 kinase which activates FOXO1 to express RAG<sup>13,20,22</sup>. Consequently, in small pre-B cells subsequent V to J rearrangements occur at *Ig L k* or  $\lambda$  light chain loci. Upon completion of a successful V to J recombined allele, the cell develops into naïve immature B cell, exposing IgM B cell receptors (BCR).

Interference of V(D)J recombination with other concurrent exogenous factors favoring DNA DSBs, like ionizing or EM irradiation can induce DNA damage which may lead to oncogenic translocations such as those described in acute lymphoblastic leukemia (ALL)<sup>23,24</sup>. Exposure of human blood lymphocytes from healthy donors to strong EMFs (2 h irradiation with sinusoidal pulses at  $4 \times 10^5$  V/m 50 Hz with a carrier wave of 10 Hz<sup>25</sup>) causes DNA DSBs and chromosomal lesions whose severity correlate with the intensity of the applied fields and the duration of exposure. However, less clear results come from studies with irradiated lymphocytes using low intensity, high radiofrequency(RF) EMFs (3 kHz–300 GHz)<sup>26</sup>. Most of these studies have assessed the levels of EMF inflicted DNA single and DSBs on lymphocytes using the microgel electrophoresis technique or 'comet assay', which detects breaks with a sensitivity limit of 50 strand events per diploid cell<sup>27</sup>. RF EM irradiation from cell phones was first studied by Phillips et al. in Molt-4 human lymphoblastoid cells exposed for 2–21 h to fields of 813.5 and 836.5 MHz with specific absorption rate (SAR) (2.4–26  $\mu$ W/g)<sup>28</sup>. Variable degree of DNA damage is reported, mainly induced by high SAR values waves (increased at 24 or 26  $\mu$ W/g and decreased at 2.4 or 2.6  $\mu$ W/g) and longer exposures (21 h versus 2 h). Another study by Mashevich et al.<sup>29</sup> reveals that continuous 72 h exposure of human peripheral blood lymphocytes to EMFs of 830 MHz waves, with SAR ranging from 1.6 to 8.8 W/kg lead to SAR dependent aneuploidy with specific abnormalities on chromosome 17. However, in vitro exposure of human blood lymphocytes for only 2 h to short pulses of 2450 MHz, at an average power of 5 mW/cm<sup>2</sup><sup>30</sup> showed no significant DNA damage as assessed by alkali comet assays. No signs of genotoxicity were found when total human blood leukocytes were in vitro exposed for 24 h either at a continuous or a pulsed-wave 1.9 GHz EMF with a SAR ranging between 0.1 and 10 W/Kg<sup>31</sup>. Absence of significant DNA damage response in human blood lymphocytes was also reported by a study by Stronati et al.<sup>32</sup> in which blood specimens were continuously exposed for 24 h at a Global System Mobile Communication generated EMF of 935 MHz with a SAR of 1 or 2 W/Kg<sup>32</sup>. Similar negative results with respect to EMF induced DNA damage was reported in a study by Hook et al.<sup>33</sup> with cultured Molt-4 human lymphoblasts exposed for 24 h to four types of frequency mobile network modulations around 815–850 MHz with SAR values ranging from 2.4 to 3.2 W/Kg<sup>33</sup>.

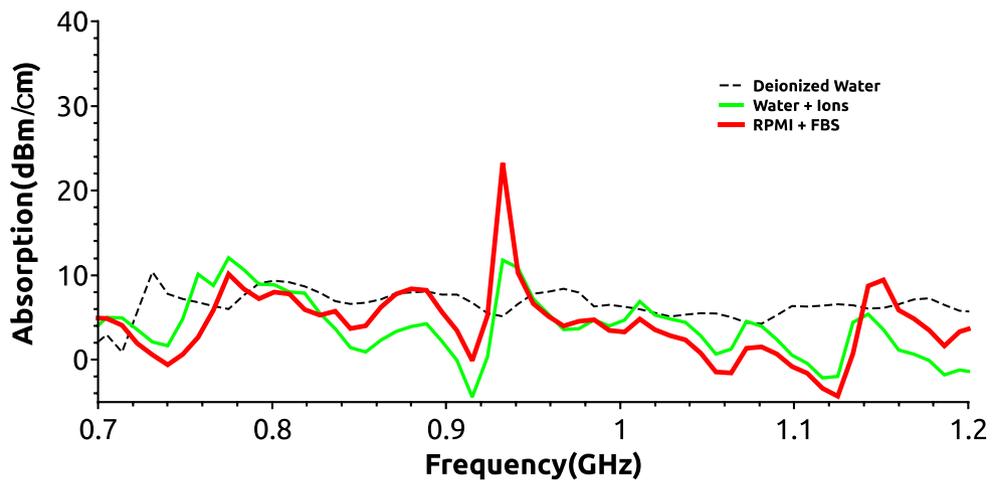
In our work we test the effects of in vitro irradiating V(D)J recombining pre-B cells with very low doses of RF EM waves. RAG stimulation is obtained either mimicking a pre-BCR stimulus with AKT inhibition, or with a stress inducible Abelson (Abl) kinase inhibitor response via STAT5 phosphorylation inhibition. For both stimuli, near 950–1000 MHz RF EMF cell irradiation, in the absence of detectable DNA DSBs, causes a four-fold reduction in recombination levels in exposed pre-Bs versus that assessed in non-irradiated cells.

## Results

**Design and specific experimental conditions used to assess *Ig k* locus rearrangements.** Our study tests how gene recombination levels are influenced by exposure to EMFs with distinct emitted frequencies and power levels (dose–response). In vitro grown vAbl transformed murine pre-B cells stimulated to recombine V(D)J are exposed to a broadband (0.8–3 GHz) emission antenna which broadcasts an EMF from a RF generator (Fig. 1A upper region). For all experiments we standardized our cellular growing conditions to control irradiation parameters (see Supplemental Material section S1 and Fig. 1Sa and b). RAG expression and V(D)J recombination can be induced in vAbl transformed pre-B cells(differentiating them in small pre-B cells) upon stimulation either with an Abl tyrosine kinase inhibitor imatinib(mesylate of imatinib)(IMA)<sup>34,35</sup>(Supplemental Material Fig. 1Sb growing dish wells 1, 2 and 3), or with an AKT inhibitor GSK-690693(GSK)<sup>19</sup>(wells 4, 5 and 6, Fig. 1Sb). Whereas IMA induces RAG by inhibiting vABL-1 tyrosine kinase via a stress-inducible GADD45 $\alpha$  action<sup>17,34,35</sup>, GSK acts as AKT inhibitor, reducing NF- $\kappa$ B and FOXO1 inhibitory phosphorylation (by CDK4) thus, mimicking a physiologic pre-BCR stimulation<sup>19</sup> (see Supplemental material section S2). Time course experiments with RAG induction in vAbl pre-B cells using both drugs show maximal RAG1 levels after 36 h of stimulation (see Supplemental material S2 and Fig. 2Sa and b). Using this finding, after 48 h post drug induction (to allow recombination), all synchronized cultured cells were harvested and their genomic DNA extracted. A previously described two-steps nested PCR (polymerase chain reaction) which assesses the recombination extent taking place at *Igk* kappa light chain locus (chromosome 6, locus schematic and primer positions shown in Fig. 1B), is templated with the equivalent genomic DNA extracted from  $2 \times 10^6$  cells from each tested culture set<sup>36,37</sup>. In the absence of V(D)J recombination (control reactions with no stimulation Fig. 1C lane 2) the variable region V and J segments in germline configuration are too far apart on the chromosome to yield appropriate amplification products. The PCR amplification products obtained only from recombined templates (Fig. 1C lane 3) are separated after electrophoretic migration on 1.5% agarose gels and visualized after fluorescent staining with SYBR green (schematic lower drawing Fig. 1A, and gel scan Fig. 1C). This typical nested PCR reaction reports *k* locus recombination events with two detectable products; the predominant one Vk-Jk2 of 280 bp (95%) and Vk-Jk1 of

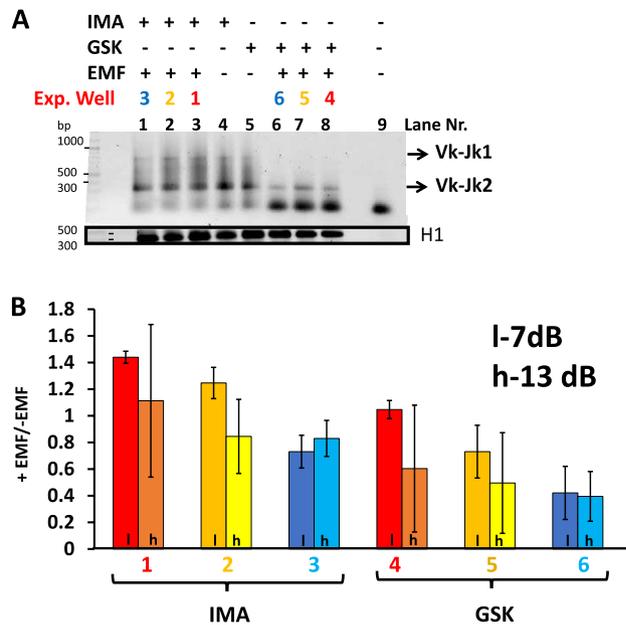


**Figure 1.** (A) Schematic depiction of the flow chart of the experimental design. Murine A-70 vAbl pre-B cells grown with or without exposure to Electromagnetic field influence(EMF), in the absence or presence of RAG stimulation either by Mesylate of Imatinib (IMA) or by GSK-690693(GSK), are harvested and their genomic DNA subjected to a two-steps PCR recombination assay identifying Vk to Jk rearrangements at their *IgL kappa* loci. The electrophoretically separated recombination products (Vk-Jk) are quantified by densitometry to assess the extent of locus rearrangements influenced by EMF. (B) Schematic configuration of *IgL kappa* locus on Mouse chromosome 6, and the positioning of the primers used in the assay. (C) PCR reactions electrophoretically separated in agarose gel stained with Sybr green identify the recombined products (arrows show Vk to Jk1 and Vk to Jk2) in lane 3 versus, control reactions lane 1 without genomic DNA, lane 2 templating genomic DNA from uninduced cells (in germline configuration). Such recombination amplified reactions are then used for densitometry quantifications. The entire gel from which (C) was cropped displaying amplifications (Vk to Jk response) from cells treated with a wide range of increasing IMA concentrations, is shown in Supplemental Material Fig. 3Sa.



**Figure 2.** Absorption spectra measurements of filtered deionized water (dashed thin black line), tap water (green thick line Water + Ions) and RPMI cell culture medium with 10% fetal calf serum (FBS)(red thick line RPMI + FBS) All measurements were done using a Keysight-AGILENT-HP N9935A spectrum analyzer as described in “Methods” section.

700 bp (5%)<sup>36</sup> (Fig. 1C lane 3). Densitometric quantifications of the DNA Vk-Jk2 recombination products allow us to assess the EMF influence on recombination (Fig. 1A lower drawing). A dose-response (recombination) effect obtained with increasing IMA concentrations in 48 h stimulated pre-B cells is shown in supplementary Fig. 3Sa, gel and quantified data from three such experiments shown in Fig. 3Sb histograms. The lowest drug concentration (3 μM for IMA and of 10 μM for GSK,) for which maximal recombination effects are obtained, is used for each drug in our irradiation assays. For linear range quantifications of the image scans each reaction uses genomic DNA template at least at three distinct dilutions from the cellular extraction stock solution and the final result may be reported as an average of the three quantified products values corrected by the histone H1 band intensity of the corresponding sample. In Supplemental material in Fig. 3Sc an 3Sd a set of nested PCR



**Figure 3.** A two steps PCR recombination assay is used to identify Vk to Jk1 or Vk to Jk2 rearrangements from pre-B cells upon RAG induction with Imatinib or GSK. **(A)** A Sybr Green stained 1.5% Agarose TBE gel in which the recombination PCR reactions templated with initial 1:5 dilutions of genomic DNA extracted from each distinct cell treatment lot ( $2 \times 10^6$  cells) are electrophoretically resolved. The cells were either unexposed (gel reaction lanes 4 and 5) or subjected for 48 h to 1 GHz EMF irradiation (lanes 1 to 3 and 6 to 8) with the generator setting at 13 dBm. The color code designating the positions of exposed EMF (exp.Well) wells in the dish is the same with the one used in supplementary Fig. 1Sb. Last lane (9) of the gel, -DNA control reaction. The bottom black box (cropped from a distinct gel) displays Histone H1 PCR reactions templated with the same amount of genomic DNA as the recombination reactions above (control genomic DNA). **(B)** Identified Vk to Jk2 recombined products were quantified from scanned gels corresponding to PCR reactions from cells +/- Irradiation and the calculated ratios of band intensities expressed +EMF/-EMF (irradiated/nonexposed) for each well (color code consistent with that shown in Fig. 1S). The histograms represent the average values of three independent quantified experiments. EMF-Electromagnetic Field, Recombination pharmacological stimuli (Imatinib, IMA) versus (GSK-690693, GSK). H1, histone H1 control reaction PCR reactions. Darker font histograms correspond to lower 7 dBm (l) and brighter to higher 13 dBm (h) generator power settings.

reactions templated with serial dilutions of input genomic DNA from IMA stimulated cells, followed by quantification of the signal are shown to illustrate that the assay responds linearly in its amplified Vk-Jk2 band intensity.

**EM wave absorption spectrum of the cell culture medium.** We measured how the EM waves with frequencies ranging from 700 to 1224 MHz are absorbed by the fetal bovine calf serum supplemented cell culture medium (RPMI + FBS in Fig. 2) in which the pre-B cells are cultured. For comparison only absorption measurements were also performed for deionized water (conductivity  $< 5 \mu\text{S}/\text{cm}$ ), and for ions containing unfiltered tap water samples (see Methods Water + ions, Fig. 2). The measurements were done using a setup in which an emission and a reception horn antenna were spaced 1 m apart with the liquid sample container positioned in the vicinity (1 cm) of the later (see Supplemental material Fig. 4S). The emission antenna was connected to a generator and signals from the receiver antenna were collected and recorded by a standard spectrum analyzer. In Fig. 2 are presented the background corrected absorption spectra per 1 cm width of each liquid sample measured. A well-defined absorption peak is observed at 938 MHz for the RPMI + FBS medium sample which is twice as large as the others measured at this frequency. All samples have similar absorption values for the rest of the tested spectral frequencies. This finding is important since the range of frequencies (720 MHz, 850 MHz, 950 MHz, 1 GHz and 1.2 GHz) to be used for cell irradiation centers our window of exposure between 950 MHz and 1 GHz, proximal to the maximal culture medium absorption peak.

To test how the cell growing medium affects the electric intensity of the exposing fields, EMF electric flux density (D displacement) measurements were made inside the incubator for each mentioned frequency, in the absence or presence of culture medium in the culture plate (Supplemental Material S3 and Fig. 5S). Values greater than one of the  $D_m/D_{\text{air-inc}}$  (1.8–1.95) ratios measured between 750 and 1000 MHz (Supplemental material section S3 and Fig. 5Sc) show in this range, the complete RPMI + FBS cell growing medium selectively potentiates the developed fields.

**EMF irradiation effects on V(D)J recombination in v-Abl pre-B cells.** Murine vAbl pre-B cells were grown under normal conditions or stimulated either with 3  $\mu\text{M}$  IMA or with 10  $\mu\text{M}$  GSK in the presence/absence of an antenna which emits a generator controlled EMF from waves of 720 MHz, 850 MHz, 950 MHz,

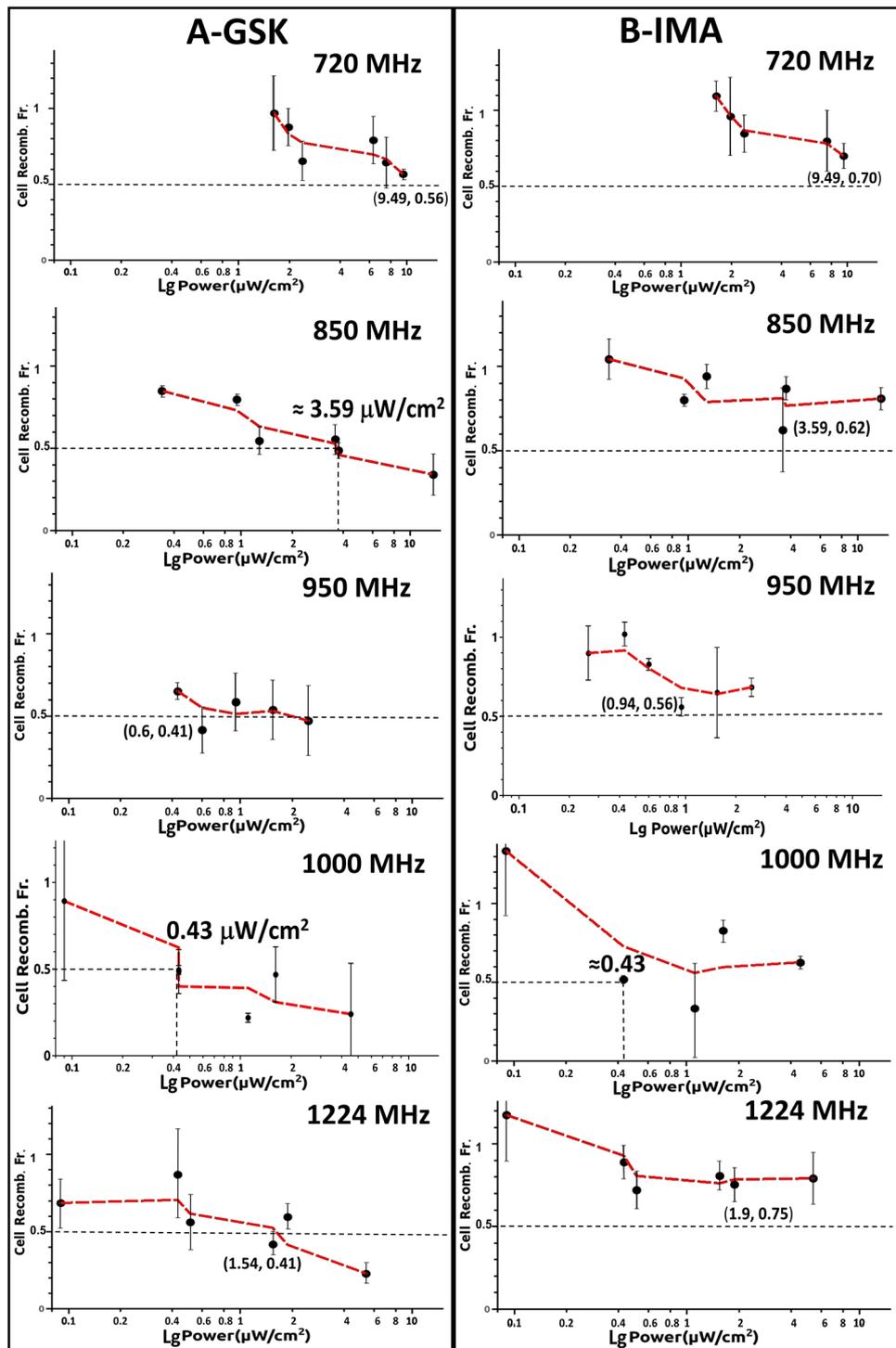
1 GHz, 1.224 GHz each with 7 or 13 dBm output power setting. For all exposures, the antenna was held at 2.4 cm above the composite 6 wells plate as depicted in Supplemental material Fig. 1Sb (lower profile drawing) consistently keeping it in the same location with respect to the incubator walls (Supplemental material S1 and Fig. 1S). Cells were grown +/- EMF constant continuous exposure for 48 h with +/- IMA or +/- GSK. In Fig. 3A is shown a gel with resolved reactions either from nonexposed cells (lanes 4 and 5) or from cells continuously subjected for 48 h to the influence of 1 GHz fields (gel for generator set at 13dBm-h), with both RAG induction treatments (plate Exp. wells IMA 1, 2, 3 and GSK- 4, 5, 6 with color code shown in Supplemental material Fig. 1Sb). Visually one can see, a reduction of Vk to Jk2 recombination products obtained in reactions from irradiated cells versus those from similarly drug induced, non-irradiated cells (see Fig. 3A compare lane 4 non-irradiated to reactions in lanes 1–3 exposed for IMA, and lane 5 unexposed to lanes 6–8 from irradiated GSK stimulated cells). The irradiating effects are most pronounced in the plate wells closest to the actively emitting antenna elements ( $\lambda/2$  for 1 GHz waves use as main element the 15 cm one located near wells 3 and 6 (Supplemental material Fig. 1Sb) hence, recombination reduction for plate Exp. wells 3, 6 > 2, 5 > 1, 4 or correspondingly gel lanes 1, 6 > 2, 7 > 3, 8). The value of the calculated ratios between recombination Vk-Jk2 PCR band intensities obtained from irradiated/non-irradiated(+EMF/-EMF) cells for all tissue culture wells are shown as histograms in Fig. 3B. Values less than one show specific Vk-Jk2 recombination reduction associated with EMF irradiation.

Similar experiments were performed with EMF exposures at 720, 850, 950, 1000 and 1224 MHz (each frequency centers on a different antenna element), generator setting either at 7 dBm or 13 dBm. To display a wider palette of EMF dose exposure values we summed up the data from all of the wells in Fig. 4 which displays cell Vk-Jk2 recombination Fractions(+EMF/-EMF -ordinates), against logarithm of measured irradiating power flux density S values ( $\mu\text{W}/\text{cm}^2$ -abscissas) at each location. Each row of the two panels is for a distinct frequency with panels for each drug located on the same column: Fig. 4A(GSK- left) and B(IMA- right). Consistently all diagrams show power dependent reduction in cellular Vk-Jk2 recombination. S values into the emitting antenna were calculated from antenna recorded voltages, circuit impedance, and antenna constructive elements dimensions and reflect S in the air inside incubator, surrounding the involved culture well. In each panel with dotted black lines we pointed the EMF power dose required to induce a two-fold Vk-Jk2 recombination reduction from that of the non-irradiated lot (+EMF/-EMF 50% reduction shown as 0.5 ratio for Vk-Jk2, Cellular Recombination Fraction). In Fig. 4 when 50% recombination reduction (exposed versus non-irradiated cells) is not reached, the minimal recombination ratios obtained and their inducing S levels are shown in parenthesis. The most remarkable finding of our study is that even for such a small window of frequencies (between 720 and 1224 MHz), the power dose-response effect is dramatically influenced by the frequency of the irradiating EMF. If at 720 MHz one reaches a 0.56/0.70 maximal recombination reduction for 9.49  $\mu\text{W}/\text{cm}^2$  exposure, similar reduction in recombination effects are obtained at 950 MHz and 1 GHz with only 1/15th respectively 1/20th (0.63 or 0.43  $\mu\text{W}/\text{cm}^2$ ) the power used at 720 MHz. The power dose-cell recombination response curves at 950 MHz and 1 GHz EMFs show by far the most accentuated measured effects (for both drugs). For GSK at 1 GHz irradiation, an almost four-fold decrease in V(D)J recombination (from 0.90 to 0.22) is observed over a moderate increase in S exposure from 0.1 to 4.53  $\mu\text{W}/\text{cm}^2$  (see second from the bottom panel in Fig. 4A GSK 1000 MHz). Both curves in Fig. 4 for 1 GHz display an abrupt recombination decrease at a small increase in S (0.25–1  $\mu\text{W}/\text{cm}^2$ ) after which the cellular effect plateaus out over a larger window of higher exposure power S values (1–4.5  $\mu\text{W}/\text{cm}^2$ ). To emphasize the influence of EMF frequency Table 1 shows how recombination fractions (+EMF/-EMF) vary at a relatively constant  $\approx 1.5 \mu\text{W}/\text{cm}^2$  irradiating power flux density S exposure level for all tested EMF frequencies. At this small irradiating power no effect is detectable at 720 MHz, whereas at 950 MHz a two-fold recombination reduction is measured reaching almost three-fold recombination inhibition at 1 GHz.

To circumvent the cellular growing medium polarization effects (which significantly change polarity at 720 MHz and above 1100 MHz, Supplemental material 3S and Fig. 5Sc) or its enhanced wave absorption at 938 MHz (Fig. 2), we intentionally represented in Fig. 5 the recombination fractions for two constant electric field intensity E exposure values, measured inside the medium; one of 0.4 V/m (Fig. 5A.) and the other of 0.6 V/m (Fig. 5B). The approximative intensity of the emitted electric field was calculated in the cell medium from the measured electric flux density ( $D_m$  displacement) values<sup>38</sup> described in the previous section, and averaged for the central plate well. For both E values and both pharmacological stimuli (IMA-red and GSK-blue) the most accentuated plots concavities (maximal irradiation induced recombination reduction effect), correspond to 950–1000 MHz. At both E values represented in Fig. 5 the recombination ratios are unaffected by EMFs at 720 MHz. In contrast, at 1000 MHz, a two-fold reduction is observed for the 0.4 V/m EMF intensity, and a three (IMA) to four-fold (GSK) decrease is measured at the stronger 0.6 V/m field exposure. The electric fields dose exposures -recombination reduction effects in Fig. 5 and those reported for EMFs power dose exposures in Fig. 4 are qualitatively similar. These data strongly suggest that exposure even to very low irradiation doses from specific 900–1000 MHz radiofrequency waves dramatically affect in irradiated pre-B cells the efficiency of V(D)J recombination at their Ig kappa locus.

### Histone H2AX phosphorylation shows no detectable DNA DSB damage cell response in EMF exposed pre-B cells.

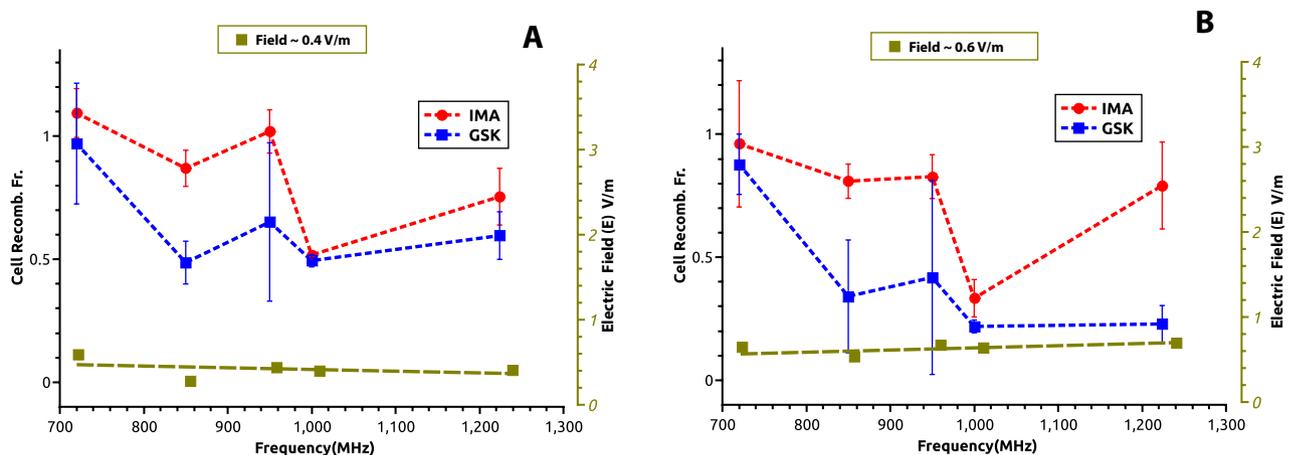
We asked whether the observed EMF irradiating effects on V(D)J recombination are due to DNA damage and presence of unrepaired DSBs. Impairment of DNA integrity can be assessed by the extent with which irradiation induces H2AX histone phosphorylation ( $\gamma\text{H2AX}$ ), a process associated with DNA DSBs and their intranuclear repair. The nuclear  $\gamma\text{H2AX}$  repair foci are the fairest indication that the NHEJ DNA repair machinery acts properly in these cells repairing DSBs caused by any DNA lesion-causing agent<sup>39,40</sup>. We grew cells under similar stimulation (+/- IMA, +/- GSK) and +/- EMF irradiation conditions (7 dBm or 13 dBm generator power settings at 950 MHz) with those described above but instead of extracting DNA, the harvested cells were fixed and doubly stained: (a) with Hoechst 33342 dye (for nuclear total DNA staining in



**Figure 4.** The EMF power dose-cell recombination response curves at 720, 850, 950, 1000 and 1224 MHz for both types of pharmacological agents stimulating RAG expression (A) (GSK-690693, GSK), and (B) (Imatinib, IMA). Cell Recomb. Fr. expresses the ratio values of measured Vk-Jk2 recombination quantified from cells grown in +EMF/-EMF (irradiated/non-exposed) conditions. Bottom abscissa displays logarithm of S power flux density values (Power  $\mu\text{W}/\text{cm}^2$ ) measured around the emitting antenna inside the CO<sub>2</sub> 5 vol%, and 95% water humidity incubator air conditions, expressed as a single range in all panels (logarithmic scale). The black dotted line denote a level of EMF induced two-fold recombination reduction (Cell recomb. Fr. = 0.5), whereas when this level is not reached in the experiment the coordinates of the lowest obtained Cell Recomb. Fr. are given. The red dotted line connecting markers is just a Moving Window Average line which accounts for the average between successive data points displaying the trend of data variation. The error bars represent standard deviation (SD) values from three independent experiments.

Fraction recombination +EMF/-EMF (EMF at $S \approx 1.5 \mu\text{W}/\text{cm}^2$ )		
Frequency (MHz)	Response stimulus	
	GSK	IMA
720	$0.97 \pm 0.2$	$1.09 \pm 0.1$
850	$0.56 \pm 0.1$	$0.8 \pm 0.1$
950	$0.53 \pm 0.2$	$0.65 \pm 0.3$
1000	$0.38 \pm 0.1$	$0.46 \pm 0.3$
1224	$0.41 \pm 0.1$	$0.8 \pm 0.1$

**Table 1.** Lists the measured cell recombination fraction (+EMF/-EMF) at a relative constant power flux density  $S$  value of  $1.5 \mu\text{W}/\text{cm}^2$  for all tested frequencies.

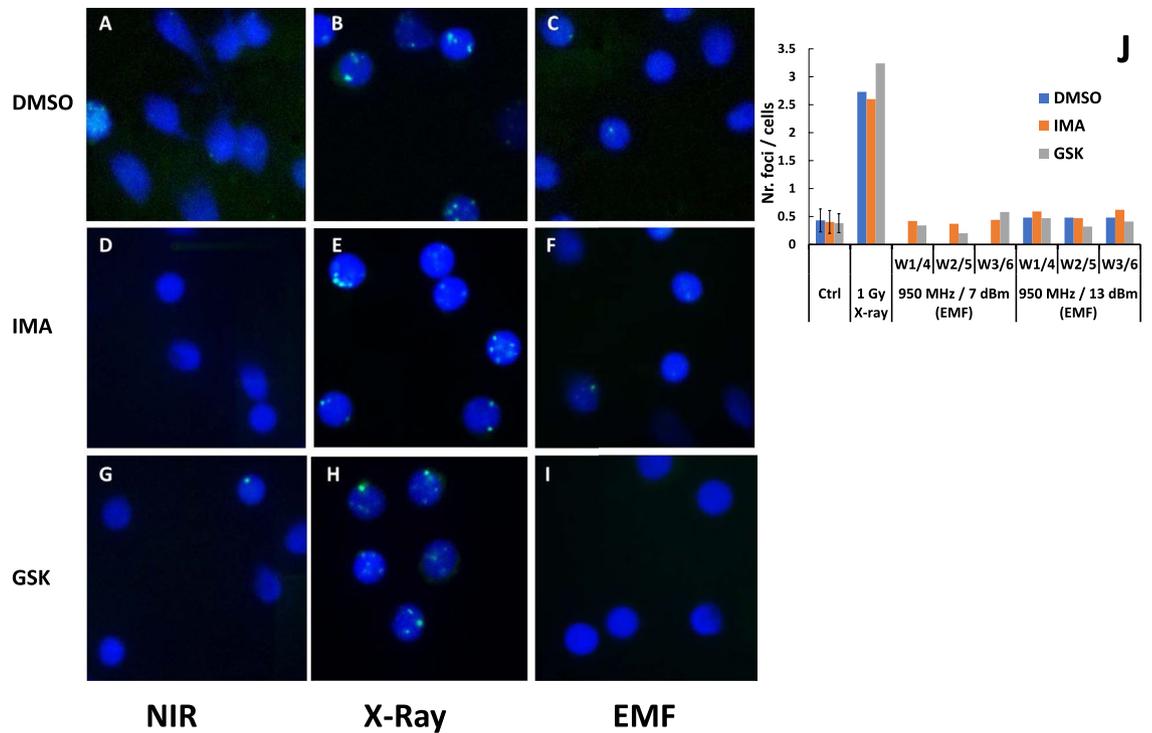


**Figure 5.** Variation of cell recombination fraction (+EMF/-EMF) with the field irradiation frequency shown in each panel for a constant receiver EMF electric intensity field  $E$  calculated in the cell culture medium. (A) EMF electric field intensity  $E$  0.4 V/m, (B) EMF electric field intensity  $E$  0.6 V/m. The pharmacological agents stimulating RAG expression GSK-690693, GSK-blue, and Imatinib, IMA-red. The pale green dotted line shows the relative constant distribution of measured electric field as a function of frequency. The error bars represent standard deviation (SD) values from three independent experiments.

blue) and (b) immunofluorescently with anti  $\gamma\text{H2AX}$  antibodies yielding a Cy2 green fluorescence which identifies DNA DSBs repairing  $\gamma\text{H2AX}$  foci<sup>40</sup>(see Methods). As a DNA DSBs control an extra lot of cells were either noninduced or similarly drug treated but instead of EMF they were subjected to a quick 1 Gy, X ray irradiation dose prior to their harvest. Nine immunofluorescent images are shown in Fig. 6 A-I where blue contours show the cell nuclei and the green dots the DNA DSBs repairing  $\gamma\text{H2AX}$  foci from: cells treated with +/- DMSO solvent control, +/- GSK, +/- IMA, +/- EMF set at 950 MHz, 7dBm exposure and the control lot of cells exposed to 1 Gy X ray. Such foci were also counted and their number reported per cell to a number of total 100 counted cells gathered from more than twenty successive field views for each experimental lot (shown as histograms in Fig. 6J) for both 7 dBm and 13 dBm generator power settings). 1 Gy dose X ray irradiated cells are shown in Fig. 6B control with DMSO solvent, E with IMA, H with GSK and in 6 J the corresponding foci/cell counted histograms. All images (Fig. 6B,E,H) and the quantified histograms from X ray irradiated cells show similar and considerable DNA DSB lesions with consequent accumulation of  $\gamma\text{H2AX}$  repair foci, regardless of the chemical stimulus used. On the contrary, the long 48 h EMF exposure experiments do not show signs of detectable unrepaired DNA DSB damage (Fig. 6C DMSO solvent, F with IMA and I with GSK, and counted foci/cells in Fig. 6J), above the background level of non-irradiated control cultures (Fig. 6A,D,G and ctrl. histograms in Fig. 6J). Exposing for 48 h cells to EMF, regardless of drug treatment, does not seem to inflict significant/ cumulative unrepaired DNA DSB lesions, (unlike those caused even by mild quick irradiation with 1 Gy dose of X rays). Only such DNA injuries could have caused a detectable accumulation of repairing  $\gamma\text{H2AX}$  foci at the time of their harvest. Indirectly, these results suggest that the significant EMF induced reduction in pre-B cells recombination reported in Figs. 3B, 4, 5 and Table 1 is probably not caused by an enhanced level of accumulated unrepaired DNA DSBs.

## Discussion

V(D)J recombination the central process in lymphocyte development physiologically generates DNA DSBs during its course, when cells become susceptible to external sources of DNA damage<sup>5</sup>. Our work tests how pre-B lymphocytes exposure to low dose EMFs of frequencies ranging from 720 MHz to 1.2 GHz, used in utilitarian purpose telecommunication, affects the efficiency of their *Igk* loci rearrangements. First, we established a setup



**Figure 6.** (A–I) Immunofluorescence detection of  $\gamma$ H2AX phosphorylated histone foci in pre-B cells exposed to EMF or X ray irradiation. The  $\gamma$  H2AX foci are shown in bright green— $\gamma$ -H2AX, whereas DNA staining is in blue – nuclei. (A) Control solvent (DMSO) treated cells Nonirradiated (NIR); (B) Control (DMSO) treated 1 Gy X-ray irradiated cells(X-Ray); (C) Control (DMSO)treated EMF exposed (waves at 950 MHz, with emission generator power setting set at 7 dBm-EMF); (D) IMA treated NIR; (E) IMA, X-ray; (F) IMA, EMF; (G) GSK, NIR; (H) GSK, X-ray; (I) GSK, EMF. (J) Number of counted foci per /cell represented as histograms. W1/4 refers to growing plate wells 1 and 4, W2/5 wells 2 and 5 and W3/6 wells 3 and 6 equivalent positions with respect to which cells were EMF irradiated, with generator power setting set at 7 dBm and 13 dBm. GSK is cells treatment with 10  $\mu$ M GSK-690693, IMA their treatment with 3  $\mu$ M Imatinib. ANDOR camera assisted by IQ Live Cell Imaging software and foci analysis with Imaris for Cell Biologists software (both from OXFORD Instruments).

to control the EMF developed inside the cellular growing medium in a typical cell culture incubator. Cultured pre-B cells synchronously recombining V(D)J were EMF exposed during a 48 h window, which starts with RAG expression and ends with the NHEJ DSBs DNA repair<sup>5</sup>. A nested PCR assay is then used to study the cellular EMF irradiation gene effects.

The measured data in Figs. 3B, 4, 5 and Table 1 consistently show, EMFs cause a dose dependent reduction in V(D)J recombination in the irradiated pre-B cells, with similar effects for both RAG inducing stimuli (IMA or GSK) used. The magnitude of effects is tightly determined by the EMF frequency. A two-fold reduction in V<sub>k</sub>-J<sub>k2</sub> recombination at *Igk* locus narrowly can be obtained by an emitted S power value of 9.49  $\mu$ W/cm<sup>2</sup> at 720 MHz (Cell Recomb. Fr. 0.56 for GSK and 0.7 for IMA), whereas this effect may be achieved by a field developing one twentieth of that S dose at 1000 MHz (0.5 Cell Recomb. Fr. for both drugs at 0.43  $\mu$ W/cm<sup>2</sup>) (Fig. 4). The recombination reduction although observed for all EMFs tested, seems to be maximal for 950 and 1000 MHz waves, a small domain where the serum containing cell culture medium displays maximal EMF absorbance (Fig. 2), and augments by its molecular polarization the EMF electric intensity (supplementary Fig. 5Sc). We measured EMF local antenna emissive S values only in the incubator air surrounding the cell culture plate. Despite this limitation we measured and calculated the average irradiated electric field intensity E, inside the culture medium. The maximal effects were measured at 950 and 1000 MHz, where *Igk* recombination levels for an EMF of E 0.4 V/m are only half (Fig. 5A), or for one of 0.6 V/m E a quarter of those reported for same E values at 720 MHz (Fig. 5B). E dose effects parallel the frequency dependency described for the antenna emitted power dose S. However, the cell medium electric properties mentioned above, (increased absorbance and polarization between 900 and 1000 MHz), cannot account for the frequency results shown in Fig. 5 for irradiations at constant electric field E values. Besides such intrinsic medium properties there must be also a major EMF frequency direct influence on the cellular components linked to recombination.

Various wireless network service providers use for mobile phone communication frequencies ranging between 700 and 2100 MHz. At 1 cm distance, during outgoing calls the measured emitted field E intensities vary with  $\pm$  5–15% from the 41.25 V/m (recommended ICNIRP value) with cell phone models, whereas their maximum output recorded power levels for a GSM1800 net varies between 0.25 and 1 W<sup>41,42</sup>.

We assessed if the low dose 48 h EMF irradiations cause DNA DSBs and detectable  $\gamma$ H2AX repair foci in exposed cells. From the levels of detected  $\gamma$ H2AX repair foci of the EMF irradiated pre-B cells we could not reveal in exposed cells above background DNA DSBs repair activity (Fig. 6 compare panel A with C, G with I, and histograms in Fig. 6j). Using chromatin immunoprecipitation (ChIP) Savic et al.<sup>43</sup> show considerable  $\gamma$ H2AX accumulation near Jk5 in IgK locus after 24 h post STI571 (Imatinib) treatment of pre-Bs, but a dramatic more than two-fold decrease in  $\gamma$ H2AX detection as cells were kept from 24 to 48 h post STI571 treatment<sup>43</sup>. We could not detect above background  $\gamma$ H2AX foci levels in IMA or GSK treated cells after 48 h culture growth. This could be due either to a considerable post RAG DSBs repair recovery, or to a reduced sensitivity of our immunofluorescence assay (less sensitive than ChIP in detecting  $\gamma$ H2AX). The onset of DNA DSBs either prior to or during pre-B cells maturation inhibits *rag1*, 2 transcription<sup>44</sup> and reduces the levels of *Igk* locus rearrangement events<sup>45</sup>. These cellular stress effects are caused by ataxia teleangiectasia mutated (ATM) kinase either via NF- $\kappa$ B, FOXO1 signaling<sup>44,46</sup> or via GADD45a inhibition<sup>45</sup>, both pathways directly targeting *rag* genes transcription levels. If very few EMF induced DSBs (below those detectable by  $\gamma$ H2AX foci assay), or breaks already repaired before our cell harvests could have reduced RAG expression in our experiments (via ATM kinase) this could explain our observed reduced recombination effects. We used in our experiments two RAG induction stimuli, IMA sensitive to ATM kinase via GADD45a inhibition<sup>44–46</sup> and the second GSK690693 AKT-inhibitor insensitive to this signaling pathway<sup>19</sup>. If very few EMF induced DNA DSBs would have reduced RAG expression prior to, or during drug action, one would have expected experiments to show a more accentuated recombination reduction for IMA than that obtained for GSK treatment. Instead, the experimental data in Figs. 3B, 4, 5 and Table 1 show for both drugs very similar EMF induced reduction of *Igk* loci rearrangement levels (if not even slightly more pronounced reduction for GSK). IMA although a more potent RAG inducer than GSK has the disadvantage that post recombination blocks cells in Go phase preventing further their division<sup>36,47–49</sup>. On the contrary, the AKT inhibitor GSK-690693 not only is a weaker RAG induction stimulus (closer to the one physiologically occurring in small pre-B cells)<sup>50,51</sup> but also enables cells to divide prior to and after *Igk* loci rearrangements and protect their progress to the next stage of development<sup>19</sup>. Because our PCR assay intentionally uses the amount of templating genomic DNA from the same number of 2 millions harvested cells, replication would have “diminished the EMF recombination reduction” in GSK treated cells in contrast to those incubated with IMA (the later, on the contrary, “freezes” the EMF effect on BCL2 maintained survivors). As pointed earlier, in treated cells, both drugs show very similar EMF induced reduction of rearrangements in treated cells. Although we cannot fully refute that the observed EMF recombination effects may have been caused in irradiated pre-B cells by undetectable DNA DSBs via ATM, the line of evidence gathered from our experiments in Figs. 4, 5, 6 and the arguments presented above for the comparative IMA/GSK treatments make this mechanism a less likely candidate for their account.

Indirectly our work addresses the longstanding question of how innocuous low dose EMF irradiation from our telecommunication devices may be and whether it may affect the immunity of our organisms. It remains only to our speculation to extend the observed recombination effects induced by small EMFs from an in vitro culture system to the in vivo situation on the ability of irradiated B cells to elicit an unaltered antibody response to antigen challenge.

## Methods

**Materials.** DNA oligonucleotides were purchased from Life Technologies and IDT DNA: Vk degenerate primer 5' GCTGCAGSTTCAGTGGCAGTGGCAGTGGRTCWGGRAC 3' where S is G or C, R is A or G, W is T or A, Jk2-1 primer 5' CAAAACCCTCCCTAGGTAGACAATTATCCCTC 3' and Jk2-2 primer 5' GGACAG TTTTCCCTCCTTAACACCTGATCTG 3'. For Histone H1 gene control amplifications the following primers were used: H1fw 5' GGCTGCTATCCAGGCAGAGAAGAACCG 3', H1rv: 5' GCTTTGGAGGCGCCTTCT TGGGCTTG 3'.

*Murine pre B cells* transformed with Abelson virus (v-Abl preB, A70 line, that harbor a E $\mu$ -Bcl2 transgene) were a kind gift from Barry Sleckman Duke University<sup>47</sup>. The cells were maintained in RPMI 1640 medium, supplemented with 10% FBS (both from GIBCO), 50  $\mu$ M 2-mercaptoethanol and induced at  $0.5 \times 10^6$  cells/ml density either with 3  $\mu$ M Imatinib Mesylate (IMA) (SIGMA-ALDRICH) or with 10  $\mu$ M GSK-690693 (GSK) (GLAXOSMITHKLINE, SELLECK-chem) in solutions with 0.1% DMSO. After 48 h the cells were collected and analyzed using the nested PCR described below.

*Pre-B Cells irradiation* was performed with a 1 Hz–1.224 GHz, 13 dBm radiofrequency generator (Hameg Instruments 1 Hz–1.2 GHz programmable synthesizer HM8134-3, used throughout our study as emission generator) using a broadband irradiating 800 MHz–3 GHz LTE ATK-LOG ALP logarithmic antenna, in a regular CO<sub>2</sub> incubator (SANYO Electric Co. MCO-17AIC), with CO<sub>2</sub> 5 vol. %, and 95% purified water humidity. Cells were grown at 37 °C in 5 ml medium in standard six flat bottom wells (16.8 ml capacity) polystyrene lidded plates (Corning Costar), which were always positioned in the same place with respect to the incubator walls (in the center of the incubator, see Supplemental material Fig. 1Sa) and the emission antenna (antenna central guiding label positioned midway between wells 3 and 6 at 2.4 cm above the mid plane of the plate, see supplementary Fig. 1S). Two parallel sets of experiments were performed with wells 1, 2, 3 containing cells stimulated with 3  $\mu$ M IMA, whereas wells 4, 5 and 6 cells were stimulated with 10  $\mu$ M GSK (Fig. 1SB).

**Two steps nested PCR reactions for K locus recombination.** Template DNA was prepared for PCR using a modified technique developed by Schlissel<sup>37</sup>. Pre-B A-70 v-Abl cells were harvested after 48 h incubation with IMA<sup>36,47</sup>, GSK<sup>19</sup> or unstimulated. Cultured cells ( $2 \times 10^6$ –2 millions) were pelleted for 15 s in a microfuge, washed once in PBS (phosphate saline buffer pH 7.2), resuspended in 200  $\mu$ l PCR lysis buffer (10 mM Tris pH 8.4, 2.5 mM MgCl<sub>2</sub>, 50 mM KCl, 200  $\mu$ g/ml gelatin, 0.45% NP40, 0.45% Tween-20 (CALBIOCHEM), and 60  $\mu$ g/ml Proteinase K (Boehringer), and incubated at 56 °C for 3 h followed by 15 min at 95 °C. Dilution of templates

was done with PCR lysis buffer without Proteinase K. Two successive PCR amplifications were done in a final volume of 50  $\mu$ l containing 2 to 5  $\mu$ l template DNA, 10 mM Tris-HCl (pH 8.4; at room temperature), 50 mM KCl, 2.5 mM MgCl<sub>2</sub>, 200  $\mu$ g/ml gelatin, 0.2 mM of all four dNTPs (all from ThermoFisher scientific), each oligonucleotide primer at 0.4  $\mu$ M (20 pmol each primer per reaction), and 1 U TAQ DNA polymerase (GoTaq PROMEGA) in nested reactions. First step PCR reactions for 25 cycles use Vk, and Jk2-1 primers. In the second step various dilutions (from 4  $\mu$ l 1:100 dilution of first PCR to 0.5  $\mu$ l of the first undiluted PCR) are individually used to template the second PCR reactions to which Vk and Jk2-2 primers are added and an additional round of 30 cycles amplification is performed. Cycling steps were: an initial 1 min denaturation at 94 °C, then repeated cycles each, 30 s at 94 °C, 0.5 min annealing at 50 °C, and 1.5 min polymerization at 72 °C. A final additional 5 min extension step was performed at 72 °C<sup>36,37</sup>. PCR products were resolved on 1.5% agarose gel, stained either with ethidium bromide or Sybr Green (THERMOFISHER scientific) and visualized using the PharosFX system (BIORAD). The bands intensities were quantified using QuantityOne software.

**Kappa locus amplification products analysis.** Each Vk-Jk2 product band density of the gel scan image is quantified and the ratio between the densitometry value of the PCR product band detected from cells grown in the presence of EMF and the corresponding one without field exposure (EMF+/EMF-, Cell Recomb. Fr., Figs. 3, 4, 5) reports the changes in V(D)J recombination occurred upon each cell treatment (IMA/GSK). To normalize for DNA extraction levels we performed similar PCR amplifications from the same amount of template DNA using a pair of primers H1fw and H1rv to specifically detect the histone H1 gene.

**$\gamma$ H2AX foci analysis for irradiation induced DNA damage cellular response.** Cells were grown under similar conditions with those described above for recombination assays. Additionally, a DNA DSBs control cell lot either uninduced or one for each RAG stimulus (IMA or GSK) was exposed to a quick 20 min X ray cumulative dose exposure of 1 Gray (X-ray irradiation with a slow rate 50-milligray /min with a Mevatron Primus 2D, 6MV, SIEMENS instrument) prior to their harvest. The samples were irradiated at 100 cm distance from the source axis, the field size being of 30  $\times$  30 cm. The dosimetry was performed using a water phantom (1 cm water depth). Symmetry and homogeneity were checked, the dose proved to be homogenous throughout the sample in the used plates. For all treatments, twenty minutes after harvest, instead of extracting DNA, the cells from each individual culture type were separately spread onto clean designated slide sets using a Cytospin Centrifuge. The cells were then fixed with paraformaldehyde, permeabilized with Triton X and then doubly stained with: (a) Hoechst 33342 dye (THERMO SCIENTIFIC) (for their nuclei-DNA total staining in blue) and (b) immunofluorescently with primary unlabeled anti  $\gamma$ H2AX antibodies of mouse antigen specificity complemented with secondary Cy2 labeled anti primary source antibodies (rat anti mouse IgG Cy2 detection antibodies-green)(both from SIGMA ALDRICH); to identify in green the DSB repairing  $\gamma$ H2AX foci<sup>40</sup>. The slides were examined with a fluorescence microscope (OLYMPUS BX60) with adequate filter for the fluorophores, and images of the nuclei and  $\gamma$ H2AX foci recorded with a camera connected to the microscope. The images were analyzed using specific analysis software to quantify the number of foci per each cell treatment type, and morphologically to indicate their level of dispersion or nuclear positioning (see Fig. 6).

*Western blot analysis* for endogenous RAG time course induction in pre-B cells (Supplemental material Fig. 2S) following IMA/GSK treatment was performed as previously described in our work using anti RAG1 and anti RAG2 mouse monoclonal antibodies (gift from Dr. David G. Schatz, Yale University), and control sample purified murine core RAG1(384–1040) and coreRAG2 (1–387) fused to Maltose binding protein (MBP-40kD) which were transiently expressed in co-transfected HEK293T cells<sup>52</sup> (source ATCC CRL-3216).

*Absorption spectra measurements* were made using two identical broadband (0.8–16 GHz) horn antennas facing each-other and placed at 1 m distance. The measurement subjected sample was placed in close proximity (1 cm) of the receiver whereas the emission antenna (supplementary Fig. 4S a and b), was coupled to the generator. The receiver antenna was connected to a commercial Spectrum Analyzer (Keysight-AGILENT-HP N9935A, 0.1- 9 GHz) on which the received signals were recorded and analyzed. The shown absorption spectra in Fig. 2 were obtained after subtraction of the background spectra with no liquid sample placed in the container in front of the receiver antenna. The deionized water used for measurement has the conductivity < 5  $\mu$ S/cm, whereas the used unfiltered tap water with ions has the following characteristic measured chemical parameters per liter (l) pH 6.5–9.5, Conductivity < 800  $\mu$ S/cm, ammonia < 0.5 mg/l, free residual Chlorine < 0.5 mg/l, Fe < 200  $\mu$ g/l, Mn < 50  $\mu$ g/l, Al < 200  $\mu$ g/l, nitrites < 0.5 mg/l, nitrates < 50 mg/l, Borate salts 1 mg/l, Chlorides 250 mg/l, Sulphates 250 mg/l, 65 mg/l calcium carbonate, Hardness < 5degrees (dGH).

Received: 29 November 2020; Accepted: 31 May 2021

Published online: 16 June 2021

## References

1. Tonegawa, S. Somatic generation of antibody diversity. *Nature* **302**, 575–581 (1983).
2. Kim, M. S., Lapkouski, M., Yang, W. & Gellert, M. Crystal structure of the V(D)J recombinase RAG1-RAG2. *Nature* **518**, 507–511. <https://doi.org/10.1038/nature14174> (2015).
3. Sadofsky, M. J. The RAG proteins in V(D)J recombination: more than just a nuclease. *Nucleic Acids Res.* **29**, 1399–1409 (2001).
4. Schatz, D. G. & Ji, Y. Recombination centres and the orchestration of V(D)J recombination. *Nat. Rev. Immunol.* **11**, 251–263. <https://doi.org/10.1038/nri2941> (2011).
5. Schatz, D. G. & Swanson, P. C. V(D)J recombination: mechanisms of initiation. *Annu. Rev. Genet.* **45**, 167–202. <https://doi.org/10.1146/annurev-genet-110410-132552> (2011).

6. Herzog, S., Reth, M. & Jumaa, H. Regulation of B-cell proliferation and differentiation by pre-B-cell receptor signalling. *Nat. Rev. Immunol.* **9**, 195–205. <https://doi.org/10.1038/nri2491> (2009).
7. Peschon, J. J. *et al.* Early lymphocyte expansion is severely impaired in interleukin 7 receptor-deficient mice. *J. Exp. Med.* **180**, 1955–1960. <https://doi.org/10.1084/jem.180.5.1955> (1994).
8. O’Shea, J. J. & Plenge, R. JAK and STAT signaling molecules in immunoregulation and immune-mediated disease. *Immunity* **36**, 542–550. <https://doi.org/10.1016/j.immuni.2012.03.014> (2012).
9. Goetz, C. A., Harmon, I. R., O’Neil, J. J., Burchill, M. A. & Farrar, M. A. STAT5 activation underlies IL7 receptor-dependent B cell development. *J. Immunol.* **172**, 4770–4778. <https://doi.org/10.4049/jimmunol.172.8.4770> (2004).
10. Yao, Z. *et al.* Stat5a/b are essential for normal lymphoid development and differentiation. *Proc. Natl. Acad. Sci. U. S. A.* **103**, 1000–1005. <https://doi.org/10.1073/pnas.0507350103> (2006).
11. Cooper, A. B. *et al.* A unique function for cyclin D3 in early B cell development. *Nat. Immunol.* **7**, 489–497. <https://doi.org/10.1038/ni1324> (2006).
12. Jiang, Q. *et al.* Distinct regions of the interleukin-7 receptor regulate different Bcl2 family members. *Mol. Cell Biol.* **24**, 6501–6513. <https://doi.org/10.1128/MCB.24.14.6501-6513.2004> (2004).
13. Ramadani, F. *et al.* The PI3K isoforms p110alpha and p110delta are essential for pre-B cell receptor signaling and B cell development. *Sci Signal* **3**, ra60. <https://doi.org/10.1126/scisignal.2001104> (2010).
14. Amin, R. H. & Schlissel, M. S. Foxo1 directly regulates the transcription of recombination-activating genes during B cell development. *Nat. Immunol.* **9**, 613–622. <https://doi.org/10.1038/ni.1612> (2008).
15. Dengler, H. S. *et al.* Distinct functions for the transcription factor Foxo1 at various stages of B cell differentiation. *Nat. Immunol.* **9**, 1388–1398. <https://doi.org/10.1038/ni.1667> (2008).
16. Eijkelenboom, A. & Burgering, B. M. FOXOs: signalling integrators for homeostasis maintenance. *Nat. Rev. Mol. Cell Biol.* **14**, 83–97. <https://doi.org/10.1038/nrm3507> (2013).
17. Kuo, T. C. & Schlissel, M. S. Mechanisms controlling expression of the RAG locus during lymphocyte development. *Curr. Opin. Immunol.* **21**, 173–178. <https://doi.org/10.1016/j.coi.2009.03.008> (2009).
18. Ozes, O. N. *et al.* NF-kappaB activation by tumour necrosis factor requires the Akt serine-threonine kinase. *Nature* **401**, 82–85. <https://doi.org/10.1038/43466> (1999).
19. Ochodnicka-Mackovicova, K. *et al.* NF-kappaB and AKT signaling prevent DNA damage in transformed pre-B cells by suppressing RAG1/2 expression and activity. *Blood* **126**, 1324–1335. <https://doi.org/10.1182/blood-2015-01-621623> (2015).
20. Ochiai, K. *et al.* A self-reinforcing regulatory network triggered by limiting IL-7 activates pre-BCR signaling and differentiation. *Nat. Immunol.* **13**, 300–307. <https://doi.org/10.1038/ni.2210> (2012).
21. Inlay, M. A., Tian, H., Lin, T. & Xu, Y. Important roles for E protein binding sites within the immunoglobulin kappa chain intronic enhancer in activating V kappa J kappa rearrangement. *J. Exp. Med.* **200**, 1205–1211. <https://doi.org/10.1084/jem.20041135> (2004).
22. Herzog, S. *et al.* SLP-65 regulates immunoglobulin light chain gene recombination through the PI(3)K-PKB-Foxo pathway. *Nat. Immunol.* **9**, 623–631. <https://doi.org/10.1038/ni.1616> (2008).
23. Lieber, M. R., Yu, K. & Raghavan, S. C. Roles of nonhomologous DNA end joining, V(D)J recombination, and class switch recombination in chromosomal translocations. *DNA Repair (Amst)* **5**, 1234–1245. <https://doi.org/10.1016/j.dnarep.2006.05.013> (2006).
24. Marculescu, R., Le, T., Simon, P., Jaeger, U. & Nadel, B. V(D)J-mediated translocations in lymphoid neoplasms: a functional assessment of genomic instability by cryptic sites. *J. Exp. Med.* **195**, 85–98 (2002).
25. Delimaris, J., Tsilimigaki, S., Messini-Nicolaki, N., Ziros, E. & Piperakis, S. M. Effects of pulsed electric fields on DNA of human lymphocytes. *Cell Biol. Toxicol.* **22**, 409–415. <https://doi.org/10.1007/s10565-006-0105-1> (2006).
26. Phillips, J. L., Singh, N. P. & Lai, H. Electromagnetic fields and DNA damage. *Pathophysiology* **16**, 79–88. <https://doi.org/10.1016/j.pathophys.2008.11.005> (2009).
27. Olive, P. L. & Banath, J. P. The comet assay: a method to measure DNA damage in individual cells. *Nat. Protoc.* **1**, 23–29. <https://doi.org/10.1038/nprot.2006.5> (2006).
28. Phillips, J. L. *et al.* DNA damage in Molt-4 T-lymphoblastoid cells exposed to cellular telephone radiofrequency fields in vitro. *Bioelectrochem. Bioenerg.* **45**, 103–110. [https://doi.org/10.1016/s0302-4598\(98\)00074-9](https://doi.org/10.1016/s0302-4598(98)00074-9) (1998).
29. Mashevich, M. *et al.* Exposure of human peripheral blood lymphocytes to electromagnetic fields associated with cellular phones leads to chromosomal instability. *Bioelectromagnetics* **24**, 82–90. <https://doi.org/10.1002/bem.10086> (2003).
30. Leal, B. Z., Szilagyi, M., Prihoda, T. J. & Meltz, M. L. Primary DNA damage in human blood lymphocytes exposed in vitro to 2450 MHz radiofrequency radiation. *Radiat. Res.* **153**, 479–486. [https://doi.org/10.1667/0033-7587\(2000\)153\[0479:pddihb\]2.0.co;2](https://doi.org/10.1667/0033-7587(2000)153[0479:pddihb]2.0.co;2) (2000).
31. McNamee, J. P. *et al.* No evidence for genotoxic effects from 24 h exposure of human leukocytes to 1.9 GHz radiofrequency fields. *Radiat. Res.* **159**, 693–697. [https://doi.org/10.1667/0033-7587\(2003\)159\[0693:nfgef\]2.0.co;2](https://doi.org/10.1667/0033-7587(2003)159[0693:nfgef]2.0.co;2) (2003).
32. Stronati, L. *et al.* 935 MHz cellular phone radiation. An in vitro study of genotoxicity in human lymphocytes. *Int. J. Radiat. Biol.* **82**, 339–346. <https://doi.org/10.1080/09553000600739173> (2006).
33. Hook, G. J. *et al.* Measurement of DNA damage and apoptosis in Molt-4 cells after in vitro exposure to radiofrequency radiation. *Radiat. Res.* **161**, 193–200. <https://doi.org/10.1667/rr3127> (2004).
34. Muljo, S. A. & Schlissel, M. S. A small molecule Abl kinase inhibitor induces differentiation of Abelson virus-transformed pre-B cell lines. *Nat. Immunol.* **4**, 31–37. <https://doi.org/10.1038/ni870> (2003).
35. Wilson, M. K., McWhirter, S. M., Amin, R. H., Huang, D. & Schlissel, M. S. Abelson virus transformation prevents TRAIL expression by inhibiting FoxO3a and NF-kappaB. *Mol. Cells* **29**, 333–341. <https://doi.org/10.1007/s10059-010-0029-8> (2010).
36. Carmona, L. M., Fugmann, S. D. & Schatz, D. G. Collaboration of RAG2 with RAG1-like proteins during the evolution of V(D)J recombination. *Genes Dev.* **30**, 909–917. <https://doi.org/10.1101/gad.278432.116> (2016).
37. Schlissel, M. S. & Baltimore, D. Activation of immunoglobulin kappa gene rearrangement correlates with induction of germline kappa gene transcription. *Cell* **58**, 1001–1007. [https://doi.org/10.1016/0092-8674\(89\)90951-3](https://doi.org/10.1016/0092-8674(89)90951-3) (1989).
38. Gregson, S. M., McCormick, J. & Parini, C. *Principles of Planar Near-Field Antenna Measurements* 35–61 (The Institution of Engineering and Technology, London, 2007).
39. Helmink, B. A. *et al.* H2AX prevents CtIP-mediated DNA end resection and aberrant repair in G1-phase lymphocytes. *Nature* **469**, 245–249. <https://doi.org/10.1038/nature09585> (2011).
40. Huang, X. & Darzynkiewicz, Z. Cytometric assessment of histone H2AX phosphorylation: a reporter of DNA damage. *Methods Mol Biol* **314**, 73–80. <https://doi.org/10.1385/1-59259-973-7:073> (2006).
41. I. C. o. N.-I. R. P. Principles for non-ionizing radiation protection. *Health Phys.* **118**, 477–482. <https://doi.org/10.1097/hp.0000000000001252> (2020).
42. Isabona, J. & Srivastava, V. M. (2017) Cellular mobile phone—a technical assessment on electromagnetic radiation intensity on human safety. *IEEE*, 271–274. Doi: <https://doi.org/10.1109/NIGERCON.2017.8281899>
43. Savic, V. *et al.* Formation of dynamic gamma-H2AX domains along broken DNA strands is distinctly regulated by ATM and MDC1 and dependent upon H2AX densities in chromatin. *Mol. Cell* **34**, 298–310. <https://doi.org/10.1016/j.molcel.2009.04.012> (2009).
44. Fisher, M. R., Rivera-Reyes, A., Bloch, N. B., Schatz, D. G. & Bassing, C. H. Immature lymphocytes inhibit Rag1 and Rag2 transcription and V(D)J recombination in response to DNA double-strand breaks. *J. Immunol.* **198**, 2943–2956. <https://doi.org/10.4049/jimmunol.1601639> (2017).

45. Steinel, N. C. *et al.* The ataxia telangiectasia mutated kinase controls Iggkappa allelic exclusion by inhibiting secondary V kappa-to-J kappa rearrangements. *J. Exp. Med.* **210**, 233–239. <https://doi.org/10.1084/jem.20121605> (2013).
46. Ochodnicka-Mackovicova, K. *et al.* The DNA damage response regulates RAG1/2 expression in pre-B cells through ATM-FOXO1 signaling. *J. Immunol.* **197**, 2918–2929. <https://doi.org/10.4049/jimmunol.1501989> (2016).
47. Bredemeyer, A. L. *et al.* ATM stabilizes DNA double-strand-break complexes during V(D)J recombination. *Nature* **442**, 466–470. <https://doi.org/10.1038/nature04866> (2006).
48. Hantschel, O., Rix, U. & Superti-Furga, G. Target spectrum of the BCR-ABL inhibitors imatinib, nilotinib and dasatinib. *Leuk. Lymphoma* **49**, 615–619. <https://doi.org/10.1080/10428190801896103> (2008).
49. Marinelli Busilacchi, E. *et al.* Immunomodulatory effects of tyrosine kinase inhibitor in vitro and in vivo study. *Biol. Blood Marrow Transplant.* **24**, 267–275. <https://doi.org/10.1016/j.bbmt.2017.10.039> (2018).
50. Borghesi, L. *et al.* B lineage-specific regulation of V(D)J recombinase activity is established in common lymphoid progenitors. *J. Exp. Med.* **199**, 491–502. <https://doi.org/10.1084/jem.20031800> (2004).
51. Grawunder, U. *et al.* Down-regulation of RAG1 and RAG2 gene expression in preB cells after functional immunoglobulin heavy chain rearrangement. *Immunity* **3**, 601–608. [https://doi.org/10.1016/1074-7613\(95\)90131-0](https://doi.org/10.1016/1074-7613(95)90131-0) (1995).
52. Ciubotaru, M. *et al.* The architecture of the 12RSS in V(D)J recombination signal and synaptic complexes. *Nucleic Acids Res.* **43**, 917–931. <https://doi.org/10.1093/nar/gku1348> (2015).

## Acknowledgements

This work was supported by the following projects: PN-III-P4-ID-PCE-2016-0502 Acronym V(D)JMYC contract 178/2017, 5/5.1/ELI-RO, RDI 2016 acronym "BIOSAFE" Contract Nr. 17/2016 and PN-III-P1-1.2-PCCDI-2017 acronym "ONCORAD" Contract Nr. 64/2018. We would also wish to acknowledge support from COMTEST srl. company from Dr. ing. Radu Mateescu in measuring probe absorption and discussions of the manuscript with Dr. Gabriel Banciu, D. Liviu Nedelcu and Dragos Geambasu.

## Author contributions

E.I. has performed all the cell irradiation experiments, maintained the pre-B cells in culture, performed PCR from genomic DNA and quantified the amplification results. A.M. and M.S. have performed all the experiments to measure the EMF parameters used in irradiation, absorption spectra, power flux density and electric field intensity measurements. M.T. has performed the experiments to detect and quantify the  $\gamma$ H2AX foci whereas D.S. helped in interpreting the results of their foci/cell analysis. M.C. has designed the experiments, performed the analysis and interpretation of the cell irradiation experiments, supervised experiments and wrote the manuscript. All authors reviewed the manuscript.

## Competing interests

The authors declare no competing interests.

## Additional information

**Supplementary Information** The online version contains supplementary material available at <https://doi.org/10.1038/s41598-021-91790-3>.

**Correspondence** and requests for materials should be addressed to M.C.

**Reprints and permissions information** is available at [www.nature.com/reprints](http://www.nature.com/reprints).

**Publisher's note** Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.



**Open Access** This article is licensed under a Creative Commons Attribution 4.0 International License, which permits use, sharing, adaptation, distribution and reproduction in any medium or format, as long as you give appropriate credit to the original author(s) and the source, provide a link to the Creative Commons licence, and indicate if changes were made. The images or other third party material in this article are included in the article's Creative Commons licence, unless indicated otherwise in a credit line to the material. If material is not included in the article's Creative Commons licence and your intended use is not permitted by statutory regulation or exceeds the permitted use, you will need to obtain permission directly from the copyright holder. To view a copy of this licence, visit <http://creativecommons.org/licenses/by/4.0/>.

© The Author(s) 2021

OPEN

# Whole-body exposures to radiofrequency-electromagnetic energy can cause DNA damage in mouse spermatozoa via an oxidative mechanism

Brendan J. Houston<sup>1,2</sup>, Brett Nixon<sup>1,4</sup>, Kristen E. McEwan<sup>1</sup>, Jacinta H. Martin<sup>1,4</sup>, Bruce V. King<sup>3</sup>, R. John Aitken<sup>1,4</sup> & Geoffrey N. De Iulius<sup>1,4\*</sup>

Artificially generated radiofrequency-electromagnetic energy (RF-EME) is now ubiquitous in our environment owing to the utilization of mobile phone and Wi-Fi based communication devices. While several studies have revealed that RF-EME is capable of eliciting biological stress, particularly in the context of the male reproductive system, the mechanistic basis of this biophysical interaction remains largely unresolved. To extend these studies, here we exposed unrestrained male mice to RF-EME generated via a dedicated waveguide (905 MHz, 2.2W/kg) for 12 h per day for a period of 1, 3 or 5 weeks. The testes of exposed mice exhibited no evidence of gross histological change or elevated stress, irrespective of the RF-EME exposure regimen. By contrast, 5 weeks of RF-EME exposure adversely impacted the vitality and motility profiles of mature epididymal spermatozoa. These spermatozoa also experienced increased mitochondrial generation of reactive oxygen species after 1 week of exposure, with elevated DNA oxidation and fragmentation across all exposure periods. Notwithstanding these lesions, RF-EME exposure did not impair the fertilization competence of spermatozoa nor their ability to support early embryonic development. This study supports the utility of male germ cells as sensitive tools with which to assess the biological impacts of whole-body RF-EME exposure.

With rapid advances in technology and increasing demand for electronic communication, mobile phone usage has become virtually ubiquitous in the developed world<sup>1</sup>. Mobile phone devices receive and emit radiofrequency-electromagnetic energy (RF-EME) to transfer information, and accordingly our exposure to this form of energy is now unprecedented. Thus there is a clear imperative to establish public safety guidelines around the use of these mobile devices. It is, however, difficult to meet this demand due to a current lack of understanding concerning how RF-EME interacts with biology. While to date, no overwhelming clinical effects have been associated with RF-EME exposure<sup>2-6</sup>, multiple studies suggest that this form of energy can elicit subtle detrimental effects on biological systems<sup>7-10</sup>. Accordingly, the International Agency for Research on Cancer have yet to dismiss the risks of RF-EME, instead classifying this form of energy as a potential carcinogen. While we continue to debate the biological effects of chronic RF-EME exposure, a growing body of evidence now proposes that acute *in vitro* RF-EME exposure can elicit oxidative stress in a range of model cell lines<sup>7,9,11-13</sup>. A leading hypothesis to account for the mechanistic basis of this response is that RF-EME targets the mitochondria, leading to perturbation of proton flux across the inner mitochondrial membrane and promoting electron leakage from the electron transport chain. The resultant formation of superoxide anion serves as a progenitor for additional reactive oxygen species generation (ROS), eventually creating a ROS imbalance and a state of oxidative stress<sup>1,12</sup>.

<sup>1</sup>Priority Research Centre for Reproductive Science, School of Environmental and Life Sciences, Discipline of Biological Sciences, University of Newcastle, Callaghan, NSW, 2308, Australia. <sup>2</sup>School of Biological Sciences, Faculty of Science, Monash University, Clayton, VIC, 3800, Australia. <sup>3</sup>School of Mathematical and Physical Sciences, University of Newcastle, Callaghan, NSW, 2308, Australia. <sup>4</sup>Hunter Medical Research Institute, Cancer Research Program, New Lambton Heights, NSW, 2305, Australia. \*email: [geoffry.deiulius@newcastle.edu.au](mailto:geoffry.deiulius@newcastle.edu.au)

The potential for this mechanism of biophysical interaction provides the impetus for well-designed studies to ascertain the effect of RF-EME following whole-body irradiation regimens that more accurately mimic human exposure. In this context, a focus on the male reproductive system is justified owing to the common practice of storing mobile phone devices in the pant pocket, placing them in close proximity to the reproductive tract. Further emphasizing the relevance of the male reproductive system is mounting evidence that male germ cells are particularly susceptible to RF-EME<sup>14</sup> and the associated production of oxidative stress<sup>7,12</sup>. Indeed, it has been shown that spermatozoa provide a sensitive model to study the specific physical and chemical responses to RF-EME<sup>15</sup>. The situation arises because of the unique architecture and metabolism of spermatozoa, which places these cells at heightened vulnerability to damage by free radicals<sup>16</sup>. Moreover, spermatozoa provide a readily assessable means of monitoring adverse biological effects, through functional parameters such as motility, or more detailed analysis that can pinpoint biochemical disruption and more subtle endpoints such as the accumulation of DNA damage. Besides serving as a sensitive model, these cells are also clinically important, since the induction of DNA damage in the male germ line contributes to infertility<sup>16</sup> and has the potential to propagate in the embryo, altering developmental trajectory and the health of the offspring<sup>16,17</sup>.

To date, a handful of studies have sought to assess the effects of RF-EME on the male germ line. However, the majority of these studies have focused on isolated spermatozoa or immature male germ cells<sup>12,15,18–21</sup>. While this approach is conducive to examination of the intricate biochemical and cellular responses to direct RF-EME exposure, the use of alternate *in vivo* rodent models is likely to present a closer clinical representation of exposure, which can also serve to extend our understanding of EME-perturbed biochemical pathways highlighted from *in vitro* studies. Whole body models afford the added advantage that they enable observation of the holistic effects of RF-EME on all stages of male germ cell development<sup>22</sup>, encompassing the differentiation of germ into spermatozoa and their subsequent functional maturation as they transit the epididymis. With a sustained interest in establishing the biophysical mechanism(s) of action for RF-EME on biology, we report the use of a mouse model to probe reproductive stress following whole-body RF-EME exposure regimens. Specifically, a dedicated waveguide machine (Fig. 1), similar to that developed by Puranen and colleagues<sup>23</sup>, was constructed to facilitate exposure of unrestrained mice to RF-EME at 905 MHz with a specific absorption rate (SAR) of 2.2 W/kg. Mice were exposed to RF-EME for 12 h per day, over a period of between 1 to 5 weeks and subsequently the testes and epididymides were collected to investigate the effects of RF-EME on spermatogenesis and sperm function.

## Results

### Whole-body RF-EME exposure does not elicit gross histological changes in the mouse testis.

Following exposure of unrestrained mice to whole-body RF-EME exposure, we first examined the effects of our varied regimens on the average growth rate (Fig. 2a) of irradiated animals over the 5 weeks; revealing no changes in rate between the sham and RF-EME exposure groups. Similarly, gross testis morphology of sham and RF-EME exposed mice also remained comparable to that of control mice (Fig. 2b), with all samples exhibiting healthy tubule growth and extensive germ cell proliferation irrespective of the duration of exposure. All mice were 8 weeks of age at the commencement of the 1, 3 and 5 week study, however, some variance in body weight between cohorts was observed on their arrival. Nevertheless, no significant change in average growth rate was recorded between exposures, over the 35-day study (Fig. 2a).

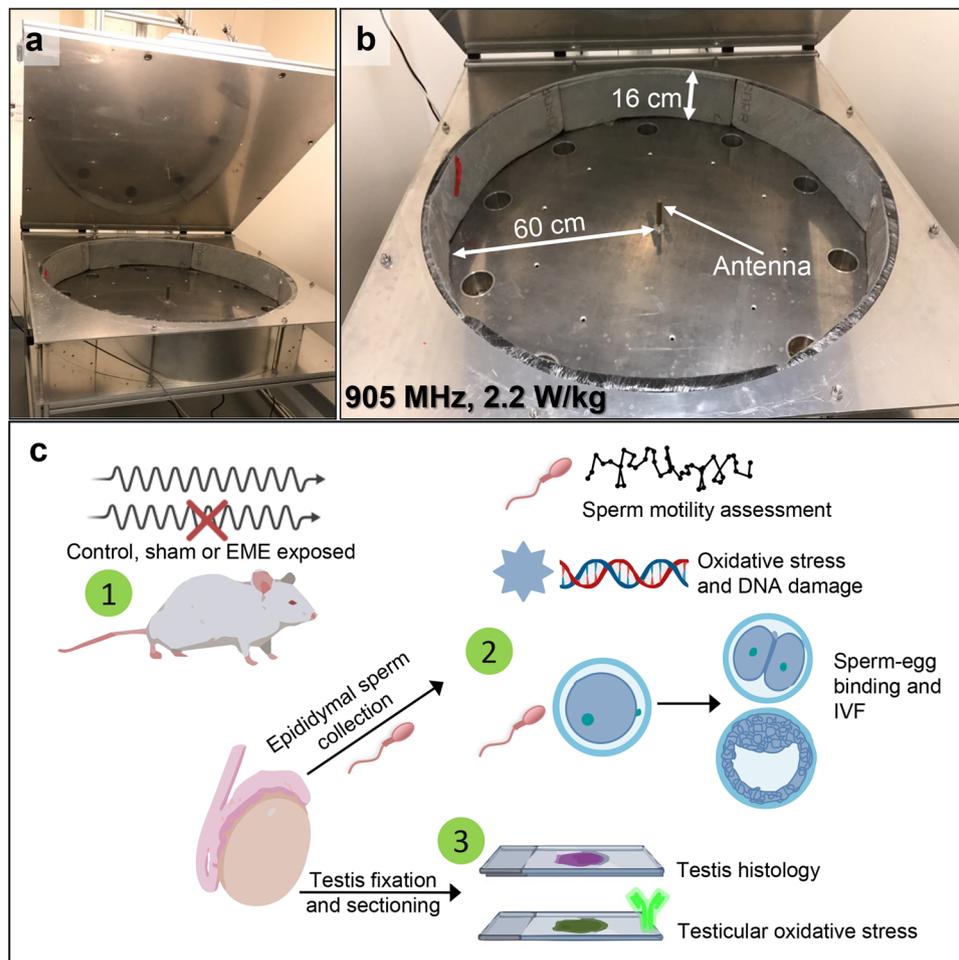
Guided by our previous studies in which we have shown that *in vitro* RF-EME exposure can induce a state of oxidative stress, leading to DNA damage in some male germ cell types<sup>7,12</sup>, we next explored the levels of DNA fragmentation and lipid peroxidation present within the testes of RF-EME exposed animals. For the former analysis, testis sections were probed with an anti- $\gamma$ H2AX antibody, a marker of DNA double strand breaks (Fig. 3). This revealed modest levels of DNA damage, which was largely restricted to meiotic germ cells within the seminiferous tubules. Furthermore, this tissue localization and levels of  $\gamma$ H2AX staining were consistent across the panel, with no effect observed due to EME exposure ( $p = 0.07$ ) or time. With regard to lipid peroxidation (Fig. 4), we documented a similar response, with no substantive increases in the lipid peroxidation product, 4-hydroxynonenal being detected within the testis sections of any RF-EME treatment group with respect to the untreated or sham controls ( $p = 0.22$ ).

### Whole-body RF-EME exposure adversely impacts the vitality and motility profiles of mature spermatozoa.

To explore the effect of *in vivo* RF-EME exposure on mature spermatozoa, we next investigated the outcomes of our irradiation regimen on sperm motility and vitality (Fig. 5). It was observed that the total number of live spermatozoa isolated from the cauda epididymis was diminished with RF-EME exposure ( $p < 0.05$ ) (Fig. 5a), an effect that was particularly evident after 5 weeks of exposure ( $p < 0.001$ ); whereas no changes were observed in our sham-exposed populations. In a similar manner, we noted a significant reduction in the percentage of motile spermatozoa isolated from RF-EME exposed mice following a treatment regimen extending over 5 weeks ( $p < 0.05$ ) (Fig. 5b). This reduction in overall sperm motility occurred commensurate with defects in the objective measurements of progressive and rapid sperm motility (Fig. 5c,d) in exposed mice. In this regard, the impact on both parameters was again most notable following 5 weeks of exposure ( $p < 0.001$ ). Conversely, spermatozoa isolated from the sham exposure groups displayed no such changes in their vitality or motility profile; with both parameters remaining indistinguishable from those documented in an unexposed control group of males.

### Whole-body RF-EME exposure elevates oxidative stress and DNA damage in mature spermatozoa.

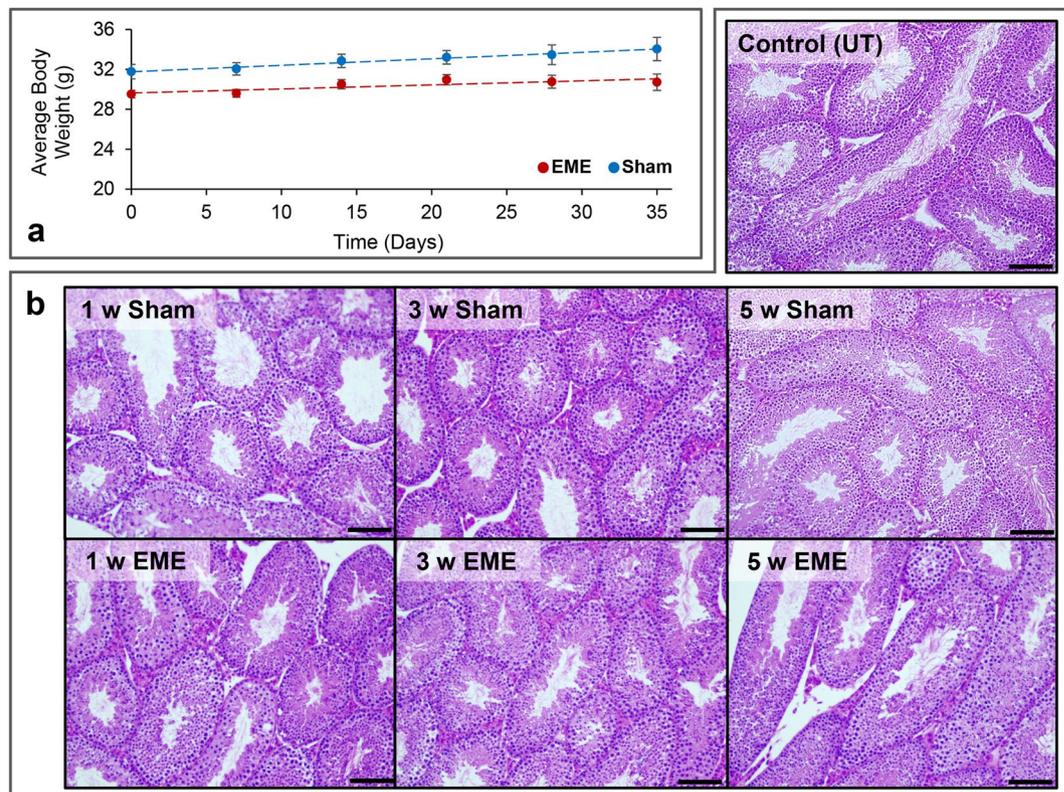
To determine whether the functional lesions in motility and vitality documented in the spermatozoa of RF-EME exposed mice were linked to the induction of oxidative stress, we next investigated the levels of cellular and mitochondrial ROS present in these cells (Fig. 6). Specifically, the dihydroethidium (DHE) fluorescent probe was utilized to provide insight into levels of cellular ROS production (Fig. 6a). Approximately 14% and 75% spermatozoa



**Figure 1.** Waveguide instrument used to deliver whole-body RF-EME exposure. Shown are (a) the complete waveguide apparatus with lid in open configuration and (b) close-up view illustrating the dimensions of the inner chamber. (c) A graphical experimental overview. (1) Mice were RF-EME or sham exposed for 1, 3 or 5 weeks and compared to a control population that did not enter the apparatus (untreated). Mice were culled and their spermatozoa were examined using sperm functional assays and a variety of oxidative stress assays (2). The testes of these mice were also examined for gross histological abnormalities and for markers of oxidative stress, via tissue sections (3).

stained positively for DHE in the negative (untreated) and positive (i.e. hydrogen peroxide exposed) control populations, respectively. When the experimental groups were analyzed, neither the sham nor the RF-EME treatment conditions resulted in a significant deviation from basal ROS generation detected by DHE labeling. This was in contrast to mitochondrial ROS production, where the MitoSOX Red (MSR) probe (Fig. 6b) revealed a significant, two-fold elevation in ROS generation within the sperm mitochondria of animals exposed to RF-EME for periods of either 1 or 3 weeks, compared to the control and sham-exposed cell populations ( $p < 0.05$ ). Intriguingly, sperm mitochondrial ROS generation had normalized to basal, control levels following 5 weeks of RF-EME exposure.

DNA damage assays were next employed to gain insight into the consequences of RF-EME induced ROS generation on the DNA integrity of mouse spermatozoa (Fig. 7). The halo assay (Fig. 7a), which evaluates DNA integrity based on the presence or absence of a halo-like stained DNA structure, revealed a modest but significant increase (i.e. ~5–6%) in the percentage of DNA-fragmented spermatozoa following 3 and 5 weeks of RF-EME exposure ( $p < 0.05$ ). Consistent with these findings, the application of an alkaline comet assay (Fig. 7b) confirmed that whole-body RF-EME exposure stimulated sperm DNA fragmentation. After 1 week, sperm DNA fragmentation was elevated by 18%, however, this increase only gained significance after 5 weeks of exposure (23% increase in fragmentation;  $p < 0.05$ ). Given the elevation in mitochondrial ROS, we next demonstrated that this DNA damage was likely oxidative in nature, highlighted by an increase in the percentage of RF-EME exposed sperm displaying positive staining for 8-hydroxy-2-deoxyguanosine (8-OH-dG; Fig. 7c); a biomarker of oxidative DNA damage. Indeed, across each of the three exposure times assessed, RF-EME induced a significant ( $p < 0.05$ ) increase in 8-OH-dG labelling relative to control and sham exposed populations. As anticipated, 8-OH-dG labelling was localized to the nuclear compartment of the sperm head and was consistently more intense in RF-EME treated spermatozoa (Fig. 7d).



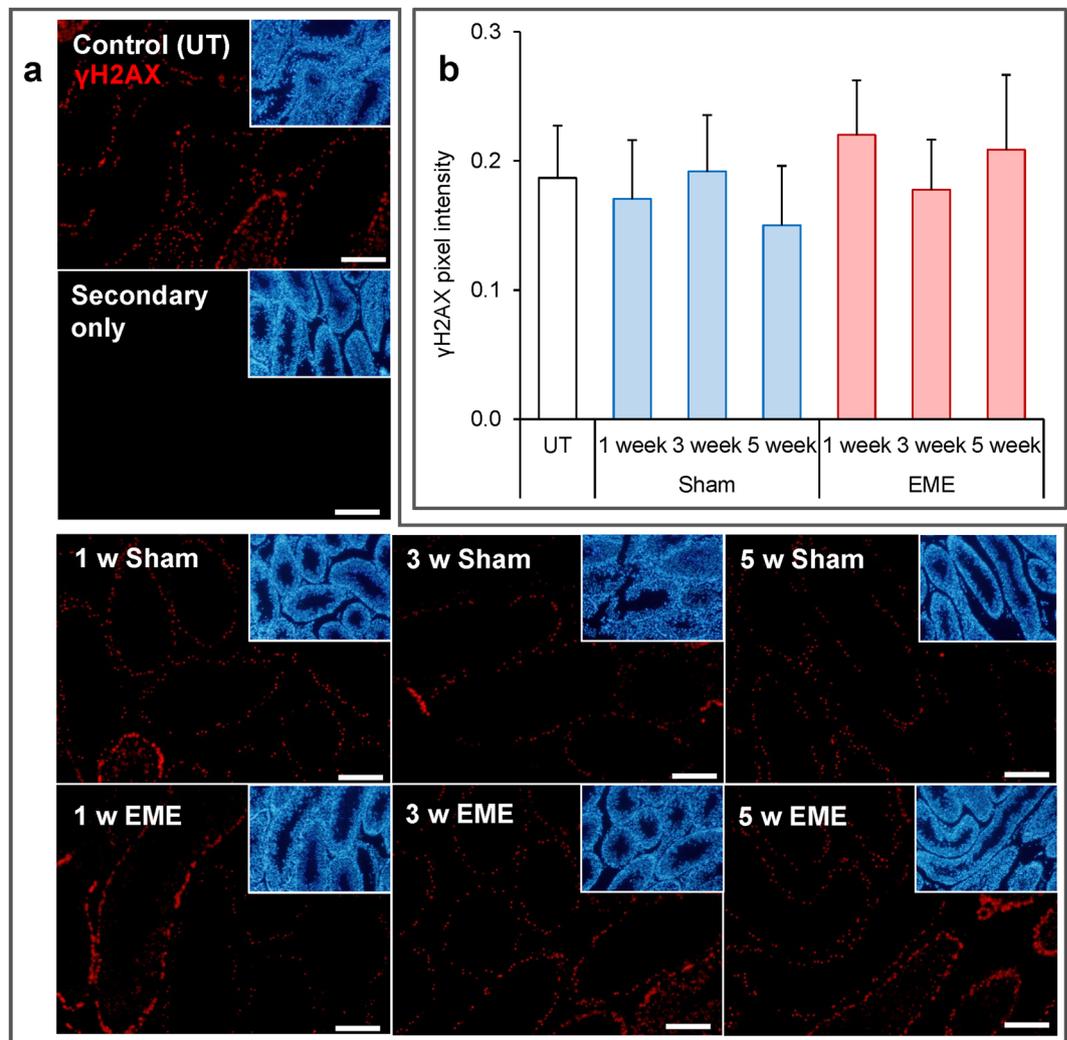
**Figure 2.** The effect of RF-EME on the growth and testis morphology of C57BL/6 mice. (a) Mice were weighed at weekly intervals to investigate the effects of RF-EME on body mass against sham exposed males ( $n = 8\text{--}20$  mice measured/treatment group). Red circles represent the mean weight of EME treated mice, whereas blue circles represent the sham exposed group (b) Haematoxylin and eosin staining of testis sections was conducted to facilitate comparison of gross seminiferous tubule morphology ( $n = 3$  mice/treatment group). Scale bar represents  $400\ \mu\text{m}$ .

### Whole-body RF-EME exposure does not impair the fertilization competence of spermatozoa.

In order to determine if RF-EME mediated induction of sperm DNA damage was sufficient to compromise the fertilization competence of these cells, we undertook an assessment of selected markers of sperm capacitation and *in vitro* fertilization success utilizing the spermatozoa from 5 week RF-EME exposed mice (Fig. 8). Of the capacitation markers assessed, neither the number of sperm displaying complete flagellum phosphotyrosine labelling (Fig. 8a) or the ability to undergo a calcium ionophore induced acrosome reaction (Fig. 8a) differed significantly between the control and RF-EME treatment groups. Similarly, the average number of spermatozoa bound to the zona pellucida of fixed oocytes was also unchanged across our control (25), sham (25) and RF-EME exposed (19) populations (Fig. 8c,d;  $p = 0.99$ ). As an extension of this assessment of sperm function, the ability of spermatozoa from all three treatment groups to achieve fertilization and progression to the blastocyst stage of development was then investigated. Exposure to RF-EME under our regime, did not exert any observable effect on fertilization rate (Fig. 8d), with all treatment groups resulting in the fertilization of between 83–87% of inseminated oocytes. Furthermore, when these zygotes were cultured through to the blastocyst stage of development (Fig. 8e), a modest increase was observed in the development rate of the RF-EME group, although this did not prove to be significantly different from the sham exposed or untreated sperm groups.

### Discussion

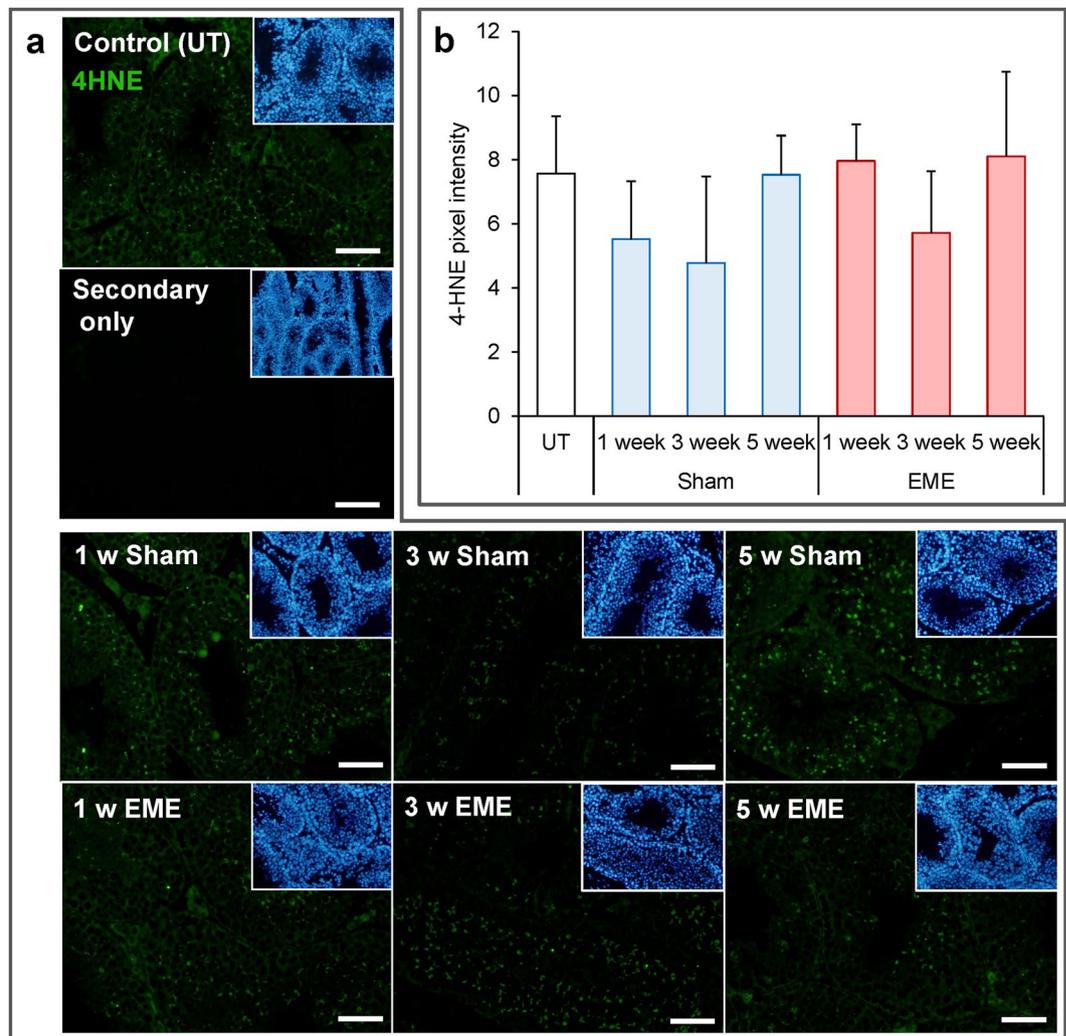
Several lines of evidence now propose RF-EME to be capable of inducing a state of oxidative stress in a variety of cell types<sup>24–27</sup>, including the male germline<sup>7,12</sup>. It is also well established that spermatozoa are particularly sensitive to oxidative insults, a phenomenon that may be traced to their surplus of oxidizable substrates and restricted antioxidant capacity<sup>16,28</sup>. What remains less certain is how RF-EME is capable of inducing such cellular responses in the absence of a thermal induction mechanism. In seeking to resolve this question, here we have utilized an *in vivo* exposure model that not only approximates the complexities of environmental RF-EME exposures, but also enables the dissection of RF-EME effects on key stages of male germ line development. Specifically, our exposure regimen enabled determination of the interaction of RF-EME with spermatozoa held exclusively within the luminal environment of the epididymis (1 week exposure), as well as those exposed during their progression through a spermatogenic cycle and transit of the epididymal tract (5 week exposure). Consistent with our previous *in vitro* investigations<sup>7,12</sup>, we here contribute data to support the dysregulation of sperm mitochondria as a pivotal target for driving RF-EME associated stresses in the male reproductive system.



**Figure 3.** RF-EME exposure does not induce  $\gamma$ H2AX expression in the testis. Testis sections from untreated control animals (UT), as well as those of the sham and RF-EME exposure groups, were probed with anti-phospho- $\gamma$ H2AX antibodies (red) to detect DNA double strand breaks. (a) Representative images are depicted, with scale bar equating to 400  $\mu$ m. A secondary antibody only control is also included. Corresponding DAPI (blue) stained images, illustrating tubule morphology are included as insets included in the upper right corner of each panel. (b) Analysis of pixel intensity was performed on the germ cell population within the seminiferous tubules in order to quantify  $\gamma$ H2AX expression levels across treatments. Graphical data are presented as mean + SEM (n = 3 mice/treatment group, with 8–25 tubules being analyzed for each testis).

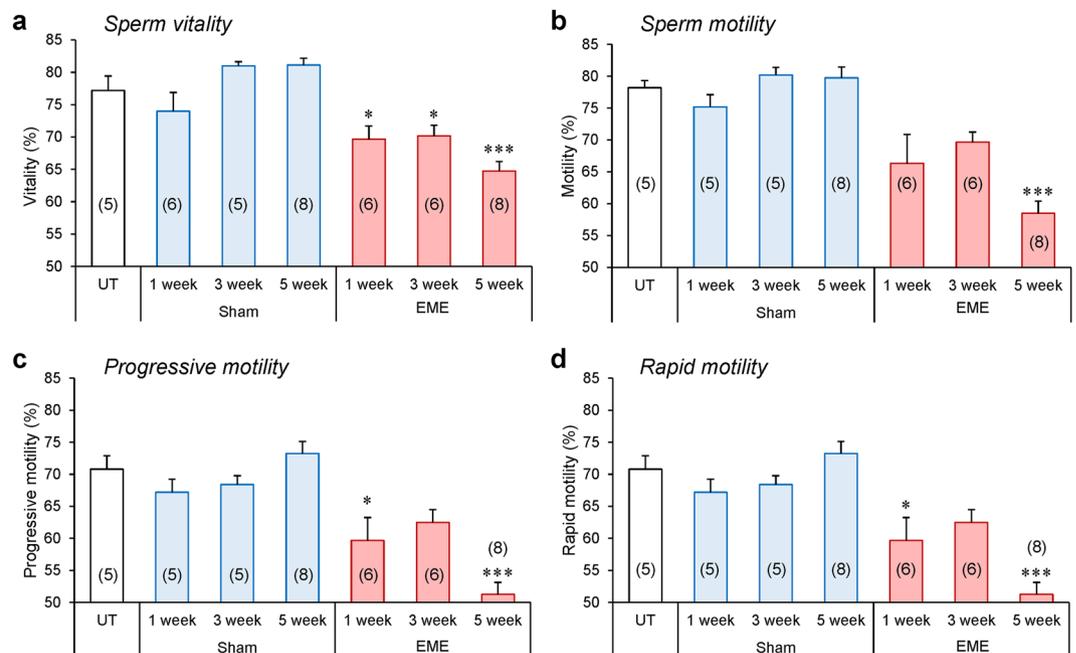
In contrast to previous reports of disorganized testicular architecture and spermatogenesis arising from whole-body RF-EME exposure<sup>29,30</sup>, the supraphysiological treatment regimen implemented here did not support these findings, with no changes to gross testicular histology observed. Similarly, both the somatic and germline tissue within the seminiferous tubules also proved recalcitrant to RF-EME induction of DNA damage or lipid peroxidation. Such findings are not entirely unexpected given the lack of robust evidence to support the ability of environmental RF-EME exposure conditions to elicit such obvious overt tissue damage<sup>1</sup>. Rather, on the basis of prevailing evidence we consider that any biophysical RF-EME interactions would likely result in more subtle phenotypic changes<sup>1</sup>, thus justifying our primary focus on the male germ line as a sensitive model cell type<sup>16</sup> to explore mechanisms of RF-EME mediated stress. Accordingly, we observed a clear attenuation of sperm motility, occurring in concert with increased mitochondrial ROS generation, after 1 and 3 weeks of whole-body RF-EME exposure. In the absence of commensurate increase in cytosolic ROS production, these data provide correlative evidence that sperm mitochondria are indeed prone to RF-EME dysregulation and that the ensuing production of ROS was sufficient to compromise the most vulnerable aspects of sperm cell function. Additional support for this model rests with a growing body of literature implicating RF-EME in the generation of a state of oxidative stress in a variety of cell types other than the male germ line<sup>11,13,31–34</sup>.

An interesting observation to arise from our study was that the induction of mitochondrial ROS generation after 1 and 3 weeks of RF-EME exposure was followed by an apparent normalization of mitochondrial ROS after an additional 2 weeks of exposure. At present, we remain uncertain what mechanism(s) could account for the



**Figure 4.** RF-EME exposure does not induce elevated 4-hydroxynonenal formation in the testis. Testis sections from untreated control animals (UT), as well as those of the sham and RF-EME exposure groups, were probed with anti-4-hydroxynonenal antibodies (green) to detect by-products of lipid peroxidation. (a) Representative images are depicted, with the scale bar equating to 200  $\mu$ m. A secondary antibody only control is also included. Corresponding DAPI stained images illustrating tubule morphology are included as insets included in the upper right corner of each panel. (b) Analysis of pixel intensity was performed on the germ cell population within the seminiferous tubules in order to quantify 4-hydroxynonenal expression levels across treatments. Graphical data are presented as mean + SEM (n = 3 mice/treatment group, with 10–20 tubules being analyzed for each testis).

mitigation of this response, but speculate they may be associated with reduced mitochondrial function in germ cells subjected to prolonged RF-EME exposure, or that these cells are capable of responding to this challenge through an elevation of intrinsic antioxidant defenses. As an extension of this hypothesis, it is possible that the male reproductive tract also mounts a protective response to chronic RF-EME via an upregulation of exogenous antioxidant production. In keeping with this notion, it has been shown that the concentrations of both vitamin A and E increase in the testis of RF-EME exposed rats<sup>35</sup>. Alternatively, this phenomenon could be linked to morphological changes in the mitochondrion during spermatogenesis<sup>36</sup>, such as the extensive vacuolization these organelles undergo during the maturation of spermatogonia to spermatocytes<sup>37</sup>. Accompanying such changes, mitochondrial activity is also elevated in spermatocyte and spermatid populations, whereas mature mouse spermatozoa are known to limit their investment into oxidative phosphorylation and instead utilize glycolysis to meet their energy demands<sup>38</sup>. Finally, there is also evidence that the mitochondria of caput epididymal spermatozoa are silenced<sup>39</sup>, which may afford some protection against perturbed mitochondrial ROS production while also identifying a dynamic sensitivity of spermatozoa. The cauda epididymal spermatozoa sampled after enduring 5 weeks of EME exposure, will comprise a mixture of cells exposed during various stages of germ cell development and maturation, however the majority of the cells will likely have encountered EME as morphologically mature spermatozoa, which may house less vulnerable mitochondria. Irrespective of the mechanism(s) responsible for suppression of ROS production, the downstream detrimental legacy of RF-EME exposure persisted in mature spermatozoa

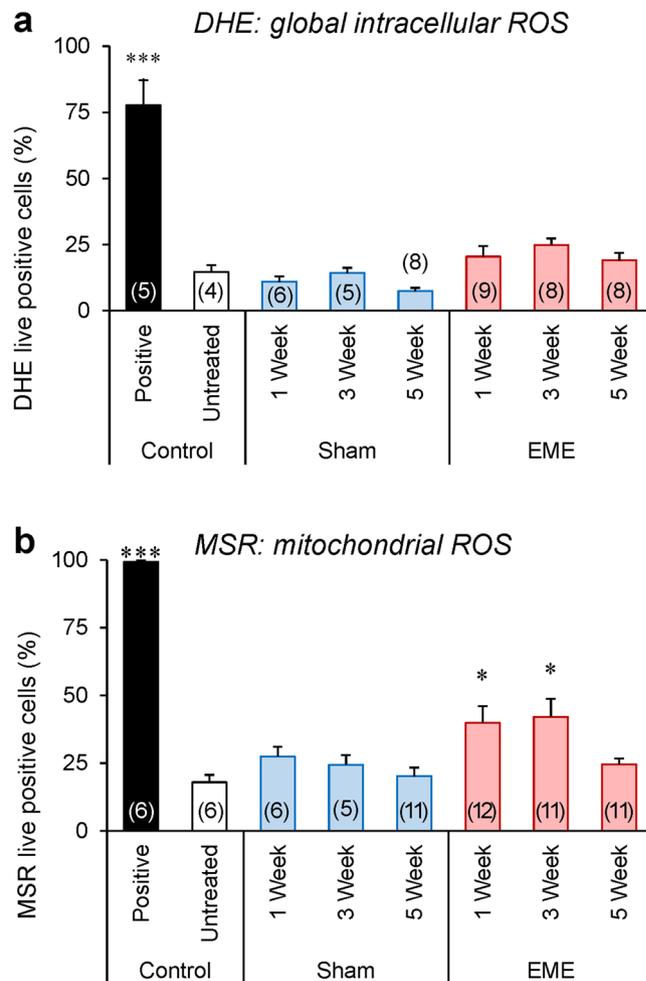


**Figure 5.** Sperm vitality and motility declines in response to RF-E-ME exposure. Spermatozoa were collected from the cauda epididymis of untreated control animals (UT), as well as those of the sham and RF-E-ME exposure groups. **(a)** Sperm vitality was assessed via the eosin-exclusion method. Next, the percentage of sperm displaying **(b)** any form of motility, **(c)** progressive motility, and **(d)** rapid motility was determined using computer assisted semen analysis. Data are presented as mean + SEM ( $n = 5-8$  mice/treatment group), with a minimum of 100 spermatozoa being analyzed from each animal). The number of biological replicates used is denoted in each bar. \* $P < 0.05$ , \*\*\* $P < 0.001$ .

after 5 weeks of treatment as evidenced by the demonstration that these cells suffered the highest losses of vitality and motility. Thus, although the production of mitochondrial ROS was ameliorated in spermatozoa after 5 weeks of RF-E-ME exposure, these cells were unable to repair the oxidative damage they sustained during prior exposure.

The identification of sperm motility as being vulnerable to RF-E-ME exposure is consistent, independent evidence that this functional attribute is among of the first to succumb to elevated levels of ROS<sup>40,41</sup>. ROS mediated lipid peroxidation is known to drive the production of reactive aldehydes, such as 4-hydroxynonenal, which causes irreversible protein modifications and alkylation of the sperm axoneme<sup>42</sup>. Where oxidative stress levels may spike at an earlier window in sperm development, limiting the amount of detectable ROS in the sperm collected at 5 weeks, these cells can retain hallmarks of this pathology, in the form of oxidized DNA lesions. Consistent with this notion, we detected an increase in the oxidative stress biomarker, 8-OH-dG, in the nuclei of sperm across all exposure regimens; indicating abundant guanosine oxidation and supporting RF-E-ME as a mediator of oxidative stress. A similar finding has been reported by Liu *et al.*<sup>19</sup>, who documented a significant elevation in 8-OH-dG formation in spermatocytes exposed to RF-E-ME. Accompanying oxidative DNA damage, we observed elevated sperm DNA fragmentation in the form of single strand breakage following whole-body RF-E-ME exposure. These data accord with the enhanced levels of DNA fragmentation documented in spermatozoa<sup>7,21,43</sup> and spermatocytes<sup>19</sup> exposed to RF-E-ME; a phenomenon that may describe a continuum of DNA damage, originating from oxidative DNA insults<sup>44</sup>. While further studies are required to pinpoint variations in the sensitivity of different germ cell populations to RF-E-ME *in vivo*, our data suggests that a window of vulnerability may extend across both testicular and post-testicular (i.e. epididymal) phases of development.

Notwithstanding an elevation in oxidative stress mediated DNA damage and an attendant reduction of motility, the spermatozoa recovered from mice exposed to 5 weeks of whole-body RF-E-ME did not display any associated lesions in their fertilization potential. Thus, these cells retained their ability to capacitate, acrosome react, and bind zona pellucidae at rates that were statistically indistinguishable from those of untreated and sham exposed mice. Moreover, these spermatozoa were capable of supporting normal rates of *in vitro* fertilization and early embryo development. In seeking to reconcile these data, a key limitation is that the *in vitro* fertilization strategy adopted in this study, introduced selection bias for the higher quality, motile spermatozoa which potentially harbour only basal levels of DNA damage. Even in the lowest motility group, after 5 weeks of EME exposure, 60% of the recovered cells remain motile. This notion is consistent with studies of human IVF patients, which have revealed that *in vitro* assays of sperm-zona pellucida binding are highly selective for spermatozoa with intact DNA and normal motility profiles<sup>45</sup>. Alternatively, it is possible that the burden of DNA damage harbored by the fertilizing spermatozoon was sufficiently resolved by the oocyte. In any case, this clearly illustrates, reassuringly, that even at the supraphysiological regimens of whole-body RF-E-ME exposure used in this study, no overt impairment to fertilization potential and early embryo development was observed. This is perhaps in alignment to the lack of overt morphological changes observed in the reproductive tissue of exposed mice, confirming



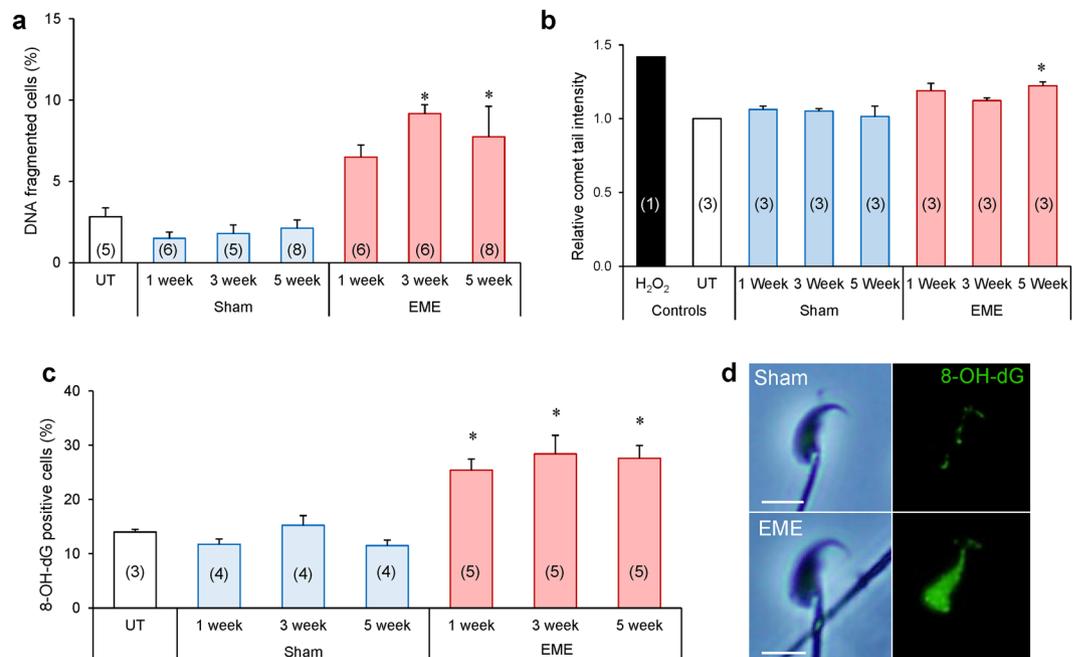
**Figure 6.** Exposure to RF-EME stimulates the generation of mitochondrial reactive oxygen species. Spermatozoa were isolated from the cauda epididymis of untreated control animals, as well as those of the sham and RF-EME exposure groups. These cells were pre-loaded with fluorescent probes and then analyzed using flow cytometry to assess their generation of reactive oxygen species (ROS). (a) Global levels of ROS generated in the sperm cell was assessed with the dihydroethidium (DHE) probe. (b) Alternatively, mitochondrial ROS generation was investigated with the MitoSOX Red (MSR) probe. In both instances, a minimum of 10,000 spermatozoa were assessed from 5–12 of animals and data are presented as mean + SEM. The number of biological replicates used is denoted in each bar. \* $P < 0.05$ .

observations that environmental RF-EME does not contribute to gross biological damages. In this context, and given the evidence of cellular oxidative impacts, we cannot yet discount the possibility of transmission of subtle phenotypic or epigenetic changes in the offspring. Thus, future studies focused on trans- and multi-generational outcomes will likely play a key role in resolving any potential for cumulative changes caused by RF-EME. While more targeted investigations into this aspect of exposure is warranted, it is perhaps comforting that whole-body chronic exposure (life-long, 24 h/day) to electromagnetic fields has been reported to elicit no harmful effects on the fertility or development of mice over four successive generations<sup>46</sup>.

In summary, our evidence supports the hypothesis that sustained whole-body RF-EME is capable of inducing a state of oxidative stress in the male germ line, a cell vulnerable to the effects of ROS. Furthermore, our data further implicate the mitochondria as the target for RF-EME biophysical interaction, with a consequential elevation of mitochondrial ROS generation being linked to reduced motility and elevated oxidative DNA damage and DNA fragmentation in the spermatozoa of exposed males. Whilst these lesions were not sufficient to compromise fertilization competence or early embryo development, it will nonetheless be of interest to investigate the trans-generational influence of whole-body RF-EME in future studies.

## Methods

**Chemical reagents.** The reagents used in this study were purchased from Sigma-Aldrich (St. Louis, MO, USA) unless stated otherwise. Fluorescent probes were purchased from Thermo Fisher Scientific (Waltham, MA, USA), unless otherwise stated. All fluorescence imaging was performed using a Zeiss Axioplan 2 fluorescence microscope (Carl Zeiss MicroImaging GmbH, Jena, Germany).



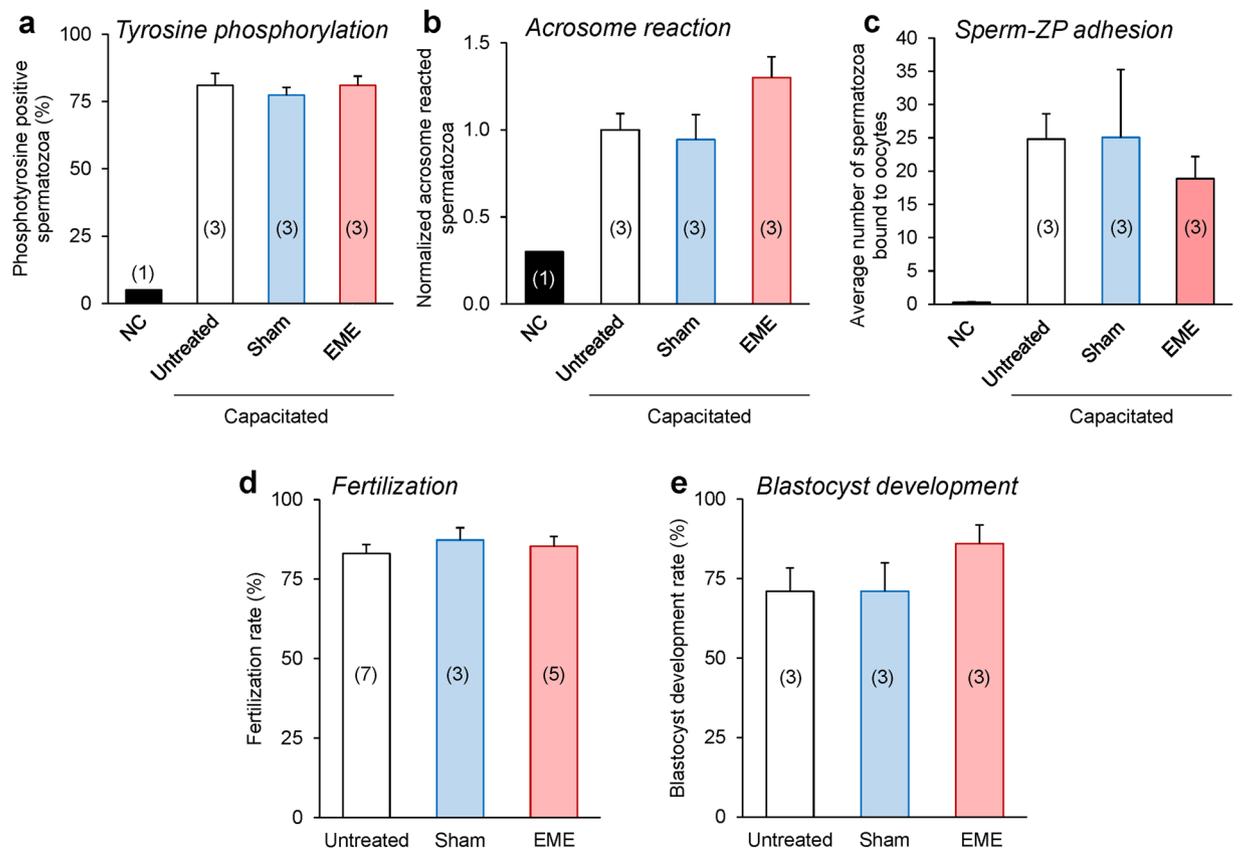
**Figure 7.** RF-EME exposure induces oxidative DNA damage in spermatozoa. Spermatozoa were isolated from the cauda epididymis of untreated control animals (UT), as well as those of the sham and RF-EME exposure groups. These cells were assessed for DNA fragmentation using (a) the halo assay showing the percentage of cells fragmented ( $n = 5-8$  mice/treatment group, each with 100 sperm assessed for each replicate) and then (b) quantified by the alkaline comet assay, expressed as percentage tail intensity and normalized to control data for each run ( $n = 3$  mice/treatment, each with 50–70 sperm cells assessed). (c) To extend this DNA integrity analysis, sperm were evaluated for oxidative DNA adducts via labelling with anti-8-hydroxy-2-deoxyguanosine (8-OH-dG) antibodies ( $n = 3-5$  mice/treatment). (d) Representative images of spermatozoa stained with the 8-OH-dG antibody from the 5 week sham and RF-EME exposed populations are included. The number of biological replicates used is denoted in each bar. Data are presented as mean + SEM. \* $P < 0.05$ , \*\* $P < 0.01$ .

**Waveguide design and whole-body RF-EME exposure regimens.** Adult (>8 weeks) male C57BL/6 mice were irradiated with 2 W/kg and 905 MHz RF-EME in a waveguide (Fig. 1) for 12 h daily, during a night (7 pm–7 am) cycle while the waveguide lid was closed. This waveguide was constructed by the Physics Department at the UON and comprises a cylindrical aluminium chamber (radius of 60 cm and depth of 16 cm) and mechanically operated lid. The chamber sides were insulated with carbon impregnated foam (RFI Industries, VIC, Australia) to prevent RF-EME reflection. Small fans were implemented for external air circulation into the chamber through the base. RF-EME was generated by a Rohde and Schwarz SMC100A signal generator (Macquarie Park, NSW, Australia), connected to a signal amplifier. Chamber lid operation was controlled by a timed motor in order to raise or lower the lid every 12 h. Mice were housed in plastic cages with Perspex lids and plastic water bottles to ensure there was no metal, which interferes with RF-EME distribution. Cages were arranged radially around a central RF-EME emitting antenna, and oriented so that the water bottle furthest from the radiation source to minimize liquid interference. When mice were removed they were replaced with ‘phantoms’ composed of a 50 ml Falcon tube filled with 142 mM NaCl in deionized water to mimic blood. Sham exposed males were placed in the waveguide under identical conditions, however, the signal generator was turned off, thus receiving no exposure to RF-EME. All treatment groups were sacrificed at three time points; 1, 3 and 5 weeks of exposure and compared to an untreated control population of mice that were not placed inside the chamber. Mice were weighed weekly throughout the treatment regime (EME or sham exposed) during the time the waveguide lid was open. The weights were recorded after mice were individually placed in a tared container on top of the weigh tray of an electronic balance.

The SAR delivered to the mice was calibrated using a NARDA NBM 520 electric field meter with an EF1891 probe to measure electric fields in the empty irradiation system. Radial electric field measurements were made as a function of distance from the vertical aerial mounted in the center of the system after the antenna length was adjusted to maximize power supplied to the system at a frequency of 905 MHz. For 1 W input to the aerial a maximum electric field of 94 V/m was measured 16 cm from the center, whereas in their slightly larger setup, Puranen *et al.*<sup>23</sup> measured a maximum electric field of 80 V/m at 15 cm from the center. The variation of E field with radial distance and the maximum electric fields in the two setups were found to be similar for the same power input.

The SAR (W kg<sup>-1</sup>) is related to the electric field, E, in a sample of conductivity  $\sigma$  (S m<sup>-1</sup>), and density  $\rho$  (kg m<sup>-3</sup>) by

$$\text{SAR} = \sigma |E|^2 / 2\rho \quad (\text{Wkg}^{-1}) \quad (1)$$



**Figure 8.** RF-EME exposure did not compromise the fertilization competence of spermatozoa. Spermatozoa were isolated from the cauda epididymis of untreated control animals, as well as those of the 5-week sham and RF-EME exposure groups. These cells were driven to capacitate and then assessed for (a) anti-phosphotyrosine labeling of the sperm flagellum, and (b) their ability to undergo a calcium ionophore induced acrosome reaction [assessed via peanut agglutinin (PNA) labeling of the sperm outer acrosomal membrane with values being normalized to the untreated control], and (c) binding to the zona-pellucida (ZP) of homologous oocytes (the average number of spermatozoa bound to ZP intact oocytes is shown). In each instance a non-capacitated (NC) population of spermatozoa from untreated animals was included as a negative control. Alternatively, spermatozoa were examined for their ability to (d) fertilize oocytes *in vitro* and subsequently (e) support early embryo development through to the blastocyst stage. In all instances, assessed spermatozoa were isolated from each of three animals and data are presented as mean + SEM, except for (d), where 3–7 mice were used. The number of biological replicates is shown in each bar. (a, b) A minimum of 100 spermatozoa from each animal were assessed for phosphotyrosine labelling of the sperm flagellum, and PNA labelling of the acrosome. (c, d) 8–10 oocytes per replicate were assessed for sperm-ZP binding and 11–30 for fertilization, and (e) 11–30 embryos were assessed for blastocyst development.

where E is the root-mean-square local electric field strength in V m<sup>-1</sup>. Puranen *et al.* (2009) measured a SAR of 0.11 W/m for the above 1 W input to the aerial. During our irradiations the input RF power was 20 W, corresponding to an average SAR of 2.2 W/kg since the geometry of our irradiation system is very similar to that of Puranen *et al.*<sup>23</sup>.

**Assessment of testis sections.** Upon dissection, testes were fixed in Bouin's solution, sectioned, dewaxed and rehydrated using standard protocols<sup>47</sup>. One section from each testis was stained with hematoxylin and eosin to investigate testis morphology, while the remainder were prepared for immunohistochemistry as previously described<sup>48</sup>. Antigen retrieval was performed by microwaving slides in 50 mM Tris (pH 10.5) for 9 min. Tissue sections were blocked (3% bovine serum albumin (BSA)-PBST, 10% goat serum) for 1 h at room temperature, washed in PBS for 5 min and labeled with appropriate pairs of primary (either anti-phospho- $\gamma$ H2AX (2  $\mu$ g/ml) or anti-4-hydroxynonenal (1/300) antibodies in 1% BSA-PBST overnight at 4 °C) and AlexaFluor-conjugated secondary antibodies (1 h at 37 °C). After washing in PBS, sections were counterstained with DAPI (0.5  $\mu$ g/ml), and viewed using fluorescence microscopy. Mean pixel intensity analysis was conducted on images using ImageJ version 1.48 V (NIH, USA). Pixel intensity determination was performed only on the seminiferous tubules, with surrounding interstitial tissue isolated from this analysis. For  $\gamma$ H2AX, meiotic germ cells were excluded from the analysis due naturally occurring high levels of double strand breaks in these cells<sup>50</sup>.

Assay/measurement	Untreated control	1 week		3 weeks		5 weeks	
		Sham	EME	Sham	EME	Sham	EME
Body weight	NA	20	20	14	14	8	8
Testis histology	3	3	3	3	3	3	3
Testis staining: $\gamma$ H2AX	3	3	3	3	3	3	3
Testis staining: 4-hydroxynonenal	3	3	3	3	3	3	3
Sperm vitality	5	6	6	5	6	8	8
Sperm motility, progressive and rapid	5	5	6	5	6	8	8
Dihydroethidium staining	4	6	9	5	8	8	8
MitoSOX red staining	6	6	12	5	11	11	11
DNA fragmentation	5	6	6	5	6	8	8
Comet tail intensity	3	3	3	3	3	3	3
8OHdG	3	4	4	4	5	5	5
Tyrosine phosphorylation	3	NA	NA	NA	NA	3	3
Acrosome reaction	3	NA	NA	NA	NA	3	3
Sperm-zona pellucida adhesion	3	NA	NA	NA	NA	3	3
Fertilization	7	NA	NA	NA	NA	3	5
Blastocyst development	3	NA	NA	NA	NA	3	3

**Table 1.** Number of replicates used for each assay.

**Preparation of spermatozoa.** Epididymides were dissected immediately after euthanasia and mature spermatozoa were collected from the caudal segment by retrograde perfusion before being resuspended in 1 ml of modified Biggers, Whiting, Whittingham media (BWW)<sup>49</sup>. Objective sperm motility was assessed by computer assisted sperm analysis (IVOS, Hamilton Thorne, Danvers, MA, USA) as previously described<sup>50</sup>, and sperm vitality was determined via eosin exclusion.

**Determination of ROS production in spermatozoa.** Spermatozoa were assessed for ROS generation via flow cytometry with the mitochondrial superoxide probe MitoSOX Red (MSR) or cytosolic superoxide probe dihydroethidium (DHE) in conjunction with Sytox Green (SYG) vitality stain as previously described<sup>51</sup>.

**Sperm chromatin dispersion (Halo) assay.** Spermatozoa were snap frozen in liquid nitrogen and stored at  $-80^{\circ}\text{C}$  prior to analysis. Spermatozoa were defrosted and mixed with 1% low melting point agarose at  $37^{\circ}\text{C}$  and applied to Superfrost slides (Thermo Fisher Scientific) pre-coated with 0.65% agarose. The slides were sealed with a coverslip and placed at  $4^{\circ}\text{C}$  to solidify for 5 min. After removing the coverslips, the slides were treated with 0.08 N HCl for 7 min in foil, followed by Halo solution 1 (pH 7.5; 0.4 M Tris, 1% SDS, 50 mM EDTA, 0.8 M DTT) for 10 min and Halo solution 2 (pH 7.5; 0.4 M Tris, 1% SDS, 2 M NaCl) for 5 min at room temperature to lyse the cells, relax and neutralize the DNA. Next, slides were exposed to Tris-boric acid-EDTA buffer (pH 7.5; 0.1 M tris, 0.09 M boric acid, 0.002 M EDTA) for 2 min, then washed in ethanol (70%, 90% then 100%) for 2 min each to dehydrate the slides. After air drying, slides were counterstained with DAPI (0.5  $\mu\text{g}/\text{ml}$ ) for 10 min at room temperature, rinsed in PBS and mounted.

**Alkaline comet assay.** The alkaline comet assay was performed as described previously<sup>52</sup>. DNA damage was analysed using Comet Assay IV software (Perceptive Instruments, Suffolk, UK). Hydrogen peroxide treatment (500  $\mu\text{M}$ , 5 min at room temperature) was utilized as a positive control. To compare sperm DNA damage between treatments, percentage tail DNA values of each cell in the treated samples were normalized to that of the average percentage tail DNA of the respective untreated control for each time point. The control itself taking on the value of 1. The normalized data for each sample then contributed to a biological replicate. The average of these replicates are then graphed. The normalization process is required to minimize the noise generated by the small fluctuations in tail intensity between independent runs and days.

**Oxidative DNA damage assay.** Oxidative DNA damage was assessed by suspending  $2 \times 10^6$  spermatozoa in Oxidative DNA/RNA damage antibody (Thermo Fisher Scientific) diluted 1/40 in PBST overnight at  $4^{\circ}\text{C}$ . Cells were then centrifuged for 5 min at  $450 \times g$  and washed in PBS before incubation in AlexaFluor-488 goat  $\alpha$  rabbit secondary (Abcam, Massachusetts, US) diluted 1/400 in PBST for 1 h at  $37^{\circ}\text{C}$ . Finally, cells were again washed and resuspended in PBS for counting and imaging via fluorescence microscopy.

**Sperm functional assays and *in vitro* fertilization.** Cauda epididymal spermatozoa were assessed for their ability to undergo capacitation-associated tyrosine phosphorylation, a calcium ionophore (A23187) induced acrosome reaction and bind zona pellucidae as previously described<sup>53,54</sup>. Alternatively,  $2 \times 10^5$  capacitated spermatozoa were inseminated into a droplet of oocytes recovered from superovulated female C57BL/6 mice<sup>55</sup>. The gametes were co-incubated for 4 h at  $37^{\circ}\text{C}$  prior to the oocytes being assessed for fertilization (i.e. extrusion of second polar body and/or pronucleus formation). Zygotes were cultured in HTF medium overnight and transferred into G1 PLUS culture medium (Vitrolife, Stockholm, Sweden) on the morning of day 2 followed by an

additional media change into G2 PLUS medium (Vitrolife) on Day 4<sup>55</sup>. The percentage of fertilized oocytes as well as embryos that had reached blastocyst stage by the morning of day 5 was calculated.

**Study design.** Twenty adult male mice were randomly assigned to three treatment groups (untreated control, sham exposure control, RF-EME exposed), determined by the number of mice that could fit in the waveguide (10 cages, 2 mice per cage). Six mice were randomly selected for the 7 and 21 day intervals, while eight mice were selected for the 35 day interval. After each interval the mice were phenotyped for male fertility. For the purpose of this study, we ran the experiment twice to generate sufficient numbers of biological replicates for certain assays, e.g. MitoSOX, where we used 11 replicates. Each of the two treatment cycles consisted of 6 males treated for 1 week, 6 males treated for 3 weeks and 8 males treated for 5 weeks. The individual number of replicates for each assay can be found within the figures and is also shown in Table 1, below. As 20 mice were utilized for end point assays over the period of 5 weeks, the reduction in the number of replicates for body weight measurements decreases with the use of these individuals at the 1 and 3 week time points accordingly.

**Statistical analysis.** Samples from each animal were considered as a single biological replicate. Experimental data was analyzed using JMP version 11 software (SAS Institute Inc., Cary, NC). Normality of datasets was assessed with the Shapiro-Wilks test ( $\alpha = 0.05$ ). A one-way ANOVA was used to compare normally distributed treatments, with a post-hoc Tukey's honest significant difference test ( $\alpha = 0.05$ ). For data not normally distributed, the Wilcoxon test was used ( $\alpha = 0.05$ ), with a post-hoc Dunn's test. Error bars represent standard error values around the mean.

**Ethics statement.** All experimental protocols were approved by the University of Newcastle (UON) Animal Care and Ethics Committee (Ethics Number 2014-447) and were performed in accordance with national and international guidelines, including the NSW Animal Research Act 1998, NSW Animal Research Regulation 2010 and the Australian Code for the Care and Use of Animals for Scientific Purposes 8th Ed.

### Data availability

All data generated or analyzed during this study are included in this published article.

Received: 14 June 2019; Accepted: 5 November 2019;

Published online: 25 November 2019

### References

- Houston, B. J., Nixon, B., King, B. V., De Iulius, G. N. & Aitken, R. J. The effects of radiofrequency electromagnetic radiation on sperm function. *Reproduction* **152**, R263–R276, <https://doi.org/10.1530/REP-16-0126> (2016).
- Dasdag, S., Akdag, M. Z., Ulukaya, E., Uzunlar, A. K. & Ocak, A. R. Effect of mobile phone exposure on apoptotic glial cells and status of oxidative stress in rat brain. *Electromagn Biol Med* **28**, 342–354, <https://doi.org/10.3109/15368370903206556> (2009).
- Demirel, S. *et al.* Effects of third generation mobile phone-emitted electromagnetic radiation on oxidative stress parameters in eye tissue and blood of rats. *Cutan Ocul Toxicol* **31**, 89–94, <https://doi.org/10.3109/15569527.2012.657725> (2012).
- Khalil, A. M., Abu Khadra, K. M., Aljaberi, A. M., Gagaa, M. H. & Issa, H. S. Assessment of oxidant/antioxidant status in saliva of cell phone users. *Electromagn Biol Med* **33**, 92–97, <https://doi.org/10.3109/15368378.2013.783855> (2014).
- Marchionni, I. *et al.* Comparison between low-level 50 Hz and 900 MHz electromagnetic stimulation on single channel ionic currents and on firing frequency in dorsal root ganglion isolated neurons. *Biochim Biophys Acta* **1758**, 597–605, <https://doi.org/10.1016/j.bbame.2006.03.014> (2006).
- Masuda, H. *et al.* Effect of GSM-900 and -1800 signals on the skin of hairless rats. I: 2-hour acute exposures. *Int J Radiat Biol* **82**, 669–674, <https://doi.org/10.1080/09553000600930079> (2006).
- De Iulius, G. N., Newey, R. J., King, B. V. & Aitken, R. J. Mobile phone radiation induces reactive oxygen species production and DNA damage in human spermatozoa *in vitro*. *PLoS One* **4**, e6446, <https://doi.org/10.1371/journal.pone.0006446> (2009).
- Narayanan, S. N., Kumar, R. S., Karun, K. M., Nayak, S. B. & Bhat, P. G. Possible cause for altered spatial cognition of prepubescent rats exposed to chronic radiofrequency electromagnetic radiation. *Metab Brain Dis* **30**, 1193–1206, <https://doi.org/10.1007/s11011-015-9689-6> (2015).
- Ozguner, F. *et al.* Mobile phone-induced myocardial oxidative stress: protection by a novel antioxidant agent caffeic acid phenethyl ester. *Toxicol Ind Health* **21**, 223–230, <https://doi.org/10.1191/0748233705th228oa> (2005).
- Salford, L. G., Brun, A., Sturesson, K., Eberhardt, J. L. & Persson, B. R. Permeability of the blood-brain barrier induced by 915 MHz electromagnetic radiation, continuous wave and modulated at 8, 16, 50, and 200 Hz. *Microsc Res Tech* **27**, 535–542, <https://doi.org/10.1002/jemt.1070270608> (1994).
- Hou, Q. *et al.* Oxidative changes and apoptosis induced by 1800-MHz electromagnetic radiation in NIH/3T3 cells. *Electromagn Biol Med* **34**, 85–92, <https://doi.org/10.3109/15368378.2014.900507> (2015).
- Houston, B. J., Nixon, B., King, B. V., Aitken, R. J. & De Iulius, G. N. Probing the Origins of 1,800 MHz radio frequency electromagnetic radiation induced damage in mouse immortalized germ cells and spermatozoa *in vitro*. *Front Public Health* **6**, 270, <https://doi.org/10.3389/fpubh.2018.00270> (2018).
- Yao, K. *et al.* Electromagnetic noise inhibits radiofrequency radiation-induced DNA damage and reactive oxygen species increase in human lens epithelial cells. *Mol Vis* **14**, 964–969 (2008).
- Adams, J. A., Galloway, T. S., Mondal, D., Esteves, S. C. & Mathews, F. Effect of mobile telephones on sperm quality: a systematic review and meta-analysis. *Environ Int* **70**, 106–112, <https://doi.org/10.1016/j.envint.2014.04.015> (2014).
- Agarwal, A. *et al.* Effects of radiofrequency electromagnetic waves (RF-EMW) from cellular phones on human ejaculated semen: an *in vitro* pilot study. *Fertil Steril* **92**, 1318–1325, <https://doi.org/10.1016/j.fertnstert.2008.08.022> (2009).
- Aitken, R. J., Smith, T. B., Jobling, M. S., Baker, M. A. & De Iulius, G. N. Oxidative stress and male reproductive health. *Asian J Androl* **16**, 31–38, <https://doi.org/10.4103/1008-682X.122203> (2014).
- Aitken, R. J. Human spermatozoa: revelations on the road to conception. *F1000Prime Rep* **5**, 39, <https://doi.org/10.12703/P5-39> (2013).
- Erogul, O. *et al.* Effects of electromagnetic radiation from a cellular phone on human sperm motility: an *in vitro* study. *Arch Med Res* **37**, 840–843, <https://doi.org/10.1016/j.arcmed.2006.05.003> (2006).
- Liu, C. *et al.* Exposure to 1800 MHz radiofrequency electromagnetic radiation induces oxidative DNA base damage in a mouse spermatocyte-derived cell line. *Toxicol Lett* **218**, 2–9, <https://doi.org/10.1016/j.toxlet.2013.01.003> (2013).

20. Liu, C. *et al.* Mobile phone radiation induces mode-dependent DNA damage in a mouse spermatocyte-derived cell line: a protective role of melatonin. *Int J Radiat Biol* **89**, 993–1001, <https://doi.org/10.3109/09553002.2013.811309> (2013).
21. Zalata, A., El-Samanoudy, A. Z., Shaalan, D., El-Baiomy, Y. & Mostafa, T. *In vitro* effect of cell phone radiation on motility, DNA fragmentation and clusterin gene expression in human sperm. *Int J Fertil Steril* **9**, 129–136 (2015).
22. Ghanbari, M., Mortazavi, S. B., Khavanin, A. & Khazaei, M. The Effects of Cell Phone Waves (900 MHz-GSM Band) on Sperm Parameters and Total Antioxidant Capacity in Rats. *Int J Fertil Steril* **7**, 21–28 (2013).
23. Puranen, L. *et al.* Space efficient system for whole-body exposure of unrestrained rats to 900 MHz electromagnetic fields. *Bioelectromagnetics* **30**, 120–128, <https://doi.org/10.1002/bem.20449> (2009).
24. Avci, B., Akar, A., Bilgici, B. & Tuncel, O. K. Oxidative stress induced by 1.8 GHz radio frequency electromagnetic radiation and effects of garlic extract in rats. *Int J Radiat Biol* **88**, 799–805, <https://doi.org/10.3109/09553002.2012.711504> (2012).
25. Irmak, M. K. *et al.* Effects of electromagnetic radiation from a cellular telephone on the oxidant and antioxidant levels in rabbits. *Cell Biochem Funct* **20**, 279–283, <https://doi.org/10.1002/cbf.976> (2002).
26. Meral, I. *et al.* Effects of 900-MHz electromagnetic field emitted from cellular phone on brain oxidative stress and some vitamin levels of guinea pigs. *Brain Res* **1169**, 120–124, <https://doi.org/10.1016/j.brainres.2007.07.015> (2007).
27. Yurekli, A. I. *et al.* GSM base station electromagnetic radiation and oxidative stress in rats. *Electromagn Biol Med* **25**, 177–188, <https://doi.org/10.1080/15368370600875042> (2006).
28. Aitken, R. J. *et al.* On methods for the detection of reactive oxygen species generation by human spermatozoa: analysis of the cellular responses to catechol oestrogen, lipid aldehyde, menadione and arachidonic acid. *Andrology* **1**, 192–205, <https://doi.org/10.1111/j.2047-2927.2012.00056.x> (2013).
29. Al-Damegh, M. A. Rat testicular impairment induced by electromagnetic radiation from a conventional cellular telephone and the protective effects of the antioxidants vitamins C and E. *Clinics (Sao Paulo)* **67**, 785–792 (2012).
30. Dasdag, S. *et al.* Whole-body microwave exposure emitted by cellular phones and testicular function of rats. *Urol Res* **27**, 219–223 (1999).
31. Evaluation of oxidant stress and antioxidant defense in discrete brain regions of rats exposed to 900 MHz radiation. *Bratisl Lek Listy* **115**, 260–266 (2014).
32. Burlaka, A. *et al.* Overproduction of free radical species in embryonal cells exposed to low intensity radiofrequency radiation. *Exp Oncol* **35**, 219–225 (2013).
33. Kahya, M. C., Naziroglu, M. & Cig, B. Selenium reduces mobile phone (900 MHz)-induced oxidative stress, mitochondrial function, and apoptosis in breast cancer cells. *Biol Trace Elem Res* **160**, 285–293, <https://doi.org/10.1007/s12011-014-0032-6> (2014).
34. Wang, H. & Zhang, X. Magnetic Fields and Reactive Oxygen Species. *Int J Mol Sci* **18**, <https://doi.org/10.3390/ijms18102175> (2017).
35. Ozorak, A. *et al.* Wi-Fi (2.45 GHz)- and mobile phone (900 and 1800 MHz)-induced risks on oxidative stress and elements in kidney and testis of rats during pregnancy and the development of offspring. *Biol Trace Elem Res* **156**, 221–229, <https://doi.org/10.1007/s12011-013-9836-z> (2013).
36. Hermo, L., Pelletier, R. M., Cyr, D. G. & Smith, C. E. Surfing the wave, cycle, life history, and genes/proteins expressed by testicular germ cells. Part 4: intercellular bridges, mitochondria, nuclear envelope, apoptosis, ubiquitination, membrane/voltage-gated channels, methylation/acetylation, and transcription factors. *Microsc Res Tech* **73**, 364–408, <https://doi.org/10.1002/jemt.20785> (2010).
37. Meinhardt, A., McFarlane, J. R., Seitz, J. & de Kretser, D. M. Activin maintains the condensed type of mitochondria in germ cells. *Mol Cell Endocrinol* **168**, 111–117 (2000).
38. Ramalho-Santos, J. *et al.* Mitochondrial functionality in reproduction: from gonads and gametes to embryos and embryonic stem cells. *Hum Reprod Update* **15**, 553–572, <https://doi.org/10.1093/humupd/dmp016> (2009).
39. Aitken, R. J. *et al.* Proteomic changes in mammalian spermatozoa during epididymal maturation. *Asian J Androl* **9**, 554–564, <https://doi.org/10.1111/j.1745-7262.2007.00280.x> (2007).
40. O'Flaherty, C. Peroxiredoxin 6: The Protector of Male Fertility. *Antioxidants (Basel)* **7**, <https://doi.org/10.3390/antiox7120173> (2018).
41. Pereira, R., Sa, R., Barros, A. & Sousa, M. Major regulatory mechanisms involved in sperm motility. *Asian J Androl* **19**, 5–14, <https://doi.org/10.4103/1008-682X.167716> (2017).
42. Baker, M. A. *et al.* Defining the mechanisms by which the reactive oxygen species by-product, 4-hydroxynonenal, affects human sperm cell function. *Biol Reprod* **92**, 108, <https://doi.org/10.1095/biolreprod.114.126680> (2015).
43. Gorpinchenko, I., Nikitin, O., Banyra, O. & Shulyak, A. The influence of direct mobile phone radiation on sperm quality. *Cent European J Urol* **67**, 65–71, <https://doi.org/10.5173/cej.2014.01.art14> (2014).
44. Smith, T. B. *et al.* The presence of a truncated base excision repair pathway in human spermatozoa that is mediated by OGG1. *J Cell Sci* **126**, 1488–1497, <https://doi.org/10.1242/jcs.121657> (2013).
45. Liu, D. Y. & Baker, H. W. Human sperm bound to the zona pellucida have normal nuclear chromatin as assessed by acridine orange fluorescence. *Hum Reprod* **22**, 1597–1602, <https://doi.org/10.1093/humrep/dem044> (2007).
46. Sommer, A. M. *et al.* Effects of radiofrequency electromagnetic fields (UMTS) on reproduction and development of mice: a multi-generation study. *Radiat Res* **171**, 89–95, <https://doi.org/10.1667/RR1460.1> (2009).
47. Zhou, W. *et al.* Developmental expression of the dynamin family of mechanoenzymes in the mouse epididymis. *Biol Reprod* **96**, 159–173, <https://doi.org/10.1095/biolreprod.116.145433> (2017).
48. Katen, A. L., Stanger, S. J., Anderson, A. L., Nixon, B. & Roman, S. D. Chronic acrylamide exposure in male mice induces DNA damage to spermatozoa; Potential for amelioration by resveratrol. *Reprod Toxicol* **63**, 1–12, <https://doi.org/10.1016/j.reprotox.2016.05.004> (2016).
49. Walsh, A. *et al.* Identification of the molecular chaperone, heat shock protein 1 (chaperonin 10), in the reproductive tract and in capacitating spermatozoa in the male mouse. *Biol Reprod* **78**, 983–993, <https://doi.org/10.1095/biolreprod.107.066860> (2008).
50. Smith, T. B., Baker, M. A., Connaughton, H. S., Habenicht, U. & Aitken, R. J. Functional deletion of Txndc2 and Txndc3 increases the susceptibility of spermatozoa to age-related oxidative stress. *Free Radic Biol Med* **65**, 872–881, <https://doi.org/10.1016/j.freeradbiomed.2013.05.021> (2013).
51. Houston, B. J. *et al.* Heat exposure induces oxidative stress and DNA damage in the male germ line. *Biol Reprod* **98**, 593–606, <https://doi.org/10.1093/biolre/iox009> (2018).
52. Katen, A. L., Chambers, C. G., Nixon, B. & Roman, S. D. Chronic Acrylamide Exposure in Male Mice Results in Elevated DNA Damage in the Germline and Heritable Induction of CYP2E1 in the Testes. *Biol Reprod* **95**, 86, <https://doi.org/10.1095/biolreprod.116.139535> (2016).
53. Nixon, B. *et al.* Elucidation of the signaling pathways that underpin capacitation-associated surface phosphotyrosine expression in mouse spermatozoa. *J Cell Physiol* **224**, 71–83, <https://doi.org/10.1002/jcp.22090> (2010).
54. Reid, A. T. *et al.* Dynamin regulates specific membrane fusion events necessary for acrosomal exocytosis in mouse spermatozoa. *J Biol Chem* **287**, 37659–37672, <https://doi.org/10.1074/jbc.M112.392803> (2012).
55. Martin, J. H., Nixon, B., Lord, T., Bromfield, E. G. & Aitken, R. J. Identification of a key role for permeability glycoprotein in enhancing the cellular defense mechanisms of fertilized oocytes. *Dev Biol* **417**, 63–76, <https://doi.org/10.1016/j.ydbio.2016.06.035> (2016).

## Acknowledgements

The authors wish to thank Hayley Boyce for accommodating all equipment within the University of Newcastle animal facility. This research was supported by a National Health and Medical Research Council of Australia Project Grant (APP1156997) to G.N.D., B.N., B.V.K. and R.J.A. and B.H. was the recipient of an Australian Postgraduate Award scholarship.

## Author contributions

B.J.H., B.N., G.N.D. and R.J.A. designed the experimental regime. B.V.K. constructed and tested the waveguide apparatus. B.J.H. performed all experiments with assistance from K.E.M. and J.H.M. B.J.H. drafted the initial manuscript, which was edited by B.N., R.J.A. and G.N.D.

## Competing interests

The authors declare no competing interests.

## Additional information

**Correspondence** and requests for materials should be addressed to G.N.D.I.

**Reprints and permissions information** is available at [www.nature.com/reprints](http://www.nature.com/reprints).

**Publisher's note** Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.



**Open Access** This article is licensed under a Creative Commons Attribution 4.0 International License, which permits use, sharing, adaptation, distribution and reproduction in any medium or format, as long as you give appropriate credit to the original author(s) and the source, provide a link to the Creative Commons license, and indicate if changes were made. The images or other third party material in this article are included in the article's Creative Commons license, unless indicated otherwise in a credit line to the material. If material is not included in the article's Creative Commons license and your intended use is not permitted by statutory regulation or exceeds the permitted use, you will need to obtain permission directly from the copyright holder. To view a copy of this license, visit <http://creativecommons.org/licenses/by/4.0/>.

© The Author(s) 2019

**Expert Report**  
**Christopher J. Portier, Ph.D.**

## Table of Contents

<b>Table of Contents</b> .....	<b>2</b>
<b>Listing of Figures</b> .....	<b>3</b>
<b>Listing of Tables</b> .....	<b>3</b>
<b>1. Charge</b> .....	<b>5</b>
<b>2. Qualifications</b> .....	<b>5</b>
<b>3. Explanation of Bradford Hill Causality Evaluation</b> .....	<b>7</b>
<b>4. Human Epidemiology</b> .....	<b>9</b>
<b>The evidence on an association between cellular phone use and the risk of glioma and/or acoustic neuroma in adults is strong</b> .....	<b>9</b>
<b>4.1 Glioma</b> .....	<b>9</b>
4.1.1 Studies in Adults .....	9
4.1.2 Studies in Children .....	29
4.1.3 Discussion .....	32
4.1.4 Ecological Epidemiology Studies of Malignant Brain Tumors and Gliomas .....	46
4.1.5 Conclusions for Gliomas .....	51
<b>4.2 Acoustic Neuromas</b> .....	<b>52</b>
4.2.1 Studies in Adults .....	52
4.2.2 Studies in Children .....	63
4.2.3 Discussion .....	63
4.2.4 Ecological Epidemiology Studies of Acoustic Neuroma .....	72
4.2.5 Conclusions for Acoustic Neuromas .....	72
<b>Laboratory Cancer Studies</b> .....	<b>72</b>
<b>5.1 Chronic Carcinogenicity Studies</b> .....	<b>72</b>
5.1.1 Mice .....	72
5.1.2 Rats .....	74
<b>5.2 Transgenic and Tumor-Prone Models</b> .....	<b>78</b>
5.2.1 E $\mu$ -pim1 transgenic mouse .....	78
5.2.2 Patched1 <sup>-/-</sup> Mice .....	79
5.2.3 AKR/j Mouse .....	79
5.2.3 C3H Mice .....	80
<b>5.3 Initiation-Promotion Studies</b> .....	<b>81</b>
5.3.1 Skin Models .....	81
5.3.2 Lymphoma Models .....	82
5.3.3 Mammary-gland Tumor Model .....	82
5.3.4 Brain tumor models .....	83
5.3.5 Liver Tumor Models .....	84
<b>5.4 Co-Carcinogenesis</b> .....	<b>85</b>
<b>5.5 Summary and Conclusions for Laboratory Cancer Studies</b> .....	<b>86</b>
<b>6. Mechanisms Related to Carcinogenicity</b> .....	<b>91</b>
6.1 Introduction .....	91
6.2 Oxidative Stress.....	92
6.2.1 Introduction .....	92

6.2.2 International Agency for Research on Cancer (IARC).....	93
6.2.3 <i>In vivo</i> Studies in Mammals, 2011-2020.....	93
6.2.4 <i>In Vitro</i> Studies in Mammalian Cells.....	100
6.2.5 Summary for Oxidative Stress .....	102
<b>6.3 Genotoxicity.....</b>	<b>103</b>
6.3.1 Introduction.....	103
6.3.2 International Agency for Research on Cancer (IARC).....	104
6.3.3 <i>In Vivo</i> Studies in Mammals .....	104
6.3.4 <i>In Vitro</i> Studies in Mammalian Cells.....	107
6.3.5 Summary for Genotoxicity.....	109
<b>6.3. Summary for Mechanisms of Carcinogenicity .....</b>	<b>109</b>
<b>7. Summary of Bradford Hill Evaluation.....</b>	<b>109</b>
<b>8. References Cited .....</b>	<b>112</b>
<b>Appendix I: Current CV: Christopher J. Portier.....</b>	<b>146</b>
<b>Appendix II: Previous Cases Resulting in Depositions and Court Appearances.....</b>	<b>175</b>
<b>Appendix III: Compensation .....</b>	<b>176</b>
<b>Certification.....</b>	<b>176</b>

## Listing of Figures

Figure 1: Forest plot and meta-analyses of regular use or ever use of cellular telephones and the risk of glioma [studies with a solid blue square either single studies that stand alone or pooled studies that encompass numerous single studies; open squares are individual studies or smaller pooled studies; red diamonds are meta-analyses] <sup>a</sup> .....	40
Figure 2: Forest plot and meta-analyses of duration of use of cellular telephones and the risk of glioma [studies with a solid blue square are either single studies that stand alone or pooled studies that encompass numerous single studies; open squares are a second analysis from that same paper; red diamonds are meta-analyses, the columns and the figure are as in Figure 1].....	41
Figure 3: Forest plot and meta-analyses of regular use or ever use of cellular telephones and the risk of acoustic neuroma [studies with a solid blue square either single studies that stand alone or pooled studies that encompass numerous single studies; open squares are individual studies or smaller pooled studies; red diamonds are meta-analyses] <sup>a</sup> .....	66
Figure 4: Forest plot and meta-analyses of duration of use of cellular telephones and the risk of acoustic neuroma [studies with a solid blue square are stand alone; red diamonds are meta-analyses, the columns and the figure are as in Figure 1] .....	68
Figure 5: Exogenous and endogenous stimuli leading to ROS generation and activation of stress-sensitive gene expression. (modified from [232]) .....	93

## Listing of Tables

Table 1: Results from epidemiology studies for ever versus never or regular versus non-regular use of a cellular telephone and the risk of glioma in adults .....	21
---	----

Table 2: Results from epidemiology studies for duration (years) of use of a cellular telephone and the risk of glioma in adults.....	22
Table 3: Results from epidemiology studies for duration (cumulative hours) of use of a cellular telephone and the risk of glioma in adults .....	24
Table 4: Results from epidemiology studies for average daily or monthly use of a cellular telephone and the risk of glioma in adults .....	25
Table 5: Results from epidemiology studies for other use measures of a cellular telephone and the risk of glioma in adults.....	26
Table 6: Results from epidemiology studies for laterality of cellular telephone use and the risk of glioma in adults .....	27
Table 7: Results from epidemiology studies for cellular telephone use and the location of glioma in adults.....	28
Table 8: Results from epidemiology studies RF and brain tumors in children and adolescents .....	31
Table 9: Meta-Regression Exposure Values for Tables 11 and 12.....	44
Table 10: Meta-Regression Analysis with Sensitivity Analysis of ORs for Five Case-Control Studies using Cumulative Hours of Use as the Exposure Metric and the Original Referent Groups.....	44
Table 11: Meta-Regression Analysis <sup>a</sup> with Sensitivity Analysis of ORs for Five Case-Control Studies using Cumulative Hours of Use as the Exposure Metric and the Alternative Referent Group for the Interphone Study .....	45
Table 12: Results from epidemiology studies for ever versus never or regular versus non-regular use of a cellular telephone and the risk of acoustic neuroma in adults .....	56
Table 13: Results from epidemiology studies for time (years) since first use of a cellular telephone and the risk of Acoustic Neuroma in adults .....	57
Table 14: Results from epidemiology studies for duration (cumulative hours) of use of a cellular telephone and the risk of acoustic neuroma in adults .....	58
Table 15: Results from epidemiology studies for average daily or monthly use of a cellular telephone and the risk of acoustic neuroma in adults .....	59
Table 16: Results from epidemiology studies for other use measures of a cellular telephone and the risk of acoustic neuroma in adults.....	60
Table 17: Results from epidemiology studies for laterality of cellular telephone use and the risk of acoustic neuroma in adults .....	61
Table 18: Meta-Regression Exposure Values for Table 19.....	70
Table 19: Meta-Regression Analysis with Sensitivity Analysis of ORs for Five Case-Control Studies using Cumulative Hours of Use as the Exposure Metric and the Original Referent Groups.....	70
Table 20: Summary of Chronic Exposure Carcinogenicity Studies for Radiofrequency Radiation.....	89
Table 21: Key characteristics of carcinogens, Smith et al. (2016)[65].....	91

Table 22: Summary conclusions for Hill's nine aspects of epidemiological data and related science.....	110
--	-----

## 1. Charge

Mobile or cellular phones, cellular towers and wi-fi base stations are sources of radiofrequency electromagnetic field (RF-EMF or simply RF) exposure to humans. This exposure falls predominantly in the range of 850 to 2500 megahertz (MHz). Epidemiological studies have suggested that exposure to RF is associated with an increased risk of brain tumors (glioma, acoustic neuroma) in humans. After evaluating the body of existing scientific research and literature including very recent studies, I have now developed the conclusions set forth in this report on whether it is feasible that RF exposure can cause specific brain tumors in humans.

## 2. Qualifications

I received an undergraduate degree in mathematics in 1977 from Nicholls State University and a Master's degree and Ph.D. in biostatistics from the University of North Carolina School of Public Health in 1979 and 1981 respectively. My Ph.D. thesis addressed the optimal way to design a two-year rodent carcinogenicity study to assess the ability of a chemical to cause cancer[1, 2]; the optimal dosing pattern from my thesis is still used by most researchers. My first employment following my doctoral degree was a joint appointment at the National Institute of Environmental Health Sciences (NIEHS) and the National Toxicology Program (NTP) to conduct research on the design and analysis of experiments generally employed in toxicology. After 5 years with NIEHS/NTP, I developed my own research group which eventually became the Laboratory of Quantitative and Computational Biology and then the Laboratory of Computational Biology and Risk Assessment (LCBRA). One highlight during this period was the development of the Poly-3 Test for survival adjustment of data from two-year carcinogenicity studies in rodents [3, 4]; this test is used as the main method of analysis of these studies by the NTP and many others. We also did a complete analysis of the historical controls animals from the NTP studies [5, 6]. The LCBRA focused on the application of computational tools to identify chemicals that are toxic to humans, to develop tools for understanding the mechanisms underlying those toxicities and to quantify the risks to humans associated with these toxicities. The main toxicological focus of the LCBRA was cancer and my laboratory developed many methods for applying multistage models to animal cancer data and implemented the use of these models in several experimental settings [7-19]. In my last few years at the NIEHS/NTP, my research focus expanded to the development of tools for evaluating the response of complex experimental and human systems to chemicals [20-24] and the name of the laboratory shifted to Environmental Systems Biology.

Over my 32 years with the NIEHS/NTP, I was involved in numerous national priority issues that went beyond my individual research activities. After Congress asked NIEHS to work with the Vietnamese government to address the hazards associated with Agent Orange use during the Vietnamese War, I was given the responsibility of working with my counterparts in Vietnam to build a research program in this area [25]. Congress also tasked NIEHS with

developing a research program (EMF-RAPID) to address concerns about the risks to humans from exposure to extremely low frequency electric and magnetic fields (ELF-EMF) from power lines and to report back to Congress on what we found. I was in charge of evaluating all research developed under this program and was responsible for the final recommendations to Congress on this issue [26-28].

While at the NIEHS/NTP, I also had administrative positions that relate to my qualifications. From 2000 to 2006 I was the Director of the Environmental Toxicology Program (ETP) at NIEHS. The ETP included all of the toxicology research laboratories within the NIEHS Intramural Research Program. It was my responsibility to ensure the research being done was pertinent to the mission of the NIEHS, addressing high priority concerns about toxic substances and human health and that the NIEHS had adequate resources to complete this research.

During this time I was also Associate Director of the NTP, a position in which I was the scientific and administrative director of the NTP (The Director of the NTP was also the NIEHS Director and gave me complete autonomy in the management and science of the NTP). These two positions were historically always combined at the NIEHS and the NTP so that one person was in charge of all toxicological research at the NIEHS/NTP. The NTP is the world's largest toxicology program, routinely having 15 to 25 active two-year carcinogenicity studies, numerous genetic toxicology studies and many other toxicological studies being conducted at any given time. The NTP two-year carcinogenicity studies and their technical reports are also considered the "gold standard" of cancer studies due to their extreme high quality, their tremendous utility in evaluating human health hazards and the rigor and transparency they bring to the evaluation of the data. All data from NTP two-year cancer studies are publicly available including data on individual animals and images from the pathology review of each animal. The NTP is also home to the Report on Carcinogens, the US Department of Health and Human Services official list of what is known or reasonably anticipated to be carcinogenic to humans. It was my responsibility to decide what items eventually went onto this list while I was Associate Director of the NTP. In 2006, I became an Associate Director of the NIEHS, a senior advisor to the director and the director of the Office of Risk Assessment Research (ORAR). ORAR focused on stimulating new research areas on the evaluation of health risks from the environment and addressed major risk assessment issues on behalf of the NIEHS/NTP. For example, in this capacity, I lead a multiagency effort to understand the health risks to humans from climate change and to develop a research program in this area [29].

I left the NIEHS/NTP in 2010 to become the Director of the National Center for Environmental Health (NCEH) at the Centers for Disease Control and Prevention and simultaneously Director of the Agency for Toxic Substances and Disease Registry (ATSDR). NCEH does research and supports activities aimed at reducing the impact of environmental hazards on public health. One well-respected research effort of the NCEH is the National Biomonitoring Program. This program tests for the presence of hundreds of chemicals in human blood and urine in a national sample of people in the United States. ATSDR advises the Environmental Protection Agency (EPA) and communities on the potential health impacts from toxic waste dump sites (superfund sites). ATSDR is required by law to produce ToxProfiles. These are comprehensive reviews of the scientific literature for specific chemicals generally found at superfund sites. They also provide an assessment of the safety of these chemicals. As part of my activities at ATSDR, I began a modernization of the

ToxProfiles to use systematic review methods in their assessments; this effort was linked to a similar effort that I had helped to implement at the NIEHS/NTP.

Aside from my official duties in my various federal jobs, I also served on numerous national and international science advisory panels. Most notable, for my qualifications for this statement, are my serving as Chair from 2005 to 2010 of the Subcommittee on Toxics and Risk of the President's National Science and Technology Council, member and chair of EPA'S Science Advisory Panel from 1998 to 2003 (focused specifically on advising their pesticides program) and chair of the International Agency for Research on Cancer (IARC) advisory group that updated and improved its rules for reviewing scientific data to ensure that conclusions on the carcinogenicity of human exposures are the best possible (Preamble) [30]. As part of my work on science advisory panels, I have served on EPA's Science Advisory Board, as an advisor to the Australian Health Council on risk assessment methods, as an advisor to the Korean Food and Drug Administration on toxicological methods and served on several World Health Organization (WHO) International Program on Chemical Safety scientific panels dealing with risk assessment. Besides the guidelines for evaluating cancer hazards used by the IARC, I have either chaired or served as a member of scientific panels developing guidance documents for other organizations including the EPA.

I have received numerous awards, most notably the Outstanding Practitioner Award from the International Society for Risk Analysis and the Paper of the Year Award (twice) from the Society of Toxicology Risk Assessment Specialty Section. I am a fellow of the American Statistical Association, the International Statistical Institute, the World Innovation Foundation and the Ramazzini Institute. I have published over 250 peer-reviewed scientific papers, book chapters and technical documents on topics in toxicology and risk assessment. Finally, I have served on numerous national and international committees tasked with evaluating the risk and/or hazard of specific environmental chemicals, including RF exposure. For example, I have contributed to risk assessments for EPA, the Food and Drug Administration, the Centers for Disease Control and Prevention, the National Institutes of Health, the WHO and IARC.

### 3. Explanation of Bradford Hill Causality Evaluation

***Most of the guidelines [31-33] used for cancer risk assessment trace their origins to a paper by Hill (1965) [34]. The IARC review of RF [35] followed guidelines derived from Hill (1965) and concluded RF exposure was "possibly carcinogenic to humans".***

The evaluation of whether RF exposure can cause brain tumors in humans requires the review and synthesis of scientific evidence from studies of human populations (epidemiology), animal cancer studies, and studies investigating the mechanisms through which chemicals cause cancer. Many different approaches[36, 37] are used to synthesize these three areas of science to answer the question "Does this chemical/agent cause cancer in humans?" In any of these three science areas, the quality of the individual studies has to be assessed and summarized to make certain the studies included in the overall assessment are done appropriately. Once the quality of the individual studies has been assessed, a judgment needs to be made concerning the degree to which the studies support a finding of cancer in humans. To do this, the EPA, IARC, the European Chemical Agency (EChA), the US Report on Carcinogens, and many others use guidelines [30, 31, 33, 38] that rely upon aspects of the criteria for causality developed by Hill (1965) [34].

Hill listed nine (9) aspects of epidemiological studies and the related science that one should consider in assessing causality. The presence or absence of any of these aspects is neither sufficient nor necessary for drawing inferences of causality. Instead, the nine aspects serve as means to answer the question of whether other explanations are more credible than a causal inference. As noted by Hill:

*“None of my nine viewpoints can bring indisputable evidence for or against the cause-and-effect hypothesis and none can be required as a sine qua non. What they can do, with greater or less strength, is to help us to make up our minds on the fundamental question — is there any other way of explaining the set of facts before us, is there any other answer equally, or more, likely than cause and effect?”*

The nine aspects cited by Hill include consistency of the observed association, strength of the observed association, biological plausibility, biological gradient, temporal relationship of the observed association, specificity of the observed association, coherence, evidence from human experimentation and analogy. These are briefly described below.

An inference of causality is strengthened when several of the studies show a **consistent positive association** between cancer and the exposure. This addresses the key issue of replication of studies which is critical in most scientific debates. If studies are discordant, differences in study quality, potential confounding, potential bias and statistical power are considered to better understand that discordance.

An inference of causality is strengthened when the **strength of the observed association** in several studies are large and precise. These large, precise associations lessen the possibility that the observed associations are due to chance or bias. A small increase in risk of getting cancer does not preclude a causal inference since issues such as potency and exposure level may reduce the ability of a study to identify larger risks. Meta-analyses provide an objective evaluation of the strength of the observed association across several studies with modest risks to help clarify strength of the observed associations.

An inference of causality is strengthened when there is data supporting **biological plausibility** demonstrated through experimental evidence. Animal carcinogenicity studies, in which tumor incidence is evaluated in experimental animals exposed to RF, play a major role in establishing biological plausibility. There are numerous types of mechanisms that can lead to cancer [39], most of which can be demonstrated through experimental studies in animals, human cells, animal cells, and/or other experimental systems. Occasionally, occupational, accidental or unintended exposures to humans allow researchers to evaluate mechanisms using direct human evidence.

An inference of causality is strengthened when there is a **biological gradient** showing a reasonable pattern of changing risk with changes in exposure (e.g. risk increases with increasing exposure or with longer exposure). In many epidemiological studies, this aspect cannot be examined due to limitations in the study design or due to a lack of clarity in the presentation of the results. When a study does address an exposure-response relationship, failure to find a relationship can be due to a small range of exposures, insufficient sample size or a changing exposure magnitude over time that has not been accounted for.

An inference of causality is strengthened when there is a **temporal relationship** in which the exposure comes before the cancer. This aspect is necessary to show causality; if it is not

present, a causal inference is not plausible. Because the latency period for cancers can be long (years), evaluation of studies should consider whether the exposure occurred sufficiently long ago to be associated with cancer development.

An inference of causality is strengthened when the exposure is **specific** for a given cancer. This would mean that the disease endpoint being studied is only due to the cause being assessed or that, even though many different cancers have been studied for an association with a given exposure, only one type of cancer shows a consistent association for the exposure of interest.

An inference of causality is strengthened when other lines of experimental evidence are **coherent** with a causal interpretation of the association seen in the epidemiological evidence. To evaluate coherence, information from animal carcinogenicity studies, and mechanistic investigations would be considered.

An inference of causality is strengthened when there is **experimental evidence in humans** supporting a causal interpretation. Seldom is this type of information available when addressing the toxicity of environmental exposures. However, experiments in which an individual reduces or limits exposures and the risk of cancer is reduced would carry considerable weight in the evaluation (e.g. studies evaluating the cancer risks of people who stop cigarette smoking compared with continuing smoking have demonstrated reduced lung cancer risks). No such data are available for RF exposures.

Finally, an inference of causality is strengthened when there are other agents with **analogous** characteristics showing similar effects in humans and/or animals and/or showing similar biological impacts in mechanistic studies.

The most logical approach to developing an inference of causality is to step through each of the aspects of causality developed **by Hill (1965)** [34] and apply them to the available data for RF exposures. This is done after a review of the relevant literature from human epidemiology studies, animal cancer studies, and mechanistic studies.

## 4. Human Epidemiology

**The evidence on an association between cellular phone use and the risk of glioma and/or acoustic neuroma in adults is strong.**

### 4.1 Glioma

#### 4.1.1 Studies in Adults

##### 4.1.1.1 Case-Control Studies

**Muscat et al. (2000)** [40] conducted a case-control study of cancers of the brain in five academic medical centers in the US from 1994-1998. Cases consisted of 469 patients with brain cancers (mainly glioma patients) and 422 controls matched from the same medical center as the cases. They basically saw no increased odds ratios for brain tumors overall or any subtype with the exception of neuroepitheliomatous tumors (14 exposed cases) where they saw an odds-ratio of 2.1 (0.9-4.7). Only 35 patients had these tumors and 14 of these used cellular phones. (Note, these are tumors arising in the neuroepithelial cells which serve as somewhat pluripotent stem cells in the brain). This study has a small number of cases, exposures were low and for short duration, they were predominantly analog

exposures and many study participants had never used a cellular phone. (Table 1) (other related papers include [41-43]).

**Inskip et al. (2001)** [44] performed a case-control study of intracranial tumors of the nervous system (brain tumors) and cellular phone use from 1994-1998 from three hospitals in the United States (Boston Brigham and Women's Hospital, Phoenix St. Joseph's Hospital and Pittsburgh Western Pennsylvania Hospital). They had 782 cases (489 with glioma, 197 with meningioma, and 96 with acoustic neuroma) and 799 matching hospital controls. Controls were predominantly hospital admissions without tumors however there were some neoplastic controls (leukemia/lymphoma patients excluded). Regular use was defined as 2 calls per week. Usage of handheld cellular phones increased dramatically during the study (e.g. controls doubled usage from 1994 to 1998 from ~20% to ~40%). The cases were older than the controls. They saw no increases in any ORs for any analysis done in the study (use/no use, frequency of use, years of use, cumulative use, year of first use) or any linkage between predominant side of use and the side on which tumors appeared. The study was basically negative in all aspects. Like the previous study, exposures were low and for short duration, they were predominantly analog exposures and many study participants had never used a cellular phone. (Table 1, Table 2, Table 3, Table 4, Table 5, Table 6)

**Auvinen et al. (2002)** [45] conducted a case-control study of brain tumors in males and females aged 20-69 in 1996 from the Finish Cancer Registry. There were 398 brain tumors (198 gliomas, 129 meningiomas, and 72 other unspecified types) and 5 age- and sex-matched controls for each case. For gliomas, there were 172 cases (86% response) and 921 controls (93% response). Each subject in the study was linked to a list of all subscribers to mobile phone networks in Finland to determine exposure. The OR for gliomas and any mobile phone subscription was 1.5 (1.0-2.4) with increasing ORs for increasing years of subscription (1.2 (0.5-3.0) for <1 year, 1.6 (0.8-2.9) for 1-2 years and 1.7 (0.9-3.5) for ≥2 years, 1.2 (1.0-1.4) increase in OR per year). The increases seen for analog phones was larger than that seen for digital phones. The major strengths of this study are their linkage to cancer records and mobile phone subscription records. It was limited by its size, inability to look at subscriptions of greater than 2 years and inability to look at the frequency of phone usage. (Table 1, Table 2)

**Gousias et al. (2009)** [46] conducted a hospital-based case-control study for cerebral gliomas and various exposures. The study included 41 cases (persons referred to the Neurosurgery and Neurology departments of University Hospital of Ioannina and surrounding hospitals) and 82 controls (2 neurosurgery patients per case matched for age, gender and district of residence with cervical myelopathy or disk herniation). They used one measure for cell phone use; minute-years of exposure (undefined). Logistic regression gave an OR of 1.00 (0.99-1.01, p=0.56). All evaluations were adjusted for alcohol consumption, smoking and history of severe cranial trauma. This is a small study with limited statistical power. (Table 1, Table 2)

**Spinelli et al. (2010)** [47] conducted a hospital-based case-control study in France on malignant primary brain tumors and various exposures. The study included 122 cases (new cases between Jan. 2005 and Dec. 2005 in the public reference hospitals in Marseilles and St. Anne's Hospital in Toulon) and 122 controls (neurosurgery patients matched for age and gender with no cancer diagnosis). They evaluated cell phone use in hour-years (number of hours of subscription per month x number of years of use in categories). They show ORs of 0.86 (0.30-2.44) for less than 4 hour-years of exposure, 1.45 (0.75-2.80) for 4 to 36 hour-

years and 1.07 (0.41-2.82) for  $\geq 36$  hour-years of exposure. All evaluations were adjusted for sex and age. This is a small study with limited statistical power. (Table 1, Table 2)

The **INTERPHONE Study (IS)** [48] is a interview-based multi-center case-control study on the use of cellular phones and histologically-confirmed cases of glioma, meningioma or acoustic neuroma. The study had 16 study centers in 13 countries with a common protocol (Australia, Canada, Denmark, Finland, France, Germany, Israel, Italy, Japan, New Zealand, Norway, Sweden, and the U.K.). Participants were mostly between 30 and 59 years of age (differing a bit by country), lived in a major metropolitan region, and were recruited from candidates over a 2-4 year timeframe from 2000 to 2004. Population controls were randomly selected from population registries (part of Canada, Denmark, Finland, Germany, Italy, Norway and Sweden), electoral lists (Australia, part of Canada, France, New Zealand), patient lists (U.K.) or random-digit dialing (part of Canada, France, Japan). Controls were either individually matched to cases or frequency matched to cases on year of birth, sex and study region. Glioma and meningioma patients had one matched control and acoustic neuroma patients had 2 controls. All patients or their proxies were interviewed in person using a questionnaire. Some centers also included a few other tumors which will not be discussed here.

Numerous publications have resulted from this study for single countries [49-62], subsets of pooled countries [58, 63-66], and pooled analyses of the entire study [48, 67]. There were also numerous papers addressing methodological issues [68-75]. I will focus on the overall pooled results.

In the **IS (2010)** [48] study, the evaluation of the data is complicated, looking at four different ways to characterize exposure, three different types of referent populations, multiple sensitivity analyses and three different evaluations of tumor location relative to phone use. During the study period, the IS identified 3115 meningioma cases, 4301 glioma cases and 14354 controls. The IS eventually included 2708 glioma cases with 2972 matched controls and 2409 meningioma cases with 2662 matched controls resulting in participation rates of 64% (range 36-92%) among cases of glioma, 78% (56-92%) among meningioma cases and 53% (42-74%) among controls. Meningioma cases were predominantly female, glioma cases were predominantly male, mean age at diagnosis was 51 years for meningioma cases and 49 years for glioma cases and gliomas were diagnosed at a younger age than meningiomas.

The OR for meningiomas for regular users versus others was 0.79 (0.68-0.91) with four countries having individual ORs greater than 1. Breaking time since start of use into 4 categories yielded ORs below 1 for all categories (0.90, 0.77, 0.76, 0.83) and for cumulative number of calls with no hands-free device, divided into 10 categories, the ORs were also all below 1 with no obvious pattern (0.95, 0.62, 0.90, 0.80, 0.60, 0.81, 0.79, 0.92, 0.81, 0.80). Only for cumulative call time with no hands-free device was there a single OR $>1$  and only in the highest percentile of cumulative use with OR=1.15 (0.81-1.62) (0.90, 0.82, 0.69, 0.69, 0.75, 0.69, 0.71, 0.90, 0.76, 1.15). Digital phone users in the highest exposure category had a significant OR 1.84 (1.17-2.88) as did those who used both digital and analog phones OR=4.43 (1.42-13.9); analog-only phone users had an OR of 0.50 (0.25-0.99). When the data were divided into use 1-4 years before reference date (date of diagnosis), 5-9 years and  $\geq 10$  years, ORs in the highest quintile of cumulative use for the most recent groupings were greater than 1.0 (4.80 [1.49-15.4] for 1-4 years, 1.03 [0.65-1.65] for 5-9 years, 0.95 [0.56-1.63] for  $\geq 10$  years). The ORs for anatomical location were generally  $<1$  for most analyses.

When analyzing for ipsilateral use or contralateral use independently, all ORs were <1.0. The ratio of ORs for ipsilateral use to contralateral use were always above 1 using any of the exposure metrics suggesting there was some degree of discernment in the results. A case-case analysis based on methods from **Inskip et al. (2001)** [44] showed an OR of 1.07 (1.00-1.16).

The OR for gliomas for regular users versus others was 0.81 (0.70-0.94) with three countries having individual ORs greater than 1. For time since start of use, ORs were below 1 for all categories (0.62, 0.84, 0.81, 0.98) and for cumulative number of calls with no hands-free device, the ORs were also all below 1 with a slightly increasing pattern (0.74, 0.71, 0.76, 0.90, 0.78, 0.83, 0.71, 0.93, 0.96, 0.96). For cumulative call time with no hands-free device two categories had ORs>1 and only in the highest tertile was it significant with OR=1.40 (1.03-1.89) (0.70, 0.71, 1.05, 0.74, 0.81, 0.73, 0.76, 0.82, 0.71, 1.40). Digital phone users in the highest exposure cumulative call time category had an increased OR 1.46 (0.98-2.17) as did those who used analog phones OR=1.95 (1.08-3.54). When the data were divided into use 1-4 years before reference date (date of diagnosis), 5-9 years and ≥10 years, ORs in the highest quintile of cumulative use for the most recent groupings were greater than 1.0 (3.77 [1.25-11.4] for 1-4 years, 1.28 [0.84-1.95] for 5-9 years, 1.34 [0.90-2.01] for ≥10 years). The ORs for anatomical location were generally <1 for most analyses except in the temporal lobe where the highest exposures in all three exposure measures were >1 (1.36 [0.88-2.11] for time since start of use, 1.87 [1.09-3.22] for cumulative call time, and 1.10 [0.65-1.85] for cumulative number of calls). When analyzing for ipsilateral use or contralateral use independently, all ORs were <1.0 except the highest exposures in all three exposure measures (1.21 [0.82-1.80] for time since start of use, 1.96 [1.22-3.16] for cumulative call time, and 1.51 [0.91-2.51] for cumulative number of calls). The ratio of ORs for ipsilateral use to contralateral use were all above 1 using any of the exposure metrics except for one category of time since first use suggesting there was some degree of discernment in the results. These ratios increased in an exposure-dependent fashion for cumulative number of calls. A case-case analysis based on methods from **Inskip et al. (2001)** [44] showed an OR of 1.27 (1.19-1.37) and was 1.55 (1.24-1.99) for the highest decile of cumulative call time.

An extensive sensitivity analysis on 13 separate factors did not substantively change the results for gliomas or meningiomas.

The reason for the low ORs seen in the various analyses could not be established. The authors examined sampling bias as a reason, arguing cases may have been missed and that controls may not have represented the study base, but concluded this was unlikely. Selection bias and participation bias may have contributed to the lower ORs, but they were unlikely to explain it all [48, 74]. When never regular users were excluded from the analysis and the lowest exposure category was used as the reference category (in an attempt to reduce participation bias), most of the ORs for gliomas increased above unity. Most notably, all three ORs for time since start of use became significant (1.7 [1.2-2.4] for 2-4 years, 1.5 [1.1-2.2] for 5-10 years, and 2.2 [1.4-3.3] for >10 years).

Some subjects reported very high cell phone use (>5h/day) and this was more common in glioma cases than controls. Truncating these at 5h/day had no effect on the resulting ORs. Thus, although there was some evidence of overestimation by heavy users [71], it is unlikely to have a large impact on the ORs.

The main strengths of the IS are the large sample size, the use of population-based controls and the extensive analyses performed on the data. One major limitation, as with most case-controls studies, is the use of a questionnaire for obtaining exposure information and the possibility of recall bias. Using a small sample of participants from three countries, the authors compared self-reported mobile-phone use with operator-recorded data and saw very little differential exposure misclassification. A second limitation was the low participation rate. There was some evidence that controls who regularly used mobile phones were more likely to participate than those who never used mobile phones; this could lead to a reduction in the ORs in the various exposure categories. The analyses using the lowest exposure category as the referent partially addressed this issue. (Table 1, Table 2, Table 3, Table 5, Table 6, Table 7)

In an effort to better refine the exposure in the IS, **Cardis et al. (2011)** [63] developed an estimate of the radio frequency (RF) dose as the amount of mobile phone RF energy absorbed at the location of a brain tumor in a selection of cases from the IS. This measure is a function of the frequency band and the types of phones the subjects had used and is multiplied by the duration of use to determine the total specific energy absorbed at the location of the tumor (TCSE, J/kg). After applying these exposure measures to the 5 countries in the IS where they could get the necessary usage information and tumor location data [63], they saw slight increases in both the glioma and meningioma ORs compared to the cumulative duration of mobile-phone use seen in the larger analysis [48]. The most significant finding was in the highest exposure group with a 7-year lag yielding an OR of 1.91 (1.05-3.47).

**Grell et al. (2016)** [76] used a model for spatial distribution of glioma occurrence developed by **Grell et al (2015)** [77] to reanalyze the tumor location data and laterality using the data from **Cardis et al. (2011)** [63]. The cases consisted of the 792 regular mobile phone users who provided data on preferred side of phone use and the center location of their tumor mass. The statistical test has the null hypothesis that the chances of getting the tumor are independent of side of use (in their parlance, the alphas for the four distances from the phone are all equal to 1 against the ordered alternative) with three different analyses based on slightly different assumptions. The p-value for the hypothesis of no association with mobile phone use was <0.01 for all three models. Dichotomizing (one variable at a time) by sex, age, tumor grade, tumor size, and years of mobile phone use yielded  $p < 0.01$  in all cases. The only weakness of this study would be if recall bias is driving the choice of which side of the brain the phone is typically used.

**Cardis et al. (2011)** [63] also conducted a case-case analysis in which mobile phone use was compared between cases whose probable tumor location was in the most exposed part of the brain region versus cases where the location of the tumor was elsewhere. The most exposed area was defined as falling within the 3 dB exposure volume of the brain regardless of laterality of use [78]. The OR for gliomas in regular users versus not regular users was 1.35 (0.64-2.87). For time since start of use, the ORs were 1.37 (0.59-3.19) for 1-4 years, 0.72 (0.27-1.90) for 5-9 years and 2.80 (1.13-6.94) for  $\geq 10$  years. A similar pattern was seen for cumulative call time. Because this uses only cases, case-case analysis is likely to have very limited recall bias but could still have exposure misclassification which is likely to be non-differential and reduce the ORs toward 1.0.

**Larajavara et al. (2011)** [79] also conducted a case-case analysis using seven European countries from the IS (Denmark, Finland, Germany, Italy, Norway, Sweden, and Southeast

England). In this analysis, distance between the midpoint of the glioma and the mobile phone axis was used to compare cases. Using the direct distance measurement, there was little difference between mean distance for various exposures categories with all p-values exceeding 0.39. Classifying tumors as  $\leq 5$  cm from midpoint of the glioma to the mobile phone axis or not yielded ORs that were below 1 for all but one situation and none were statistically significant. They also did a case-specular analysis of these same data. In a case-specular analysis, a mirror image of the location of the glioma is projected across the midpoint of the axial and coronal planes to use as the control. An association of cell phone usage with gliomas would exist if the ORs increased with increasing exposure; this was not seen. Using distance instead of exposure dose could lead to greater exposure misclassification since most exposures occur in the area of the brain closest to the ear and is not evenly distributed along the phone axis [63].

**Hardell and colleagues** conducted five separate case-control studies in Sweden on the risks of malignant brain tumors and exposure to cellular telephones [80-85]. All of the studies used self-administered questionnaires to ascertain mobile phone use followed by supplementary phone interviews to verify information provided in the questionnaire. All studies obtained matching controls for living cases from the Swedish Population Registry matching on gender and 5-year age group, and matching controls for deceased cases were obtained from the Death Registry of Sweden matched for year of death, gender, 5-year age group and medical region. The first study, **Hardell et al. (1999)** [85], was a small study with 233 patients identified from records in two regions of Sweden from 1994 to 1996. This study was effectively negative, probably due to the short latency periods for cellular phone use (Table 1, Table 6).

The next two studies were conducted back-to-back and used the same basic methodology. **Hardell et al. (2002)** [83] was conducted on males and females, aged 20-80 years, who developed a malignant brain tumor between 1997-2000 in Uppsala-Orebro, Stockholm, Linköping and Göteborg; this study included 588 cases and 581 controls. Only cases that were alive at the time of the study were included in the evaluation. Ever use of an analog mobile phone showed an elevated OR for ipsilateral use of 1.85 (1.16-2.96) for malignant brain tumors. Digital phones showed a smaller OR for ipsilateral use of 1.59 (1.05-2.41). Multivariate analysis showed an elevated risk for all types of phones with confidence bounds that included 1. **Hardell et al. (2006a)** [81] was conducted in the same manner from 2000 to 2003 in Uppsala-Orebro and Linköping and included 317 cases and 692 controls. No participants in this study overlapped with the previous study [83] and, as before, only cases alive at the time of the study were included. The use of analog cell phones yielded an OR for malignant brain tumors of 2.6 (1.5-4.3) and increased to 3.5 (2.0-6.4) for >10-year latency and 6.2 (2.5-15) for >15-year latency. The use of digital cell phones yielded an OR of 1.9 (1.3-2.7) and increased to 2.9 (1.6-5.2) for >10-year latency. Other exposure metrics were provided, some of which were also significant. A third case-control study [80] was conducted using those who had died prior the start of the previous two studies. Deceased cases were matched with two controls, one who had died of cancer and one who had died of another cause. The study included 346 cases (75% response rate, 314 cases of glioma) and 619 controls (67% response rate, 74% response rate from cancer controls). The OR for all malignant brain tumors and use of a mobile phone was 1.3 (0.9-1.9) increasing to 2.4 (1.4-4.1) with a latency of >10 years. They saw increasing ORs with increasing cumulative lifetime use (1.2 [0.8-1.8] for 1-1,000h, 2.6 [0.9-8.0] for 1,001-2,000h, and 3.4 [1.5-8.1] for

≥2,000h). The ORs were the same in the low exposure and high exposure groups regardless of whether cancer controls or other controls were used but differed in the middle exposure group with analyses using cancer controls showing no increased OR and using non-cancer controls showing an OR very similar to the analysis using all controls.

These three case-control studies [80, 81, 83] were combined in a pooled analysis in **Hardell et al. (2006)** [86]. The final study included 1,251 cases and 2,438 controls. This constitutes a response rate of 85% for cases and 84% for controls. For mobile phone usage and 1-year latency, they reported an OR for gliomas of 1.3 (1.1-1.6) that stayed at 1.3 (0.99-1.6) for 5-10-year latency and rose to 2.5 (1.8-3.3) for >10-year latency; the numbers were slightly higher if only a mobile phone was used (no cordless phone). They also saw a clear exposure-response relationship for lifetime use in hours where the OR was 1.2 (1.03-1.5) for 1-1000 hours of use, 1.8 (1.2-2.8) for 1001-2000 hours of use and 3.2 (2.0-5.1) for >2000 hours of use. The OR increase per 100 hours of use was 1.023 (1.013-1.034). In a follow-up to this study, **Hardell and Carlberg (2013)** [87] evaluated the survival of glioma patients until death or May 30, 2012 using Cox's proportional hazards model adjusted for age, gender, year of diagnosis, socioeconomic status and study. Exposed patients were those using a phone at least 1 year prior to tumor development, unexposed were all other patients. The hazard ratio (HR) for users of mobile phones was 1.1 (0.9-1.2) and increased with latency (0.9 [0.8-1.1] for 1-5 years; 1.1 [0.9-1.4] for 5-10 years; 1.3 [1.0005-1.6] for >10 years), and tertiles of cumulative use (0.9 [0.7-1.1] for T1; 1.0 [0.8-1.3] for T2; 1.3 [1.05-1.6] for T3). For lower grade astrocytomas (I and II), all HRs were below 1, for grade III astrocytomas, most HRs were below 1 and for grade IV, all HRs were greater than 1, but none were significant.

The fourth case-control study, **Hardell et al. (2013)** [82], covered all of the administrative regions of Sweden and included males and females aged 18-75 years who were diagnosed with a brain tumor between 2007 and 2009 (there were some differences by region). Deceased cases were excluded from the study. The study eventually included 593 cases (87% response rate) and 1368 controls (85% response rate). There were more female controls responding than males although there were more male cases than female cases. The OR for use of a mobile phone for more than 1 year and malignant brain tumors was 1.6 (0.99-2.7) with very little change by latency until a latency of 20-25 years where the OR was 1.9 (1.1-3.5) and >25 years where the OR was 2.9 (1.4-5.8). They conducted a novel analysis where they used meningioma patients as the controls and saw similar patterns but slightly higher ORs. The OR for ipsilateral use was slightly increased from the overall OR with a value of 1.7 (1.01-2.9). Analyses were also conducted separately for use of analog mobile phones with an OR of 1.8 (1.04-3.3), second-generation (2G) digital mobile phones 1.6 (0.996-2.7) and third-generation (3G) phones 1.2 (0.6-2.4). All of these had the highest ORs in the longest latency group. They also broke exposure to wireless phones (combined exposure to mobile phones and cordless phones) in the controls into quartiles and, using these categories, calculated ORs for malignant tumors and use of mobile phones. Regardless of phone type, the highest ORs were seen in the highest quartile of exposure and analog, 2G and the combined analysis of all mobile phones displayed significant trends with increasing ORs across quartiles. They also did a separate analysis for malignant tumors located in temporal and overlapping lobes and saw a similar pattern with latency, but higher ORs. Finally, they did a separate analysis for exclusive use of each type of phone, but numbers were small in most cases and this does not relate well to phone use (e.g. there

were no users of only analog phones since every phone user had moved on to digital phones by the time of this study).

**Hardell and Carlberg (2015)** [88] pooled the data on glioma patients from all of their case-control studies into one large study; they excluded deceased cases from all of the studies in this analysis. Cases and controls are described above. The pooled cases of malignant tumors number 1498 (89% response rate total) with 817 males and 563 females with gliomas. There are 3530 controls (87% total response rate) with 1492 males and 2038 females. The median latency time for use of mobile phones in glioma patients was 9 years (range 2-28 years). All analyses were adjusted for age at diagnosis, gender, socio-economic index, and year of diagnosis. Ever use (>1 year) of analog phones gave an OR of 1.6 (1.2-2.0), ever use of 2G phones gave an OR of 1.3 (1.1-1.6), ever use of 3G phones gave an OR of 2.0 (0.95-4.4), ever use of any 2G or 3G digital phone gave an OR of 1.3 (1.1-1.6) and ever use of any mobile phone gave an OR of 1.3 (1.1-1.6). For any use of mobile phones, all latency groups showed significantly increased ORs except for the >1-5 years group (OR=1.2, 0.98-1.5) and all phone groupings had their highest ORs for the longest latencies. Ipsilateral use of mobile phones gave an OR of 1.8 (1.4-2.2) whereas contralateral use gave an OR of 1.1 (0.8-1.4). Using the method of **Inskip et al. (2001)** [44] gave a relative risk (RR) of 1.5 with  $p < 0.001$ . Dividing hours of exposure into quartiles (as done in [82]) yielded significant trends for use of any mobile phone as well as analog and 2G phones. Age at first use of a mobile phone was significant in all categories with <20 years showing the highest OR=1.8 (1.2-2.8) and the highest ipsilateral OR of 2.3 (1.3-4.2). Using meningiomas as the referent group led to similar results. Multivariate analysis yielded increases per 100 hours of cumulative use for analog mobile phones (1.025, 1.010-1.041) and 2G phones (1.009, 1.005-1.014) but not 3G phones (0.980, 0.944-1.017). Multivariate analysis also yielded increases per year of latency for analog mobile phones (1.056, 1.036-1.076) and 2G phones (1.030, 1.009-1.052) but not 3G phones (1.127, 0.955-1.329).

The greatest strengths of these studies are their use of population-based controls and the high participation rates of cases and controls. One major limitation, as with most case-controls studies, is the use of a questionnaire for obtaining exposure information and the possibility of recall bias. Overall, the studies show little indication of recall bias, especially since the meningioma cases used as the referent population showed little change in the ORs. (Table 1, Table 2, Table 3, Table 5, Table 6)

**Baldi et al. (2011)** [89] conducted a case-control study (CEREPHY) of brain tumors in the area of Gironde, France. Eligible cases were patients aged 16 and older diagnosed with a brain cancer from May 1, 1999 to April 30, 2001. The study had 221 (70% participation rate) cases and 442 (69% participation) controls matched on age, sex and residence. Gliomas were seen in 105 cases (26 ever used a cellular phone) and the OR for ever versus never use of a cellular telephone was 0.82 (0.53-1.26). The use of a cellular telephone exceeded 10 years for 1 user and 5 years for 12 users. (Table 1)

The CERENAT study by **Coureau et al. (2014)** [90] is a multicenter case-control study conducted in four areas of France. Cases were defined as all subjects aged 16 and over diagnosed between June 2004 and May 2006 and living in one of four French areas (Gironde, Calvados, Manche, Herault) with a benign or malignant brain tumor (with specific ICDO-3 codes). These tumors were verified either through neuropathological, clinical or radiological assessment. For each case, two controls with no history of CNS tumors were randomly selected from electoral rolls and matched on age ( $\pm 2$  years), sex and department

of residence. Exposures were determined through non-blinded, face-to-face application of questionnaires; proxies were given a simplified questionnaire. Regular users were defined as people who were phoning at least once per week for 6 months or more and at least one-year prior to diagnosis. An adjustment was made for subjects using hands-free calling or sharing their phones with others. The analyses for gliomas included 253 cases and 504 controls with a participation rate of 66% for gliomas and 45% for controls. The OR for regular users versus others was 1.24 (0.86-1.77) adjusted for level of education and exposure to ionizing radiation. Exposure-response analyses were conducted for time since first use ( $p=0.17$ ,  $\geq 10$  years 1.61, 0.85-3.09), average calling time per month ( $p<0.001$ ,  $\geq 15$  hours 4.21, 2.00-8.87), average number of calls per day ( $p=0.04$ , 5-9 calls 2.74, 1.33-5.65,  $\geq 10$  calls 1.78, 0.88-3.59), cumulative duration of calls ( $p=0.02$ ,  $\geq 896$  hours 2.89, 1.41-5.93) and cumulative number of calls ( $p=0.41$ ,  $\geq 18,360$  calls 2.10 (1.03-4.31). Analyses excluding proxies saw almost the same results. Among the heaviest users ( $\geq 896$  hours cumulative duration of calls), the OR for 5-year latency was 5.30 (2.12-13.23), for occupational users the OR was 3.27 (1.45-7.35) and for exclusive use in an urban setting the OR was 8.20 (1.37-49.07). Ipsilateral use (0.70, 0.46-1.07) was higher than contralateral use (0.30, 0.17-0.52), however, these findings were questioned by **Hardell and Carlberg (2015)** [91] because the approach used was different than that used in their analyses and in the Interphone Study. The authors responded [92] and, using the same method as **Hardell and Carlberg (2015)** [88], obtained an OR for ipsilateral use of 4.21 (0.70-25.52) and for contralateral use of 1.61 (0.36-7.14). They also applied the same method used in **Inskip et al. (2001)** [44] and obtained an OR of 2.40 (1.002-5.73). The major weaknesses of this study are the response rates and the use of questionnaire data for exposure. The authors addressed concern for recall bias by carefully assessing exposure in the highest exposed individuals. They found that there may be some small concern for exposure misclassification, but it is likely to be non-differential and is unlikely to have affected the final results. (Table 1, Table 2, Table 3, Table 4, Table 5, Table 6, Table 7)

**Yoon et al. (2015)** [93] conducted a case-control study in five areas of Korea (Seoul, Gyeonggi-do, Gyeongsang-do, Jeolla-do, Chungcheong-do, Gangwon-do, and Jeju-do). Cases (285 participated, 142 refused, 465 had excessive pain and 5 had no matched control) were identified as glioma patients between the ages of 15 and 69 years of age and controls (285 participated, 354 refused, 7 had excess pain and 405 had no matched case). Cases and controls came from the recruiting hospitals and were given a questionnaire during the initial interview. Cases were also excluded if they died during the course of the study. There were some significant differences between cases and controls (residential region, education, patient or proxy, use of dye, alcohol use, computer use and use of electric blankets). Users were defined as having more than 1 year of cellular phone use. The OR for users was 1.17 (0.63-2.14) for all respondents and 0.94 (1.46-1.89) for self-respondents. The largest group of users had used both analog and digital phones and they had an OR of 1.89 (0.96-3.81). Lifetime years of use, cumulative hours of use, average number of calls received daily, average number of calls sent daily and average duration of calls had ORs that were generally greater than 1.0, included 1.0 in the 95% confidence interval, and did not appear to show dose-response although no test was done. Using the method of **Inskip et al. (2001)** [44] gave a relative risk (RR) of 1.26 ( $p=0.05$ ) for all respondents and 1.43 ( $p=0.01$ ) for self-respondents. ORs for ipsilateral versus contralateral use were very mixed and seldom included the OR from the original evaluation as falling between the ORs for the two sides (it appears they used the same method as the **CERENAT study (2014)** [90] but this cannot be

verified). Besides the usual possibility of recall bias in these types of studies, this study's weaknesses include poor reporting of the methods, an unusual exclusion of patients due to pain and very high refusal rates for both cases and controls. (Table 1, Table 2, Table 3, Table 6)

#### 4.1.1.2 Cohort Studies

**Schuz et al. (2006)** [94] extended the evaluation of a retrospective cohort study in Denmark [95]. They identified 723,421 cellular telephone subscribers in Denmark from 1982 to 1995, 420,095 of whom could be identified as individuals and became part of the cohort. The other 303,326 were excluded because the user was listed as a corporation (200,507) or excluded for other reasons (102,819). Approximately 85% of the cohort members were males. Only first cancer diagnoses were used in this analysis and the ending date of follow-up is December 31, 2002. The observed cancers in the cohort were compared to the expected numbers in the Danish population using the Danish Cancer Registry after subtracting the number of cancer case patients and person-years observed in the cohort from those in the registry.

There was a significant decrease in all cancers for males (RR 0.93, 0.92-0.95) and a marginally significant increase in females (1.03, 0.99-1.07). All of the RRs for cancers in males, including brain and CNS tumors (0.96, 0.87-1.05), lacked statistical significance with 14 of the 20 grouped organ sites having RRs below 1. In females, all smoking-related sites, cervix/uteri and kidney tumors showed significantly increased RRs with brain and CNS tumors non-significant (1.03, 0.82-1.26). For males and females combined, gliomas (1.01, 0.89-1.14), meningiomas (0.86, 0.67-1.09) and cranial nerve sheath tumors (0.73, 0.50-1.03) were all non-significant. There was no increase with years on use in both males and females for brain and CNS tumors ( $p=0.51$ ) or leukemias ( $p=0.69$ ).

**Frei et al. (2011)** [96] conducted an update of the Danish cohort study using the same information on cellular phone subscriptions (1982-1995); hence the update is only with regard to tumor rates and contains no information on cellular phone subscriptions post 1995. Only first cancer diagnoses were used in this analysis and the ending date of follow-up is December 31, 2007. To obtain information on socioeconomic factors, they used the CANULI cohort study data [97] which includes all Danes aged 30 or older born after 1925 in Denmark. Because of eligibility requirements for CANULI, the number of subscribers was reduced by 54,350; thus, the follow-up contained 358,403 subscription holders.

There was a significant decrease in all cancers for males with subscriptions (RR 0.96, 0.95-0.98) and a marginally significant increase in females (1.02, 0.98-1.06). There were slight increases in central nervous system tumors for both males (1.02; 0.94-1.10) and females (1.02; 0.86-1.22) with no apparent increase in risk as years of subscription increased. There was a stronger increase for gliomas alone in males (1.08; 0.96-1.22) but not in females (0.88; 0.69-1.40) with the highest RRs in males for only 1-4 years of subscription (1.20; 0.96-1.50) and the lowest for  $\geq 13$  years of subscription (0.98; 0.70-1.36); there was no exposure response in females. There is a chance some of the gliomas could have fallen in the "other and unspecified" category and those saw RRs above 1 for both males (1.12; 0.95-1.33) and females (1.19; 0.85-1.67). For men, RRs for mobile phone use and tumors in the frontal lobe (1.13; 0.89-1.45), temporal lobe (1.13; 0.86-1.48), occipital lobe (1.47; 0.87-2.48) and other or unspecified brain regions (1.35; 1.05-1.75) were above 1. (Table 1, Table 2, Table 7)

**Schuz et al. (2009)** [98] also looked at central nervous system diseases in this same cohort. They looked for hospital contacts for migraine (RR 1.2, 1.1-1.3), vertigo (1.1, 1.1-1.2), alzheimer's (0.7, 0.6-0.9), vascular dementia (ns), other dementia (0.7, 0.6-0.8), Parkinson (0.8, 0.7-0.9), ALS (ns), MS (ns), epilepsy in men (0.7, 0.7-0.7) and women (ns).

The biggest concern with all these studies [94, 96, 98, 99] are the various sources of misclassification that could be differential and/or non-differential. By their own count, 303,326 phone contracts could not be assigned to specific users and were classified into the non-user category. In addition, a member of the cohort may have been the owner of the account but not the primary user of the cellular phone (e.g. parents or spouses paying for the account). Using information from a separate case-control study [49], it was estimated that 16% of the non-users could have been frequent users; this was used to suggest the potential impact of this bias on the overall RRs will be low; no sensitivity analysis was provided. No phone data past 1995 was used for any of these analyses. According to the World Bank (2020) [100], there were 15.714 subscriptions to mobile phones per 100 people in Denmark in 1995 against a population of 5,233,373 [101]. To compare, 723,421 subscriptions in Denmark from 1982 to 1995 would be 13.82 per 100 people (very close to the World Bank numbers). By 2002, when the Schuz et al. (2006) [94] follow-up ended, there were 83.341 subscriptions per 100 people (5.3x increase) and by 2007 when Frei et al. (2011) [96] follow-up ended, there were 115.322 per 100 people (7.3x increase); in 2018, there are 125.119 subscriptions per 100 people in Denmark. Thus, of the 1853 male and 1455 female non-subscribers who had gliomas, most of them will have had subscriptions of some sort by 2007. Hence, the exposure misclassification is extreme with many cellular phone users in the non-subscription category who are undoubtedly using mobile phones. Finally, in the **Frei et al (2011)** [96] update, the use of the CANULI database required dropping all cell phone users below the age of 30 before 1995 which appears to be the 54,350 subscribers they lost; hence the youngest phone users before 1995 were excluded from the study.

**Benson et al. (2013)** [102] used data from the Million Women Study (MWS; for details, see [103, 104]) to evaluate the linkage between brain tumors and mobile phone use. Researchers recruited 1.3 million middle-aged women in the UK into the MWS during the period of 1996-2001. Women completed an initial survey on lifestyle factors, sociodemographic factors and medical history and are resurveyed every 3-4 years. Questions on mobile phone use were asked in 1999-2005 and again in 2009. Information about incident cases of brain tumors were obtained through linkage to Hospital Episode Statistics in England and Scottish Morbidity Records. Of the 866,525 women who answered the questionnaire between 1999 and 2005, numerous women were excluded from the analysis (14,387 got a questionnaire without cell phone usage, 11,981 did not answer the cell phone usage question, 48,531 had CNS tumors at baseline and 6 had a genetic predisposition to get neurological tumors); eventually leaving 791,710 women in the study. Average follow-up time was 7 years (follow-up was through December 31, 2009 except for 1 region where the date was December 31, 2008). Cell phone usage was assessed with two questions: 1) About how often do you use a mobile phone? Never/less than once a day/every day; 2) For how long have you used one? Responses to mobile phone usage questions in 2009 were used to assess the repeatability of earlier questions for the 31,110 women who answered both; however, the questions were different and consistency is not easy to assess. Approximately half of those who reported no use of a mobile phone in the

first survey reported use in 2009. There were a number of demographic differences between mobile phone users and non users, including age, affluence, exercise, alcohol and smoking. In addition, the phone users saw less incident cancers (6.05%) than did non-users (7.32%) during the follow-up period. In total, there were 571 gliomas in this cohort. Risk ratios (RRs) for phone use were ever/never 0.91 (0.76-1.08), daily use 0.80 (0.56-1.14), <5 years 0.93 (0.71-1.21), 5-9 years 0.92 (0.75-1.13) and 10+ years of use 0.78 (0.55-1.10) (all adjusted for socioeconomic status, region, age (in 3-year groupings), height, BMI, alcohol intake, exercise and hormone therapy). In a letter responding to a letter by **de Vocht (2014)** [105], **Benson et al. (2014)** [106] updated their follow-up to 2011 but did not update cellular phone usage (still relying on the 1999-2005 response) and saw the RR for glioma for ever/never users of 0.86 (0.75-0.99). Note that with 7 years average follow-up, they saw 571 gliomas or 82/year but adding 2010 and 2011 increased the gliomas by over 100 per year. The main limitations of this study are the rapidly changing exposures to mobile phones and the short follow-up period. Both of these factors likely pushed the results toward the null. In essence, this study creates considerable challenges in terms of misclassification of exposure. For example, a case answering the question in 2005 with 1 year of usage would have 6 years of exposure. In contrast, a woman answering in 1999 with no cell phone usage who then gets a phone in 2000 has 10 years of use but is considered a non-user. This problem is exacerbated by the rapid increase in cellular phone usage in the UK during this period. Cellular phone usage in the UK increased dramatically during the actual study period as well as the recruiting period with rates per 100 people of 9.901 (1995), 12.473 (1996), 78.281 (2001), 108.598 (2005) and 121.73 (2009) [107] so some of the cases with no exposure are likely to have been exposed. They attempted to address these issues by excluding women who reported phone use in 1999-2000 since many of these will have changed their status but this discards the longest exposed individuals and removed 73 glioma patients with cellular phone usage (21.8%). In addition, the fact that the use of a cellular phone is associated with a significant reduction in all invasive neoplasms (e.g. ever use 0.97 [0.95-0.99]) could indicate a difference between the groups that is not being addressed in the analysis. (Table 1, Table 2)

Table 1: Results from epidemiology studies for ever versus never or regular versus non-regular use of a cellular telephone and the risk of glioma in adults

Author (year)	Study Type	Years, Country	Age (years), sex	Tumor Type	Sample Size for all endpoints (% resp.)	Exposed (%) Cases	OR (95% CI)	Comparison group
Hardell et al. (1999)	CC	1994-1996, Sweden	20-80, Both	All Malignant Astrocytoma, glioblastoma	272 (90%) Gliomas 439 (91%) Controls	53 (19.5) 36 (38.3)	0.98 (0.63-1.50) 1.09 (0.64-1.84)	>1 year, all malignant (mostly gliomas, 4 NUD) >1 year, astrocytoma & glioblastoma (L&R match)
Muscat et al. (2000)	CC	1994-1998, US	18-80, Both	Astrocytic tumor Oligodendroglioma	354 cases 55 cases	41 (11.6) 9 (16.4)	0.8 (0.5-1.2) 0.9 (0.4-2.1)	Has subscription
Inskip et al. (2001)	CC	1994-1998, US	≥18, Both	Glioma	782 (92%) Cases 799 (86%) Controls	201 (41.4) 121 (24.7)	1.0 (0.7-1.4) 0.9 (0.7-1.4)	Any use >5 times use
Auvinen et al. (2002)	CC	1996, Finland	20-69, Both	Glioma	198 (100%) Gliomas 989 (100%) Controls	32 (16.3)	1.5 (1.0-2.4)	Has subscription
Gousias et al. (2009)	CC	2005-2007, Greece	22-82, Both	Glioma	36 (ND) Gliomas 82 (ND) Controls	ND (ND)	1.0 (0.99-1.01)	ND
Spinelli et al. (2009)	CC	2005, France	≥18, Both	Glioma	122 (17.2%) Gliomas 122 (90.2%) Controls	85 (69.7)	ND (ND)	Used a phone
INTERPHONE (2010)	CC	2000-2004, 13 countries	30-59, Both	Glioma	2765 (64%) Gliomas 7658 (53%) Controls	1,666 (61.5)	0.81 (0.70-0.94)	Avg 1 call per week for 6 mo (lag 1 yr)
Baldi et al. (2011)	CC	1999-2001, France	≥16, Both	Glioma	221 (70%) Brain 442 (69%) Controls	26 (24.8)	0.82 (0.53-1.26)	Ever versus never use
Coureau et al. (2014)	CC	2004-2006, France	≥16, Both	Glioma	596 (73%) Cases 1192 (45%) Controls	142 (57.0) Excluding proxies 123 (21.6)	1.24 (0.86-1.77) 1.33 (0.89-1.98)	Avg 1 call per week for 6 mo
Hardell et al. (2015)	CC	1997-2003, 2007-2009, Sweden	20-80, Both	Glioma	1498 (89%) Gliomas 3530 (87%) Controls	945 (68.5)  Per year of latency	1.3 (1.1-1.6)  1.032 (1.017-1.046)	>1 year
Yoon et al. (2015)	CC	2002-2007, Korea	15-69	Glioma	285 (32%) Gliomas 285 (27%) Controls Excluding proxies 219 Gliomas 273 Controls	235 (83.9)  191 (87%)	1.17 (0.63-2.14)  0.94 (0.46-1.89)	>1 year (maybe also non-regular user)
Frei et al. (2011)	Cohort	1990-2007, Denmark	≥30 at time of entry	Glioma	358,403	324 (17.5) Male 32 (2.2) Female	1.08 (0.96-1.22) 0.98 (0.69-1.40)	Subscription >1 year between 1982 and 1995 Phone use only for before 1995
Benson et al. (2013)	Cohort	1999-2009, UK	Middle-aged women	Glioma	791,710 (65%)  Follow-up to 2011	334 (58.5) Ever use 36 (6.3) Daily use Exclude first 3 years 261 (63.3) Follow-up to 2011	0.91 (0.76-1.08) 0.80 (0.56-1.14)  0.83 (0.68-1.02)	Ever used (asked 1999-2005) Every day (asked 1999-2005)  Ever used (asked 1999-2005)

Benson et al. (2014)		1999-2011, UK			875 glioma cases vs 571 in 2009	Not given	0.86 (0.72-1.02)	Ever used (asked 1999-2005)
----------------------	--	---------------	--	--	---------------------------------	-----------	------------------	-----------------------------

Table 2: Results from epidemiology studies for duration (years) of use of a cellular telephone and the risk of glioma in adults

Author (year)	Study Type	Years, Country	Age (years), sex	Tumor Type	Duration	Exposed Cases	OR (95% CI)	P Trend	Comments
Inskip et al. (2001)	CC	1994-1998, US	≥18, Both	Glioma	<0.5 years 0.5-3 years ≥3 years ≥5 years	24 31 30 11	0.6 (0.3-1.1) 0.9 (0.5-1.6) 0.9 (0.5-1.5) 0.6 (0.3-1.4)	ND	Any use 2+ calls/w
Auvinen et al. (2002)	CC	1996, Finland	20-69, Both	Glioma	<1 year 1-2 years >2 years	ND	1.2 (0.5-3.0) 1.6 (0.8-2.9) 1.7 (0.9-3.5)	ND	Has subscription Increase in OR per year 1.2 (1.0-1.4)
Gousias et al. (2009)	CC	2005-2007, Greece	22-82, Both	Glioma	Minute-years	ND	1.0 (0.99-1.01)	0.56	undefined
INTERPHONE (2010)	CC	2000-2004, 13 countries	30-59, Both	Glioma	1-1.9 years 2-4 years 5-9 years ≥10 years 1-1.9 Years as referent 2-4 years 5-9 years ≥10 years	156 644 614 252 460 468 190	0.63 (0.46-0.81) 0.84 (0.70-1.00) 0.81 (0.60-0.97) 0.98 (0.76-1.26) 1.68 (1.16-2.41) 1.54 (1.06-2.22) 2.18 (1.43-3.31)	ND	Avg 1 call per week for 6 mo (lag 1 yr), no hands-free  Excludes hands-free usage
Coureau et al. (2014)	CC	2004-2006, France	≥16, Both	Glioma	1-4 years 5-9 years ≥10 years Excluding proxies 1-4 years 5-9 years ≥10 years	49 66 22 47 58 14	0.88 (0.56-1.39) 1.34 (0.87-2.06) 1.61 (0.85-3.09) 1.04 (0.64-1.69) 1.45 (0.91-2.33) 1.45 (0.68-3.08)	0.17  0.36	Avg 1 call per week for 6 mo
Hardell et al. (2015)	CC	1997-2003, 2007-2009, Sweden	20-80, Both	Glioma	1-5 years 5-10 years 10-15 years 15-20 years 20-25 years >25 years	262 301 211 92 50 29	1.2 (0.98-1.5) 1.5 (1.2-1.8) 1.4 (1.1-1.9) 1.6 (1.1-2.2) 2.1 (1.3-3.2) 3.0 (1.7-5.2)	ND	>1 year
Yoon et al. (2015)	CC	2002-2007, Korea	15-69	Glioma	1-5 years 5-8 years >8 years Excluding proxies 1-5 years 5-8 years >8 years	97 70 70 37 76 76	1.28 (0.62-2.64) 1.27 (0.63-2.56) 1.04 (0.52-2.09) 0.94 (0.42-2.13) 1.01 (0.45-2.23) 0.90 (0.40-2.02)	ND	>1 year (maybe also non-regular user)

Frei et al. (2011)	Cohort	1990-2007, Denmark	≥30 at time of entry	Glioma	Male 1-4 years 5-9 years ≥10 years 10-12 years ≥13 years Females 1-4 years 5-9 years ≥10 years	Male 85 122 117 80 37 Females 8 14 10	Males 1.20 (0.96-1.50) 1.05 (0.87-1.26) 1.04 (0.85-1.26) 1.06 (0.85-1.34) 0.98 (0.70-1.36) Females 0.87 (0.43-1.75) 1.02 (0.60-1.72) 1.04 (0.56-1.95)	ND	Subscription >1 year between 1982 and 1995 Phone use only before 1995
Benson et al. (2013)	Cohort	1999-2009, UK	Middle-aged women	Glioma	<5 years 5-9 years ≥10 years Excluding first 3 years <5 years 5-9 years ≥10 years	89 185 40 66 148 29	0.93 (0.71-1.21) 0.92 (0.75-1.13) 0.78 (0.55-1.10) 0.77 (0.57-1.06) 0.86 (0.68-1.09) 0.75 (0.49-1.13)	ND	Ever used (asked 1999-2005)
Benson et al. (2014)		1999-2011, UK			Follow-up to 2011 <5 years 5-9 years ≥10 years	Not given	0.96 (0.75-1.23) 0.86 (0.72-1.02) 0.77 (0.62-0.96)		Ever used (asked 1999-2005)



Table 4: Results from epidemiology studies for average daily or monthly use of a cellular telephone and the risk of glioma in adults

Author (year)	Study Type	Years, Country	Age (years), sex	Tumor Type	Measure	Exposed Cases	OR (95% CI)	P Trend	Comparison group
Inskip et al. (2001)	CC	1994-1998, US	≥18, Both	Glioma	Average daily <3 minutes 3 to 15 minutes ≥15 minutes ≥60 minutes	53 64 51 24	0.9 (0.5-1.6) 1.0 (0.6-1.6) 0.5 (0.3-1.0) 0.7 (0.3-1.7)	ND	Any use 2+ calls/w
Coureau et al. (2014)	CC	2004-2006, France	≥16, Both	Glioma	Average monthly <2 hours 2-4 hours 5-14 hours ≥15 hours Excluding proxies <2 hours 2-4 hours 5-14 hours ≥15 hours	40 19 36 29 36 16 33 25	0.91 (0.57-1.46) 0.57 (0.30-1.10) 1.70 (0.97-2.99) 4.21 (2.00-8.87) 1.01 (0.61-1.69) 0.59 (0.29-1.21) 1.78 (0.99-3.22) 4.04 (1.84-8.86)	<0.001    <0.001	Avg 1 call per week for 6 mo

Table 5: Results from epidemiology studies for other use measures of a cellular telephone and the risk of glioma in adults

Author (year)	Study Type	Years, Country	Age (years), sex	Tumor Type	Measure	Exposed Cases	OR (95% CI)	P Trend	Comments
Inskip et al. (2001)	CC	1994-1998, US	≥18, Both	Glioma	Year use began			ND	Any use 2+ calls/w
					1995-1998	61	0.8 (0.4-1.5)		
					1993-1994	60	1.0 (0.6-1.6)		
					≤1992	50	0.6 (0.3-1.1)		
					<1990	23	0.3 (0.1-1.0)		
INTERPHONE (2010)	CC	2000-2004, 13 countries	30-59, Both	Glioma	Cumulative use by recency of starting use			ND	Avg 1 call per week for 6 mo (lag 1 yr), no hands-free
					1-4 years before reference date				
					<5 hours	127	0.68 (0.50-0.93)		
					5-114.9 hours	449	0.82 (0.67-0.99)		
					115-359.9 hours	121	0.74 (0.52-1.03)		
					360-1639.9 hours	80	0.75 (0.50-1.13)		
					≥1640 hours	23	3.77 (1.25-11.4)		
					5-9 years before reference date				
					<5 hours	10	0.86 (0.32-2.28)		
					5-114.9 hours	180	0.86 (0.66-1.12)		
					115-359.9 hours	156	0.71 (0.53-0.95)		
					360-1639.9 hours	174	0.72 (0.54-0.95)		
					≥1640 hours	94	1.28 (0.84-1.95)		
					≥10 years before reference date				
					<5 hours	4	1.13 (0.16-7.79)		
					5-114.9 hours	20	0.63 (0.32-1.25)		
					115-359.9 hours	41	0.89 (0.53-1.50)		
					360-1639.9 hours	94	0.91 (0.63-1.31)		
					≥1640 hours	93	1.34 (0.90-2.01)		
Coureau et al. (2014)	CC	2004-2006, France	≥16, Both	Glioma	Cumulative # of calls			0.41	Avg 1 call per week for 6 mo
					<660	23	1.06 (0.59-1.91)		
					(660-2219)	27	1.06 (0.59-1.91)		
					(2220-7349)	28	1.48 (0.79-2.76)		
					(7350-18359)	12	1.30 (0.60-2.83)		
					≥18359	21	2.10 (1.03-4.31)		
					Excluding proxies (weighted)			0.14	
					<476	19	0.80 (0.43-1.47)		
					(476-1649)	26	1.26 (0.70-2.28)		
					(1650-6269)	35	1.71 (0.95-3.09)		
					(6270-14699)	11	1.14 (0.52-2.53)		
					≥14,700	20	2.11 (1.03-4.33)		
					Occupational use	45	3.27 (1.45-7.35)		
					Urban use only	16	8.20 (1.37-49.07)		
Hardell et al. (2015)	CC	1997-2003, 2007-2009, Sweden	20-80, Both	Glioma	Age				>1 year
					<20 years old	69	1.8 (1.2-2.8)		
					20-49 years old	605	1.3 (1.1-1.6)		
					≥50 years old	271	1.3 (1.1-1.6)		

Table 6: Results from epidemiology studies for laterality of cellular telephone use and the risk of glioma in adults

Author (year)	Study Type	Years, Country	Age (years), sex	Tumor Type	Location or laterality	Ipsilateral OR (95%CI)	Contralateral OR (95% CI)	Inskip P.value	Comparison group
Hardell et al. (1999)	CC	1994-1996, Sweden	20-80, Both	All Malignant Astrocytoma, glioblastoma	Right side + right ear Left side + left ear Right side + right ear Left side + left ear	1.43 (0.70-2.90) 0.58 (0.17-1.92) 1.30 (0.54-3.13) 0.35 (0.07-1.81)			>1 year
Inskip et al. (2001)	CC	1994-1998, US	≥18, Both	Glioma	Inskip method Left Right	0.9 (0.6-1.5) 0.8 (0.5-1.3)		0.77	2 or more calls/week + 6 months latency
INTERPHONE (2010)	CC	2000-2004, 13 countries	30-59, Both	Glioma	Regular use ≥10 years since start ≥1640 hours cumulative ≥270 calls (hundreds)	0.84 (0.69-1.04) 1.21 (0.82-1.80) 1.96 (1.22-3.16) 1.51 (0.91-2.51)	0.67 (0.52-0.87) 0.70 (0.42-1.15) 1.25 (0.64-2.42) 0.61 (0.32-1.18)		Avg 1 call per week for 6 mo (lag 1 yr)
Coureau et al. (2014)	CC	2004-2006, France	≥16, Both	Glioma	Regular use Cumulative duration of calls (Interphone method) <43 43-112 113-338 339-895 ≥896 Inskip method	2.11 (0.73-6.08)  0.29 (0.11-0.80) 0.44 (0.16-1.23) 0.78 (0.27-2.24) 1.69 (0.52-5.49) 4.21 (0.70-25.52) 2.40 (1.002-5.73)	0.66 (0.23-1.89)  0.25 (0.07-0.95) 0.33 (0.10-1.08) 0.25 (0.06-1.02) 0.23 (0.05-1.11) 1.61 (0.23-1.89)		Avg 1 call per week for 6 mo
Hardell et al. (2015)	CC	1997-2003, 2007-2009, Sweden	20-80, Both	Glioma	Regular use Meningioma cases as referent Latency groups 1-5 years 5-10 years 10-15 years 15-20 years 20-25 years >25 years Age groups <20 years old 20-49 years old ≥50 years old Inskip method	1.8 (1.4-2.2) 1.4 (1.1-1.8)  1.6 (1.3-2.1) 1.9 (1.4-2.5) 1.7 (1.2-2.3) 2.2 (1.5-3.4) 2.3 (1.3-4.1) 4.6 (2.1-10)  2.3 (1.3-4.2) 1.8 (1.4-2.3) 1.7 (1.3-2.2) 1.5 (ND)	1.1 (0.8-1.4) 1.0 (0.7-1.4)  0.9 (0.7-1.2) 1.3 (0.9-1.8) 1.3 (0.9-2.0) 1.0 (0.6-1.7) 2.2 (1.1-4.6) 3.2 (1.2-8.6)  1.9 (0.9-3.7) 1.1 (0.8-1.5) 1.1 (0.8-1.5)		>1 year
Yoon et al. (2015)	CC	2002-2007, Korea	15-69	Glioma	Total respondents Inskip method Self respondents (Inskip) Cumulative hours of use <300 300-900 >900	0.95 (0.50-1.83) 1.26 1.43 0.96 (0.37-2.47) 1.04 (0.45-2.40) 1.77 (0.32-1.84)	0.90 (0.43-1.89)   1.20 (0.43-3.29) 1.09 (0.36-3.28) 0.63 (0.24-1.65)	0.05 0.01	>1 year (maybe also non-regular user)

Table 7: Results from epidemiology studies for cellular telephone use and the location of glioma in adults

Author (year)	Study Type	Years, Country	Age (years), sex	Tumor Type	Location or laterality	Exposed Controls	OR (95%CI)	Comparison group
INTERPHONE (2010)	CC	2000-2004, 13 countries	30-59, Both	Glioma	Temporal lobe	509	0.86 (0.66-1.13)	Avg 1 call per week for 6 mo (lag 1 yr)
					≥10 years since start	94	1.36 (0.88-2.11)	
					≥1640 hours cumulative	78	1.87 (1.09-3.22)	
					≥270 calls (hundreds)	61	1.10 (0.65-1.85)	
					Parietal lobe	871	0.77 (0.62-0.95)	
					≥10 years since start	129	0.92 (0.65-1.30)	
					≥1640 hours cumulative	105	1.25 (0.81-1.91)	
					≥270 calls (hundreds)	86	1.02 (0.67-1.57)	
					Other locations	248	0.79 (0.51-1.23)	
					≥10 years since start	32	0.41 (0.16-1.08)	
≥1640 hours cumulative	18	0.91 (0.33-2.51)						
≥270 calls (hundreds)	19	0.42 (0.13-1.33)						
Coureau et al. (2013)	CC	2004-2006, France	≥16, Both	Glioma	Temporal lobe	68	3.94 (0.81-19.08)	Avg 1 call per week for 6 mo
Frontal lobe	76	1.87 (0.62-5.64)						
Other locations	87	3.61 (1.00-12.96)						
Hardell et al. (2015)	CC	1997-2003, 2007-2009, Sweden	20-80, Both	Glioma	Temporal Lobe	367	4.3 (2.0-9.3)	
Frei et al. (2011)	Cohort	1990-2007, Denmark	≥30 at time of entry	Glioma	Cerebrum	52	0.90 (0.67-1.22)	Subscription >1 year between 1982 and 1995 Phone use only before 1995
					Frontal lobe	79	1.13 (0.89-1.45)	
					Temporal lobe	65	1.13 (0.86-1.48)	
					Parietal lobe	33	0.73 (0.50-1.05)	
					Occipital lobe	18	1.47 (0.87-2.48)	
					Other and unspecified	77	1.35 (1.05-1.75)	

#### 4.1.2 Studies in Children

**Elliott et al. (2010)** [108] conducted a case-control study of cancers in children aged 0-4 in Great Britain looking at a linkage to mobile phone base stations. Cases were all registered children with cancer in 1999-2001 (1926 cases) and four controls for each case were chosen from the national birth registry matched by sex and date of birth. Birth addresses (or approximate addresses) were needed for each case and each control leaving a total of 1397 cases and 5588 controls. Three exposure metrics were used, distance from the nearest mobile phone base station, total output from all base stations within 700 meters, and a modeled power density (dBm) from all base stations within 1400 meters of the birth address (modeling was based upon surveys and then validated against later additional survey data). Of the 1397 cases, there were 251 brain cancers (1004 controls). None of the mean exposures for any of the three metrics were different between cases and controls. ORs were very close to 1 for all exposure metrics when exposure was broken into tertiles and the referent group was the first tertile. Similar results were seen in an analysis using the continuous exposure measure directly. The same patterns were true for all cancers and leukemias. (Table 8)

The CEFALO study (**Aydin et al. (2012)** [109]) is an international case-control study conducted in Denmark, Norway, Sweden and Switzerland of children and adolescents aged 7-19 years at time of diagnosis of a brain cancer. Cases had brain tumors with a specific ICD-10 classification and were identified by a combination of factors. Controls were matched on year and month of birth or just year of birth (Norway) with two cases per control. The study included 352 cases (83.2% response) and 646 controls (71.1% response); 213 of the cases had gliomas. Exposure was obtained by personal interviews with mobile phone use 6 months prior to diagnosis excluded from the analyses. Cases were asked for permission to access usage data from mobile phone operators. In Denmark and Sweden, data covered the entire period of usage whereas in Switzerland, data was only kept for 6 months so data were only available for after diagnosis; data from providers in Norway was not obtained. The OR for regular use (one call per week for at least 6 months) versus not was 1.36 (0.92-2.02). All ORs for time since first use were above 1 (1.35 (0.89-2.04) for <3.3 years, 1.47 (0.87-2.49) for 3.3-5.0 years, 1.26 (0.70-2.28) for > 5 years). Similar patterns were seen for cumulative duration of subscriptions ( $\leq 2.7$  years, 1.34 [0.89-2.01]; 2.8-4 years, 1.45 [0.83-2.54]; >4 years, 1.58 [0.86-2.91]), cumulative duration of calls ( $\leq 35$  hours, 1.33 [0.89-2.01]; 36-144 hours, 1.44 [0.85-2.44]; >144 hours, 1.55 [0.86-2.82]) and cumulative number of calls ( $\leq 936$  calls, 1.34 [0.89-2.02]; 937-2638 calls, 1.47 [0.86-2.51]; >2638 calls, 1.42 [0.79-2.53]). Stratifying the analysis for only gliomas yielded an OR of 1.14 (0.66-1.97) but only included 192 cases (it appears they excluded the 21 ependymomas even though these are gliomas). When they analyzed brain tumors using the operator-recorded data (35% of cases, 34% of controls), they saw a significant trend for time since first subscription ( $p=0.001$ ) with the highest exposure group (>2.8 years) having a statistically significant OR of 2.15 (1.07-4.29). The same analysis using self-reported use had a trend test with  $p=0.22$  and an OR in the highest exposure class of 1.47 (0.81-2.67). Other exposure metrics saw generally higher ORs using the operator-recorded use data than self-reported use; this is likely due to some degree of differential exposure misclassification since a study showed cases overestimated their numbers of calls (9%) and duration of calls (52%) much less than controls (34% and 163% respectively) [110]. The OR for ipsilateral use (1.74, 0.91-3.33) was not larger than that for contralateral use (2.07, 0.95-4.52), although the definition used for ipsilateral and contralateral was unique to this study [111]. For ipsilateral and contralateral use, exposure-response relationships were seen for all exposure measures and the highest exposure groups had the biggest ORs, many statistically significant. The major strengths of this study include the participation rates and the

exposure information. The major weaknesses include a failure to analyze all gliomas and to do the ipsilateral analysis and operator-generated usage on the gliomas alone. There were other criticisms of this paper [112]. (Table 8)

**Li et al. (2012)** [113] conducted a population-based case-control study of incident cases of all cancers in Taiwan in children and adolescents <15 years of age between 2003 and 2007. Thirty controls were randomly selected for each case and matched on year of birth. The annual power density (APD; watt-year/km<sup>2</sup>) for each township was calculated from the 71,185 mobile phone base stations in Taiwan. Exposure was calculated as the average APD five years prior to diagnosis for cases and prior to July 1 for the controls in the year their matched case was admitted. For brain tumors there were 394 cases and 11,820 controls. OR for above median versus below median exposure was 1.09 (0.88-1.36) for the crude estimate and 1.14 (0.83-1.55) for the adjusted estimate (calendar year, age, gender, high-voltage transmission line, and urbanization of township). When the exposures were divided into tertiles, there was an indication of a trend (crude: 1.01 [0.84-1.42] T2, 1.09 [0.77-1.32] T3; adjusted: 1.03 [0.73-1.45] T2, 1.14 [0.70-1.85] T3), but no test for trend was used. The major limitation of this study is that the exposure metric does not pertain to the individual's exposure, but exposure to anyone in the township. Nearness to a tower, use of a cellular telephone, and other sources of RF that might have been related to disease incidence were not assessed. Thus, this study is closer to using an ecological exposure measurement than an individual personal exposure measurement. (Table 8)

**Feltbower et al. (2014)** [114] conducted a pilot case-control study of children and young adults ages 0-24 in two UK cancer treatment centers. Eligible cases were 0-24 years of age presenting with a diagnosis of intracranial tumor during an unspecified period. At one center, cases were matched by age and sex with a target of 2 controls per case and randomly selected from the general practice. At the second center, 3 friend controls were envisioned but the researchers were unable to attain any controls. Eventually, they were able to interview 49 cases (52% response) and 78 controls (32% response). The study was designed to be compatible with the CEFALO study [109]. The OR for brain cancer and having spoken on a mobile phone more than 20 times was 0.9 (0.2-3.3). The main weaknesses of this study are its size, response rate, and failure to get controls from the second center. (Table 8)

Table 8: Results from epidemiology studies RF and brain tumors in children and adolescents

Author (year)	Study Type	Years, Country	Age (years), sex	Tumor Type	Sample Size for all endpoints (% resp.)	Exposed (%) Cases	Group	OR (95% CI)	P trend	Comparison group
Elliott et al. (2010)	CC	1999-2001, Great Britain	0-4, Both	Brain and CNS tumors	251 (ND) Brain and CNS	85 81 251 56 45 251 80 78 251	Base station distance Medium High 15-18 centile change Total power Medium High 15-18 centile change Modelled Power Medium High 15-18 centile change	0.95 (0.67-1.34) 0.95 (0.65-1.38) 1.12 (0.91-1.39) 1.02 (0.72-1.46) 0.83 (0.54-1.25) 0.89 (0.73-1.09) 0.97 (0.69-1.37) 0.76 (0.51-1.12) 0.82 (0.55-1.22)		Referent is lowest exposure group  Most adjusted analyses
Aydin et al. (2012)	CC	1999-2001, Denmark, Norway, Sweden, Switzerland	7-19, Both	Brain and CNS tumors	352 (83.2%) cases 646 (71.1%) controls	194 95 53 46 19 19 24 94 45 52 13 10 11 94 48 49 14 11 9 83 75 84 74	Regular use Years since first use ≤3.3 3.3-5.0 >5.0 Operator-recorded first use ≤1.8 years 1.8-2.8 years >2.8 years Cumulative years use ≤2.7 2.8-4.0 >4.0 Operator-recorded cumulative use ≤1.8 years 1.9-3.3 years >3.3 years Cumulative hours ≤35 36-144 >144 Operator-recorded cumulative use ≤11 hours 12-27 hours >27 hours Tumor Location Temporal, frontal, occ. Other Morphology Glioma Other	1.36 (0.92-2.02) 1.35 (0.89-2.04) 1.47 (0.87-2.49) 1.26 (0.70-2.28) 0.78 (0.43-1.40) 1.71 (0.85-3.44) 2.15 (1.07-4.29) 1.34 (0.89-2.01) 1.45 (0.83-2.54) 1.58 (0.86-2.91) 1.14 (0.55-2.37) 1.73 (0.71-4.20) 1.84 (0.74-4.58) 1.33 (0.89-2.01) 1.44 (0.85-2.44) 1.55 (0.86-2.82) 1.24 (0.61-2.55) 1.95 (0.81-4.73) 1.38 (0.53-3.61) 1.00 (0.58-1.72) 1.92 (1.07-3.44) 1.14 (0.66-1.97) 1.65 (0.93-2.93)	0.37    0.001   0.14   0.15   0.42   0.36	>1 call per week, 6 months lag
Li et al. (2012)	CC	2003-2007, Taiwan	<15 years	Brain tumors	394 (ND) Cases 11820 (ND) Controls	174 106 121 394	RF exposure density ≥median 1 <sup>st</sup> -2 <sup>nd</sup> tertile ≥2 <sup>nd</sup> tertile Per 1 SD exposure density	1.14 (0.83-1.55) 1.03 (0.73-1.45) 1.14 (0.70-1.85) 1.09 (0.95-1.25)	0.426 0.875 0.599 0.230	Referent <median Referent 1 <sup>st</sup> tertile  Most adjusted analyses
Feltblower et al. (2014)	CC	2007-2010, UK	0-24, Both	Brain tumors	49(52%) Brain tumors 78 (32%) Controls	26	Cumulative speaking on phone >20 ties	0.9 (0.2-3.3)		Referent spoken on phone ≤20 times

#### 4.1.3 Discussion

The strongest evidence for an effect of RF on the risks of glioma come from the case-control studies. Case-control studies are designed to compare the exposure characteristics of cases (people who have or have had a glioma) against a collection of controls (people without a history of gliomas). In evaluating the results from case-control studies, researchers must consider two possible sources of bias; selection bias and recall bias. Selection or participation bias occurs when the people who are selected to be a part of the study (both cases and controls) are not willing to participate and that participation is related to both the status of the person (case versus control) and to the exposure (cellular phones) being investigated. For example, if participants that do not use a cellular phone are less willing to participate than participants who do use a cellular phone and that controls are less likely to participate than cases, this can reduce the odds ratio<sup>1</sup> (OR) and hide a potential risk.

Case-control studies rely on measures of exposure that are generally obtained through a questionnaire administered to both the cases and the controls about their past exposures. Because they are recalling past exposures, there is a possibility that this recall may be linked in some way to their status as a case or a control. This is recall bias. For example, if cases are more likely to say they have used a cellular phone than controls or they are more likely to overestimate their cellular phone usage, this could increase the ORs and lead to an overestimation of the risk from cellular phone use. The recall must be different for the cases than the controls for this to cause a bias; errors in recalling past exposures that are similar for both cases and controls would not be recall bias.

Cohort studies generally do not have these two problems since they are asked about their exposure prior to getting the disease of interest. Cohort studies are usually aimed at identifying causes for disease in a large population of people who are followed over time. As the diseases appear in the population, an analysis is done to evaluate the risk ratio<sup>2</sup> (RR) in order to find exposures that are associated with the disease. Exposure is generally determined using a questionnaire administered during the course of the study where participants are asked about their exposures. Disease status (e.g. presence or absence of a glioma) is usually determined through periodic evaluations of cancer registries and publication of the results; thus the study has a baseline date (the date a participant enters into the study) and a follow-up date (the last date of update of the cancer registry or the date the participant got the tumor or the date the participant left the study). In evaluating the results from cohort studies, researchers must consider a different source of bias; exposure

---

<sup>1</sup> The odds ratio (OR) is calculated as the proportion of exposed cases with disease to exposed controls divided by the proportion of non-exposed cases to non-exposed controls. For rare diseases, this value approximates the population risk ratio (PRR) which is the probability of having the disease in exposed individuals divided by the probability of having the disease in non-exposed individuals. If the PRR is 1, then there is no difference in the probability of having the disease regardless of your exposure. Values of PRR greater than 1 imply the risk is higher in the exposed population. Because the OR is an estimate of the PRR for rare diseases, it is usually accompanied by a 95% confidence interval that describes the probable range of the estimate. If the OR is greater than 1, then the exposure is associated with the disease. If the lower 95% confidence bound for the OR is greater than 1, this is typically used to say the association is statistically significant.

<sup>2</sup> The rate ratio (RR) is estimated as the incidence in the exposed population divided by the incidence in the unexposed population. Incidence is calculated as the number of events in a fixed period of time divided by the person years at risk. Unlike the OR, the RR does not require the assumption of a rare disease to serve as a good estimate of the population risk ratio (PRR). Like the OR,  $RR > 1$  implies an association between the disease and the exposure.

misclassification. Exposure misclassification occurs when the exposure for participants is incorrectly applied. For example, if a participant is asked on Tuesday about their cellular phone use and they do not use a cellular phone, they would be classified as a non-user. If on Wednesday, they go to the store and purchase a phone, they are now a user, but if they do not get asked again about their use prior to the follow-up date, they would be misclassified in any evaluations. Non-differential exposure misclassification occurs when the probability of an error in determining whether an individual is exposed or not is the same for both those with the disease and for those without the disease. Non-differential exposure misclassification generally results in RRs that are closer to 1 than the true underlying risk would imply and can hide risks that are really there. Differential exposure misclassification occurs when there is a difference in the exposure misclassification between those with the disease and those without. Depending on the direction of the misclassification relative to disease status, this can either hide risks or inflate risks. For example, if those with the disease are more likely to be misclassified as non-exposed, the estimated RRs will be smaller than they should be and this would result in a reduced estimate of the risk.

Finally, one other problem to be carefully considered is confounding. Confounding occurs when exposure is correlated with another factor that is also associated with the disease of interest. For example, if age is associated with the incidence of gliomas and is also correlated with cellular phone usage, failure to recognize this potential confounding could lead to an association between cell phone usage and the incidence of gliomas that is spurious. To avoid this, researchers, when evaluating their data, will “adjust” the analysis for other potential confounders. Thus, in evaluating the findings from these studies, it is important to evaluate what adjustments were made for potential confounders in the analysis. This problem can affect both case-control studies and cohort studies.

In evaluating the epidemiological evidence, there are three areas that need to be carefully explored: consistency of the association, the existence of an exposure-response relationship (definitions to follow), and the strength of the association.

#### *4.1.3.1 Consistency of the Association*

I will focus on the main studies listed in Table 1. All of these studies did a reasonable job of addressing confounders in their analyses and so this problem will not be discussed further. First, we should consider timing of the study. According to the **World Bank** [115], 0.001% of people globally had subscriptions to mobile phones in 1980. By 1990, that was 0.2% and by 2000 it was 12%. In the US, by 1990, 2% of people had subscriptions and by 2000, 39% had cellular phones. Thus, for studies in the 1990s, we are looking at a rare exposure and trying to associate it with a rare disease (gliomas) and probably with very little time from the beginning of exposure to disease onset. Thus, it is unlikely that studies like **Hardell et al. (1999)** [85], **Muscat et al. (2000)** [40], **Inskip et al. (2001)** [44], and **Auvinen et al. (2002)** [45] would show much of an association. And that is basically the case, with these studies producing ORs of approximately 1.0 except for **Auvinen et al. (2002)** [45] with an OR of 1.5 (1.0-2.4). Thus, the later studies are more likely to show an effect if one exists than are the earlier studies and these should be given greater weight.

The size of a study will also matter since studies with greater numbers of cases and controls (especially exposed cases) will generally have smaller confidence bounds and have a greater chance of seeing an effect if one exists. Thus, the studies by **Gousias et al. (2009)** [46] and **Baldi et al. (2011)** [89] will carry less weight in an overall evaluation.

There are also studies where the referent group was “never used a mobile phone” versus studies where the referent group was “not a regular user of mobile phones” defined by different measures. Less weight should be given to studies with comparisons to “never used” simply because the “ever used” group could include people who used a phone only a few times.

Given these caveats, there are 4 case-control studies that should carry the greatest weight; **Interphone (2010)** [48], **Coureau et al. (2014)** [90], **Hardell et al. (2015)** [88] and **Yoon et al. (2015)** [93]. Three of these studies show ORs >1 for regular use of a cellular phone with only one showing a significantly increased OR (**Hardell et al. (2015)** [88], 1.3 (1.1-1.6)).

The largest study, **Interphone (2010)**, has an OR<1 and more cases and controls than the other three studies combined. The ORs also did not increase with increasing duration of the use of a mobile phone (Table 1). This study used cases that were both living and, by proxy information, those who had died before interview. However, in the Interphone study there was some degree of participation bias [48, 116] that could have resulted in a reduction of the ORs by as much as 10% according to some analyses [74, 116]. For example, just looking at the cases and controls from Canada in the Interphone study, the OR for regular use of a cellular phone went from 1.0 (0.7-1.5) to 1.1 (1.0-1.2) when this bias was theoretically corrected [116]. Applying this same bias correction to the Interphone study yields an OR of 0.9, still below 1. Another correction one could use to account for participation bias, and to some degree recall bias, is to use the lowest category of usage as the reference category rather than the non-regular user category. When this was done for the Interphone study, using the lowest duration of use as the reference group, all longer durations were significantly greater than 1.0 (Table 2). Analyses of recall bias in the Interphone study showed very little impact of recall bias on the evaluation of regular usage [74, 116].

The studies demonstrating the greatest ORs for regular use are the studies that went into the pooled analysis by **Hardell et al. (2015)** [88]. Their pooled study showed an overall OR of 1.3 (1.1-1.6) for regular use. In addition, all of the 5-year groupings of duration of use were greater than 1 and all usage longer than 5-years was significantly greater than 1 (Table 2). Only living cases were included. Their response rate was high enough that participation bias is unlikely to have lowered the OR values. It is possible that participation bias could have occurred from the use of only live cases, but in a separate analysis from a subset of the pooled studies, they saw no important differences between their analyses using live cases when compared to analyses using only deceased cases. On the other hand, recall bias could have increased the ORs. In one of the original case-control studies [117] used in their pooled analysis, they evaluated this issue and saw little indication of recall bias. In addition, in their pooled analysis, they used meningioma cases as the reference group since they were likely to have the same recall bias as the glioma cases if recall bias was a problem. The OR from the population-based reference group was 1.3 (1.1-1.6) and dropped slightly to 1.2 (0.97-1.5) with the meningioma reference group. It is unlikely recall bias explains these results.

**Spinelli et al. (2010)** [47] is also a very small study, but they provided no information on ever versus never use of mobile phones.

**Coureau et al. (2014)** [90] is about 12 times smaller than the Interphone study and about 7 times smaller than **Hardell et al. (2015)** [88]. Their evaluation showed an overall OR for regular users of 1.24 (0.86-1.77) which rose slightly to 1.33 (0.89-1.98) if proxies are removed. Duration of use was weakly associated with duration of cellular phone use but had the highest OR (1.61 [0.85-3.09]) in the longest duration group ( $\geq 10$  years) (Table 2). This study used cases that were both living and, by proxy information, those who had died before interview. This study had a lower participation rate

than the other two studies and a large difference in participation between cases (66%) and controls (45%). They did not have a questionnaire for non-participants so there is no information on whether participation bias is a problem in this study. Exposure from mobile phones was done by interview using a standardized questionnaire which limits mistakes, but does nothing to control for potential recall bias. The fact that ORs for analyses with proxies versus those without proxies gave equivalent results helps to reduce the possibility of recall bias, but the number of proxy respondents was small.

**Yoon et al. (2015) [93]** has about twice as many exposed cases as **Coureau et al. (2014) [90]**. The OR for regular use was 1.17 (0.63-2.14) dropping to 0.94 (0.46-1.89) if proxy responders are removed. The OR for duration of use was >1 for all categories but showed no obvious pattern and dropped slightly when proxies were removed. The participation rates in this study were very low (32% cases, 27% controls) mostly due to cases refusing to participate or not participating due to excess pain. Participation bias and recall bias are certainly possible from this study.

One way in which to evaluate the consistency of these findings across the various studies is by means of a meta-analysis. A meta-analysis is a technique of synthesizing research results by using various statistical methods to retrieve, select, and combine results from previous separate but related studies. There have been numerous meta-analyses on the relationship between cell phone use and gliomas [118-125]. The three most recent studies are worth a quick review. **Roosli et al. (2019) [118]** explored the risks of glioma using the two cohort studies [96, 102] and 10 case-control studies [40, 44, 45, 47, 48, 85, 88-90, 93] based upon an inclusion criteria of 1) a clearly defined source population, 2a) provide a comparison of ever versus never use of a mobile phone (they also included regular use) and/or 2b) allow for an evaluation of long-term use ( $\geq 10$  years of use before glioma diagnosis) and 3) where there are multiple publications on the same data or subsets of the same data, they included the most recent comprehensive analysis. Where there were multiple publications of subgroups of studies (e.g. Interphone), they did sensitivity analyses to examine the impact of using the subgroups rather than the pooled publications. Meta-estimates of glioma risks (mRRs) were calculated using a random-effects model using the DerSimonian and Laird method using Stata (version 11.2, Stata Corp, College Station, Texas). Unless noted otherwise, all of the meta-analyses used the same method of a random-effects model and the DerSimonian and Laird method).

The main analysis from **Roosli et al. (2019) [118]** is shown in their Figure 1 and give the mRRs for the analyses of studies showing ORs for  $\geq 10$  years exposure. For the case-control studies, they get an mRR of 1.30 (0.90-1.87). For the Cohort studies, they show an mRR of 0.92 (0.72, 1.16) and for all studies combined they get 1.11 (0.85-1.46). Entering their numbers into Stata (v 16.2 for MAC), I am able to reproduce their mRRs, however, they had to first calculate an mRR for  $\geq 10$  years in the study by Hardell et al. (2015) [88] by combining results from multiple 5-year categories. They list this combination as giving an mRR for  $\geq 10$  years for that study of 1.69 (1.40-2.03) whereas when I do the same analysis, I get 1.81 (1.35-2.43). The only way I was able to achieve the same results as **Roosli et al. (2019) [118]** for the mRR was to use a fixed-effects model rather than a random-effects model (this appears to be a mistake in the paper). They also did a meta-analysis of ever versus never use for all 10 case-control studies (1.03 [0.86-1.22]) and the cohort studies (0.97 [0.82-1.15]) with a combined mRR of 1.00 (0.89-1.13). They also conducted a cumulative meta-analysis of the studies with  $\geq 10$  years of use splitting the Hardell group studies into those from 1997-2003 and 2007-2009 yielding a slightly higher mRR (1.24 [0.93-1.66]) for all studies combined. They also did several other analyses of ever versus never use with no appreciable changes in the results. One problem with these meta-analyses is that they give very little weight to the largest studies. For

example, in their analysis of the 12 ever versus never studies, **The Interphone (2010)** [48] study with 1666 exposed cases got a relative weight of 13%, **Hardell et al. (2015)** [88] with 945 exposed cases got a relative weight of 11.6% and the remaining studies with a total of 1586 exposed cases got a relative weight of >75%. In addition, all of these analyses showed highly significant heterogeneity. **Roosli et al. (2019)** [118] did not consider laterality or tumor location in the brain.

**Wang et al. (2018)** [119] did a meta-analysis like that done by **Roosli et al. (2019)** [118] for ever versus never use, but did not include the **Spinelli et al. (2010)** [47] study (no reason given) and instead of using all malignant brain tumors from **Muscat et al. (2000)** [40], they included separate ORs for astrocytic tumors (0.80 [0.50-1.20]) and oligodendrogliomas and mixed gliomas (0.90 [0.40-2.10]). They also included wireless telephones from **Hardell et al. (2015)** [88] in their analyses. Their analysis resulted in an mRR of 1.03 (0.92-1.16). They also did meta-analyses on the data for 0-5 years (0.92 [0.77-1.09]), 5-10 years (1.07 [0.88-1.30]) and  $\geq 10$  years (1.33 [1.05-1.67]). Their  $\geq 10$  years category was done differently than **Roosli et al. (2019)** [118] in that they did not include **Yoon et al. (2015)** [93] and the 4 exposure categories for **Hardell et al. (2015)** [88] were entered directly into the analysis rather than being pooled first. All of these analyses showed significant heterogeneity which they said was reduced by removing either the Interphone study or the study by **Hardell et al. (2015)** [88]. For ipsilateral tumors and ever versus never use, they saw an mRR of 1.26 (0.87-1.84) in comparison to contralateral use that showed an mRR of 1.10 (0.85-1.42). Finally, evaluating gliomas located in the temporal lobe, again for ever versus never use, they saw an mRR of 1.61 (0.78-3.33) [Note that in the text of the manuscript rather than their table, they list this mRR as 0.93 (0.69-1.24); I was able to verify the mRR of 1.61 but could not find a reasoning behind the number in the text]. The relative weights for the individual studies also fail to match the sample sizes in these evaluations.

**Yang et al. (2017)** [120] also performed a meta-analysis on some of the studies included in this review. Their analysis excluded both the **Hardell et al. (2015)** [88] pooled analysis and the **Interphone (2010)** [48] pooled analysis. Instead, they included the **Hardell et al. (2011)** [126] study that included the pooled analysis of the 1997-2003 studies with the inclusion of deceased cases and individual Interphone studies from separate countries [49, 52, 54, 55, 59, 61] or a pooled analysis from 5 countries [64]. For ever versus never use, they saw an mRR of 0.98 (0.88-1.10) and for  $\geq 10$  years duration of use, the mRR was 1.44 (1.08-1.91); both evaluations showed substantial heterogeneity. For ipsilateral use and ever/never exposures, the mRR was 0.97 (0.88-1.06) whereas for contralateral use it was 0.75 (0.65-0.87) with marginal heterogeneity. For  $\geq 10$  years use, the ipsilateral mRR was 1.46 (1.12-1.92) and contralateral use was 1.12 (0.81-1.55) with no heterogeneity. The studies on laterality did not include the study by **Hardell et al. (2011)** [126] for low-grade (1.11 [0.87-1.42] ever/never, 2.22 [1.69-2.92]  $\geq 10$  years) and high grade (0.82 [0.68-0.99] ever/never; 1.16 [0.85-1.59]  $\geq 10$  years) gliomas.

The remaining meta-analyses are older and use fewer and fewer of the individual studies. One meta-analysis worth mentioning is the one done by **Hardell et al. (2013)** [127] directly comparing the results of **Hardell et al. (2011)** [128] with the results from the pooled **Interphone (2010)** [48] study. For a latency of  $\geq 10$  years, they saw the following mRRs: all users 1.48 (0.65-3.35); ipsilateral 1.84 (0.80-4.25); contralateral 1.23 (0.40-3.73); temporal lobe 1.71 (1.04-2.81). For a cumulative use  $\geq 1640$  hours, they saw the following mRRs: all users 1.74 (1.07-2.83); ipsilateral 2.29 (1.56-3.37); contralateral 1.52 (0.90-2.57); temporal lobe 2.06 (1.34-3.17). An important point of this report is that the **Interphone (2010)** [48] study included adults 30-59 years of age and **Hardell et al. (2011)** [128] extracted the same group from their 1997-2003 pooled analysis [86] and adjusted the exposure groupings to match the Interphone groupings. They did not present these numbers in

their meta-analysis, but that can be done. The results of the same random-effects modeling as done by **Hardell et al. (2011)** [128] yields the following results:  $\geq 10$  years 1.30 (0.72-2.33);  $\geq 1640$  hours 1.48 (1.13-1.92);  $\geq 1640$  hours ipsilateral 2.03 (1.37-3.00);  $\geq 1640$  hours contralateral 1.32 (0.76-2.28).

It is clear from these numerous meta analyses, that the choice of which studies to use, how to enter the multiple studies by Hardell et al. and whether to use the pooled analysis from the Interphone study or some of the single analyses can have an impact on the final values. To provide a better view of the results, Figure 1 is a forest plot of all of the ORs from individual publications that evaluated regular use versus minimal or never use or ever use versus never use (if both were given in a study, regular use is shown). The column labeled "Study" provides the reference to the publication and the years in which cases and controls were collected for case control studies and the years when phone use information was collected for cohort studies and the year in which follow-up ended. Some studies are pooled evaluations of multiple other studies, so the other studies are indented. For example, the **Interphone (2010)** [48] study (Study F) is the pooled analysis of studies from 13 countries. **Lahkola et al. (2007)** [64] (Study F3) is a pooled analysis of the data from 5 of those countries and **Christenson et al (2005)** [49] (Study F3a) is the publication for data from one of those 5 countries. The column labeled "RR" is the risk ratio (OR, RR or mRR) from the study, "Lower" and "Upper" are the lower and upper bound on a 95% confidence interval around the RR. The graphic on the right simply plots the RR as a square or diamond with the "whiskers" (blue line running through the box) showing the width of the 95% confidence interval. The vertical line passing through 1 represents no effect. If the box and both whiskers are to the right of this line (greater than 1) and not touching it, this finding is statistically significant with a positive effect; if they fall completely to the left of the vertical line (below 1), then the risk is significantly reduced. The blue boxes that are filled in are major studies, the blue boxes that are white in the middle are the sub-studies and the red diamonds are all meta-analyses.

The graphic in Figure 1 is very useful for examining these types of data in a single view. Looking just at the filled in blue blocks (Studies A,B,C,D,E,F,G,H,I,J,K,L), it is clear some studies (D, I) fall clearly above the vertical line and demonstrate statistically significant increased risk. One study (F) shows a significant reduction in risk. The remaining studies show increases (H, J, K) or decreases (A, B, E, G, L) or no risk (C). The question to be addressed is what is the overall tendency of these data? The meta-analyses address this issue. The first meta-analysis (Meta Analysis A,B,C,D,E,F,G,H,I,J,K,L) combines the information from all of the major studies to produce an mRR of 1.01 (0.92-1.11) for ever versus never exposure suggesting that all of the positives and negatives balance out to give no overall effect. This meta-analysis also shows these studies are very different (Homogeneity Test:  $p=0.01$ ) which suggests the combination is not accounting for all of the variability in the RRs. However, as mentioned earlier, the newer, larger studies represent longer exposures, so I have also done meta-analyses on four large, recent case-control studies (F,H,I,J) and the two cohort studies (K,L) which should carry the greatest weight in any decision. Combining the four case-control studies (Meta Analysis F,H,I,J) results in a mRR of 1.09 (0.8-1.49), a slight increase in risk from the use of a mobile phone, but still heterogenous across studies. The combined cohort studies yield a mRR of 0.97 (0.74-1.27) suggesting no risk, and no heterogeneity ( $p=0.84$ ). Combining the 4 case-control studies and the 2 cohort studies (Meta Analysis F,H,I,J,K,L) yields an mRR of 1.03 (0.86-1.24) again suggesting no risk but with significant heterogeneity ( $p=0.00$ ).

As mentioned earlier, the Interphone study did an alternate set of analyses where the referent group was different depending upon the exposure metric being used (Appendix 2 Table, **Interphone (2010)**). It is possible to use meta-analysis to combine these results to get a pseudo regular/not

mRR for each exposure metric<sup>3</sup>. The rows labelled F6, F7 and F8 are the mRR values for these meta-analyses: F6 is an estimate of  $\geq 2$  years since start of regular use compared to 1-2 years of regular use [mRR 1.75 (1.40-2.18)], F7 is  $\geq 5$  hours of cumulative hands-free use compared to  $< 5$  hours [mRR 1.16 (1.00-1.35)], and F8 compares  $\geq 1500$  cumulative calls to  $< 1500$  cumulative calls [mRR 1.12 (0.96-1.30)]. To evaluate the sensitivity of the meta-analyses to the use of this alternative set of reference groups, I applied the least significant evaluation (F8) to the meta-analyses as a replacement for the Interphone study value (F). For the full analysis (Meta Analysis A,B,C,D,E,F8,G,H,I,J,K,L), the mRR becomes almost statistically significant; mRR 1.06 (0.98-1.15). Using just the larger and recent case-control studies (Meta Analysis F8,H,I,J), the mRR is significant [mRR 1.19 (1.07-1.33)] as is the combination of these case-control studies with the cohort studies [mRR 1.12 (1.01-1.24)]. None of these meta-analyses substituting F8 for F show significant heterogeneity. Thus, the meta-analysis is highly sensitive to the use of the reference group for the Interphone study.

Figure 2 is a forest plot of all of the ORs from individual publications that reported on duration of use  $\geq 8$  years or more. There are 6 studies; 5 of these studies show groupings of 1-4 years, 5-9 years and  $\geq 10$  years and one study with groupings of 1-5 years, 5-8 years and  $\geq 8$  years. For the study by **Hardell et al. (2015)** [88], groupings of 10-14, 15-19, 20-24 and  $\geq 25$  years were combined by meta-analysis to get a single mRR for  $\geq 10$  years. For **Frei et al. (2011)** [96], individual male and female RRs were combined by meta-analysis to get a single mRR for males and females combined. There are 4 groups of meta-analyses each with three separate meta-analyses for 1-4 years, 5-9 years and  $\geq 10$  years (combined with 1- $< 5$  years, 5-8 years and  $\geq 8$  years respectively for **Yoon et al. (2015)** [93]). The four groups are case-control studies, case-control studies and cohort studies, then the same two groups substituting the original analysis in the Interphone study with their alternative analysis using 1-1.9 years as the referent group. A few things are noticeable in the Forest plot; with the exception of **Yoon et al. (2015)** (D), all of the case-control studies (A, B and C) show increasing ORs with increasing duration of use. The cohort studies (E and F) generally have decreasing RRs with increasing duration. In the meta-analyses, regardless of how the data are combined, there are increasing mRRs with increasing duration. The case-control studies generally show larger mRRs than the case-control and cohort studies combined and using the alternative referent group from the Interphone study yielded the largest mRRs with the highest 2 categories of duration being statistically significant for case-control studies using the alternate referent group.

The studies in adults are consistent.

**Aydin et al. (2012)** is the only study in children that looked at regular use of a mobile telephone and saw an OR of 1.36 (0.92-2.02). For years since first use, they saw ORs of 1.35 (0.89-2.04), 1.47 (0.87-2.49) and 1.26 (0.70-2.28) for lag times of  $\leq 3.3$  years, 3.3-5 years and  $> 5$  years respectively. When they used operator-recorded first use and lag times of  $\leq 1.8$  years, 1.8-2.8 years and  $> 2.8$  years, they saw a significant increasing risk ( $p=0.001$ ) and ORs of 0.78 (0.43-1.40), 1.71 (0.85-3.44) and 2.15 (1.07-4.29) respectively. When they divided the tumors into gliomas or other tumors, they saw an OR for gliomas of 1.14 (0.66-1.97) and for other of 1.65 (0.93-2.93). They saw no

---

<sup>3</sup> To build this combination, a meta-analysis is done on all of the risk ratios for a specific exposure metric (e.g. 1-5 years, 5-10 years and  $\geq 10$  years latency). To check if this yields reasonable mRRs, meta-analyses were used to combined the various categories under the three exposure metrics in the cases where the referent group is non-regular users. This analysis yielded OR=0.81 (0.70-0.94) whereas doing a meta-analysis to get an equivalent estimate yielded mRR=0.84 (0.72-0.99) for latency years, mRR=0.82 (0.72-0.94) for cumulative hours and mRR=0.82 (0.75-0.90) for cumulative number of call.

relationship with the temporal lobe (1.00 (0.58-1.72). **Feltblower et al. (2014)** saw an OR of 0.9 (0.2-3.3) for young adults who used a mobile phone more than 20 times.

Study	RR	Lower	Upper
A: Handet et al. (1989) (1984-1996)	0.99	0.67	1.50
B: Muscat et al. (2000) (1984-1998)	0.80	0.50	1.20
C: Inskip et al. (2001) (1984-1995)	1.00	0.70	1.40
D: Avloneh et al. (2002) (1996)	1.50	1.00	2.40
E: Oksanen et al. (2008) (2005-2007)	1.00	0.69	1.51
F: INTERPHONE (2010) (2000-2008)	0.81	0.70	0.94
F1: Sams et al. (2006) (2000-2005)	0.88	0.74	1.04
F2: Lohkova et al. (2007) (2000-2004)	0.78	0.68	0.91
F3a: Christenson et al. (2005) (2000-2002)	0.71	0.52	0.97
F3b: Klashore et al. (2007) (2001-2002)	0.60	0.40	0.90
F3c: Lonn et al. (2008) (2000-2002)	0.80	0.60	1.00
F3d: Hoppworth et al. (2006) (2000-2004)	0.84	0.70	1.13
F4: Taketayashi et al. (2008)	1.22	0.62	2.37
F5: Carols et al. (2011) (2000-2004)	0.92	0.70	1.13
F5a: Hours et al. (2007) (2001-2003)	1.10	0.60	2.00
F6: Referent 1-4 hours (meta)	1.70	1.40	2.10
F7: Referent <4 hours (meta)	1.16	1.00	1.35
F8: Referent <150 calls (meta)	1.12	0.90	1.30
G: Hadd et al. (2011) (1989-2001)	0.82	0.53	1.26
H: Coureau et al. (2014) (2004-2008)	1.24	0.66	1.77
I: Handet et al. (2015) (1987-2002, 2017-2008)	1.00	1.10	1.00
I1: Handet et al. (2006) (1987-2000)	1.40	1.10	2.10
I1a: Handet et al. (2006) (1987-2000) Analog	1.10	0.80	1.60
I1a: Handet et al. (2006) (1987-2000) Digital	1.00	0.80	1.20
I1b: Handet et al. (2006) (2000-2003) Analog	2.00	1.50	4.00
I1b: Handet et al. (2006) (2000-2003) Digital	1.80	1.30	2.70
I2: Handet et al. (2013) (2007-2009)	2.80	0.20	9.90
J: Youn et al. (2015) (2000-2007)	1.17	0.60	2.14
K: Frei et al. (2011) (1980-1995, and 2007) meta	1.07	0.90	1.20
K1: Frei et al. (2011) Mexico	1.08	0.90	1.20
K2: Frei et al. (2011) Venezuela	0.90	0.60	1.40
K3: Schuz et al. (2006) (1987-1995, and 2002)	1.61	0.90	3.14
K4: Johnsen et al. (2001) (1987-1995, and 1996)	0.94	0.72	1.20
L: Benson et al. (2013) (1989-2005, follow-up 2008)	0.80	0.50	1.14
<b>Meta Analysis A,B,C,D,E,F,G,H,I,J,K,L</b> <small>Heterogeneity Test: I<sup>2</sup>=64.4% p=0.01</small>	<b>1.01</b>	<b>0.92</b>	<b>1.11</b>
<b>Meta Analysis F,H,I,J</b> <small>Heterogeneity Test: I<sup>2</sup>=53.3% p=0.00</small>	<b>1.09</b>	<b>0.80</b>	<b>1.49</b>
<b>Meta Analysis K,L</b> <small>Heterogeneity Test: I<sup>2</sup>=55.9% p=0.02</small>	<b>0.97</b>	<b>0.74</b>	<b>1.27</b>
<b>Meta Analysis F,H,I,J,K,L</b> <small>Heterogeneity Test: I<sup>2</sup>=64.4% p=0.00</small>	<b>1.00</b>	<b>0.80</b>	<b>1.24</b>
<b>Meta Analysis A,B,C,D,E,F,G,H,I,J,K,L</b> <small>Heterogeneity Test: I<sup>2</sup>=42.9% p=0.11</small>	<b>1.06</b>	<b>0.98</b>	<b>1.15</b>
<b>Meta Analysis F8,H,I,J</b> <small>Heterogeneity Test: I<sup>2</sup>=30.0% p=0.02</small>	<b>1.19</b>	<b>1.07</b>	<b>1.33</b>
<b>Meta Analysis F8,H,I,J,K,L</b> <small>Heterogeneity Test: I<sup>2</sup>=57.9% p=0.01</small>	<b>1.12</b>	<b>1.01</b>	<b>1.24</b>

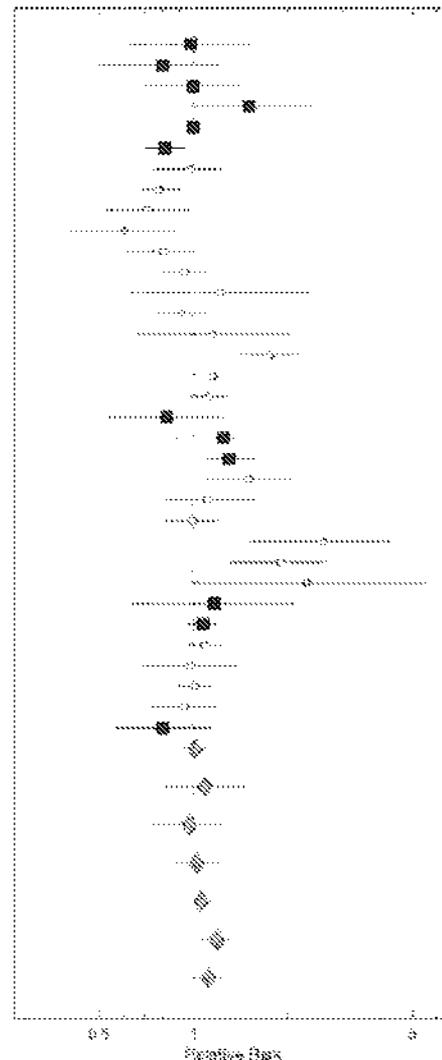


Figure 1: Forest plot and meta-analyses of regular use or ever use of cellular telephones and the risk of glioma [studies with a solid blue square either single studies that stand alone or pooled studies that encompass numerous single studies; open squares are individual studies or smaller pooled studies; red diamonds are meta-analyses]<sup>a</sup>

<sup>a</sup> - The column labeled "Study" provides the reference to the publication and the years in which cases and controls were collected for case control studies and the years when phone use information were collected for cohort studies and the year in which follow-up ended. Some studies are pooled evaluations of multiple other studies, so the other studies are indented. For example, the Interphone study (Study F) is the pooled analysis of studies from 13 countries. Lohkova et al. (2007) (Study F3) is a pooled analysis of the data from 5 of those countries and Christenson et al (2005) (Study F3a) is the publication for data from one of those 5 countries. The column labeled "RR" is the risk ratio (OR, RR or mRR) from the study, "Lower" and "Upper" are the lower and upper bound on a 95% confidence interval around the RR. The graphic on the right simply plots the RR as a square or diamond with the "whiskers" (blue line running through the box) showing the width of the 95% confidence interval. The vertical line passing through 1 represents no effect. If the box and both whiskers are to the right of this line (greater than 1) and not touching it, this finding is statistically significant with a positive effect; if they fall completely to the left of the vertical line (below 1), then the risk is significantly reduced. The blue boxes that are filled in are major studies, the blue boxes that are white in the middle are the sub-studies and the red diamonds are all meta-analyses. "Homogeneity Test" provides the I<sup>2</sup> statistic and the p-value for the Q-test.

Study	RR	Lower	Upper
A1: Interphone (2010) 1-4 years (meta)	0.74	0.56	0.98
A2: 5-9 years	0.81	0.60	0.97
A3: ≥10 years	0.95	0.76	1.26
AM1: 2-4 years (1-1.9 referent)	1.88	1.16	2.41
AM2: 5-9 years (1-1.9 referent)	1.54	1.06	2.22
AM3: ≥10 years (1-1.9 referent)	2.18	1.43	3.31
B1: Coureau et al. (2014) 1-4 years	0.88	0.56	1.39
B2: 5-9 years	1.34	0.87	2.06
B3: ≥10 years	1.51	0.85	3.09
C1: Hardež et al. (2016) 1-4 years	1.29	0.98	1.50
C2: 5-9 years	1.59	1.20	1.80
C3: ≥10 years (meta)	1.81	1.35	2.43
D1: Yoon et al. (2015) 1-5 years	1.22	0.62	2.64
D2: 5-8 years	1.27	0.63	2.56
D3: ≥6, >8 years	1.04	0.52	2.09
E1: Frei et al. (2011) 1-4 years (meta)	1.17	0.94	1.44
E2: 5-9 years (meta)	1.05	0.88	1.25
E3: ≥10 years (meta)	1.04	0.86	1.25
F1: Benson et al. (2013) <5 years	0.92	0.71	1.21
F2: 5-9 years	0.92	0.75	1.13
F3: ≥10 years	0.78	0.55	1.10
<b>Meta Analysis: A1,B1,C1,D1 (case-control)</b>			
Homogeneity Test: $I^2=0.06$ $p=0.95$	0.97	0.73	1.31
<b>Meta Analysis: A2,B2,C2,D2 (case-control)</b>			
Homogeneity Test: $I^2=0.24$ $p=0.90$	1.18	0.82	1.72
<b>Meta Analysis: A3,B3,C3,D3 (case-control)</b>			
Homogeneity Test: $I^2=1.38$ $p=0.91$	1.32	0.90	1.94
<b>Meta Analysis: A1,B1,C1,D1,E1,F1 (all)</b>			
Homogeneity Test: $I^2=5.49$ $p=0.97$	1.01	0.85	1.20
<b>Meta Analysis: A2,B2,C2,D2,E2,F2 (all)</b>			
Homogeneity Test: $I^2=1.98$ $p=0.99$	1.09	0.89	1.35
<b>Meta Analysis: A3,B3,C3,D3,E3,F3 (all)</b>			
Homogeneity Test: $I^2=1.17$ $p=0.93$	1.14	0.88	1.47
<b>Meta Analysis: AM1,B1,C1,D1 (case-control)</b>			
Homogeneity Test: $I^2=0.21$ $p=0.93$	1.24	0.97	1.59
<b>Meta Analysis: AM2,B2,C2,D2 (case-control)</b>			
Homogeneity Test: $I^2=0.00$ $p=0.99$	1.47	1.25	1.73
<b>Meta Analysis: AM3,B3,C3,D3 (case-control)</b>			
Homogeneity Test: $I^2=9.07$ $p=0.25$	1.77	1.40	2.23
<b>Meta Analysis: AM1,B1,C1,D1,E1,F1 (all)</b>			
Homogeneity Test: $I^2=8.73$ $p=0.19$	1.16	0.98	1.36
<b>Meta Analysis: AM2,B2,C2,D2,E2,F2 (all)</b>			
Homogeneity Test: $I^2=6.67$ $p=0.97$	1.22	1.00	1.48
<b>Meta Analysis: AM3,B3,C3,D3,E3,F3 (all)</b>			
Homogeneity Test: $I^2=7.52$ $p=0.90$	1.31	0.94	1.83

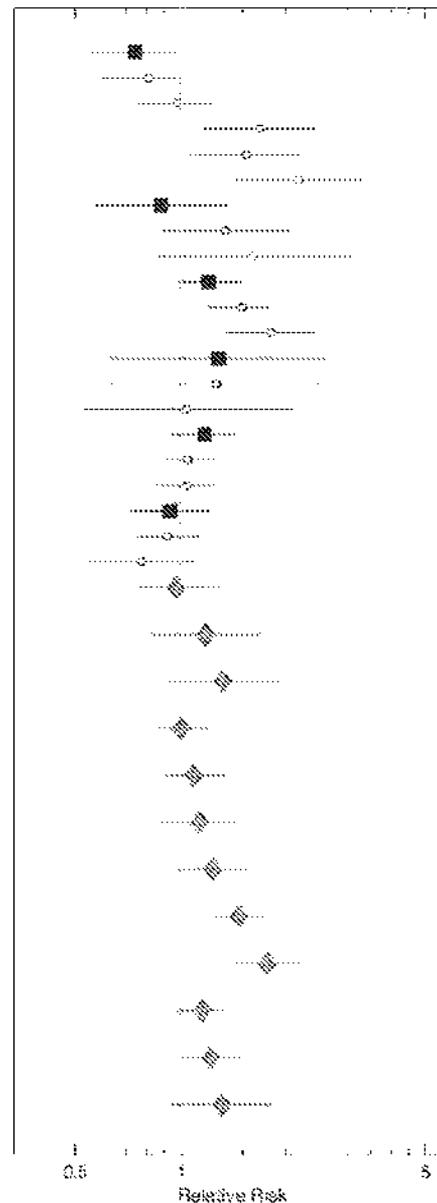


Figure 2: Forest plot and meta-analyses of duration of use of cellular telephones and the risk of glioma [studies with a solid blue square are either single studies that stand alone or pooled studies that encompass numerous single studies; open squares are second analysis from that same paper; red diamonds are meta-analyses, the columns and the figure are as in Figure 1].

#### 4.1.3.2 Exposure-Response Relationship

The best measure for exposure-response relationships is the cumulative hours of use of a cellular telephone since it includes both the frequency of use and the duration of use. While duration of use is also a form of exposure-response, it is more likely that, similar to ionizing radiation, total

accumulated exposure is related to the risk of glioma if a relationship exists. Table 3 provides the results for all of the epidemiology studies with estimates of the cumulative use of cellular phones.

**Inskip et al. (2001)** shows no consistent exposure-response and has all of the ORs below 1. **Spinelli et al. (2005)** show an increase in the OR for use of 48-432 cumulative hours, but this drops for  $\geq 432$  hours. In addition, their measure of cumulative hours is different from the remaining studies in that they calculated frequency of use based upon the number of hours allowed in the subscription rather than the actual usage as recounted by the user. This could lead to misclassification of exposure and may have affected the ORs. The **Interphone study (2010)** basically shows flat exposure-response for the entire study until the largest exposure category, that is significantly elevated in risk with an OR of 1.40 (1.03-1.89). Using greater than 0 but less than 5 hours as the referent group, they see higher ORs with a slight increasing pattern and again the highest exposure group significantly elevated. **Coureau et al. (2014)** saw a clearly increasing exposure-response pattern with ORs below 1 in the low exposure categories and becoming marginally significant in the second highest exposure group [1.78 (0.98-3.24)] and significant in the highest exposure category [2.89 (1.41-5.93)]. Excluding proxies did not change this pattern. **Hardell et al. (2015)** saw a clear pattern of increasing risk with increasing exposure with all of their categories statistically significant. They also did a regression resulting in an OR of 1.013 (1.009-1.017) per hundred cumulative hours of use with a  $p < 0.0001$ . Finally, **Yoon et al. (2015)** saw a similar up-down pattern as **Spinelli et al. (2009)**, but with lower ORs and none of them significant.

It is not possible from the published results to find categories of exposure that match across the various studies in order to do a simple meta-analysis by category. However, it is possible to do a meta-regression where the exposure categories are turned into a single exposure and the meta-regression tests to see if the slope of the data from the various studies is increasing with exposure. In order to do this analysis, I set the exposure for each category equal to the center of the interval defined for the category (e.g., if the category is 512-1486 hours, the midpoint exposure is  $(512+1486)/2=999$  hours). For **Inskip et al. (2001)**, the last category is  $\geq 100$  hours and had 54 cases and  $\geq 500$  hours had 27, so I chose 500 for the highest exposure. For the remaining studies, it is not clear how to choose the exposure of the highest category. To follow the same pattern seen with **Inskip et al. (2001)**, I chose 5x the lower limit of the last category as the regression point for that category. **Hardell et al. (2015)** did a regression through their data and saw an OR of 1.013 (1.009-1.017) per 100 hours; doing a meta-regression using only the **Hardell et al. (2015)** data with the highest category dose set at  $5 \times 1486 = 7430$  hours yields an mRR of 1.011 (1.005-1.018), similar to the result seen by **Hardell et al. (2015)**. A second dosing approach for the last category was to take the difference between the middle of the second largest category and the lower bound of that category and add it to the upper end of the second highest category to get the exposure for the highest category (e.g. if 512-1486 hours is the second highest category and the last category is  $\geq 1486$  hours, I set the center of the highest category as  $(512+1486)/2-512+1486=1973$  hours). The exposures for all of the categories of the studies entering into the main meta-regression are shown in Table 9. The study results from **Spinelli et al. (2009)** are excluded from the meta-regression because of the difference in their exposure metric.

Table 10 provides the results of the meta-regression for the 5 case-control studies with duration of exposure where all of the ORs are a comparison against non-regular users. There is a significant association between exposure and risk with an mRR of 1.007 (1.002-1.012,  $p=0.004$ ). Dropping the **Interphone (2010)** study from the meta-regression results in a highly significant trend (1.011 [1.005-1.017];  $p < 0.001$ ), almost doubling of the risk, and reduced heterogeneity between the studies. In contrast, dropping the study by **Hardell et al. (2015)** reduces the risk by almost half

(1.004 [0.998-1.010; p=0.184) but the heterogeneity remains. Dropping any of the other studies has little impact on the findings. The alternate dosing strategy for the highest dose yielded the same pattern but mRRs that are roughly 3 times higher than those presented in Table 10 (not shown). (Table 10)

To examine the sensitivity of the analysis to the use of a different referent population in the Interphone study, their analysis using greater than 0 and <5 hours of cumulative exposure as the referent group was plugged into the same analysis. Table 11 provides the results of the meta-regression for the 5 case-control studies with duration of exposure using the alternative referent group. There is an increase in the mRR to 1.010 (1.006-1.014) per 100 hours of use. This fit demonstrated less heterogeneity with  $I^2=33.95$ . None of these results change substantially if any one study is dropped from the meta-regression. The alternative high dose yielded the same pattern but higher ORs per 100 hours (not shown). (Table 11)

There were other measures of exposure used in the various studies that are worth mentioning. **Inskip et al. (2001)** used average daily exposure and saw no exposure-response relationship (Table 4). **Coureau et al. (2014)** used average monthly exposure and saw a fairly clear exposure-response relationship (Table 4). **Inskip et al. (2001)** also considered the year that cellular telephone use began and again saw no exposure-response (Table 5). The **Interphone Study (2010)** considered cumulative use by years of duration of use (1-4 years, 5-9 years and  $\geq 10$  years). In each duration category, they saw the same pattern of flat exposure-response except for the highest cumulative exposure group that was increased in all categories. The shortest duration had the highest OR in the highest cumulative use category, but also had only 25 exposed cases with that much usage (to get greater than 1640 hours of usage in 4 years would require >1 hour of usage every day) (Table 5). **Coureau et al. (2014)** considered cumulative number of calls and saw a non-significant increasing risk with increasing exposure (Table 5). **Hardell et al. (2015)** used age and saw no pattern (Table 5).

**Elliott et al. (2010)** compared distance to power station, total power and modeled power to evaluate the contributions of mobile phone towers on the rates of brain and central nervous system tumors in young adults and basically saw no relationship. **Li et al. (2012)** did something similar but calculated exposure for an entire township instead of individuals. They saw slightly increased ORs for different types of divisions of the data and an increase in the risk of brain tumors of 1.09 (0.95-1.25) per standard deviation of their exposure density measure.

**Aydin et al. (2013)** looked at total cumulative years of use of a mobile phone by self-reporting and operator recorded cumulative years of use and saw marginal increases in risk with increasing exposure (p=0.14 and p=0.15 respectively, (Table 8)). When they also looked at cumulative hours of use for the self-reported and operator-recorded data, they saw no relationship although all ORs were greater than 1.

Table 9: Meta-Regression Exposure Values for Tables 11 and 12

Author (year)	Exposures (times 100 hrs)
Inskip et al. (2001)	0.065, 0.57, 5.00
Interphone (2010)	0.025, 0.09, 0.22, 0.46, 0.88, 1.575, 2.80, 5.475, 11.875, 82
Coureau et al. (2014)	0.215, 0.775, 2.255, 6.27, 44.8
Hardell et al. (2015)	0.615, 3.17, 9.99, 74.3
Yoon et al. (2015)	1.50, 6.00, 45

Table 10: Meta-Regression Analysis with Sensitivity Analysis of ORs for Five Case-Control Studies using Cumulative Hours of Use as the Exposure Metric and the Original Referent Groups

Meta Regression Studies <sup>a,b</sup>	Coefficient	P> Z	95% Confidence Interval		I <sup>2</sup>	pQ
All	1.007	0.004	1.002	1.012	68.18	<0.001
drop Inskip et al. (2001)	1.007	0.004	1.002	1.012	71.34	<0.001
drop Interphone (2010)	1.011	<0.001	1.005	1.017	54.36	0.006
drop Coureau et al. (2014)	1.006	0.02	1.001	1.011	71.65	<0.001
drop Hardell et al. (2015)	1.004	0.184	0.998	1.010	61.27	0.001
drop Yoon et al. (2015)	1.008	0.001	1.003	1.013	69.85	<0.001

a – studies included in the analysis are Inskip et al. (2001), Interphone (2010), Coureau et al. (2014), Hardell et al. (2015), Yoon et al. (2015); b - Interphone Study uses <1 year duration of use as the referent group

Table 11: Meta-Regression Analysis<sup>a</sup> with Sensitivity Analysis of ORs for Five Case-Control Studies using Cumulative Hours of Use as the Exposure Metric and the Alternative Referent Group for the Interphone Study

Meta Regression Studies <sup>a,b</sup>	Coefficient	P> Z	95% Confidence Interval		I <sup>2</sup>	pQ
All	1.010	<0.001	1.006	1.014	33.95	0.054
drop Inskip et al. (2001)	1.010	<0.001	1.006	1.014	38.66	0.037
drop Interphone (2010)	1.011	<0.001	1.005	1.017	54.36	0.006
drop Coureau et al. (2014)	1.009	<0.001	1.005	1.013	35.34	0.065
drop Hardell et al. (2015)	1.008	0.003	1.003	1.013	0.49	0.451
drop Yoon et al. (2015)	1.011	<0.001	1.007	1.014	27.65	0.118

a – studies included in the analysis are Inskip et al. (2001), Interphone (2010), Coureau et al. (2014), Hardell et al. (2015), Yoon et al. (2015); b - Interphone Study uses greater than 0 and <5 hours cumulative use as the referent group

#### 4.1.3.3 Strength of the Association

The strength of the association is tied to the magnitude of the response and the statistical significance of that response. For all of these studies, the actual magnitude of the RRs seen in the studies are small, in many cases falling below 1. It is clear from [Figure 2](#), that the longer the duration, the larger the mRR and the more statistical significance to the risk. It is also clear from [Figure 2](#) that the actual analysis used from the **Interphone study (2010)** can make a difference in the magnitude of the response. This is a strong set of findings.

In addition, laterality matters for addressing the strength of the association. Laterality seems to become more pronounced with a longer duration of exposure or greater cumulative hours of use. For ≥10 years of usage, the **Interphone study (2010)** has an ipsilateral RR of 1.21 (0.82-1.80) and a contralateral RR of 0.70 (0.42-1.15) whereas **Hardell et al. (2015)** saw an ipsilateral mRR of 2.24 (1.61-3.11) (pooling all categories above 10) and contralateral of 1.52 (0.99-2.34). Combining these by meta-analysis yields an mRR of 1.66 (0.91-3.04) for ipsilateral and 1.04 (0.49-2.23) for contralateral with significant heterogeneity (not shown). For cumulative duration of use in the highest category, the **Interphone study (2010)** has ipsilateral 1.96 (1.22-3.15) and contralateral 1.25 (0.64-2.43), **Coureau et al. (2014)** has ipsilateral 4.21 (0.70-25.42) and contralateral 1.61 (0.56-4.62), and **Yoon et al. (2015)** has ipsilateral 1.77 (0.32-1.84) and contralateral 0.63 (0.24-1.65). Combining these by meta-analysis yields an mRR of 1.99 (1.33-3.00) for ipsilateral and 1.11 (0.68-1.80) for contralateral with no heterogeneity (not shown). These results are surprisingly consistent and suggest a strong effect on laterality.

Finally, since the temporal lobe gets some of the highest fields when using a mobile phone, many researchers have looked at whether this location seems to associate with the use of mobile phones.

The Interphone study evaluated this for  $\geq 10$  years duration [1.36 (0.88-2.11)] and for  $\geq 1640$  hours cumulative use [1.87 (1.09-3.22)]. **Hardell et al. (2015)** did not address this issue for longer latency, but in one of their earlier studies, **Hardell et al. (2013)**, they found the following : 10-15 years latency 1.6 (0.7-4.1), 15-20 years 2.0 (0.8-5.2), 20-25 years 2.7 (1.02-7.3) and  $\geq 25$  years (4.8 (1.7-14). A meta-analysis of these numbers from **Hardell et al. (2013)** yields mRR 2.41 (1.49-3.89) (no heterogeneity) which, when combined with **Interphone (2010)** yields an mRR of 1.79 (1.02-3.14) (some heterogeneity,  $pQ=0.08$ ). Regretfully, no other study looked at this issue for the highest exposure categories. However, 4 studies addressed this for the evaluation of ever versus never exposure and saw ORs of 0.86 (0.66-1.13) (Interphone), 3.94 (0.81-19.08) (Coureau), 4.30 (1.99-9.27) (Hardell) and 1.13 (0.86-1.48) (Frei.). The combined mRR for these 4 is 1.56 (0.88-2.77) with significant heterogeneity (not shown).

#### 4.1.4 Ecological Epidemiology Studies of Malignant Brain Tumors and Gliomas

Ecological epidemiology studies attempt to look at trends of disease in a population and relate this to a particular exposure that changes over time or space in the population. The main difference between an ecological epidemiology study and the studies discussed up to this point (case-control and cohort studies) is that the unit of observation is a population, not an individual. Thus, ecological studies do not ask the individuals about their exposures but instead infer that exposure based upon other information. All of the ecological studies regarding cellular telephone use are based upon the idea that cellular telephone use has been increasing over time and this would imply that glioma rates in a population will be increasing in time as well. To be able to do this type of analysis, one would need to know the statistics on the use of cell phones in this population; something that is seldom known and must be inferred from statistics on ownership of a cellular phone or from the control populations in the case-control studies or from the usage seen in the cohort studies.

Usage data from the cohort studies, if obtained in a timely manner, would be a good estimate of usage in the general population. Regretfully, the two cohort studies in adults obtained these data early on in the use of cellular telephones (1982-1995 in Denmark and 1999-2005 in the UK) and their usage has increased dramatically since that time. Thus, it is hard to extrapolate from the usage in these populations to usage today. In the case-control studies, one can make assumptions of how well the cases and controls represent the general population, but these assumptions generally cannot be tested and may be wrong.

It is also required to have accurate information on cancers in a population. This type of information is usually derived from routinely collected national or regional statistics from cancer registries. Cancer registries can be notoriously inaccurate in the actual diagnosis of the cancer, gaps in coverage of a region or time and other problems. Because of all of these problems, ecological epidemiology studies are often affected by confounding or ecological fallacy (this occurs when inferences about what is happening at the individual level are derived from correlations seen in groups or populations). For these reasons, ecological studies are considered very weak in identifying or excluding risk factors that might be important in a population.

The ecological studies relevant to this review can be broken down into three categories: ecological studies on brain tumors in general, ecological studies on specific types of malignant brain tumors, and ecological studies on acoustic neuromas. In this section, I will review ecological studies on brain tumors and gliomas.

**Deltour et al. (2009)** [129] investigated temporal trends in glioma incidence rates in Denmark, Finland, Norway and Sweden using data from the national cancer registries. These data are intended to cover the populations incidence for 100% of the Nordic population and there is no discussion about limitations of the data for gliomas. They restricted their analysis to the years 1974-2003. They did a change-point analysis and saw no statistically significant change in incidence rates from 1998-2003, when they claimed changes caused by cell phones would be visible. They concluded any increase in gliomas caused by cell phones, if it exists, is not observable in this population. This is an extension of an earlier paper [130].

**Inskip et al. (2010)** [131] examined temporal trends in brain cancer incidence rates in the United States using data from the Surveillance, Epidemiology, and End Results (SEER) Program. For this analysis, they used SEER data from 9 cancer-registries which cover about 10% of the US population, restricted their analysis to Caucasians, and covered the years 1992-2006. They only saw increases in the 20-29 year age group in females. They also looked at specific locations in the brain and saw increases in both males and females in frontal lobe tumors. They concluded these findings do not support the view that use of cellular telephones increase cancer risks.

**de Vocht et al. (2011)** [132] examined temporal trends in brain cancer incidence rates in England using data from the UK Office of National Statistics. These data should cover 100% of the UK population, but there are gaps maybe as high as 35%. They restricted their analysis to the years 1998-2007. They saw no increases in any age group. They also looked at specific locations in the brain and saw increases in both males and females in temporal lobe tumors and in men only, frontal lobe tumors. They concluded these findings do not indicate a pressing need to implement a precautionary principle to reduce RF exposures.

**Ding and Wang (2011)** [133] investigated temporal trends in brain and nervous tissue cancer incidence rates in Shanghai using data from the Shanghai Cancer Registry. These data should cover 100% of the Shanghai population; gaps were not discussed. They restricted their analysis to the years 1983-2007. They saw a doubling of brain cancer incidence in this period with no statistically significant changes in the increasing rate at any specific time. They concluded the study did not support an increase in brain and nervous system tumors due to RF exposures because the trend began before the widespread use of cellular phones.

**Aydin et al. (2011)** [109] compared hypothetical incidence trends generated from the ORs seen in their study of childhood brain tumors to incidence data on brain tumors in children and adolescents aged 5-19 years between 1990 and 2008 from the Swedish Cancer Registry. They concluded the patterns did not match and that this indicates that short-term mobile phone use does not cause an increase in brain cancers in children. **Soderqvist et al. (2011)** [112] had concerns regarding the interpretation of these findings and suggested there could still be an effect. **Aydin et al. (2012)** [134] responded, basically reiterating their original arguments.

**Deltour et al. (2012)** [135] investigated temporal trends in glioma incidence rates in Denmark, Finland, Norway and Sweden using data from the national cancer registries. These data are intended to cover the populations incidence for 100% of the Nordic population and there is no discussion about limitations of the data for gliomas. In this period, incidence rates have increased slightly in men and women, mostly in older populations. Using simulation studies, various relative risks and various induction periods, they simulated the results of a cohort study on the entire population of men aged 40-59 years over this period (with complete follow-up). They then looked to see if they had a significant RR change in that population and equated that to being able to see a change in the incidence rates in the data from the cancer registries. The probability of seeing the

change ranged from 2.9 % to 100% depending on the underlying simulation parameters. They concluded that many increased or decreased risks reported in case-control studies are implausible, implying that biases and errors in the self-reported use of mobile phone have likely distorted the findings. This conclusion is at best speculative because the simulations do not actually match the incidence data they are looking at or the analyses they did with the data.

**Little et al. (2012)** [136] examined temporal trends in brain cancer incidence rates in the United States using data from the Surveillance, Epidemiology, and End Results (SEER) Program. For this analysis, they used SEER data from 12 cancer-registries (coverage of the US population is unknown). They restricted their analysis to non-Hispanic white people and the years 1992-2008. Using the findings from **Interphone (2010)** and **Hardell et al. (2011)**, they predicted what the tumor incidence rates in 2008 should have been by using 1992-1996 as a baseline rate and US subscription data to drive the temporal change. They concluded that the results from **Hardell et al. (2011)** are not consistent with the US SEER data but that the results from the Interphone (2010) study are.

**Barchana et al. (2012)** [137] examined temporal trends in brain cancer incidence in Israel using data from the Israel National Cancer Registry. These data should cover 100% of the Israeli population and is 95% complete for brain tumors. They restricted their analysis to the years 1989-2009. They focused on high-grade versus low-grade gliomas in males and females. They also examined changes in laterality. They found a decrease in low-grade gliomas over this period and an increase in high-grade gliomas. They also saw an increase in laterality towards more left-sided tumors. They concluded the decrease in low-grade gliomas correlated with the introduction of mobile phone technology in Israel.

**Hsu et al. (2013)** [138] examined temporal trends in malignant brain cancer incidence rates and death rates in Taiwan using data from the Taiwan National Cancer Registry. There was no discussion of the quality of this cancer registry. They restricted their analysis to the years 2000-2009. Their entire evaluation consisted of a side-by-side comparison in a histogram of deaths, incidence and cell phone usage. No statistical evaluations were performed. They concluded there was no detectable correlation between morbidity/mortality of malignant brain tumors and cell phone use in Taiwan.

**Kim et al. (2015)** [139] investigated temporal trends in primary brain cancer incidence rates in New Zealand using data from the New Zealand Cancer Registry. These data should cover 100% of the NZ population and there is some discussion about changes in histological classification that could produce a false-negative finding. They restricted their analysis to the years 1995-2010. In general, they saw a decrease in brain tumors over this period with a larger decrease in women than in men. They saw a significant increase in all brain tumors in females aged 30-49, with increases in glioma of the parietal and temporal lobe. This finding was not consistent over other age groups or with the rates in men. They saw increases in the 70+ years group in most categories, but attributed that to better diagnosis, but with no justification. They concluded there has been no increase in primary brain tumors over this period.

**Sato et al. (2016)** [140] investigated temporal trends in malignant neoplasms of the central nervous system incidence rates in Japan using nationwide estimates of cancer incidence developed by the regional cancer registries. These estimates are intended to cover the populations incidence for 100% of the Japanese population and there is some discussion about limitations of the estimates. They restricted their analysis to the years 1993-2010. They focused on men and women in their 20s and 30s and used data from a survey of cellular phone use to determine if these increases could be due to cellular phone use using the highest response category from the **Interphone (2010)** study as

the expected change in risk ratio. In general, they saw an increase in brain tumors over this period with a larger increase in men than in women. They were able to show that the observed increases were greater than what would be predicted for only heavy users and the **Interphone (2010)** OR of 1.4. They then went on to show that using ORs of 6 for men and 12 for women in their 20s and 4 for men and 7 for women in their 30s came close to matching the data. They then concluded that increases in cancers by sex, age and period are inconsistent with sex, age and period usage of mobile phones and thus cannot be explained by the mobile phones.

**Chapman et al. (2016)** [141] examined temporal trends in brain cancer incidence rates in Australia using data from the Australian Institute of Health and Welfare. These data should cover 100% of the Australian population, but there is no discussion of the quality of the data. They restricted their analysis to the years 1982-2012. They suggested incidence has risen slightly in males and remained steady in females. They then used cellular phone usage data from Australia and created hypothetical curves for a RR of 1.5 for users and a 10-year lag and a second hypothetical curve with a RR of 2.5 for heavy users (defined as >896 hours of cumulative use and assumed for 19% of all users) and a 10-year lag. They concluded the hypothetical curves were significantly different from the observed curves. They cited **Dobes et al. (2011)** [142] as showing no rise in brain tumors in Australia, however, this study concluded there was a significant rise in glioblastoma in Australia from 2000-2008 at an annual rate of 2.5%.

**de Vocht (2016)** [143] examined temporal trends in brain cancer incidence counts (not standardized rates) in England using data from the UK Office of National Statistics. These data should cover 100% of the UK population, but there are gaps maybe as high as 35% and a 5-year lag in getting complete data. He restricted the analysis to the years 1985-2014. He obtained cellular phone subscription data from the ITU. He built a Bayesian counterfactual model of glioma, glioblastoma, parietal lobe tumors and temporal lobe tumors with covariates annual cancer incidence, population size, median age, cigarette smoking, urbanization rate and a factor to account for data quality in a specific period. The counterfactual model was compared to a model including cell phone subscription rates with several cut points to allow for lag times. He concluded that for glioma, glioblastoma and malignant tumors of the parietal lobe, cell phone usage did not differ from the counterfactual model. For malignant tumors of the temporal lobe, he found cell phone usage could be a causative factor for these tumors. There was a major error in the data used for this analysis and a correction was published [144]. The author claimed it had no impact on the findings although it changed the directions of the effects seen. **de Vocht (2019)** [145] repeated this analysis for glioblastoma in specific brain regions and for meningiomas and acoustic neuromas. Excess of the counterfactual were seen for glioblastomas in the frontal and temporal lobe, but were predominantly in the highest age groups. No excesses were seen for acoustic neuromas or meningiomas. He concluded cell phones are unlikely to be causative for these tumors.

**Hardell and Carlberg (2017)** [146] demonstrated that the rates of brain tumors of unknown type obtained from the Swedish Inpatient Register were increasing in the years from 1998-2015. In contrast, brain tumor diagnoses confirmed by cytology/histology increased in the Swedish Cancer Registry. Brain tumors diagnosed by MRI and CT are not always reported to the Swedish Cancer Registry. This suggests an under-reporting of brain cancers in the cancer registry and they suggest caution in using cancer registry data to understand any linkage between cellular phone usage and brain cancers. This was also suggested in an earlier evaluation by this group [147].

**Phillips et al. (2018)** [148] examined temporal trends in brain cancer incidence in England using data from the UK Office of National Statistics. These data should cover 100% of the UK population,

but there are gaps maybe as high as 2% and a multi-year lag in getting complete data. They restricted their analysis to the years 1995-2015. They looked at a number of different forms of brain tumors and locations. They saw an increase in glioblastomas for 2011-2015 relative to 1995-1999 by age groups, with the largest increases in the higher age groups. The greatest increases were tumors in the frontal and temporal lobes. They suggest that widespread environmental or lifestyle factors may be responsible, but did not draw any conclusions regarding cellular phones.

**Keinan-Boker et al. (2018)** [149] examined temporal trends in brain cancer incidence in Israel using data from the Israel National Cancer Registry. These data should cover 100% of the Israeli population and is 95% complete for brain tumors. They restricted their analysis to the years 1990-2015. They focused on benign versus malignant tumors by age and sex. In general, they saw a mixed set of effects that changed over these categories. In conclusion, they found the results to be not consistent with the penetrance of cellular phones in Israel over this period.

**Karipidis et al. (2018)** [150] examined temporal trends in brain and central nervous system tumor incidence rates in Australia using data from the Australian Institute of Health and Welfare. These data should cover 100% of the Australian population, but there is no discussion of the quality of the data. They restricted their analysis to the years 1982-2013 and cases aged 20-59 years. There is no discussion of standardizing the rates. Percent of the population with mobile phone subscriptions was obtained from the Australian Communications and Media Authority. They used a very simple model to predict incidence rates from subscription data using regular users and heavy users (19%) and various lag times. They concluded that there was no evidence that mobile phone use correlated with any brain tumor histological type or subtype.

**Nilsson et al. (2019)** [151] examined temporal trends in glioma incidence rates in Sweden using data from the Swedish Cancer Registry. These data should cover 100% of the Swedish population. They restricted their analysis to the years 1980-2012 because problems with the registry starting in 2013. They saw no increases in age-standardized incidence rates over time and a significant decrease in low-grade gliomas. They concluded these findings do not indicate any effect of RF exposures on gliomas incidence.

**Natukka et al. (2019)** [152] examined temporal trends in glioma incidence rates in Finland using data from the Finnish Cancer Registry. These data should cover 100% of the Finnish population. They restricted their analysis to the years 1990-2016 with cases reclassified from 1990 to 2006 to match modern classifications. The data for 2007-2016 could not be classified by sex or age grouping. They discussed several major limitations of their analyses including misclassification, limitations to the analysis and small sample sizes. They saw no increases in age-standardized incidence rates for gliomas over 1990-2006 but could not do this analysis beyond then. There were no major changes in tumor locations over time.

These studies use a variety of different cancer registries and a variety of different methods to evaluate the relationship between temporal changes in brain cancer incidence and the use of mobile phones. Most studies find the relationship between increasing mobile phone use and incidence of brain tumors are inconsistent. However, all of these studies suffer from a variety of problems that are common with ecological studies. In most studies, the surrogate for individual exposure is derived from subscription data and not from actual cellular phone use data. Even in cases where exposure is used (such as high cumulative use), the exposure is simply expressed as a simple percentage of the population. The choice of tumor to examine can have a major impact on the trend as can the statistical model used to examine the data (this is clearly exemplified by the studies using the same UK data and seeing very different results). In many cases, the tumor

incidence rates are increasing, but there was insufficient statistical power to identify if the increase matches the increase in cellular phone usage and these were uniformly interpreted as showing no relationship. Finally, the cancer registries themselves have limitations and flaws that may also lead to ecological fallacies regarding their linkage to cellular phone usage.

#### 4.1.5 Conclusions for Gliomas

The evidence on an association between cellular phone use and the risk of glioma in adults is quite strong. While there is considerable difference from study to study on ever versus never usage of cellular phones, 5 of the 6 meta-analyses in Figure 1 are positive and two are significantly positive. Once you consider latency, the meta-analyses in Figure 2 clearly demonstrate an increasing risk with increasing latency. The exposure response meta-regressions in Table 10 and Table 11 clearly indicate that risk is increasing with cumulative hours of exposure, especially in the highest exposure groups. There is a strong tendency toward gliomas appearing on the same side of the head as the phone is generally used and the temporal lobe is strongly suggested as a target. These findings do not appear to be due to chance. The cohort studies appear to show less of a risk than the case-control studies, but one study is likely to be severely impacted by differential exposure misclassification (Frei et al., 2007) and the other (Benson et al., 2012) is likely to have a milder differential exposure misclassification. The case-control studies are possibly impacted by recall bias although that issue has been examined in a number of different evaluations. Selection bias could have been an issue for the Interphone study, but their alternative analysis using different referent groups reduces that concern. Confounding is not an issue here. In conclusion, an association has been established between the use of cellular telephones and the risk of gliomas and chance, bias and confounding are unlikely to have driven this finding. The ecological studies are of insufficient strength and quality to fully negate the findings from the observational studies.

The data in children is insufficient to draw any conclusions.

## 4.2 Acoustic Neuromas

### 4.2.1 Studies in Adults

#### 4.2.1.1 Case-Control Studies

**Hardell et al. (1999)** [85] did an analysis of acoustic neuromas in their study and saw an OR of 0.78 (0.14-4.20) based on 13 cases. No other information is provided. (Table 12)

**Inskip et al. (2001)** [44] saw no increases for acoustic neuromas in their study described on page 10. (Table 12, Table 13, Table 14, Table 15, Table 16, Table 17)

**Muscat et al. (2002)** [153] conducted a case-control study of acoustic neuromas from two hospitals in New York city as part of their larger study on brain tumors described on page 9. Cases were 18 years of age or older with histologically confirmed acoustic neuromas from 1997 to 1999. There were 90 cases (response rate appears to be 100%) and 86 hospital-based controls matched on age (5-years), sex, race and hospital. Interviewer-based structured questionnaires were used. Regular use was determined by simply asking the patient if they were a regular user. No OR was provided on regular users, but ORs were calculated for years of use, hours/month of use, and total hours. No obvious pattern existed for any of these categories. Ipsilateral use was evaluated using the **Inskip et al. (2001)** [44] method with an OR of 0.9,  $p=0.07$ . The main weakness in this study is the potential for recall bias, small sample size, and the short latency. (Table 13, Table 14, Table 15, Table 17)

**Warren et al. (2003)** [154] conducted a case-control study of intratemporal facial nerve tumors (age not given) in a tertiary care medical center from July 1, 1995 to July 1, 2000 in the United States. As matched controls, and to serve as an alternative case group, they chose 51 acoustic neuroma patients from the same facility. They also had rhinosinusitis controls, dysphonia or gastroesophageal reflux controls and two non-tumor control groups. Matching was based on age ( $\pm 6$  years), sex and race. Cellular telephone usage was assessed via a detailed questionnaire. The study had 51 cases of acoustic neuroma matched with 141 rhinosinusitis, dysphonia or gastroesophageal reflux controls (participation rates were not provided). Ever use of a handheld cellular phone had an OR of 1.2 (0.6-2.2) and use of a handheld cellular phone for more than 1 call per week had an OR of 1.0 (0.4-2.2). They assessed use of tote phones and car phones as well. This is a very small study with limited details. (Table 12)

**Baldi et al. (2011)** [89] saw no increases for acoustic neuromas in their study. (Table 12)

The **Interphone Study Group (2011)** [67] also did a case-control study on acoustic neuromas using the same protocol as their brain cancer study [48] shown on page 11. As for brain tumors, there were a number of publications from individual countries and/or sub-groups of countries for acoustic neuromas [50, 53, 54, 57, 58, 60, 66, 155, 156]. The odds ratio (OR) of acoustic neuroma with ever having been a regular mobile phone user was 0.85 (95% confidence interval 0.69–1.04). The OR for  $\geq 10$  years after first regular mobile phone use was 0.76 (0.52–1.11). There was no trend of increasing ORs with increasing cumulative call time or cumulative number of calls, with the lowest OR (0.48 (0.30–0.78)) observed in the 9<sup>th</sup> decile of cumulative call time. In the 10<sup>th</sup> decile ( $\geq 1640$  h) of cumulative call time, the OR was 1.32 (0.88–1.97); there were, however, implausible values of reported use in those with  $\geq 1640$  h of accumulated mobile phone use. With censoring at 5 years before the reference date the OR for  $\geq 10$  years after first regular mobile phone use was 0.83 (0.58–1.19) and for  $\geq 1640$  h of cumulative call time it was 2.79 (1.51–5.16), but again with no trend in the lower nine deciles and with the lowest OR in the 9<sup>th</sup> decile. In general, ORs were not greater in subjects who reported usual phone use on the same side of the head as their tumor than in those

who reported it on the opposite side, but it was greater in those in the 10<sup>th</sup> decile of cumulative hours of use. [partially copied from abstract] (Table 12, Table 13, Table 14, Table 16, Table 17)

**Han et al. (2012)** [157] conducted a case-control study on patients with acoustic neuromas who underwent surgery from 1997 to 2007 at the University of Pittsburgh medical center. The cases were sent questionnaires in 2009-2010 and then interviewed over the phone. Controls were from the outpatient clinic for degenerative spinal disorders at the same medical center, but during the years of 2009-2010. There were eventually 343 (59% response) cases and 343 (response rate not given) controls matched on sex and age (+/- five years). If age-matching was done based on the time of diagnosis for the case or at the time of the questionnaire administration, there should be no problem, but if age-matching was done as diagnosis for the patient matched to current age of the control, this would be a problem for the analysis of cell phone usage. Their main interest was in the relationship between dental x-rays and AN, but they asked about cell-phone usage as a side issue in order to adjust their main analyses on x-rays for cell phone usage. It is not clear exactly how exposure to cellular phones was assessed. If it was done right, regular usage was assessed at the time of the AN patient's diagnosis and the matching control was assessed the same way. The same would need to be true for the duration of use. Any other way in which exposure was assessed would render the interpretation of this study difficult. The questionnaire was not available to address these questions and the write-up does not explicitly make this clear. Assuming the case matching was done correctly and exposure was done correctly, they saw no increased OR [0.95 (0.58-1.58)] for regular use (defined as 1 call per week for 6 months or more) or for use ≤10 years [0.79 (0.45-1.37)] and saw an increased OR for ≥10 years of use [1.29 (0.69-1.63)]. Regular use of a cellular phone was a significant confounder (p=0.006) in their analysis of X-rays and AN. (Table 12, Table 13)

As for malignant brain tumors, **Hardell and colleagues** have published a number of studies on acoustic neuromas and cell phone usage [82, 158-160]. **Hardell et al. (2013)** [82] used data collected at the same time as their pooled case-control study on malignant brain tumors [88], described on page 16, to do a pooled case-control study on acoustic neuromas and cellular phone usage. ORs tended to increase with years of latency with the highest ORs in the longest latency group (>20 years), ORs tended to increase with cumulative use with the largest OR in the highest exposure quartile (>1486 hours cumulative use), ipsilateral ORs were larger than contralateral ORs and changes in tumor volume seemed to be associated with cumulative use. (Table 12, Table 13, Table 14, Table 16, Table 17)

**Corona et al. (2012)** [161] identified cases of unilateral AN in people ≥18 years of age residing in the municipalities of Salvador and Feira de Santana in Brazil from 2000 to 2010. For each case, they selected 3 controls from the same outpatient clinics as the cases and had visited the doctor "immediately after each case visit". They identified 85 AN patients and 181 controls of which 44 (51.8%) of the cases participated and 104 (57.4%) of the controls participated. There was no description of whether cases and controls were matched on any factor other than clinic. Exposure and demographic information was obtained by interview-administered questionnaire for both cases and controls. For regular use of a mobile phone (defined as one call per week for 6 months), the OR was 1.38 (0.61-3.14). For <6 years of phone use, the OR was 1.14 (0.42-3.08) and for ≥6 years it was 1.81 (0.73-4.47). They also looked at minutes of use per day (≤10, 11-30, >30) and saw increased ORs (1.49 [0.59-3.77], 1.77 [0.62-5.06], 1.15 [0.33-4.08]). Ipsilateral use showed an OR of 1.40 (0.65-3.04) and contralateral use showed an OR of 0.57 (0.23-1.43). (Table 12, Table 13, Table 15, Table 17)

**Pettersson et al. (2014)** [156] identified incident cases of acoustic neuroma (n = 542) between 20 and 69 years of age at diagnosis from September 2002 to August 2007 in Sweden. Controls (n=1095) were randomly selected from the Swedish population register, matched on age, sex and health-care region. Of these, 451 (83%) cases and 710 (65%) controls participated. The controls were assigned a reference date that corresponded to the date of diagnosis of their matched case. Self-reported exposure information was collected through postal questionnaires, sent to cases and their matched controls simultaneously, starting in October 2007. The referent group was regular users defined as having made or received on average at least one call per week over the last 6 months. Analyses were conducted on all cases and controls and then on cases and their matched controls for which the case was histologically confirmed (47% of cases). The OR for regular use is 1.18 (0.88-1.59). For duration of use, they saw an elevated OR for 5-9 years [1.40 (0.98-2.00)], but not for < 5 years [1.04 (0.72-1.52)] or ≥10 years [1.11 (0.76-1.61)]. Cumulative hours of use saw an exposure-response pattern with the highest OR [1.46 (0.98-2.17)] in the highest exposure group. Cumulative calls saw a similar pattern. When ORs are evaluated for any analog phone usage, the ORs generally increased and the pattern for time since first regular use began is decreasing with years. For digital phones, the pattern is the same as for all phones, with slightly larger ORs. The ORs for histologically-confirmed cases only generally has smaller ORs. ORs for ipsilateral use were generally lower than for contralateral use and near or below 1.0. Over half of the cases who were regular users noted they changed their preferred side of mobile use, mostly due to hearing loss. They attempted to evaluate this issue, but their definition of ipsilateral (having held the mobile phone on the tumor side or on both sides during any period before the reference date) would make it virtually impossible to see an increase in ipsilateral use [NOTE: most studies ask which is the usual hand for holding the mobile phone]. Contralateral was also defined using both sides (or opposite side). This problem is best seen when they looked at laterality over time; at the time of filling in the questionnaire, ipsilateral was 0.31 (0.18-0.53) and contralateral was 2.09 (1.45-3.00) whereas at five years before the reference date, ipsilateral was 0.97 (0.66-1.42) and contralateral was 1.33 (0.89-2.27). They evaluated the potential for recall bias for start year and found no systematic errors that were different between cases and controls [162]. (Table 12, Table 13, Table 14, Table 16, Table 17)

#### *4.2.1.2 Case-Case Studies*

**Sato et al. (2011)** [163] conducted a case-case study of mobile phone use and acoustic neuromas in Japan. Inclusion criteria were all verified cases occurring between January, 2000 and December, 2006 in 22 hospitals recruited to be in the study (32.4% of those asked). Phone usage and other information were obtained by written questionnaire sent to the patient. A total of 1589 cases met the inclusion criteria of which 787 (49.5%) eventually were included in the analysis. Reference dates were set at 1 year and 5 years before diagnosis. The case-case analysis is based upon three assumptions: (1) there was no risk from mobile phones to the contralateral side; (2) risk to the ipsilateral side was the same for left- and right-sided users; and (3) for non-users, incidence of left- and right-sided tumors was the same. Hence, contralateral cases served as controls. Weighted average number of calls per day, weighted average duration of one call and weighted average daily call duration at 5 years prior to diagnosis were all significantly increased (0.043, 0.017, and 0.004 respectively). In addition, patients with an age at diagnosis of <40 years (41 patients) had a significantly increased OR (1.72 [1.08-3.10]). Heavy users (>20 minutes per day) had increased ORs regardless of whether that heavy use was for 1 (2.7 [1.2-7.9]) or 5 (3.1 [1.5-7.4]) years or both (5.0 [1.4-24.8]) or only 5 years (1.9 [0.9-5.8]) before diagnosis, but not for only the period 1 year before diagnosis (0.9 [0.6-2.6]). Tumor sizes tended to be smaller with ipsilateral use compared to

contralateral use. The main weaknesses of this study are the potential for recall bias due to the mail-in questionnaire and the low response rate. (Table 17)

#### *4.2.1.3 Cohort Studies*

**Schuz et al. (2011)** [99] used the same cohort as **Frei et al. (2011)** [96] to evaluate the incidence of acoustical neuromas in humans associated with mobile telephone use (description of the cohort on page 19). The cohort was updated to include follow-up to 2006. The results pertain only to people who used phones for greater than 11 years (because of the 1995 cut-off for knowledge of who had a cellular phone subscription) and the referent group is all non-users and people who got phones after 1995. They saw no association (men 0.88, 0.52-1.48, no observed tumors in female users). They also saw no impact of long-term mobile phone use on the size of the tumors. This study has the same limitations of other evaluations with this cohort. There are earlier publications on this cohort [94, 95]. (Table 12)

**Benson et al. (2013)** [102] also studied acoustic neuromas in their cohort study described on page 19. Relative risks (RRs) for phone use were ever/never 1.44 (0.91-2.28), daily use 1.44 (0.91-2.28), <5 years 1.0 (0.54-1.82), 5-9 years 1.80 (1.08-3.03) and 10+ years of use 2.46 (1.07-5.64) (all adjusted for socioeconomic status, region, age (in 3-year groupings), height, BMI, alcohol intake, exercise and hormone therapy). In a letter responding to a letter by **de Vocht (2014)** [105], **Benson et al. (2014)** [106] updated their follow-up to 2011 but did not update cellular phone usage (still relying on the 1999-2005 response) and saw OR for acoustic neuroma for ever/never users of 1.19 (0.81-1.75). Note that with 7 years average follow-up, they saw 96 acoustic neuromas or 13.7/year but adding 2010 and 2011 increased the acoustic neuromas by 15 per year. The same limitations mentioned on page 19 also apply here. (Table 12, Table 13)

Table 12: Results from epidemiology studies for ever versus never or regular versus non-regular use of a cellular telephone and the risk of acoustic neuroma in adults

Author (year)	Study Type	Years, Country	Age (years), sex	Tumor Type	Sample Size for all endpoints (% resp.)	Exposed (%) Cases	OR (95% CI)	Comparison group
Hardell et al. (1999)	CC	1994-1996, Sweden	20-80, Both	Acoustic Neuroma	13 (ND) Cases ND (ND) Controls	ND (ND)	0.78 (0.14-4.20)	>1 year
Inskip et al. (2001)	CC	1994-1998, US	≥18, Both	Acoustic neuroma	782 (92%) Cases 799 (86%) Controls 96 Acoustic Neuromas	40 (41.7%) 30 (31.2%)	0.8 (0.5-1.4) 1.0 (0.5-1.9)	Any use >5 times use
Warren et al. (2003)	Case-Control	1995-2000	ND	Acoustic Neuroma	51 (ND) Cases 141 (ND) Controls	21 (41.2%) 11 (21.6%) 6 (11.8%) 7 (13.7%) 5 (9.8%)	1.2 (0.6-2.2) 1.0 (0.4-2.02) 1.0 (0.4-2.7) 1.2 (0.5-3.8) 2.1 (0.6-7.0)	Ever use >1 call per week "tote" phone Automobile phone Automobile phone >1 call/week
INTERPHONE (2010)	CC	2000-2004, 13 countries	30-59, Both	Acoustic neuroma	1105 (82%) Cases 2145 (53%) Controls	643 (58.2%) 304 (27.5%)	0.85 (0.69-1.04) 0.95 (0.77-1.17)	Avg 1 call per week for 6 mo (lag 1 yr) Avg 1 call per week for 6 mo (lag 5 yr)
Han et al. (2012)	CC	1997-2007, US	Age not given, Both	Acoustic Neuroma	343 (59%) Cases 343 (ND) Controls	203 (59.2%)	0.95 (0.58-1.58)	Avg 1 call per week for 6 mo
Corona et al. (2012)	CC	2006-2010, Brazil	18, Both	Acoustic Neuroma	44 (51.8%) 104 (57.4%)	34 (77.3%)	1.38 (0.61-3.14)	Avg 1 call per week for 6 mo
Pettersson et al. (2014)	Case-Control	Sweden	20-69, Both	Acoustic Neuroma	451 (83%) 710 (65%)	302 (67.0%) 143 (70.8%)	1.18 (0.88-1.59) 0.99 (0.65-1.52)	All, Once per week ≥6 months Histopathologically confirmed, Once per week ≥6 months
Hardell et al. (2013)	CC	1997-2003, 2007-2009, Sweden	20-80, Both	Acoustic neuroma	316 (93%) Cases 3530 (87%) Controls	200 (63.3%)	1.6 (1.2-2.2)	>1 year
Schuz et al. (2011)	Cohort	1998-2006, Denmark	≥30 at time of entry	Acoustic neuroma	2,883,665 404 cases	15 (0.38) Male 0 (0) Female	0.87 (0.52-1.46)	Subscription > 11 years prior Phone use only for before 1995
Benson et al. (2013)	Cohort	1999-2009, UK	Middle-aged women	Acoustic neuroma	791,710 (65%)  2009 – 96 cases	67 (69.8) Ever use 8 (8.3) Daily use Exclude first 3 years 31 (32.3)	1.44 (0.91-2.28) 1.37 (0.61-3.07)  1.96 (0.96-4.02)	Ever used (asked 1999-2005) Every day (asked 1999-2005)  Ever used (asked 1999-2005)
Benson et al. (2014)		1996-2011, (UK)			2011 – 126 cases		1.19 (0.81-1.75)	Ever used (asked 1999-2005)

Table 13: Results from epidemiology studies for time (years) since first use of a cellular telephone and the risk of Acoustic Neuroma in adults

Author (year)	Study Type	Years, Country	Age (years), sex	Tumor Type	Duration	Exposed Cases	OR (95% CI)	P Trend	Comments
Inskip et al. (2001)	CC	1994-1998, US	≥18, Both	Acoustic Neuroma	<0.5 years 0.5-3 years ≥3 years ≥5 years	4 8 10 5	0.3 (0.1-1.3) 1.8 (0.7-4.5) 1.4 (0.6-3.4) 1.9 (0.6-5.9)	ND	Any use ≥4 calls/w
Muscat et al. (2002)	CC	1997-1999, New York City	≥18, Both	Acoustic neuroma	1-2 years 3-6 years	7 11	0.5 (0.2-1.3) 1.7 (0.5-5.1)	0.84	Referent was asked if they were a regular user
INTERPHONE (2010)	CC	2000-2004, 13 countries	30-59, Both	Acoustic neuroma	1-1.9 years 2-4 years 5-9 years ≥10 years Exposure up 5 years 5-9 years ≥10 years	63 276 236 68 236 68	0.73 (0.49-1.09) 0.87 (0.69-1.10) 0.90 (0.69-1.16) 0.76 (0.52-1.11) 0.99 (0.78-1.24) 0.83 (0.58-1.19)	ND	Avg 1 call per week for 6 mo (lag 1 yr), no hands-free  Excludes hands-free usage
Han et al. (2012)	CC	1997-2007, US	Age not given, Both	Acoustic Neuroma	<10 years ≥10 years	111 92	0.79 (0.45-1.37) 1.29 (0.69-2.43)		Avg 1 call per week for 6 mo
Corona et al. (2012)	CC	2006-2010, Brazil	18, Both	Acoustic Neuroma	<6 years ≥6 years	12 23	1.14 (0.42-3.08) 1.81 (0.73-4.47)	ND	Avg 1 call per week for 6 mo
Pettersson et al. (2014)	Case-Control	Sweden	20-69, Both	Acoustic Neuroma	<5 years 5-9 years ≥10 years Histologically confirmed <5 years 5-9 years ≥10 years	81 119 102 47 55 41	1.04 (0.72-1.52) 1.40 (0.98-2.00) 1.11 (0.76-1.61) 0.96 (0.58-1.61) 1.10 (0.65-1.84) 0.93 (0.54-1.60)		Avg 1 call per week for 6 mo (lag 1 yr), weighted hands-free
Hardell et al. (2013)	CC	1997-2003, 2007-2009, Sweden	20-80, Both	Acoustic Neuroma	1-5 years 5-10 years 10-15 years 15-20 years >20 years Per year of latency	65 77 34 12 12	1.3 (0.9-1.8) 2.3 (1.6-3.3) 2.1 (1.3-3.5) 2.1 (1.02-4.2) 4.5 (2.1-9.5) 1.060 (1.031-1.089)	ND	>1 year
Benson et al. (2013)	Cohort	1999-2009, UK	Middle-aged women	Acoustic Neuroma	<5 years 5-9 years ≥10 years Excluding first 3 years <5 years 5-9 years ≥10 years	19 38 8 4 20 6	1.0 (0.54-1.82) 1.80 (1.08-3.03) 2.46 (1.07-5.64) 1.80 (0.55-5.90) 1.89 (0.87-4.08) 3.11 (1.08-8.95)	0.03	Ever used (asked 1999-2005)
Benson et al. (2014)		1999-2011, UK			<5 years 5-9 years ≥10 years	No data	0.94 (0.53-1.66) 1.46 (0.94-2.27) 1.17 (0.60-2.27)	0.30	Ever used (asked 1999-2005)

Table 14: Results from epidemiology studies for duration (cumulative hours) of use of a cellular telephone and the risk of acoustic neuroma in adults

Author (year)	Study Type	Years, Country	Age (years), sex	Tumor Type	Cumulative use	Exposed Cases	OR (95% CI)	P Trend	Comparison group
Inskip et al. (2001)	CC	1994-1998, US	≥18, Both	Acoustic neuroma	<13 hours 13-100 hours >100 hours >500 hours	5 8 9 1	0.7 (0.2-2.3) 1.2 (0.5-3.1) 1.4 (0.6-3.5) 0.4 (0.0-3.3)	ND	Any use 2+ calls/w
Muscat et al. (2002)	CC	1997-1999, New York City	≥18, Both	Acoustic neuroma	1-60 hours >60 hours	9 9	0.9 (0.3-3.1) 0.7 (0.2-2.6)	0.53	Referent was asked if they were a regular user
INTERPHONE (2010)	CC	2000-2004, 13 countries	30-59, Both	Acoustic neuroma	1-year lag <5 hours 5-12.9 hours 13-30.9 hours 31-60.9 hours 61-114.9 hours 115-199.9 hours 200-359.9 hours 360-734.9 hours 735-1639.9 hours ≥1640 hours 5-year lag <5 hours 5-12.9 hours 13-30.9 hours 31-60.9 hours 61-114.9 hours 115-199.9 hours 200-359.9 hours 360-734.9 hours 735-1639.9 hours ≥1640 hours	58 63 80 66 74 68 50 58 49 77 42 30 40 36 21 22 29 26 22 36	0.77 (0.52-1.15) 0.80 (0.54-1.18) 1.04 (0.71-1.52) 0.95 (0.63-1.42) 0.96 (0.66-1.41) 0.96 (0.65-1.42) 0.60 (0.39-0.91) 0.72 (0.48-1.09) 0.48 (0.30-0.78) 1.32 (0.88-1.97) 1.07 (0.69-1.68) 1.06 (0.60-1.87) 1.32 (0.80-2.19) 0.86 (0.52-1.41) 0.63 (0.35-1.13) 0.71 (0.39-1.29) 0.83 (0.48-1.46) 0.74 (0.42-1.28) 0.60 (0.34-1.06) 2.79 (1.51-5.16)		Avg 1 call per week for 6, no hands-free
Pettersson et al. (2014)	Case-Control	Sweden	20-69, Both	Acoustic Neuroma	<38 38-189 190-679 ≥680 Histologically confirmed <38 38-189 190-679 ≥680	70 73 66 89 30 39 34 37	1.09 (0.73-1.62) 1.12 (0.74-1.69) 1.13 (0.75-1.70) 1.46 (0.98-2.17) 0.97 (0.55-1.71) 0.91 (0.51-1.60) 1.03 (0.57-1.87) 1.14 (0.63-2.07)		Avg 1 call per week for 6 mo (lag 1 yr), weighted hands-free
Hardell et al. (2013)	CC	1997-2003, 2007-2009, Sweden	20-80, Both	Acoustic Neuroma	Per 100 cumulative hours of use Quartiles 1-122 hours 123-511 hours 512-1,486 hours >1,486 hours	NA 91 37 42 30	1.009 (1.001-1.017) 1.6 (1.1-2.2) 1.5 (0.9-2.3) 2.4 (1.5-3.8) 2.6 (1.5-4.4)	0.052	>1 year

Table 15: Results from epidemiology studies for average daily or monthly use of a cellular telephone and the risk of acoustic neuroma in adults

Author (year)	Study Type	Years, Country	Age (years), sex	Tumor Type	Measure	Exposed Cases	OR (95% CI)	P Trend	Comparison group
Inskip et al. (2001)	CC	1994-1998, US	≥18, Both	Acoustic neuroma	Average daily <3 minutes 3 to 15 minutes ≥15 minutes ≥60 minutes	7 10 5 1	1.0 (0.4-2.9) 1.4 (0.6-3.2) 0.9 (0.3-2.8) 0.3 (0.0-2.7)	ND	Any use 2+ calls/w
Muscat et al. (2002)	CC	1997-1999, New York City	≥18, Both	Acoustic neuroma	Average monthly 1-2.5 hours >2.5 hours	11 7	1.1 (0.4-2.9) 0.6 (0.2-1.7)	0.40	Referent was asked if they were a regular user
Corona et al. (2012)	CC	2006-2010, Brazil	18, Both	Acoustic Neuroma	Minutes/day ≤10 11-30 >30	19 11 5	1.49 (0.59-3.77) 1.77 (0.62-5.06) 1.15 (0.33-4.08)	ND	Avg 1 call per week for 6 months

Table 16: Results from epidemiology studies for other use measures of a cellular telephone and the risk of acoustic neuroma in adults

Author (year)	Study Type	Years, Country	Age (years), sex	Tumor Type	Measure	Exposed Cases	OR (95% CI)	P Trend	Comments
Inskip et al. (2001)	CC	1994-1998, US	≥18, Both	Acoustic neuroma	Year use began 1995-1998 1993-1994 ≤1992 <1990	7 9 6 2	0.7 (0.3-2.0) 1.5 (0.6-3.6) 1.2 (0.4-3.4) 1.3 (0.2-6.6)	ND	Any use 2+ calls/w
INTERPHONE (2010)	CC	2000-2004, 13 countries	30-59, Both	Acoustic neuroma	Cumulative use by recency of starting use <i>1-4 years before reference date</i> <5 hours 5-114.9 hours 115-359.9 hours 360-1639.9 hours ≥1640 hours <i>5-9 years before reference date</i> <5 hours 5-114.9 hours 115-359.9 hours 360-1639.9 hours ≥1640 hours <i>≥10 years before reference date</i> <5 hours 5-114.9 hours 115-359.9 hours 360-1639.9 hours ≥1640 hours	54 198 57 26 4 4 77 55 64 36 0 8 6 17 37	0.81 (0.53-1.24) 0.92 (0.71-1.20) 0.74 (0.49-1.13) 0.55 (0.29-1.03) 0.63 (0.14-2.80) 0.84 (0.21-3.40) 0.97 (0.67-1.41) 0.95 (0.62-1.45) 0.74 (0.49-1.12) 1.05 (0.62-1.78) - 0.81 (0.30-2.14) 0.28 (0.09-0.86) 0.39 (0.20-0.74) 1.93 (1.10-3.38)	ND	Avg 1 call per week for 6 mo (lag 1 yr), no hands-free
Pettersson et al. (2014)	Case-Control	Sweden	20-69, Both	Acoustic Neuroma	Cumulative # calls <1,100 1,100-4,400 4,400-13,850 ≥13,850	72 71 79 75	1.21 (0.82-1.78) 1.07 (0.71-1.61) 1.22 (0.83-1.80) 1.20 (0.79-1.82)		Avg 1 call per week for 6 mo (lag 1 yr), weighted hands-free

Table 17: Results from epidemiology studies for laterality of cellular telephone use and the risk of acoustic neuroma in adults

Author (year)	Study Type	Years, Country	Age (years), sex	Tumor Type	Location or laterality	Ipsilateral OR (95%CI)	Contralateral OR (95% CI)	Inskip P.value	Comparison group
Inskip et al. (2001)	CC	1994-1998, US	≥18, Both	Acoustic neuroma	Inskip method	0.9		0.63	2 or more calls/week + 6 months latency
Muscat et al. (2002)	CC	1997-1999, New York City	≥18, Both	Acoustic neuroma	Inskip Method	0.9		0.07	Asked if they were a regular user
INTERPHONE (2010)	CC	2000-2004, 13 countries	30-59, Both	Acoustic neuroma	1-year lag Regular use ≥10 years since start ≥1640 hours cumulative ≥270 calls (hundreds)	0.77 (0.59-1.02) 1.18 (0.69-2.04) 2.33 (1.23-4.40) 1.67 (0.90-3.09)	0.92 (0.70-1.22) 0.69 (0.33-1.42) 0.72 (0.34-1.53) 0.52 (0.21-1.26)		Avg 1 call per week for 6 mo (lag 1 yr)
					5-year lag Regular use ≥10 years since start ≥1640 hours cumulative ≥270 calls (hundreds)	0.98 (0.73-1.30) 1.05 (0.65-1.68) 3.53 (1.59-7.82) 2.00 (0.89-4.51)	0.93 (0.68-1.27) 0.58 (0.30-1.11) 1.69 (0.43-6.69) 1.40 (0.43-4.53)		
Corona et al. (2012)	CC	2006-2010, Brazil	18, Both	Acoustic Neuroma	Regular Users	1.40 (0.65-3.04)	0.57 (0.23-1.43)		Avg 1 call per week for 6 mo
Pettersson et al. (2014)	Case-Control	Sweden	20-69, Both	Acoustic Neuroma	Regular users Duration of use (years) <5 5-9 ≥10 Cumulative hours of use <38 38-189 190-679 ≥680	0.98 (0.68-1.43) 1.05 (0.62-1.78) 0.95 (0.57-1.58) 1.01 (0.61-1.68) 0.78 (0.45-1.38) 1.18 (0.63-2.20) 0.98 (0.52-1.84) 1.20 (0.69-2.08)	1.33 (0.89-1.99) 1.41 (0.80-2.48) 1.51 (0.92-2.49) 1.09 (0.63-1.88) 1.69 (0.94-3.05) 1.05 (0.56-1.95) 1.31 (0.74-2.32) 1.26 (0.70-2.25)		Avg 1 call per week for 6 mo (lag 1 yr), weighted hands-free
Sato et al. (2011)	Case-Case	2000-2006, Japan	Any age, Both	Acoustic neuroma	l/l & r/r (97 cases) l/l & r/r (86 cases) Duration ≤5 years 5-10 years >10 years ≤5 years 5-10 years >10 years Weighted average daily call ≤3 minutes 1-3 minutes 10-20 minutes >20 minutes ≤3 minutes 1-3 minutes 10-20 minutes >20 minutes Weighted avg duration 1 call ≤1 minute 1-3 minutes 3-5 minutes >5 minutes ≤1 minute 1-3 minutes 3-5 minutes >5 minutes	1.08 (0.93-1.28) 1.14 (0.96-1.40) 1.06 (0.88-1.31) 1.05 (0.82-1.45) 1.62 (0.79-4.77) 1.11 (0.92-1.38) 1.56 (0.90-3.34) 1.00 (0.59-3.23) 1.18 (0.93-1.57) 0.89 (0.72-1.21) 0.82 (0.65-1.19) 2.74 (1.18-7.85) 1.11 (0.85-1.55) 0.89 (0.71-1.21) 0.84 (0.62-1.44) 3.08 (1.47-7.41) 1.13 (0.89-1.51) 0.91 (0.75-1.21) 1.11 (0.76-1.95) 1.51 (0.95-2.75) 1.02 (0.79-1.43) 1.04 (0.81-1.44) 1.37 (0.83-2.74) 1.68 (1.00-3.28)		0.240 0.300 0.230 0.004 0.230 0.017	Avg 1 call per week for 6 mo (lag 1 yr) Avg 1 call per week for 6 mo (lag 5 yr) Avg 1 call per week for 6 mo (lag 1 yr) Avg 1 call per week for 6 mo (lag 5 yr) Avg 1 call per week for 6 mo (lag 1 yr) Avg 1 call per week for 6 mo (lag 5 yr) Avg 1 call per week for 6 mo (lag 1 yr)
Hardell et al. (2013)	CC	1997-2003, 2007-2009, Sweden	20-80, Both	Acoustic Neuroma	Regular users	1.8 (1.3-2.6)	1.5 (0.98-2.2)		>1 year usage



#### 4.2.2 Studies in Children

I could not identify any studies on acoustic neuromas in children and exposure to RF or cellular telephones.

#### 4.2.3 Discussion

As for gliomas, I will focus on three areas of interest from the epidemiology studies of acoustic neuromas (AN); consistency of the association, the existence of an exposure-response relationship, and the strength of the association.

##### *4.2.3.1 Consistency of the Association*

The studies to be considered are listed in Table 12 and **Muscat et al. (2002)** in Table 13. All of these studies did a reasonable job of addressing confounders in their analyses and so this problem will not be discussed further. First, we should consider timing of the study. As mentioned earlier, for studies in the 1990s, we are looking at a rare exposure and trying to associate it with a rare disease (AN) and probably with very little time from the beginning of exposure to disease onset. Thus, it is unlikely that **Hardell et al. (1999)** [85], **Inskip et al. (2001)** [44], **Muscat et al. (2002)** [153], **Warren et al. (2003)** [154], and **Baldi et al. (2011)** [89] would show much of an association. And that is basically the case, with these studies producing ORs of approximately 1.0. The later studies are more likely to show an effect if one exists than these early studies and these should be given greater weight.

The size of a study will also matter since studies with greater numbers of cases and controls (especially exposed cases) will generally have smaller confidence bounds and have a greater chance of seeing an effect if one exists. Thus, the studies by **Hardell et al. (1999)** [85], **Inskip et al. (2001)** [44], **Muscat et al. (2002)** [153], **Warren et al. (2003)** [154], **Baldi et al. (2011)** [89], **Corona et al. (2012)** [161], **Benson et al. (2013)** [102] and **Schuz et al. (2011)** [94] will carry less weight in an overall evaluation.

There are also studies where the referent group was “never used a mobile phone” versus studies where the referent group was “not a regular user of mobile phones” defined by different measures. Less weight should be given to studies with comparisons to “never used” simply because the “ever used” group could include people who used a phone only a few times.

Given these caveats, there are five case-control studies that should carry the greatest weight: **Interphone (2010)** [67], **Hardell et al. (2013)** [160], **Han et al. (2012)** [157], **Corona et al. (2012)** [161], and **Pettersson et al. (2014)** [162]. Three of these 4 studies have ORs greater than 1.0 for regular usage of a cellular phone with 1 (**Hardell et al. (2013)** [160]) being significantly >1 [1.6 (1.2-2.2)].

The largest study, **Interphone (2010)** [67] has an OR for regular use of 0.85 (0.69-1.04). The difference in the response rate for cases (82%) versus controls (53%) could lead to problems with selection bias as was suggested for the brain tumor data from the Interphone study [74]. This study demonstrated no increases in OR with duration of use, even with a 5-year latency. (Table 12, Table 13)

The next largest study, and **Pettersson et al. (2014)** [162], had approximately half the number of exposed cases as **Interphone (2010)** [67] and showed an OR for regular use of

1.18 (0.88-1.59). They saw an increased OR for 5-9 years duration of use [1.39 (0.97-1.97)] which dropped for  $\geq 10$  years durations [1.09 (0.75-1.59)]. They had a non-responder questionnaire which was answered by 93 controls and 7 cases. Of the 93 control non-responders, 62 (67%) were regular mobile phone users compared to 442 (69%) out of 643 responding controls. There were only 7 non-responder cases who replied to the questionnaire and 4 were regular phone users. Thus, even though there are a larger number of non-responders in controls, there is no obvious suggestion of selection bias. (Table 12, Table 13)

**Hardell et al. (2013)** [160] was the next largest study with roughly 1/3 of the number of exposed cases as **Interphone (2010)** [67]. They saw an OR for regular use of 1.6 (1.2-2.2) and an increasing risk with increasing duration of use. In addition, all of the 5-year groupings of duration of use were greater than 1 and all usage longer than 5-years was significantly greater than 1 (Table 13). Only living cases were included. Their response rate was high enough that participation bias is unlikely to have lowered the OR values. Recall bias could have increased the ORs. In one of the original case-control studies [117] used in their pooled analysis, they evaluated this issue and saw little indication of recall bias with regard to malignant brain tumors (no information on AN). (Table 12, Table 13)

**Han et al. (2012)** [157] also was about 1/3 of the number of exposed cases as **Interphone (2010)** [67]. They saw an OR for regular use of 0.95 (0.58-1.58) and an increasing risk with increasing duration. It is impossible to judge the potential for selection bias since they gave no indication of the response rates for controls. In addition, it is also impossible to judge the quality of the exposure metrics since there was insufficient detail to understand how they related controls to cases in obtaining this information. (Table 12, Table 13)

**Corona et al. (2012)** [161] had 34 exposed cases or about 20x smaller than **Interphone (2010)** [67]. They saw increased ORs (non-significant) for all categories of usage. The response rates for cases and controls were moderate but not remarkably different suggesting no problem with selection bias although there was no follow-up with non-respondents. It is not possible to judge recall bias in this small study. (Table 12, Table 13)

**Sato et al. (2014)** [163] is the next largest study; but being a case-case study, it is more relevant to the issue of laterality and will be discussed later.

**Schuz et al. (2011)** [99], with only 15 exposed cases, is a cohort study with limitations due to potential differential exposure misclassification (discussed earlier). They saw an OR for subscriptions from 11 years prior to reference date of 0.86 (0.52-1.46). (Table 12)

**Benson et al. (2013)** [102], with only 8 cases that are daily users, saw an OR of 1.37 (0.61-3.07). They had 67 ever users in the cases and these had an OR of 1.44 (0.91-2.28). Using never use as the reference category, they looked at duration of use and saw clearly increasing ORs with increasing duration. This study may also have problems with exposure misclassification (discussed earlier). (Table 12, Table 13)

**Roosli et al. (2019)** [118] also did a meta-analysis of AN and cellular phones. They give mRRs for the analyses of studies showing ORs for  $\geq 10$  years exposure. For the case-control studies, they get an mRR of 1.29 (0.74-2.23). For the Cohort studies, they show an mRR of 0.98 (0.65, 1.48) and for all studies combined they get 1.19 (0.80-1.79). Entering their numbers into Stata (v 16.2 for MAC), I can reproduce their findings. They also did a meta-analysis of ever versus never use for all 9 case-control studies (1.05 [0.84-1.32]) and the

cohort studies (0.93 [0.57-1.50]) with a combined mRR of 1.02 (0.84-1.24). They show a number for regular use from **Muscat et al. (2002)** [153] which is not in the paper and appears to be the unadjusted crude OR. They give no reason for using **Shuz et al. (2006)** [94] instead of **Schuz et al. (2011)** [99] for this analysis although they used **Frei et al. (2011)** [96] for their analysis of gliomas. I am also unable to match the number they use for **Benson et al. (2013)** [102] which they list as 1.19 (0.81-1.75) but the paper lists as 1.37 (0.61-3.07). They also conducted a cumulative meta-analysis of the studies with  $\geq 10$  years of use. They also did several other analyses of ever versus never use with no appreciable changes in the results. One problem with these meta-analyses is that they give very little weight to the largest studies. They did not consider laterality or tumor location in the brain.

The remaining meta-analyses are older and use fewer and fewer of the individual studies.

To provide a better evaluation of the results, **Figure 3** is a forest plot of all of the ORs from individual publications that evaluated regular use versus minimal or never use or ever use versus never use (if both were given in a study, regular use is shown). The column labeled "Study" provides the reference to the publication and the years in which cases and controls were collected for case control studies and the years when phone use information was collected for cohort studies and the year in which follow-up ended. Some studies are pooled evaluations of multiple other studies, so the other studies are indented. The column labeled "RR" is the risk ratio (OR, RR or mRR) from the study, "Lower" and "Upper" are the lower and upper bound on a 95% confidence interval around the RR. The graphic on the right simply plots the RR as a square or diamond with the "whiskers" (blue line running through the box) showing the width of the 95% confidence interval. The vertical line passing through 1 represents no effect. If the box and both whiskers are to the right of this line (greater than 1) and not touching it, this finding is statistically significant with a positive effect; if they fall completely to the left of the vertical line (below 1), then the risk is significantly reduced. The blue boxes that are filled in are major studies, the blue boxes that are white in the middle are the sub-studies and the red diamonds are all meta-analyses.

The graphic in **Figure 3** is very useful for examining these types of data in a single view. Looking just at the filled in blue blocks (Studies A,B,C,D,E,F,G,H,I,J,K), 5 studies have their ORs below 1, two are equal to 1 and four are above 1. One study (I) shows a significant increase in risk. The first meta-analysis (Meta Analysis A,B,C,D,E,F,G,H,I,J,K) combines the information from all of the studies to produce an mRR of 1.06 (0.88-1.29) suggesting that all of the positives and negatives balance out to a small, non-significant increased risk. However, as mentioned earlier, the newer, larger studies represent longer exposures, so I have also done meta-analyses on the five case-control studies that collected cases after 2002 (E,F,G,H,I) and the two cohort studies (J,K). Combining the five case-control studies (Meta Analysis E,F,G,H,I) results in a mRR of 1.13 (0.87-1.48), a slight increase in risk from the use of a mobile phone, but heterogenous across studies. The combined cohort studies yield a mRR of 0.99 (0.64-1.53) suggesting no risk, and no heterogeneity ( $p=0.35$ ). Combining the 5 case-control studies and the 2 cohort studies (Meta Analysis E,F,G,H,I,J,K) yields an mRR of 1.11 (0.88-1.39) again suggesting marginal risk but with significant heterogeneity ( $p=0.04$ ).

**Figure 4** is a forest plot of all of the ORs from individual publications that reported on duration of use  $\geq 5$  years or more. There are 8 studies; 5 of these studies show groupings of 1-4 years, 5-9 years and  $\geq 10$  years, one study with groupings of  $<6$  years, and  $\geq 6$  years, one study with  $\geq 5$  years and one study with  $<10$  years and  $>10$  years. For the study by **Hardell et**

al. (2013) [160], groupings of 10-14, 15-19 and  $\geq 20$  years were combined by meta-analysis to get a single mRR for  $\geq 10$  years. There are 2 groups of meta-analyses each with three separate meta-analyses for 1-4 years, 5-9 years and  $\geq 10$  years (combined with only  $\geq 10$  years for Han et al. (2012) [157] and  $< 6$  years for Corona et al. (2012) [161]). The first group of 3 meta-analyses combines the case-control studies and the second group of 3 meta-analyses adds in the cohort studies. In order to accommodate the study by Inskip et al. (2001) [44] with only a  $\geq 5$  year grouping and the study by Corona et al. (2012) [161] with  $\geq 6$  years, all studies with 5-9 and  $\geq 10$  years were combined in the last 2 meta-analyses to yield mRRs for  $\geq 5-6$  years for the case-control studies and all of the studies. The mRRs for  $< 5$  years are all near 1. The mRRs for 5-10 years are all elevated and close to statistical significance. The mRRs for  $\geq 10$  years are elevated, but less than for 5-10 years. Finally, both of the mRRs for  $\geq 5$  years are significantly elevated.

The studies in adults of an association between cellular phone use and acoustic neuroma are consistent enough to conclude an association exists.

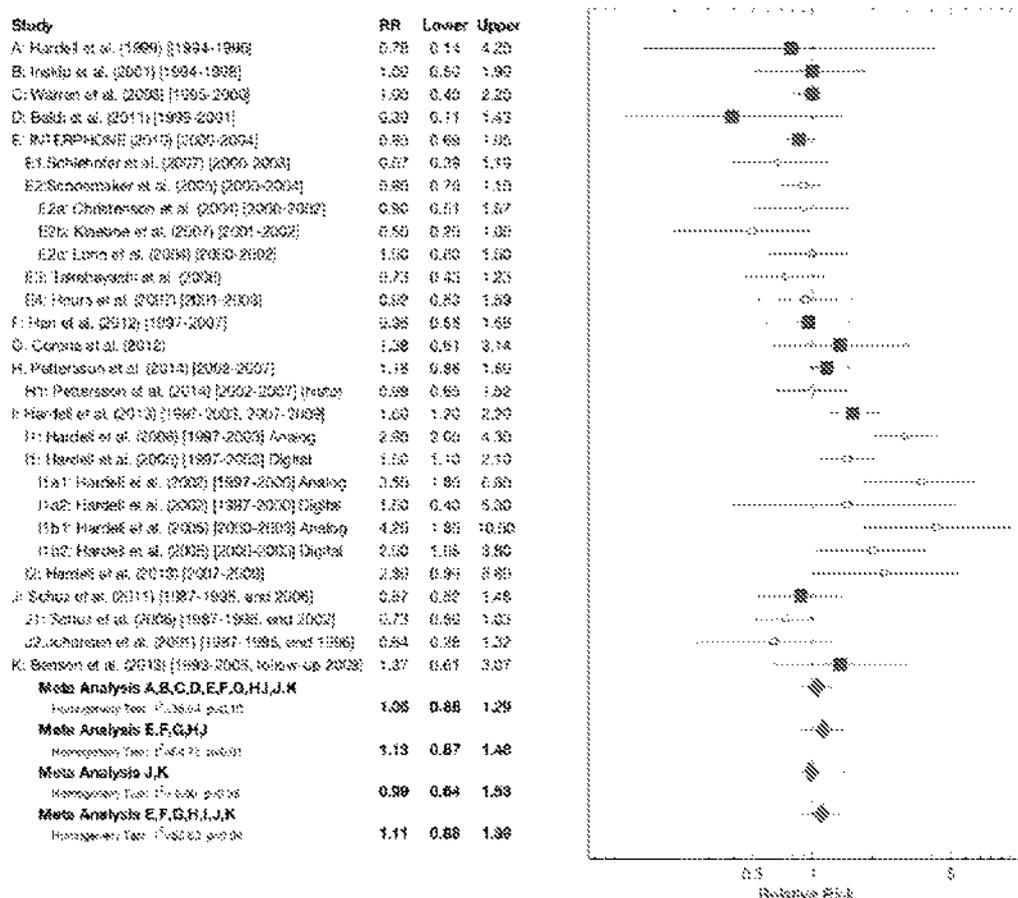


Figure 3: Forest plot and meta-analyses of regular use or ever use of cellular telephones and the risk of acoustic neuroma [studies with a solid blue square either single studies that stand alone or pooled studies that encompass numerous single studies; open squares are individual studies or smaller pooled studies; red diamonds are meta-analyses]<sup>a</sup>

<sup>a</sup> - The column labeled "Study" provides the reference to the publication and the years in which cases and controls were collected for case control studies and the years when phone use information were collected for cohort studies and the year in which follow-up ended. Some studies are pooled evaluations of multiple other studies, so the other studies are indented. The column labeled "RR" is the risk ratio (OR, RR or mRR) from the study, "Lower" and "Upper" are the lower and upper bound on a 95% confidence interval around the RR. The graphic on the right simply plots the RR as a square or diamond with the "whiskers" (blue line running through the box) showing the width of the 95% confidence interval. The vertical line passing through 1 represents no effect. If the box and both whiskers are to the right of this line (greater than 1) and not touching it, this finding is statistically significant with a positive effect; if they fall completely to the left of the vertical line (below 1), then the risk is significantly reduced. The blue boxes that are filled in are major studies, the blue boxes that are white in the middle are the sub-studies and the red diamonds are all meta-analyses. "Homogeneity Test" provides the  $I^2$  statistic and the p-value for the Q-test.

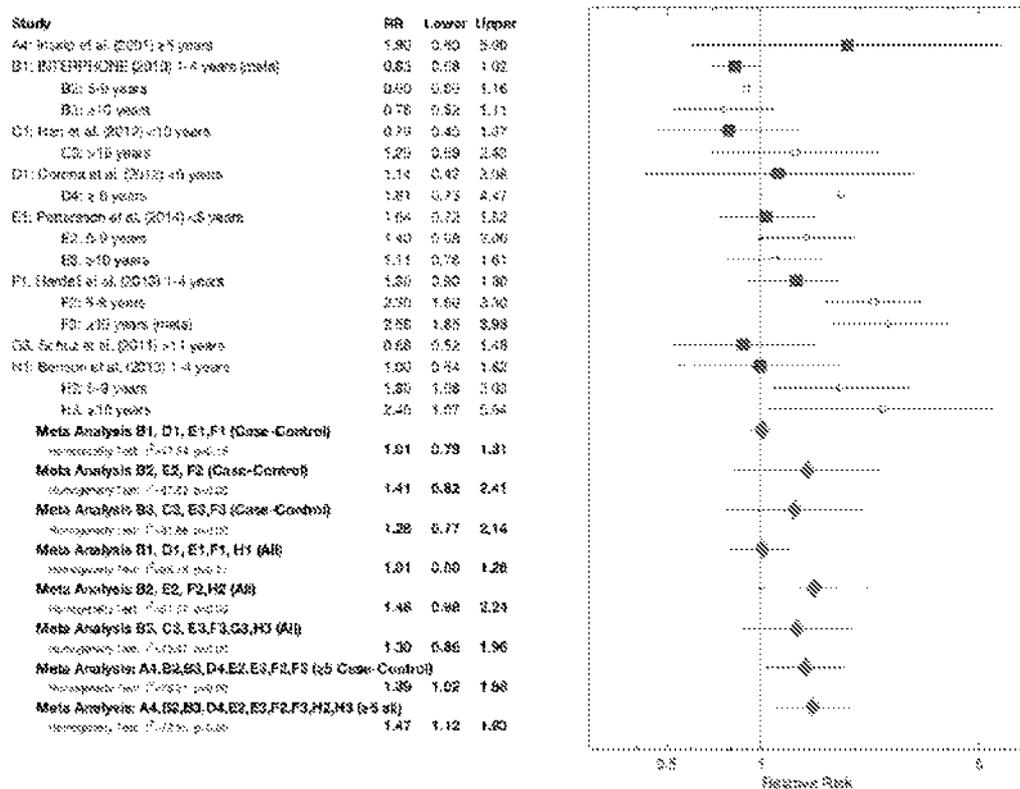


Figure 4: Forest plot and meta-analyses of duration of use of cellular telephones and the risk of acoustic neuroma [studies with a solid blue square are stand alone; red diamonds are meta-analyses, the columns and the figure are as in Figure 1]

#### 4.2.3.2 Exposure-Response

As for gliomas, the best measure for exposure-response relationships is the cumulative hours of use of a cellular telephone since it includes both the frequency of use and the duration of use. While duration of use is also a form of exposure-response, it is more likely that, similar to ionizing radiation, RF is likely to have an association between total accumulated exposure and the risk of AN if a relationship exists. Table 14 provides the results for all of the epidemiology studies with estimates of the cumulative use of cellular phones.

**Inskip et al. (2001)** [44] shows consistent exposure-response and has two of the three ORs above 1. **Muscat et al. (2002)** [153] shows no increased risk. **Interphone (2010)** [67] basically shows flat exposure-response for the entire study until the largest exposure category, that is elevated in risk with an OR of 1.32 (0.88-1.97). The same pattern holds with a 5-years lag although the highest exposure group is now statistically significant with an OR of 2.79 (1.51-5.16). **Pettersson et al. (2014)** [162] saw a clearly increasing exposure-response pattern with ORs above 1 in all exposure categories and becoming almost significant in the highest exposure category [1.46 (0.98-2.17)]. **Hardell et al. (2013)** [160] saw a pattern of increasing risk with increasing exposure with 3 of their 4 categories statistically significant. They also did a regression resulting an OR of 1.009 (1.001-1.017) per hundred cumulative hours.

It is not possible from the published results to find categories of exposure that match across the various studies in order to do a simple meta-analysis by category. However, it is possible to do a meta-regression where the exposure categories are turned into a single exposure and the meta-regression tests to see if the slope of the data from the various studies is increasing with exposure. As for glioma (Section 1.3.2, page 41), I set the exposure for each category equal to the center of the interval defined for the category and or the last category, which is generally expressed as  $\geq$  some number of hours, I used the difference between the middle of the second largest category and the lower bound of that category and added it to the upper end of the second highest category to get the exposure for the highest category. The exposures for all of the categories of the studies entering into the meta-regression are shown in Table 18. As a check, a meta-regression was performed of just the **Hardell et al. (2013)** [160] study; the mRR is 1.015 (1.000-1.030) per 100 hours with  $p=0.05$  compared to 1.009 (1.001-1.016) per 100 hours seen by **Hardell et al. (2013)** [160] using the original data.

Table 18 provides the results of the meta-regression for the 5 case-control studies with duration of exposure where all of the ORs are a comparison against non-regular users. There is a significant association between exposure and risk with a mRR of 1.007 (1.001-1.013,  $p=0.017$ ). This is almost identical to what was seen by **Hardell et al. (2015)** [1.009 (1.001-1.016)]. The test of heterogeneity is significant ( $pQ<0.001$ ) and an  $I^2$  of 57.31. Removing **Interphone (2010)** [67] doubles the mRR to 1.014 (1.066-1.024) and reduces heterogeneity. Removing **Pettersson et al. (2014)** [162] results in no change in the mRR and slightly wider confidence intervals that barely include 1. Removing **Hardell et al. (2013)** [160] cuts the mRR in half and leads to a non-significant risk (1.003 [0.998-1.009;  $p=0.250$ ]) and reduces heterogeneity. The alternative high dose yielded the same pattern but higher mRRs per 100 hours, larger confidence bounds, less statistical significance and less heterogeneity (not shown). (Table 18)

There were other measures of exposure used in the various studies that are worth mentioning. **Inskip et al. (2001)** [44] used average minutes/day and saw no exposure-response relationship (Table 15). **Corona et al. (2012)** [161] also used average minutes/day and saw an increasing exposure response in the first 2 groupings and a lower OR in the highest grouping, all increased but with lower confidence bounds below 1 (Table 15). **Muscat et al. (2002)** [153] used hours/month and saw no pattern (Table 15). **Inskip et al. (2001)** [44] also considered the year that cellular telephone use began and again saw no exposure-response (Table 16). **Interphone (2010)** [67] considered cumulative use by years of duration of use (1-4 years, 5-9 years and  $\geq 10$  years). In 1-4 years and 5-9 years duration categories, they saw flat exposure-response. The highest cumulative use,  $\geq 1640$  hours, in the highest duration of use category,  $\geq 10$  years, was significantly increase (1.93 [1.10-3.38]) (Table 16). **Pettersson et al. (2014)** [162] considered cumulative number of calls and saw a flat exposure-response with all ORs above 1.0 (Table 16).

Table 18: Meta-Regression Exposure Values for Table 19

Author (year)	Exposures (times 100 hrs)
Inskip et al. (2001)	0.065, 0.57, 1.435
Muscat et al. (2002)	0.30, 3 (0.90 <sup>a</sup> )
Interphone (2010)	0.025, 0.09, 0.22, 0.46, 0.88, 1.575, 2.80, 5.475, 11.875, 82 (20.925 <sup>a</sup> )
Pettersson et al. (2014)	0.19, 2.08, 4.345, 34 (9.245 <sup>a</sup> )
Hardell et al. (2013)	0.615, 3.17, 9.99, 74.3 (19.73 <sup>a</sup> )

<sup>a</sup> alternative exposure for highest exposure group

Table 19: Meta-Regression Analysis with Sensitivity Analysis of ORs for Five Case-Control Studies using Cumulative Hours of Use as the Exposure Metric and the Original Referent Groups

Meta Regression Studies <sup>a</sup>	Per 100 hours Use	P> Z	95% Confidence Interval		I <sup>2</sup>	pQ
All	1.007	0.017	1.001	1.013	57.31	<0.001
drop Inskip et al. (2001)	1.007	0.021	1.001	1.013	62.4	<0.001
drop Muscat et al. (2002)	1.007	0.019	1.001	1.013	60.91	<0.001
drop Interphone (2010)	1.014	0.001	1.006	1.022	42.36	0.053
drop Petterson et al. (2014)	1.007	0.053	1.000	1.014	64.21	<0.001
drop Hardell et al. (2013)	1.003	0.25	0.998	1.009	29.45	0.111

#### 4.2.3.3 Strength of the Association

The strength of the association is tied to the magnitude of the response and the statistical significance of that response. For all of these studies, the actual magnitude of the RRs seen in the studies are small, in many cases falling below 1. It is clear from Figure 4, that the longer the duration, the larger the mRR and the more statistical significance to the risk.

Laterality matters for addressing the strength of the association. For regular users versus non-regular users, **Interphone (2010)** [67] and **Pettersson et al. (2014)** [162] saw ipsilateral ORs smaller than the contralateral ORs [Note that **Pettersson et al. (2014)** [162] define ipsilateral differently, including people who used both hands in the ipsilateral category]. In contrast, **Corona et al. (2012)** [161] and **Hardell et al. (2013)** [160] saw ipsilateral ORs greater than the contralateral ORs. Laterality seems to become more pronounced with a longer duration of exposure or greater cumulative hours of use in **Interphone (2010)** [67] but not in **Pettersson et al. (2014)** [162].

In the case-case study by **Sato et al. (2014)** [163], they calculated ORs for the grouping left-handed users with left side ANs (l/l) and right-handed users with right-side ANs (r/r) against all miss-matched tumors (l/r and r/l). For a 1-year lag they saw an OR of 1.08 (0.93-1.28) and for a 5-year lag they saw an OR of 1.14 (0.96-1.40). When they examined this for duration of use, they saw generally increasing ORs that were >1, but not statistically significant. For weighted average minutes per day of use, they saw significant ORs for 1-year lag (2.74 [1.18-7.85]) and 5-year lag (3.08 [1.47-7.41]) and significantly increasing ORs for the 5-year lag group ( $p=0.004$ ). For the average duration of a call, they saw the same basic pattern.

#### 4.2.4 Ecological Epidemiology Studies of Acoustic Neuroma

**Benson et al. (2013)** [102] examined temporal trends in acoustic neuroma incidence rates in England using data from the UK Office of National Statistics. They restricted their analysis to the years 1998-2008. They provided no analysis of these data, only a plot of incidence over time.

Several studies are also mentioned in Section 1.4.

#### 4.2.5 Conclusions for Acoustic Neuromas

The evidence on an association between cellular phone use and the risk of acoustic neuromas in adults is strong. While there is considerable difference from study to study on ever versus never usage of cellular phones, 3 of the 4 meta-analyses in Figure 3 are above 1 although none-significantly. The meta-analyses in Figure 4 demonstrate an increased risk in the highest 2 latency groups for the case-control studies that gets slightly higher when the cohort studies are added. For latency  $\geq 5$  years, the mRRs are significantly elevated for the case-control studies and the combined case-control and cohort studies. The exposure response meta-regressions in Table 19 indicates that risk is increasing with cumulative hours of exposure, especially in the highest exposure groups. This finding, however, is sensitive to the inclusion of the **Hardell et al. (2013)** [160] study. There is a strong tendency toward ANs appearing on the same side of the head as the phone is generally used, especially as the exposure increases. These findings do not appear to be due to chance. The cohort studies appear to show less of a risk than the case-control studies, but one study is likely to be severely impacted by differential exposure misclassification (**Schuz et al. (2011)** [99]) and the other (**Benson et al. (2013)** [102]) is likely to have a milder differential exposure misclassification. Both studies have very few cases. The case-control studies are possibly impacted by recall bias and this cannot be ruled out for the ANs. Selection bias could have been an issue for **Interphone (2010)** [67], and, unlike their analysis of the glioma data, they have not looked at an alternate referent population for their analyses of AN. Confounding is not an issue here. In conclusion, an association has been established between the use of cellular telephones and the risk of ANs and chance and confounding are unlikely to have driven this finding. Potential recall bias and selection bias may still be an issue with some of these findings.

## Laboratory Cancer Studies

**There is sufficient evidence from laboratory studies to conclude that RF can cause tumors in experimental animals with strong findings for gliomas, heart Schwannomas and adrenal pheochromocytomas in male rats and harderian gland tumors in male mice and uterine polyps in female mice.**

### 5.1 Chronic Carcinogenicity Studies

#### 5.1.1 Mice

**Tillmann et al. (2007)** [164] Exposed groups of 50 male and female B6C3F<sub>1</sub> mice to four exposure levels (whole body averaged specific absorption rates (SAR) of 0.0, 0.4, 1.3 and 4.0 mW/g) of two different radiofrequency radiation (RF) exposures (902 MHz GSM and 1747

MHz DCS modulated frequencies) for 2 hours per day, 5 days per week for 2 years using head-only exposure in a Ferris wheel/tube-restrained exposure system. The two hours of exposure was done in three phases imitating exposures classified as “basic”, “talk” and “environment”. All test animals were given a full necropsy and both gross and microscopic lesions identified and characterized. They reported no increases in tumor incidences for any lesion. They did report a significant exposure-related decrease in hepatocellular adenomas in males in the highest exposure group for both GSM ( $p=0.048$ ) and DCS ( $p=0.015$ ) exposures. Tumor count data was provided for Pituitary gland, Harderian gland, lungs, liver, adrenals, uterus and hematopoietic/lymphoreticular tissues. Brain tumor data was described as negative but counts were not provided. They reported no difference in survival by treatment group. All data presented were reanalyzed using a one-sided Fisher’s exact test for pairwise comparisons and the one-sided exact Armitage linear trend test for increasing or decreasing risk with exposure [165]. The reanalysis showed a decrease in the GSM data in all three treated groups in females in Harderian gland adenomas ( $p=0.045$ ,  $<0.01$ ,  $0.011$ ; trend test  $p=0.047$ ), in alveolar/bronchiolar carcinomas at the two lowest exposures ( $p=0.008$ ,  $0.008$ ) and adenomas at the highest exposure ( $p=0.045$ ), and increased trend in liver adenomas ( $p=0.033$ ) and a significant increase in uterus endometrial stromal polyps at the two lowest exposures ( $p=0.004$ ,  $0.046$ ) with no increased trend. In the DCS data for females, there was significant effect at the highest exposure for uterus glandular polyps ( $p=0.013$ ) with a significant trend ( $p=0.002$ ). In the male GSM exposure groups, Harderian gland adenomas were increased in all groups ( $p=0.027$ ,  $0.003$ ,  $0.001$ ) with a significant trend ( $p=0.004$ ) and a significant decreased trend in liver adenomas ( $p=0.001$ ) and decreases at all three exposures ( $p=0.014$ ,  $0.014$ ,  $<0.01$ ). In the male DCS exposure groups, Harderian gland adenomas were decreased for all exposure groups ( $p=0.001$ ,  $0.001$ ,  $0.001$ ) with a significant decreased trend ( $p=0.018$ ), a decrease in liver adenomas at the two highest groups ( $p=0.03$ ,  $<0.01$ ) with significant negative trend ( $p<0.01$ ), and a significant increase in lymphomas in all exposure groups ( $p=0.004$ ,  $0.046$ ,  $0.046$ ) with no trend. The increases in Harderian gland adenomas in the male GSM studies may be due to the exposure, but this was not explored by the authors. The large control response for Harderian gland adenomas in males in the DCS exposure studies suggests the incidence for this tumor in these studies is highly variable.

**National Toxicology Program (2018)** [166] exposed groups of 90 5-6 week old male and female B6C3F1/N mice to sham, GSM-modulated RF (2.5, 5 or 10 W/kg 9 hours/day, 7 days/week) or CDMA-modulated RF (2.5, 5 or 10 W/kg 9 hours/day, 7 days/week) for 106 (males) or 108 (females) weeks. The 9 hours and 10 minutes of exposure was achieved by cycling the fields 10 minutes on and 10 minutes off for 18 hours and 20 minutes each day. The mice exposed GSM-modulated and CDMA-modulated RF used the same sham controls. Exposures were conducted in reverberation chambers and animals were housed in individual cages. Full pathology was conducted on all animals. **GSM Study:** Survival was significantly higher for the 5 W/kg males than the sham controls; all other groups were not different from controls. There were no body weight differences between exposed animals and controls. They saw a marginal increase in skin fibrosarcoma, sarcoma or malignant fibrous histiocytoma in male mice ( $p=0.093$ ) (mostly occurring in the tails of these animals), a significant increase in alveolar/bronchiolar adenomas and carcinomas in male mice ( $p=0.040$ ) but not for adenomas and carcinomas separately, and significant increases in malignant lymphomas in the two lowest exposure groups for females, but the trend test was not significant and the control numbers were substantially smaller than historical

controls. To clarify the significance of the lung tumors in males, the NTP historical control data described in the technical report [166] was obtained electronically online, and using Tarone's test for historical controls [167], yields  $p=0.072$ . **CDMA Study:** Survival was significantly higher for the 2.5 W/kg females than the sham controls; all other groups were not different from controls. There were no body weight differences between exposed animals and controls. There were sporadic positive pairwise comparisons that were significant for liver tumors in male mice, but none of these demonstrated any pattern of exposure-response. Also, significant increases in malignant lymphomas in the lowest exposure group for females with increases in all groups, but the trend test was not significantly increased and the control numbers were substantially smaller than historical controls. Two adenomas and 1 carcinoma of the pars distalis in the pituitary gland occurred in the 5 W/kg group but not the other groups (these tumors were not seen in the historical controls). After 14 weeks of exposure, **Smith-Roe et al (2020)** [168] evaluated genotoxicity in several tissues of mice included in these studies for this purpose using the alkaline comet assay (three brain regions, liver, peripheral blood) and the micronucleus assay (peripheral blood). Significant increases in DNA damage were seen in the frontal cortex of male mice (DCMA and GSM) and leukocytes of female mice (CDMA only). NTP uses 5 levels of evidence for classifying the findings of carcinogenicity studies. Equivocal evidence is defined as *"Equivocal evidence of carcinogenic activity is demonstrated by studies that are interpreted as showing a marginal increase of neoplasms that may be test agent related."* In this study, for GSM-exposed mice, they labeled the skin tumors and lung tumors in males as equivocal and the malignant lymphomas in females as equivocal. For CDMA-exposed mice, they labeled the liver hepatoblastomas in males and the malignant lymphomas in females as equivocal. All of these conclusions seem reasonable. (Note: some text copied directly from **NTP (2018)** [166]).

#### 5.1.2 Rats

**Chou et al. (1992)** [169] exposed groups of 100 male Sprague-Dawley rats to pulsed microwave radiation at 2450 MHz at 800 pulses per second with a pulse width of 10  $\mu$ s for 21.5 hours per day, 7 days per week, for 25 months with an appropriate sham control. The exposure was intended to match a military-grade radar system and provide a whole body SAR of about 0.4 w/kg. They saw no changes in survival, body weight, or a number of other measures in the exposed animals and no increased tumor risk in any one organ. They did see a statistically significant increase in total tumors ( $p<0.001$ ), but it is not clear if this evaluation included multiple findings from the same animal or not (the statistical method used may have been incorrect).

**La Regina et al. (2003)** [170] exposed groups of 80 male and female Fisher 344 rats (aged 6 weeks) to sham, 835.6 MHz FDMA RF (SAR 1.3 W/kg) or 847.7 MHz CDMA RF (SAR 1.3 W/kg) for 4 hours/day, 5 days/week for 24 months in a tube-restrained Ferris-wheel exposure system. The exposure was predominantly to the head, but all tissues were examined. There were no differences in survival or body weight across appropriate comparison groups. They reported no significant tumor findings.

**Anderson et al. (2004)** [171] exposed groups of pregnant Fischer 344 rats to RF at 1620 MHz for 2 hours per day, 5 days per week from day 19 of gestation to weaning. At approximately 5 weeks of age, groups of 90 male and female offspring were exposed to the same RF using tubes with predominantly head only exposure for 2 hours per day, 5 days per weeks for 24

months. Targeted head exposure was sham, 0.16 and 1.6 mW/g. They reported no statistically increased differences in reproductive index, litter size, body weight or other clinical signs. There was a slight increase in survival in the highest exposure group in females relative to the sham exposed group. They noted there were no exposure-related significant increases in any tumors and that the highest exposure group of males had a significant increase in mesothelioma of the testis, but that this was within the range of historical controls. A reanalysis of the data presented results in the same findings as those presented by **Anderson et al. (2004)** and also showing a significant trend for mesothelioma of the testis ( $p=0.003$ ). **Anderson et al. (2004)** compared the oligodentroglioma data in males to the NTP historical control data presented by **Haseman et al. (1990)** [172], however, NTP has a set of controls more closely linked in time to this study that is more appropriate [173] showing the same range of responses (0-2%). Using the range of historical controls is inappropriate in this type of analysis [32, 33, 174] and a direct method of testing, Tarone's historical control test [167], is more appropriate; this test yields a p-value of  $p<0.001$  for the oligodentrogliomas in males. For the mesotheliomas in the testes, the NTP database contains no entries and the source cited by **Anderson et al. (2004)** has a range of 0-2% while the observed response in the highest exposure group was  $6/90=6.7\%$ , so well outside the range.

**Smith et al. (2007)** [175] duplicated the exposure system of **Tillmann et al. (2007)** [164] for groups of 50 male and female Wistar rats. They reported no survival differences and no significant increases in tumors in any tissue evaluated. For the tissues they reported in the paper, a re-analysis using the Armitage linear trend test shows an increase in the incidence of C-cell adenomas in female rats for both GSM ( $p=0.025$ ) and DCS ( $p=0.043$ ) exposures, but not for c-cell carcinomas ( $p=0.50$  and  $p=0.37$ ) and it remains significant for the combined adenomas and carcinomas ( $p=0.028$  and  $p=0.044$ ).

**Bartsch et al. (2010)** [176] conducted four separate RF studies in female Sprague-Dawley rats; two long-term (I and II) and two life-long (III and IV) experiments were conducted exposing animals to a low-intensity GSM-like signal (900 MHz pulsed with 217 Hz, 100  $\mu\text{W}/\text{cm}^2$  average power flux density, 38–80 mW/kg mean specific absorption rate for whole body). Health and survival of unrestrained female Sprague-Dawley rats kept under identical conditions was evaluated. Radiofrequency (RF)-exposure was started at 52–70 days of age and continued for 24 (I), 17 (II) and up to 36 and 37 months, respectively (III/IV). In the first two experiments 12 exposed and 12 sham-exposed animals each were observed until they were maximally 770 or 580 days old (animals either died of natural causes or were sacrificed because they were moribund). In experiment I, no adverse health effects of chronic RF-exposure were detectable, neither by macroscopic nor detailed microscopic pathological examinations. In experiment II no apparent macroscopic pathological changes due to treatment were apparent and microscopic analyses were not conducted. Reductions in pituitary tumors were seen for both experiment I and II but no increases were reported. In experiments III and IV, 30 animals per group showed a significant reduction in survival in the RF-exposed groups relative to the sham-exposed groups and both groups in experiment III showed a significant reduction in survival compared to experiment IV. A reduction in mammary tumors were seen in the RF-exposed animals compared to sham, but this may be due to the survival differences (authors did not evaluate this issue). This study did not perform full pathology, had limited sample sizes and presents very little tumor data.

**NTP (2018)** [177] exposed groups of 56 time-mated F<sub>0</sub> female Sprague-Dawley rats, housed in specially designed reverberation chambers, to whole-body exposures GSM-modulated cell phone RF or CDMA-modulated RF at power levels of 0 (sham control), 1.5, 3, or 6 W/kg for 7 days per week, continuing throughout gestation and lactation. Exposure was up to 18 hours and 20 minutes per day with continuous cycling of 10 minutes on and 10 minutes off during the exposure periods. At weanling, groups of 90 5-6 week old male and female Sprague-Dawley rats were exposed the same exposures as their F<sub>0</sub> dams for 105 weeks. The rats exposed to GSM-modulated and to CDMA-modulated RF used the same sham controls. Exposures were conducted in reverberation chambers and animals were housed in individual cages. Full pathology was conducted on all animals. GSM Exposures: In F<sub>0</sub> females, there were no exposure-related effects on pregnancy status, maternal survival, or the percentage of animals that littered. During gestation, mean body weight gains of 6 W/kg females were significantly lower than those of the sham controls from GD 15 through 18 and during the overall gestation period (GD 6 through 21). During lactation, the mean body weights of 3 and 6 W/kg females were significantly lower than those of the sham controls for the period of PND 4 through 21. In F<sub>1</sub> offspring, there was no effect on litter size, pup mortality or survival. During lactation, mean pup weights were significantly lower at most timepoints in the 3 W/kg groups and at all timepoints in the 6 W/kg groups. At the end of 2 years, survival of all exposed male groups was significantly greater than that of the sham control group due to the higher severity of chronic progressive nephropathy in the kidney of sham control males (note, almost all male rats had chronic progressive nephropathy). Survival of exposed female groups was similar to that of the sham controls. The mean body weights of all exposed males and females were similar to those of the sham control groups. There were no exposure-related clinical observations. In the heart at the end of the 2-year studies, malignant schwannoma was observed in all exposed male groups and the 3 W/kg female group, but none occurred in the sham controls. Endocardial Schwann cell hyperplasia also occurred in a single 1.5 W/kg male and two 6 W/kg males. There were also significantly increased incidences of right ventricle cardiomyopathy in 3 and 6 W/kg males and females. In the brain of males, there were increased incidences of malignant glioma and glial cell hyperplasia in all exposed groups, but none in the sham controls. There was also increased incidences of benign or malignant granular cell tumors in all exposed groups. There were significantly increased incidences of benign pheochromocytoma and benign, malignant, or complex pheochromocytoma (combined) of the adrenal medulla in males exposed to 1.5 or 3 W/kg. In the adrenal medulla of females exposed to 6 W/kg, there were significantly increased incidences of hyperplasia. In the prostate gland of male rats, there were increased incidences of adenoma or adenoma or carcinoma (combined) in 3 W/kg males and epithelium hyperplasia in all exposed male groups. In the pituitary gland (pars distalis), there were increased incidences of adenoma in all exposed male groups. There were also increased incidences of adenoma or carcinoma (combined) of the pancreatic islets in all exposed groups of male rats, but only the incidence in the 1.5 W/kg group was significant. In female rats, there were significantly increased incidences of C-cell hyperplasia of the thyroid gland in all exposed groups, and significantly increased incidences of hyperplasia of the adrenal cortex in the 3 and 6 W/kg groups. CDMA Exposures: In F<sub>0</sub> females, there were no exposure-related effects on pregnancy status, maternal survival, or the percentage of animals that littered. During gestation, the mean body weights and mean body weight gains of exposed groups were similar to those of the sham controls. During lactation, mean body weights were significantly lower than those of the sham controls at

most time points in the 6 W/kg group, at several time points in the 1.5 and 3 W/kg groups, and the mean body weight gains for the period as a whole (PND 1 through 21) were significantly lower in the 3 and 6 W/kg groups. In F<sub>1</sub> offspring, there were no effects on litter size on PND 1. On PND 7 through 21, there were significant decreases in live litter size in the 6 W/kg group when compared to the sham controls. Throughout lactation, the male and female pup mean body weights in the 6 W/kg groups were significantly lower than those of the sham controls. At the end of 2 years, survival in all exposed male groups was greater than that of the sham control group due to the effects of chronic progressive nephropathy in the kidney of the sham control males. In females, there was a small, but statistically significant increase in survival in the 6 W/kg group. Although there were some differences in mean body weights in exposed male groups, at the end of the study, the mean body weights of exposed male and female groups were similar to those of the sham controls. There were no exposure-related clinical observations. At the end of the 2-year study, malignant schwannoma of the heart occurred in all exposed male groups and the incidence in the 6 W/kg group was significantly increased; this neoplasm did not occur in the sham controls. There was also an increased incidence of endocardial Schwann cell hyperplasia in 6 W/kg males. In females, malignant schwannoma occurred in two animals each in the 1.5 and 6 W/kg groups. In the brain, malignant glioma occurred in 6 W/kg males and 1.5 W/kg females; none occurred in the sham control groups. Glial cell hyperplasia also occurred in 1.5 and 6 W/kg males and 3 and 6 W/kg females. In males, there was a significantly increased incidence of pituitary gland (pars distalis) adenoma in the 3 W/kg group, and increased incidences of hepatocellular adenoma or carcinoma (combined) in the liver of all exposed groups. In the adrenal medulla of females, there were increased incidences of benign, malignant, or complex pheochromocytoma (combined) in all exposed groups, but only the incidence in the 1.5 W/kg group was significantly increased compared to the sham controls. In the prostate gland of male rats, there were increased incidences of epithelial hyperplasia in all exposed groups, but only the incidence in the 6 W/kg group was significantly increased compared to the sham control group. After 14 weeks of exposure, **Smith-Roe et al (2020)** [168] evaluated genotoxicity in several tissues of rats included in these studies for this purpose using the alkaline comet assay (three brain regions, liver, peripheral blood) and the micronucleus assay (peripheral blood). Significant increases in DNA damage were seen in the hippocampus of male rats (CDMA-only). For the NTP, clear evidence of carcinogenic activity is *“demonstrated by studies that are interpreted as showing a exposure-related (i) increase of malignant neoplasms, (ii) increase of a combination of malignant and benign neoplasms, or (iii) marked increase of benign neoplasms if there is an indication from this or other studies of the ability of such tumors to progress to malignancy.”* For GSM exposures in males, NTP classified the malignant schwannomas of the heart, the malignant gliomas and the pheochromocytomas of the adrenal medulla as “clear evidence of carcinogenicity” and the granular cell tumors of the meninges, prostate gland tumors, pituitary gland tumors and pancreas islet-cell tumors as “equivocal findings”. In females, the NTP classified the malignant schwannomas of the heart as equivocal. For the CDMA exposures in males, NTP classified the malignant schwannomas of the heart and the malignant gliomas as “clear evidence of carcinogenicity” and the pituitary tumors and liver tumors as “equivocal evidence”. In females, the NTP classified the malignant schwannomas of the heart, the malignant gliomas and the pheochromocytomas of the adrenal medulla as equivocal. Given the glial hyperplasia, cardiomyopathy in the right ventricle and the magnitude of the effect in the adrenal gland, I

agree with the calls by the NTP. It is also worth noting that, when compared to the historical controls (Tarone's test), the lowest exposure CDMA group had a significant (0.016) increase in malignant gliomas. (Note: some text copied from **NTP (2018)** [177]).

**Falcioni et al. (2018)** [178] exposed groups (number not given) of F<sub>0</sub> female Sprague-Dawley rats, housed in specially designed cages, to whole-body exposures 1.8 GHz GSM-modulated cell phone RF at power levels of 0 (sham control), 5, 25 and 50 V/m for 7 days per week, from PD-12 continuing throughout gestation and lactation. Exposure was for 19 hours per day. At weaning, groups of approximately 200 (highest 2 exposures) or 400 (sham controls and low exposure) 5-6 week old male and female Sprague-Dawley rats were exposed the same exposures as their F<sub>0</sub> dams for 105 weeks (equivalent to 0.001, 0.03 and 0.1 W/kg SAR). Exposures were conducted in circular cage array with an antenna in the middle and animals were housed in individual chambers (5 per cage). Full pathology was conducted on all animals. This report only details the findings in the brain and the heart. They noted non-significant increases in Schwann cell hyperplasia at the high exposure for both males and females and an increase in malignant Schwannomas of the heart in males in the highest treatment group (p=0.037) and, using the Armitage linear trend test, yielded a significant trend (p=0.037). They noted that the rate of schwannomas in untreated males from their historical controls was 19/3160 (0.6%) and they observed 3/207 (1.4%). Heart schwannomas in females showed no trend. There were no increases in premalignant or malignant lesions in the brain for males or females in this study. The females had a slight positive trend in gliomas (p=0.118) but it was clearly not significant.

## 5.2 Transgenic and Tumor-Prone Models

### 5.2.1 Eμ-pim1 transgenic mouse

The Eμ-pim1 transgenic mice are prone to getting lymphomas.

**Repacholi et al. (1997)** [179] exposed groups of 100 to 101 female heterozygous Eμ-pim1 mice to GSM modulated RF at 900 MHz for up to 18 months with SAR values ranging from 0.13 to 1.4 W/kg depending upon animal sizes and the number in a cage. Mice were exposed for 30 minutes twice a day in cages grouped around a central antenna. There were no differences in weight by exposure, but there was a difference in deaths prior to study termination with 44/100 sham animals terminated early and 70/101 exposed animals terminated early. They reported a significant increase in the incidence of all lymphomas (p<0.001) and of non-lymphoblastic lymphomas (p=0.002) as a function of exposure. The statistical analysis of the data were unusual with analysis of only animals that died during the course of the study (terminal sacrifice animals were not examined histopathologically) and using a competing risk logistic regression model that is not fully explained in addition to the standard Fisher's exact test. The assumption that animals that did not die prior to terminal sacrifice were free of lymphomas makes this study difficult to interpret.

**Utteridge et al. (2002)** [180] attempted to replicate the study of Repacholi et al. (1997) [179] but with several differences. They used 120 animals per group, they included groups of wild-type C57BL/6N female mice, their GSM signal was 898.4 MHz, they used a restrained Ferris wheel design, exposed for 1 hour per day, 5 days per week for 104 weeks, and did full histopathological analysis on all mice regardless of survival. They used four different exposure groups at 0.25, 1.0, 2.0 and 4.0 W/kg. No exposure-related differences in body weight or survival were seen. They reported no exposure-related increases in any tumors

from this study. The longer duration of this study makes the direct comparison to Repacholi et al. (1997) difficult since most animals in this study had lymphomas at 104 weeks.

**Oberto et al. (2007)** [181] used the same exposure system as Utteridge et al. (2002) [180] to repeat the study of Repacholi et al. (1997) [179] by exposing groups of 50 male and female heterozygous  $E\mu$ -pim1 mice to 900 MHz pulsed RF fields for 18 months at whole-body SAR levels of 0.5, 1.4 and 4.0 W/kg. Exposures were for 30 minutes, twice daily, 7 days per week. Survival was reduced for male mice in all exposures and for female mice exposed at 0.5 W/kg; there were no significant differences in body weights. They reported no significant changes in lymphomas in males or females and a significant increase in Harderian gland adenomas in males that was exposure-dependent ( $p=0.028$ ). Using the Armitage linear trend test, the data show the change in Harderian gland adenomas in males ( $p=0.007$ ), liver vascular tumors in males ( $p=0.015$ ) and lung alveolar/bronchiolar adenomas ( $p=0.045$ ) in males. The largest difference between Repacholi et al. (1997) (22%) and Oberto et al. (2007) (44%) was in the number of sham controls with lymphomas and this was not due to only looking at decedents since Oberto et al. (2007) provided this analysis as well.

#### 5.2.2 Patched1<sup>+/-</sup> Mice

The Patched1 heterozygous ( $Ptc1+/-$ ) knockout mice are prone to getting tumors of the brain and are hypersensitive to ionizing radiation.

**Saran et al. (2007)** [182] exposed groups of 23-36 male and female  $Ptc1+/-$  mice and groups of 22-29 male and female wildtype CD1 mice to 900 MHz RF at whole-body SAR of 0.4 W/kg from postnatal days 2-6 for 30 minutes, twice per day and then followed for their lifespan with full necropsy at death or moribund sacrifice. Exposures were done in a system that constrained the mice during exposure. There were no survival differences with regard to exposure. The authors reported no increases in any tumors as a function of exposure. They reported an increase in Rhabdomyosarcoma in male and female combined in exposed versus sham which was marginally significant when evaluated using the one-sided trend test ( $p=0.053$ ). This study used a fairly low exposure for a very short exposure window.

#### 5.2.3 AKR/j Mouse

The AKR/j mouse is known to rapidly develop hematopoietic tumors, especially thymic lymphoblastic lymphoma, in the first year of life.

**Sommer et al. (2004)** [183] exposed groups of 160 female AKR/j mice to either sham or 900 MHz GSM-like RF (0.4 W/kg) for 24 hours/day, 7 days/wk until 46 weeks of age. Mice were housed 6-7 per cage in a Ferris wheel design. There was a significant difference in relative weight change but not in absolute change. There were no survival differences. There were no differences in death from lymphoblastic lymphoma between the sham and RF exposed groups. In a second study using the same design, **Sommer et al. (2007)** [184] used 1966 MHz UMTS RF (0.4 W/kg). There were no significant weight changes, no changes in survival or the incidence of lymphomas although there was a marginal reduction in the number of animals surviving to study end in the RF exposed group ( $p=0.055$ ).

**Lee et al. (2011)** [185] exposed groups of 40 male and 40 female AKR/j mice to sham or a combination of 848.5 MHz CDMA (2 W/kg) and 1950 MHz WCDMA (2 W/kg) RF for 45

min/day, 5 days/week for up to 42 weeks. Animals were housed 5 per cage during exposure in a reverberation chamber. No differences in body weight, survival or tumor incidence were observed.

### 5.2.3 C3H Mice

The C3H mouse carries a virus passed through breast milk that induces tumors of the mammary gland.

**Szmigielski et al. (1982)** [186] exposed groups of 40 female C3H/HeA mice to 2450 MHz RF from 6 weeks to 12 months at levels of 0, 2-3 W/kg and 6-8 W/kg. Exposure was carried out in an anechoic chamber for 2 hours per day, 6 days per week. The presence of mammary gland tumors was determined by palpation every two weeks. The authors noted a exposure-related increase in the number of mammary tumors ( $p < 0.01$ ) and a exposure-related decrease in the time to onset of mammary tumors ( $p < 0.05$ ) in their experiments. By their analysis, no other tumors were significantly increased as a function of exposure to the RF.

**Toler et al. (1997)** [187] exposed groups of 200 female C3H/HeJ mice for 21 months (22 h/day, 7 days/week) to a horizontally polarized 435 MHz pulse-wave (1.0 microsecond pulse width, 1.0 kHz pulse rate) RF environment with an SAR of 0.32 W/kg. An additional 200 mice were sham-exposed. All animals were necropsied and subject to full histopathological analysis. The exposure facility used 50 single housing cages around a central antenna facility to produce uniform circular fields. No survival differences were observed between the groups. There were no significant differences between the two groups with respect to latency to tumor onset, tumor growth rate and overall tumor incidence for mammary tumors. The only significant difference between groups for tumors in other organs was for bilateral ovarian epithelial stromal tumors ( $p = 0.03$  by their analysis,  $p = 0.023$  by mine) but became nonsignificant when all animals with stromal tumors were considered ( $p = 0.24$  by their analysis,  $p = 0.12$  by mine).

**Frei et al. (1998)** [188] exposed groups of 100 female C3H/HeJ mice for 18 months to 2450 MHz microwave radiation for 20 hours per day, 7 days per week. Exposure was via the CWG system with 2 animals per cage distributed around a circular field. The SAR targeted in this study was 0.3 W/kg. There were no differences in body weight or survival in the two groups. There were no significant differences between the two groups with respect to latency to tumor onset, tumor growth rate and overall tumor incidence for mammary tumors. There were no significant increases in tumors at any site but they also saw a slight increase in bilateral ovarian stromal tumors. **Frei et al. (1998)** [189] repeated this study using an SAR of 1 W/kg, again seeing no increases in any tumor as a function of exposure. In this second study, mammary tumors in sham-treated animals were much lower (30%) than in the previous study (54%).

**Jauchem et al. (2001)** [190] exposed groups of 100 female C3H/HeJ mice to pulses composed of an ultra-wideband (UWB) of frequencies, including those in the RF range (rise time 176 ps, fall time 3.5 ns, pulse width 1.9 ns, peak E-field 40 kV/m, repetition rate 1 kHz) at an SAR of 0.0098W/kg for 2 minutes per week for 12 weeks with a follow-up of 64 weeks. They saw no neoplastic changes associated with exposure. [This study uses an incredibly small SAR for a very short period.]

### 5.3 Initiation-Promotion Studies

In general, initiation promotion studies use two stages of exposure to determine if a particular exposure starts the cancer process (initiates tumors) or makes tumors grow faster or appear more readily (promotion). In most cases in the literature that follows, researchers are testing for the promotional impacts of RF using a known initiator (chemical that starts the cancer process).

#### 5.3.1 Skin Models

The usual initiation-promotion study in skin involves the application of an initiator chemical (7,12-dimethylbenz[a]anthracene (DMBA) or benzo[a]pyrene (BAP)) once to the shaved skin of a mouse followed by frequent exposures to a promotor (in this case RF) for a long period of time. The studies also typically use a known promotor as a positive control (e.g. 12-O-tetradecanoylphorbol-13-acetate or TPA) to demonstrate the experimental setting is working appropriately. The tumors that appear on the back of the animals are tracked over time and the endpoints of interest (tumor frequency and multiplicity) recorded daily.

**Chagnaud et al. (1999)** [191] exposed groups of 8-18 female Sprague-Dawley rats to GSM 900 MHz RF at an SAR of 75 mW/kg starting 20, 40 or 75 days after initiation by BaP (2 mg) for 2 hours per day, 5 days per week for two weeks. In addition, GSM 900 MHz RF at an SAR of 270 mW/kg was administered 40 days after exposure to BaP (2 mg) for 2 hours per day, 5 days per week for two weeks. The study was terminated approximately 160 days after the BaP exposure. There was no impact of any RF exposure on the survival or time to tumor in these experiments.

**Mason et al. (2001)** [192] exposed groups of 27-55 female Sencar rats to DMBA (initiator, 2.56  $\mu\text{g}$ ) followed by a single 10 second exposure to 94 GHz RF at 1 W/cm<sup>2</sup> or to infrared radiation (IR) at 1.5 W/cm<sup>2</sup>, both designed to raise skin temperature by 13-15° C. The animals were followed for 23 weeks and there was no indication of a promotion affect on these animals. In a second experiment using the same basic protocol, exposures of 10 seconds twice per week for 12 weeks to RF at 333 mW/cm<sup>2</sup> and IR at 600 mW/cm<sup>2</sup> (designed to raise skin temperature by 4-5° C) and followed to 25 weeks. There was no indication of a promotion effect of RF in this experiment. The authors also conducted a co-promotional study where the RF and IR exposures were given along with TPA to see if the RF enhanced the TPA promotional effect; this study was also negative.

**Imaida et al. (2001)** [193] exposed exposed groups of 48 female ICR mice to DMBA (initiator, 100  $\mu\text{g}$ ) followed by a TDMA RF field at 1.49 GHz (50 pulse per second) for 90 minutes per day, 5 days per week for 19 weeks at an SAR of 2 W/kg. There was no promotion of tumors by RF in this study.

**Huang et al. (2005)** [194] exposed a group of 20 male ICR mice to DMBA (initiator, 100  $\mu\text{g}$ ) followed by a CDMA signal at 849 MHz for 45 minutes twice per day, 5 days per week for 19 weeks at an SAR of 0.4 W/kg. They exposed a second group of 20 males to CDMA signal at 1763 MHz for 45 minutes twice per day, 5 days per week for 19 weeks at an SAR of 0.4 W/kg. There was no promotion of tumors by RF in this study.

**Paulraj and Behari (2011)** [195] exposed groups of 10 male Swiss albino mice to DMBA (initiator, 100  $\mu\text{g}$ ) to 112 MHz amplitude modulated (AM) at 16 Hz (power density 1.0 mW/cm<sup>2</sup>, SAR 0.75 W/kg) or to 2.45 GHz radiation (power density of 0.34 mW/cm<sup>2</sup>, SAR,

0.1 W/kg), 2 h/day, 3 days a week for a period of 16 weeks. There was no promotion of tumors by RF in this study. In a second experiment, mice were transplanted intraperitoneally (ip) with ascites  $8 \times 10^8$  (Ehrlich-Lettre ascites, strain E) carcinoma cells per mouse followed by the same 2 radiation exposures for 14 days. They saw a non-significant increase in the number of ascites in the treated groups compared to the appropriate controls. This study suffers from a very small sample size.

### 5.3.2 Lymphoma Models

Here, the initiator is ionizing radiation.

**Heikkinen et al. (2001)** [196] exposed groups of 50 female CBA/S mice to Xrays (initiation, 4-6 MV, 3 weekly exposures of 1.333 Gy) followed by exposure to NMT900-type frequency-modulated RF at 902.5 MHz and a nominal SAR of 1.5 W/kg for 1.5 hours/day, 5 days per week, for 78 weeks. A second group with the same initiation was exposed to GSM-type RF at 902.5 MHz (pulse frequency 217 Hz) at an SAR of 0.35 W/kg with the same exposure pattern. They saw a increase in the median corpuscular hemoglobin concentration in both RF exposure groups ( $p=0.008$  NMT900 and  $p=0.026$  GSM). There were no survival differences. There were several changes in preneoplastic hyperplastic markers related to RF exposure, but no significant increases in tumors related to RF. There was a significant reduction in pheochromocytomas in the adrenal glands in both RF exposure groups. There were no changes in lymphoma incidence.

### 5.3.3 Mammary-gland Tumor Model

This model typically involves female Sprague-Dawley rats initiated by DMBA.

**Bartsch et al. (2002)** [197] sequentially conducted three identical studies where groups of 60 female Sprague-Dawley rats were given DMBA as an initiator (50 mg/kg/day) followed by either sham exposure or exposure to GSM RF at 900 MHz (pulse 217 Hz) for 23 hours per day, 7 days per week for 259-334 days. Exposures were in group-housed cages and ranged from 15 to 130 mW/kg depending upon the age of the animals. There were no differences between sham and exposed animals in terms of numbers of benign or malignant tumors at study termination in all three experiments although the experiments themselves differed significantly in overall tumor incidence. In the first experiment, malignant mammary tumors appeared much more rapidly in sham-exposed animals, but this was not reproduced in the two replicates.

**Anane et al. (2003)** [198] conducted 2 experiments using a GSM signal at 900 MHz with female Sprague-Dawley rats in cages in a chamber for 2 hours/day, 5 days/week for 9 weeks and followed without exposure for 2 more weeks. Initiation was done using DMBA (10 mg) and RF exposures began 10 days after initiation. In the first exposure, 16 animals per group were exposed to 0, 1.4, 2.2 or 3.5 W/kg SAR RF and in the second were exposed to 0, 0.1, 0.7 and 1.4 W/kg SAR RF. The first experiment saw a reduction in time to tumor for the 1.4 W/kg group, a lesser, but still significant reduction in time to malignant tumor for the 2.2 W/kg group and no difference from sham-exposed for the 3.5 W/kg group. This was not seen in the second experiment. The second experiment also saw substantially reduced tumor counts in the treated groups compared to the first experiment.

**Yu et al. (2006)** [199] exposed four groups of 99-100 female Sprague-Dawley rats to DMBA (initiator, 35 mg/kg) followed by sham exposure or exposure to 900MHz GSM signal RF for 4 hours/day, 5 days/week for 26 weeks in a Ferris wheel tube-restrained exposure system. The four exposures were 0, 0.44, 1.33 and 4.0 W/kg SAR. No differences in body weight, incidence, latency, multiplicity or size of mammary gland tumors was seen in this experiment as a function of RF exposure.

**Hruby et al. (2008)** [200] conducted an experiment almost identical to that of Yu et al. (2006). Four groups of 100 female Sprague-Dawley rats were exposed to DMBA (initiator, 17 mg/kg) followed by sham exposure or exposure to 900MHz GSM signal RF for 4 hours/day, 5 days/week for 26 weeks in a Ferris wheel tube-restrained exposure system. The four exposures were 0, 0.4, 1.3 and 4.0 W/kg SAR. The results showed a significant shift from benign mammary tumors to malignant mammary tumors for animals with exposure to RF. The highest exposure group saw a significant increase in malignant tumors relative to the sham controls and all three RF exposure groups saw a significant reduction in benign tumors compared to the sham exposure group. No differences in volume or time-to-palpable tumor were seen.

#### 5.3.4 Brain tumor models

Brain tumor initiation-promotion studies generally use rats (Fischer 344 or Sprague-Dawley) initiated for brain tumors using N-ethyl-N-nitrosourea (ENU) in-utero using a single intravenous exposure to the dam.

**Adey et al. (1999)** [201] exposed two groups of 9 pregnant Fisher 344 rats to ENU (4 mg/kg) on day 18 of gestation and two groups of 9 to sham exposure. Starting on day 19 of gestation to post-natal day (PND) 21, two groups of dams and offspring (one with ENU [denoted EF for ENU-Field] and the other without [denoted SF for Sham-Field]) were exposed in cages to far field TDMA (836.55 MHz) for 2 hours/day, 7 days/week (SAR not provided) and two groups (no enu [denoted SS] and with ENU [denoted ES]) were given sham exposure to RF. Starting on PND 33 until two years of age, groups of 30 male and 30 female mice were exposed to near-field TDMA exposures at 836.55 MHz in the same groups as with the dams (SS, ES, SF, EF). Near field exposures (animals held in tubes with predominantly head exposure) had an SAR from 1.1-1.6 W/kg. Animals administered ENU had a reduction in survival in all groups and animals with RF exposure survived longer than their respective controls in all groups (not statistically significant). All RF exposed groups had reduced central nervous system tumors relative to their appropriate controls except for meningiomas (without ENU there was 1 tumor in RF exposed and no tumors in control and with ENU there were 2 tumors in RF exposed and none in control) and granular cell tumors (without ENU there was 1 tumor in RF exposed and no tumors in control). A reanalysis of the data using the exact trend statistic (one-sided) shows a significant reduction in CNS tumors with RF exposure with ( $p=0.036$ ) and without ( $p=0.016$ ) ENU, almost entirely due to glial tumors. No numbers were provided for any differences by sex.

**Adey et al. (2000)** [202] repeated this study with a larger number of offspring (45 males and 45 females) in each of the exposure groups and using an FM signal (836.55 MHz). The survival patterns were the same as for their previous study. Unlike the previous study, RF exposure yielded approximately the same incidence as sham exposure for all CNS and brain tumors. Differences between sexes were not provided.

**Zook and Simmens (2001)** [203] exposed pregnant female Sprague-Dawley rats to ENU at a exposure of 0, 2.5 or 10 mg/kg on day 15 of gestation. At 8 weeks of age, groups of 30 male and 30 female rats with in-utero ENU exposure were exposed to sham, pulsed-wave RF exposure (860 MHz) at a brain SAR of 1 W/kg or pulsed-wave RF exposure (860 MHz) at a brain SAR of 1 W/kg for 6 hours per day, 4 days per week for 22 months. The exposure was 'head only' and used a tube-restrained system in a Ferris wheel design. Results were presented for males and females combined. There were no significant findings in the brain or central nervous system. There was a significant increase in thyroid tumors in males ( $p=0.016$ , all sham controls grouped and all ENU exposures grouped) and a marginal increase in female mammary tumors ( $p=0.057$ ).

**Zook and Simmons (2006)** [204] repeated this experiment where they exposed pregnant female Sprague-Dawley rats to ENU at a exposure of 6.35 or 10 mg/kg on day 15 of gestation. At 8 weeks of age, groups of 90 male and 90 female rats with in-utero ENU exposure were exposed to sham or pulsed-wave RF exposure (860 MHz) at a brain SAR of 1 W/kg for 6 hours per day, 4 days per week for 22 months. The exposure was 'head only' and used a tube-restrained system in a Ferris wheel design. Results were presented for males and females combined. There were no significant findings in the brain or central nervous system.

**Shirai et al. (2005)** [205] exposed pregnant female Fisher 344 rats to ENU as done in Adey et al. (1999). At 5 weeks of age, groups of 50 male and 50 female rats with in-utero ENU exposure were exposed to sham, TDMA RF exposure (1439 MHz) at a brain SAR of 0.67 W/kg or at a brain SAR of 2 W/kg for 90 minutes per day, 5 days per week until age 104 weeks. The exposure was "head only" as in Adey et al. (1999). In females, there was a non-significant increase in survival with RF exposure but not in males. The authors reported no significant changes in any CNS tumors in the RF-exposed animals relative to sham-exposed animals. However, a reanalysis of the data using the Armitage linear trend test shows a marginal decrease in any type of brain tumor in females ( $p=0.057$ ) that is driven by a reduction in astrocytomas ( $p=0.032$ ). This was not seen in males. They noted a significant reduction in pituitary tumors in the highest exposure group for males, but tumor numbers were not provided.

**Shirai et al. (2007)** [206] used the exact same exposure scenario to examine the effects of WCDMA RF at 1.95 GHz at SAR 0.67 W/kg and 2.0 W/kg. There were no obvious survival differences among the treated groups and the sham controls and some mild organ weight differences in females but none in males. The authors reported no significant changes in tumor rates for any organ however they did not do trend tests. Using the Armitage linear trend test, female rats saw a significant increase in any brain tumor ( $p=0.030$ ) driven primarily by an increase in astrocytomas ( $p=0.027$ ). Males saw an increase in astrocytomas that was not statistically significant ( $p=0.181$ ).

### 5.3.5 Liver Tumor Models

**Imaida et al. (1998)** [207] exposed groups of 48 five-week old male Fisher 344 rats to a single exposure of 200 mg/kg diethylnitrosamine (DEN) followed two-weeks later by exposure to 1.439 GHz TDMA RF at a whole body SAR of 0.453-0.680 W/kg 90 minutes a day, 5 days/week for six weeks. At three weeks the rats received a 2/3 partial hepatectomy and at the end of the six weeks of RF exposure, the study was terminated and all rats

examined in their liver for the number and size of glutathione S-transferase placental form positive focal lesions that are considered precursors for liver cancer. They saw significant increases in corticosterone ( $p < 0.001$ ), melatonin ( $p < 0.05$ ) and adrenocorticotrophic hormone ( $p < 0.001$ ) and a significant reduction ( $p < 0.05$ ) in the number of GST-positive foci/cm<sup>2</sup>. Similar findings were seen for the exact same experimental design using 929.2 MHz TMDA RF with whole body SARs between 0.58-0.80 W/kg [208].

#### 5.4 Co-Carcinogenesis

Co-carcinogenesis studies are conducted by administering RF exposure along with another substance already known to be carcinogenic to see if the RF exposure enhances the carcinogenic findings. Usually, these models are targeted to a specific type of cancer.

**Szmigielski et al. (1982)** [186] exposed groups of 40 6-week old male Balb/c mice to 5% solution of 3,4-benzopyrene (BP) on depilated skin every second day for 5 months. Groups of these mice were exposed to 2450 MHz microwaves for 2 hours/day for the same 5 months at exposure of 5 mW/cm<sup>2</sup> or 15 mW/cm<sup>2</sup>. Two other groups of mice were exposed to 1 or 3 months of the same RF exposure of 5 mW/cm<sup>2</sup> followed by exposure to BP until 5 months. All animals were observed until 10 months. Exposures were in anechoic chamber. The target of these exposures was skin tumors. There were clear exposure-related and age-related increases in skin tumors in all RF-exposed groups compared to their sham-exposed groups. It is not clear if the sham-exposed controls in the 1- and 3-month RF exposure experiments were properly done. In addition, the presentation of the results from this study are sufficiently confusing that misinterpretation of the findings is possible.

**Szudzinski et al. (1982)** [209] performed a similar experiment to that done by Szmigielski et al. (1982) (they are in the same research group). They exposed groups of 100 6-week old male Balb/c mice to 1% solution of 3,4-benzopyrene (BP) on depilated skin every second day for 6 months. Groups of these mice were exposed to 2450 MHz microwaves for 2 hours/day for the same 6 months at exposures of 2 mW/cm<sup>2</sup> or 6 mW/cm<sup>2</sup>. Three other groups of mice were exposed to 1, 2 or 3 months of the same RF exposure of 4 mW/cm<sup>2</sup> followed by exposure to BP until 6 months. All animals were observed until 10 months of age. Exposures were in anechoic chambers. The target of these exposures was skin tumors. There were clear exposure-related and age-related increases in skin tumors in all RF-exposed groups compared to their sham-exposed groups. It is not clear the sham-exposed controls in the 1-, 2- and 3-month RF exposure experiments were properly done. In addition, the presentation of the results from this study are sufficiently confusing that misinterpretation of the findings is possible.

**Wu et al. (1994)** [210] exposed two groups of 26-32 male and 26-32 female BALB/c mice to dimethylhydrazine for 14 weeks (15 mg/kg subcutaneous injection once per week) and then an additional 8 weeks (20 mg/kg subcutaneous injection once per week). Three weeks after the first injection, one group of mice was sham exposed and the other exposed to 2450 MHz RF (10-12 W/kg SAR) for 3 hours/day, 6 days/week for 5 months. The focus was on colon tumors and there was no difference between groups.

**Heikennen et al. (2003)** [211] exposed groups of female K2 transgenic mice (overexpressing human ornithine decarboxylase gene) and their wild-type littermates (strain not provided) were exposed to UV radiation (240 J/m<sup>2</sup>) 3 times per week for 52 weeks. The separate groups were exposed to sham RF, D-AMPS RF (849 MHz, 0.5 W/kg SAR) or GSM RF (902.4

MHz, 0.5 W/kg SAR) 1.5 hours/day, 5 days/week for 52 weeks. The target of the experiment was skin lesions. There were no survival differences when compared to appropriate controls in transgenic or wild-type RF-treated animals and no changes in skin lesion incidence was observed.

**Heikennen et al. (2006)** [212] exposed groups of 72 female Wistar rats (age 7 weeks) to 3-chloro-4-(dichloromethyl)-5-hydroxy-2(5H)-furanone (MX) via drinking water at a exposure of 1.7 mg/kg/day for 104 weeks. Separate groups were exposed to pulsed RF at 900 MHz (pulse frequency 217 Hz) in a circular array of small cages for 2 hours per day, 5 days per week, for 104 weeks at whole body SARs of 0 (sham), 0.3 or 0.9 W/kg. There were no survival differences, body weight gain differences or MX consumption differences between sham-exposed and RF-exposed rats. By Peto's test, the combined incidence of vascular tumors in the mesenteric lymph nodes was significantly increased in trend ( $p=0.036$ ). Using the Armitage linear trend test, the combined incidence was also significant ( $p=0.001$ , one-sided) driven by the increase in hemangiomas ( $p=0.023$ ). The authors argued this was not significant since the incidence in the cage controls was higher than the sham controls. There was a significant increase in vacuolated foci in the liver by the Armitage linear trend test ( $p=0.002$ ) but no increases in tumors in the liver.

**Tillmann et al. (2010)** [213] exposed pregnant B6C3F1 mice and 54-60 of their female offspring to whole-body UMTS RF at 1966 MHz (4.8 W/m<sup>2</sup> or 48 W/m<sup>2</sup>) from GD6 to 2 years of age. The dams exposed to 4.8 W/m<sup>2</sup> also received a exposure of 40 mg/kg ENU on GD 14 as did a group with sham exposure to the RF. A full necropsy was performed on each animal. No differences in survival were seen between RF-exposed groups and their appropriate controls. The 48 W/m<sup>2</sup> group did not show any increases in tumors relative to the appropriate controls although they did see a significant increase in liver focal lesions ( $p=0.002$  one-sided). The ENU-treated groups were terminated after 75 weeks due to mortality and all animals necropsied. The RF-exposed group saw an increase in bronchiolar-alveolar carcinomas ( $p=0.005$ ), adenomas ( $p=0.032$ ), adenomas or carcinomas combined (0.017) and a marginal increase in hyperplasias ( $p=0.098$ ). They also saw an increase in liver adenomas ( $p<0.001$ ), not carcinomas or blastomas, but an increase in combined adenomas/carcinomas/blastomas ( $p=0.023$ ) and an increase in liver foci ( $p=0.005$ ). There were no increases in brain tumors in any treated groups. Tumor multiplicity in both the lung and the liver was increased as was the incidence of metastasizing lung tumors.

### 5.5 Summary and Conclusions for Laboratory Cancer Studies

The central question to ask of animal cancer studies is "Can RF increase the incidence of tumors in laboratory animals?" The answer, with high confidence, is yes. Table 20 summarizes the findings from the chronic exposure carcinogenicity studies for RF.

For rats, the **NTP (2018)** [177] chronic exposure bioassay in male Sprague-Dawley rats, including in-utero exposure, is clearly positive for acoustic neuromas of the heart, malignant gliomas of the brain and pheochromocytomas of the adrenal gland. These findings are further supported by the presence of preneoplastic lesions and tissue toxicity in the heart, brain glial cells and adrenal glands. The less convincing findings in the study by **Falcioni et al. (2018)** [178] of heart acoustic neuromas in male Sprague-Dawley rats and a marginal increase in malignant gliomas in females provides additional support for this finding. The study by **Anderson et al. (2004)** [171] with a significant increase in oligodendrogliomas in

male Fischer 344 rats when compared against historical controls provides additional strong support for an increase in gliomas from exposure to RF. This study also saw an increase in testis mesothelioma which may have been due to exposure. The lack of any brain pathology or tumors in any organ or tissue within the study by **La Regina et al. (2003)** [170], which was also in Fischer 344 rats, weakens the findings from the Anderson et al. (2004) study, but cannot fully negate them since these are different exposures at different frequencies. The **Bartsch et al. (2010)** [176] study, done using Sprague-Dawley rats, is too limited to challenge the findings of the **NTP (2018)** study. Finally, the lack of brain and heart tumors in the **Smith et al. (2007)** [175] study, done in Wistar rats, could easily be due to the different strain of rat. This study did see an exposure-related increase in thyroid C-cell tumors that was not seen in the other studies in rats.

In B6C3F<sub>1</sub> mice (the only strain tested for chronic exposure), the strongest findings are for the Harderian gland tumors in males for GSM but not DCS RF and the increase in uterine polyps in females for both GSM and DCS in the **Tillmann et al. (2007)** study [164] and the increase in rare tumors of the pars distalis in the pituitary of females in the **NTP (2018)** [166] study which were also seen for the male rats in the other NTP study [177]. The variability of the Harderian gland increases and decreases between males and females and the different types of RF in the **Tillmann et al. (2007)** study suggest that the Harderian gland is a sensitive target in these animals or that the response is highly variable in these mice for these tumors. The NTP historical controls [214] for Harderian gland tumors for this period include 29 studies and range between 6% and 26% with a mean of 16% for adenomas and carcinomas combined; the exposed groups in the **Tillmann et al. (2007)** GSM study showed responses of 24%, 32% and 36% for the low, medium and high male exposure groups, beyond the range of the historical control data supporting the conclusion this is a real, exposure-related finding. The **NTP (2018)** study did not see an increase in Harderian gland tumors in males nor an increase in uterine polyps in females. However, this study used a very different exposure system and this may have contributed to the differences.

The studies in transgenic and tumor-prone mice show mixed results. The initial positive finding of lymphomas in E $\mu$ -pim1 transgenic mice by **Repacholli et al. (1997)** [179] were not seen in two subsequent studies [180, 181] that used better designs and better methods. It is interesting to note that the **Oberto et al. (2007)** study [181] saw an increase in Harderian gland tumors in male mice, supporting the finding from **Tillmann et al. (2007)** [164]. The one study in Patched1 $\pm$  transgenic mice was negative for brain tumors but saw a marginal increase in Rhabdomyosarcomas. The two studies in AKR/j mice were negative. The study with the highest SAR exposure levels in C3H mice [186] was positive for mammary tumors, but the remaining four [187-190] were not. It is of note that two of these studies [187, 188] saw increases in uterine stromal polyps supporting the findings from **Tillmann et al. (2007)** [164].

The initiation-promotion studies in skin [191-195] were uniformly negative as was the one study using a lymphoma model [196]. The initiation-promotion studies using a mammary tumor model [197-200] were also uniformly negative although the study by **Hruby et al. (2006)** [200] saw an exposure-related shift from benign mammary tumors to malignant tumors. The initiation-promotion studies using ENU-based brain tumor models [201-206] were negative for brain tumors with the exception of one study [206] showing an increase in brain tumors driven by an increase in astrocytomas. One of these studies [203] saw an increase in thyroid tumors in males as a function of exposure that supports the one finding

in the chronic study by **Smith et al. (2007)** [175] who saw an increase in thyroid tumors in females. The one initiation-promotion study using a liver tumor model [207] saw increases in liver foci and several changes in endocrine hormones, but no liver tumors.

Four of the co-carcinogenesis studies were positive [186, 209, 212, 213] and two were negative [210, 211]. Two of the positive studies [186, 209] showed skin tumors (not surprising since the co-carcinogen was BP applied to the skin) and another positive study [212] showed increases in lymph nodes and blood vessel tumors. Another positive study [213] saw increases in lung tumors and liver tumors in female mice exposed in-utero supporting findings seen in the **Tillmann et al. (2007)** [164] study and the **NTP (2018)** [166] study.

In conclusion, there is sufficient evidence from these laboratory studies to conclude that RF can cause tumors in experimental animals with strong findings for gliomas, heart Schwannomas and adrenal pheochromocytomas in male rats and harderian gland tumors in male mice and uterine polyps in female mice. There is also some evidence supporting liver tumors and lung tumors in male and possibly female mice.

Table 20: Summary of Chronic Exposure Carcinogenicity Studies for Radiofrequency Radiation

Study	Species/Strain	RF Exposure	Sex	Tumor Finding	Notes	
Tillmann et al. (2007) [164]	Mouse B6C3F <sub>1</sub>	GSM 902 MHz	M	Harderian Gland ↑ Liver Adenoma ↓	All exposures, no trend  Two lowest exposures, no trend	
			F	Harderian Gland ↓ Lung Tumors ↓ Liver adenomas ↑ Uterus polyps ↑		
		DCS 1747 MHz	M	Harderian Gland ↓ Liver Adenoma ↓ Lymphomas ↑		All exposure groups, no trend
			F	Uterus polyps ↑		
National Toxicology Program (2018) [166]	Mouse B6C3F <sub>1</sub>	GSM 1.9 GHz	M	Lung tumors ↑	Lowest 2 exposures, no trend  Sporadic, no trend or pattern  Low group, increased in all, no trend Rare tumor	
			F	Malignant lymphomas ↑		
		CDMA 1.9 GHz	M	Liver tumors ↑		
			F	Malignant lymphomas ↑ Pituitary pars distalis ↑		
Chou et al. (1992) [169]	Rats S-D	Pulsed 2450 MHz	M	Total tumors ↑	No individual tumor findings	
La Regina et al. (2003) [170]	Rats F344	FDMA 835.6 MHz	M		No tumor findings	
			F		No tumor findings	
		CDMA 847.7 MHz	M		No tumor findings	
			F		No tumor findings	
Anderson et al. (2004) [171]	Rats F344	Iridium 1.62 GHz	M	Testis mesothelioma ↑ Oligodentroglioma ↑	Using HC, p<0.001	
			F		No tumor findings	
	Rats	GSM	M		No tumor findings	

<b>Smith et al. (2007)</b> [175]	Wistar	902 MHz	F	C-cell tumors ↑	Adenomas & combined, not carc.
		DCS 1747 MHz	M		No tumor findings
			F	C-cell tumors ↑	Adenomas & combined, not carc.
<b>Bartsch et al. (2010)</b> [176]	Rats S-D	GSM 900 MHz	F		No tumor findings (four separate experiments, small sample sizes, not full pathology)
<b>NTP (2018)</b> [177]	Rats S-D	GSM 900 MHz	M	Heart schwannoma ↑ Brain glioma ↑ Adrenal pheochromocytoma ↑ Brain meninges ↑ Prostate gland ↑ Pituitary pars distalis ↑ Pancreas islets ↑	Rare tumor, biological call  Lowest 2 exposures, no trend Biological call Rare tumor, biological call No trend, extensive hyperplasia Low exposure group, no trend
			F	Heart schwannoma ↑	One exposure only, rare tumor
		CDMA 900 MHz	M	Heart schwannoma ↑ Brain glioma ↑ Pituitary pars distalis ↑ Liver tumors ↑F	Rare tumor, biological call One exposure, no trend Rare tumor, increased but not significant
				Heart schwannoma ↑ Brain glioma ↑ Adrenal pheochromocytoma ↑	Marginal finding Rare tumor, 3 in lowest group, no sig, no trend  Low exposure only, no trend
<b>Falcioni et al. (2018)</b> [178]	Rats S-D	GSM 1.8 GHz	M	Heart schwannoma ↑	
			F		No tumor findings (slight ↑ in malignant gliomas)

## 6. Mechanisms Related to Carcinogenicity

**There is sufficient evidence to suggest that both oxidative stress and genotoxicity are caused by exposure to RF and that these mechanisms could be the reason why RF can induce cancer in humans.**

### 6.1 Introduction

Many human carcinogens act via a variety of mechanisms causing various biological changes, taking cells through multiple stages from functioning normally to becoming invasive with little or no growth control (carcinogenic). **Hanahan and Weinberg (2011)**[215] identified morphological changes in cells as they progress through this multistage process and correlated these with genetic alterations to develop what they refer to as the “hallmarks of cancer.” These hallmarks deal with the entire process of carcinogenesis and not necessarily with the reasons that cells begin this process or the early stages in the process where normal protective systems within the cells remove potentially cancerous cells from the body. While tumors that arise from a chemical insult to the cell may be distinct from other tumors by mutational analysis, they all exhibit the hallmarks as described by **Hanahan and Weinberg (2011)**.

Systematic review of all data on the mechanisms by which a chemical causes cancer is complicated by the absence of widely accepted methods for evaluating mechanistic data to arrive at an objective conclusion on human hazards associated with carcinogenesis. Such systematic methods exist in other contexts [216], but are only now being accepted as a means of evaluating literature in toxicological evaluations [32, 217-220].

In this portion of the report, I am focusing on the mechanisms that can cause cancer. **Smith et al. (2015)** [39] discussed the use of systematic review methods in identifying and using key information from the literature to characterize the mechanisms by which a chemical causes cancer. They identified 10 “Key Characteristics of Cancer” useful in facilitating a systematic and uniform approach to evaluating mechanistic data relevant to carcinogens. These 10 characteristics are presented in Table 21 (copied from Table 1 of **Smith et al. (2015)** [39]). While there is limited evidence on RF exposure for most of the key characteristics, genotoxicity (characteristic two) and oxidative stress (characteristic five) have sufficient evidence to warrant a full review.

Table 21: Key characteristics of carcinogens, Smith et al. (2016)[65]

Characteristic	Examples of relevant evidence
1. Is electrophilic or can be metabolically activated	Parent compound or metabolite with an electrophilic structure (e.g., epoxide, quinone), formation of DNA and protein adducts
2. Is genotoxic	DNA damage (DNA strand breaks, DNA–protein cross-links, unscheduled DNA synthesis), intercalation, gene mutations, cytogenetic changes (e.g., chromosome aberrations, micronuclei)

3. Alters DNA repair or causes genomic instability	Alterations of DNA replication or repair (e.g., topoisomerase II, base-excision or double-strand break repair)
4. Induces epigenetic alterations	DNA methylation, histone modification, microRNA expression
5. Induces oxidative stress	Oxygen radicals, oxidative stress, oxidative damage to macromolecules (e.g., DNA, lipids)
6. Induces chronic inflammation	Elevated white blood cells, myeloperoxidase activity, altered cytokine and/or chemokine production
7. Is immunosuppressive	Decreased immunosurveillance, immune system dysfunction
8. Modulates receptor-mediated effects	Receptor in/activation (e.g., ER, PPAR, AhR) or modulation of endogenous ligands (including hormones)
9. Causes immortalization	Inhibition of senescence, cell transformation
10. Alters cell proliferation, cell death or nutrient supply	Increased proliferation, decreased apoptosis, changes in growth factors, energetics and signaling pathways related to cellular replication or cell cycle control, angiogenesis

Abbreviations: AhR, aryl hydrocarbon receptor; ER, estrogen receptor; PPAR, peroxisome proliferator-activated receptor. Any of the 10 characteristics in this table could interact with any other (e.g., oxidative stress, DNA damage, and chronic inflammation), which when combined provides stronger evidence for a cancer mechanism than would oxidative stress alone.

## 6.2 Oxidative Stress

### 6.2.1 Introduction

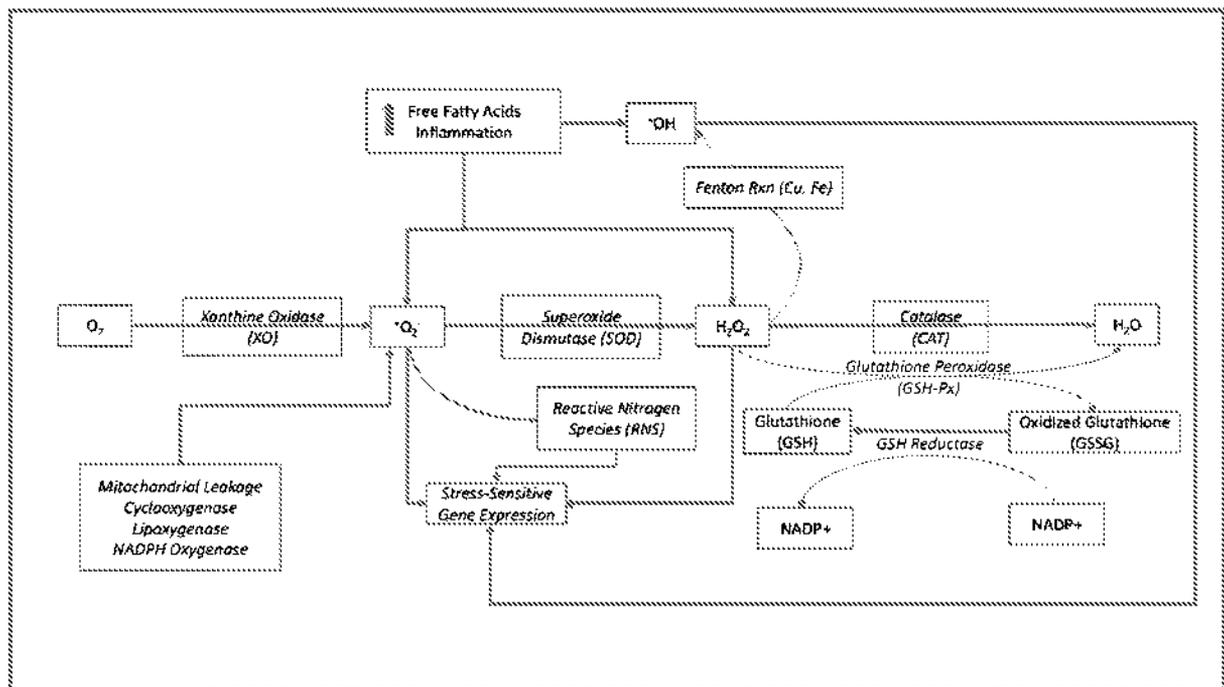
Oxidative stress refers to an imbalance between the production of reactive oxygen species (free radicals) in a cell and the antioxidant defenses the cell has in place to prevent this. Oxidative stress has been linked to both the causes and consequences of several diseases [221-226] including cancer [39, 227-231]. Multiple biomarkers exist for oxidative stress; the most common being increased antioxidant enzyme activity, depletion of glutathione or increases in lipid peroxidation. In addition, many studies evaluating oxidative stress used antioxidants following exposure to RF to demonstrate that the effect of the oxidative stress can be diminished.

Measuring oxidative stress can be difficult due to redundant pathways of a highly interconnected system. Molecular oxygen is essential to the proper function of a cell. During the course of normal oxidative phosphorylation, between 0.4 and 4% of all oxygen consumed is converted into the free radical superoxide ( $\text{O}_2^-$ ). This  $\text{O}_2^-$  can be converted into other ROS and reactive nitrogen species (RNS) and is normally eliminated by antioxidant defenses.  $\text{O}_2^-$  molecules are quickly converted to hydrogen peroxide ( $\text{H}_2\text{O}_2$ ) by superoxide dismutase (SOD).  $\text{H}_2\text{O}_2$  is then either detoxified to  $\text{H}_2\text{O}$  and  $\text{O}_2$  by glutathione peroxidase or diffuses into the cytosol and is detoxified by catalase. However, in the presence of reduced transition metals such as copper (Cu) or iron (Fe),  $\text{H}_2\text{O}_2$  can be converted to the highly reactive hydroxyl radical ( $\text{OH}^\bullet$ ). These linkages are illustrated in Figure 5.

The three reactive oxygen species (ROS) in the cell ( $\text{O}_2^-$ ,  $\text{OH}^\bullet$ ,  $\text{H}_2\text{O}_2$ ) can be measured directly, changes in the activity of the major enzymes (XO, SOD, CAT, GSH-Px, GSH

reductase) can be measured, changes in GSH or GSSG can be measured, changes in gene expression can be measured, changes in nitrogen oxide (NO) can be measured and changes in other enzymes (e.g. cyclooxygenase) can be measured. No one study measures all of these components. Most studies measure two or more components of this system in animals or cells exposed to RF to see if they have changed due to the RF exposure.

Figure 5. Exogenous and endogenous stimuli leading to ROS generation and activation of stress-sensitive gene expression. (modified from [232])



### 6.2.2 International Agency for Research on Cancer (IARC)

The IARC reviewed the potential for carcinogenicity from RF in 2011 [35]. They evaluated the scientific literature prior to 2011 and concluded “there was weak evidence that exposure to RF radiation affects oxidative stress and alters the levels of reactive oxygen species.” This conclusion was driven by methodological shortcomings in the studies, lack of a sham-controlled group in some studies, use of mobile phones for exposures and poor dosimetry. Having looked over the IARC review (I was an *Invited Specialist*<sup>4</sup> for this review), I agree with their assessment of these data and will not discuss any studies prior to 2010.

### 6.2.3 *In vivo* Studies in Mammals, 2011-2020

#### 6.2.3.1 Humans

Five studies evaluated the effects of RF on humans, two studies using blood, two using saliva and one using seminal plasma. Gulati et al. (2018) [232] compared 116 individuals in India living near cellular towers to 106 controls living more than 800 meters from towers. They saw significant decreases in SOD, CAT and a significant increase in lipid-peroxidation

<sup>4</sup> *Invited Specialists* are experts who have critical knowledge and experience but who also have a conflict of interest that warrants exclusion from developing or influencing the evaluations of carcinogenicity.

(LP) in plasma associated with being close to cellular towers. **Zothansiyama et al. (2017)** [233] studied 40 people living close to cellular towers (<80 meters) with people living further away (>300 meters) in a different population in India and measured RF power-density in the bedrooms of all of the participants. They saw the same changes in SOD, CAT and LP. In addition, increasing power-density measurements were associated with increased micronuclei (MN) in peripheral blood lymphocytes. **Khalil et al. (2014)** [234] and **Abu et al. (2015)** [235] reported on the same set of 12 individuals whose saliva was sampled before and after 15 and 30 minutes of use of a specific cellular phone (1800 MHz Nokia with an SAR of 1.09). They saw an increase in SOD, but no change in malondialdehyde (MDA) or 8-hydroxydeoxyguanosine (8-OHdG, a measure of oxidative damage). **Malini (2017)** [236] compared usage in 47 males in India in groupings of 1-5 hours/day (20 men), 5-10 hours/day (22 men) and >10 hours/day (5 men) and saw no changes in ROS, ROS scavengers or DNA damage in semen.

### 6.2.3.2 Mouse

In the discussion that follows, unless otherwise mentioned, SAR values used in the studies are generally less than 1 W/kg either whole body or tissue specific. Details can be found in Supplemental Table 1.

#### 6.2.3.2.1 BALB/c Mice

**Khalil et al. (2011)** [237] saw no changes in oxidative stress in brain, spleen or serum in BALB/c mice exposed for 30 days to 900 MHz RF at 1 W/kg SAR. **Bahreyni et al. (2018)** [238] saw changes in reactive oxygen species (ROS) and/or ROS-scavenging enzymes in heart, liver, kidney, cerebellum and hippocampus in the dams and heart, liver, kidney, and cerebellum of their offspring from pregnant female BALB/c mice exposed for 20 days to joint 900/1800 MHz RF for which the SAR was not provided.

#### 6.2.3.2.2 Parkes Mice

**Shahin et al. (2013)** [239] saw the expected changes in ROS and ROS-scavenging enzymes (SOD, CAT, GST) in the liver, kidney ovaries and blood of pregnant Parkes mice exposed for 45 days to 0.023 W/kg of 2450 MHz RF and saw associated DNA damage in the brains from the same exposure.

#### 6.2.3.2.3 Swiss Mice

**Shahin et al. (2014)** [240] saw an increase in ROS and associated changes in ROS scavengers in the hypothalamus, liver, kidney and testis of male Swiss mice exposed for 30 days to 0.018 W/kg 2450 MHz RF and saw significant tissue toxicity in the testis. **Shahin et al. (2017)** [241] also saw an increase in ROS and associated changes in ROS scavengers in the hypothalamus, uterus and ovaries of female Swiss mice exposed for 100 days to an unknown SAR from a 1800 MHz cellular phone. They also saw significant tissue changes in the uterus and a modification of reproductive hormones. **Shahin et al. (2018)** [242] saw changes in stress-related hormones and associated markers in the hippocampus and blood of male Swiss mice exposed for 15, 30 or 60 days to 0.0146 W/kg 2450 MHz RF. These stress changes, probably associated with induced nitrous oxide, led to reductions in learning and spatial memory in these mice. **Shahin et al. (2018)** [243] saw an increase in ROS and

associated changes in ROS scavengers, increased apoptosis, and tissue toxicity in the testis of male Swiss mice exposed for 120 days to 0.05 W/kg 1800 MHz (using a mobile phone).

**Pandey et al. (2017)** [244] saw mitochondrial damage, other cellular damage and DNA damage in spermatocytes of male Swiss mice exposed for 35 days to 0.0045-0.0056 W/kg 900 MHz RF; they attributed these changes to oxidative stress.

**Esmekaya et al. (2016)** [245] exposed Swiss mice with chemically-induced epileptic seizures (induced by pentylenetetrazole) for 15 or 30 minutes to a 900 MHz cellular phone with a head SAR of 0.301 W/kg and saw changes in ROS and ROS scavengers in the brain.

#### 6.2.3.2.4 ICR Mice

**Zong et al. (2016)** exposed male ICR mice for 7 days to 0.05 W/kg 900 MHz RF and saw no changes in ROS in liver, lung and blood. **Zong et al. (2015)** [246] exposed male mice to 0.05 W/kg 900 MHz RF for 4 hours/day for 7 days and saw no significant changes in ROS, ROS scavengers or DNA damage in liver, lung and blood.

#### 6.2.3.2.5 C57BL/6 Mice

**Jeong et al. (2018)** exposed 14-month-old female C57BL/6 mice for 8 months to 5 W/kg 1950 MHz RF and saw no changes in ROS, apoptosis or DNA damage in the brain and no change in locomotor activity.

#### 6.2.3.2.6 Summary in Mice

The best-studied strain of mouse is the Swiss-albino mouse and all studies using these mice demonstrated indications of oxidative stress induced by RF in multiple studies in the brain and testis and in single studies to the uterus, ovaries, liver and kidney at multiple frequencies and very low SARs. Three of the seven studies in Swiss mice used cellular phone exposure systems. In BALB/c mice, there is one negative study in brain, serum and spleen at 1 W/kg SAR, 900 MHz and 1 positive study in brain, heart, liver and kidney at 900/1800 MHz but an unknown SAR. One study in Parkes mice shows clear oxidative stress in liver, kidney and ovaries, DNA damage in the brain and changes in blood chemistry for a low SAR at 2450 MHz. In ICR mice, there is one study showing no changes in oxidative stress in liver, lung and blood at a low SAR at 900 MHz. Finally, in C57BL/6 mice, there is one study with no indication of oxidative stress in the brain at a much higher SAR at 1950 MHz.

In summary, RF can cause oxidative stress in the brain, testis, liver, kidney, uterus, heart and ovaries of Swiss-albino mice and the liver, kidney, ovaries and brain of ICR mice. There is insufficient data to support a causal linkage between RF exposure and oxidative stress in other strains of mice.

#### 6.2.3.3 Rats

In the discussion that follows, unless otherwise mentioned, SAR values used in the studies are generally less than 1 W/kg either whole body or tissue specific. Details can be found in Supplemental Table 1.

##### 6.2.3.3.1 Wistar Rats

There are 60 studies of RF in Wistar rats of which 35 used laboratory exposure systems

and 23 used cellular phones. These can be further divided by frequency and by organ to provide a summarized view of the findings. Fifteen (15) studies with laboratory exposure systems used 900-915 MHz RF, 1 used 1500 MHz, 11 used 1800 MHz, 4 used 2100 MHz, 18 used 2450 MHz, 1 used 2600 MHz and 1 used 2856 MHz (NOTE, this adds up to more than 33 studies because some studies used multiple frequencies). Seven (7) of the studies using cell phones or wifi devices used 900 MHz, 2 used cell phones with joint 900/1800 MHz, 2 used cell phones with joint 900/1800/1900 MHz, 1 used 1910.5 MHz, 3 used a 2450 MHz device, 1 used 2115 MHz and one used 2437 MHz.

All of the 8 studies in Wistar rats using laboratory systems at 900-915 MHz that evaluated oxidative stress in the brain showed changes in both ROS and ROS scavengers [247-254] with three examining and demonstrating tissue changes in the brain [250, 251, 253] (none examined DNA damage) and 2 examining and demonstrating behavioral changes [252, 253]. All 3 of the studies at only 900 MHz using a cellular phone showed changes in both ROS and ROS scavengers [255-257] with one examining and demonstrating tissue changes in the brain [256] but no significant change in DNA damage. One study at 1500 MHz showed decreases in SOD in the brain, changes in learning and spatial memory and brain tissue toxicity [258].

All of the 5 studies in Wistar rats using laboratory systems at 1800 MHz that evaluated oxidative stress in the brain showed changes in ROS and/or ROS scavengers [249-251, 259, 260] with three examining and demonstrating tissue changes in the brain [250, 251, 260] (none examined DNA damage). The one study at 900/1800 MHz using a cellular phone showed changes only in catalase activity with no other changes in either ROS or ROS scavengers [261] although they did see changes in animal behavior. Two studies in Wistar rats using laboratory systems at 2450 MHz that evaluated oxidative stress in the brain showed changes in ROS but not ROS scavengers [262, 263], one saw both change [254], one saw both change with brain toxicity [251], and one study showed no changes in ROS but used an unusual marker that appears to be focused entirely on nitrous oxides [264]. Two studies using 2450 MHz devices (wifi) were positive for both ROS and ROS scavengers with one showing changes in spatial memory from prenatal exposure [265] and the other not showing behavioral changes using adult exposure [266]. Studies were also clearly positive for the brain at 2100 MHz [267], 2115 MHz [268, 269] and 2856 MHz [258].

Sixteen (16) studies in Wistar rats looked at oxidative stress in the testis or sperm. Four (4) studies using laboratory-created 900 MHz saw changes in ROS and/or ROS scavengers (depending on what was measured) [270-273] and one saw changes in ROS but not ROS scavengers [274], two measured and demonstrated changes in tissue [272, 273] and one measured and demonstrated damage to DNA [272]. The two studies using 900 MHz cellular phones saw changes in ROS and ROS scavengers [275, 276] with one measuring and demonstrating both tissue damage and DNA damage [275]. One study with laboratory-generated 1800 MHz RF had no statistically significant change in ROS, but did see changes in ROS scavengers and apoptosis [277] and one study saw both ROS and ROS scavengers changed [271]. The one study using a 900/1800 MHz cellular phone saw changes in ROS and ROS scavengers and tissue toxicity [278]. One study with a combined 900//1800/1900 MHz cellular phone examined only ROS scavengers and saw changes and tissue toxicity [279]. The one study with a laboratory generated 2450 MHz signal saw changes in both ROS and ROS scavengers [271]. Single studies at 1950 MHz [280], 2100 MHz [281] and 2437 MHz

[282] saw changes to both ROS and ROS scavengers with two examining and demonstrating tissue toxicity [280, 282].

Heart tissue was examined in 4 studies. One, using 2450 MHz saw changes in ROS and ROS scavengers, tissue toxicity and apoptosis [283]. Another, also at 2450 MHz, saw changes in ROS and ROS scavengers, but not for all markers examined [284], and another at 2450 MHz saw changes in ROS but not ROS scavengers. The final study used laboratory generated 900 MHz and saw changes in ROS and ROS scavengers [270].

Liver tissue was examined in 7 studies in Wistar rats. Two studies using laboratory-created 900 MHz [249, 270] and one using a 900 MHz cellular phone [285] saw changes in ROS and ROS scavengers. One study at 1800 MHz saw changes in ROS and ROS scavengers [249] while another showed no significant changes [286]. The one study using laboratory-created 2450 MHz showed an increase in ROS and tissue toxicity but did not look for changes in ROS scavengers [287] and another using laboratory-created 2600 MHz saw no significant change in ROS or ROS scavengers but did see tissue changes [288]. The one study using 1910.5 MHz saw an increase in ROS (scavengers not evaluated) and increased DNA damage.

Kidney tissue was examined in 3 studies; two were positive for changes in both ROS and ROS scavengers, one using 2450 MHz [289] and the other examining the frequencies of 900, 1800 and 2450 MHz [271]. One study showed no change in ROS (ROS scavengers not examined) using 1800 MHz [286].

Three studies evaluated the effect of RF in the eye epithelium of Wistar rats and all were effectively negative [290-292].

One study using laboratory-generated 2450 MHz saw increased ROS in the spleen (ROS scavengers were not examined) [287]. One study using laboratory-generated 900 MHz saw changes in ROS and ROS scavengers in the lung [270]. The Laryngotracheal mucosa was examined in one study using 2450 MHz showing increased ROS but no significant change in ROS scavengers [293]. The ovary was examined in one study using 2450 MHz showing increased ROS (ROS scavengers were not examined) [294]. One study using the three frequencies 900, 1800 and 2450 MHz saw changes in ROS for all three frequencies but no significant changes in ROS scavengers [295] in uterus and blood. A single study using 900 MHz saw changes in ROS and ROS scavengers in lymphoid tissues and blood [296]. A cell phone at 900 MHz only was used for one study and at a combined 900/1800/1900 MHz phone for one other study. Finally, one study used a combined 848.5/1950 MHz signal that was laboratory generated.

#### 6.2.3.3.2 Sprague-Dawley Rats

There are 37 studies in Sprague-Dawley (SD) rats. Laboratory-generated RF at 900 MHz was used in 21 studies, 1800 MHz in 4 studies, 2100 MHz in 2 studies, and 2450 MHz in 5 studies [297-301].

Five studies evaluated oxidative stress in the brain using a laboratory-generated 900 MHz signal, and all of them demonstrated some degree of stress. Three studies demonstrated changes in both ROS and ROS scavengers [297, 299, 301] with 2 also demonstrating tissue changes in the brain [299, 301]. One study [298] saw no significant change in ROS but changes in ROS scavengers and tissue toxicity and one only examined a single ROS scavenger (significantly decreased) and saw changes in learning, spatial memory and the

blood-brain barrier. One study [302] using laboratory-generated 900, 1800 and 2100 MHz saw changes in ROS and ROS scavengers at all three frequencies in the brain and significant DNA damage at 2100 MHz. One last study [303] using laboratory-generated 2450 MHz RF saw changes in gene expression and protein levels in the brain linked to oxidative stress and tissue response.

Three studies [304-306] examined oxidative stress in the testis or sperm using a laboratory-generated 900 MHz signal with all showing changes to ROS and ROS scavengers and 2 examining and demonstrating tissue changes and increased apoptosis [304, 306]. One study using a 900 MHz cellular phone demonstrated changes in ROS, ROS scavengers, tissue toxicity and apoptosis [307], whereas another using a 900/1800/1900 MHz cellular phone failed to demonstrate any significant changes in ROS, ROS scavengers or tissue toxicity [308]. A single study using a laboratory-generated 2450 MHz signal with a moderate SAR (3.21 W/kg) demonstrated increases in ROS, decreases in ROS scavengers and increased tissue toxicity [309]. The final study evaluating oxidative stress in the testis used a combined 848.8/1950 MHz signal and a moderate SAR (4 W/kg) and failed to see any changes in ROS or tissue toxicity (ROS scavengers were not evaluated) [310].

Four studies examined oxidative stress in the kidney using laboratory-generated 900 MHz signals, 2 saw changes in ROS, ROS scavengers and tissue toxicity [299, 311], one saw increased ROS, tissue toxicity and apoptosis (ROS scavengers not examined) [312], and one saw no significant changes in ROS or ROS scavengers although they did see kidney toxicity [313]. One other study in the kidney used 2100 MHz and demonstrated changes in ROS, ROS scavengers, tissue toxicity and apoptosis [314]. **Turedi et al. (2017)** [312] also examined the bladder and saw clear changes in oxidative stress.

Four studies examined oxidative stress in the liver using laboratory-generated 900 MHz signals, 2 saw changes in ROS, ROS scavengers and tissue toxicity [299, 315], one saw increased ROS and decreased ROS scavengers (tissue toxicity not examined) [316], and one saw no significant changes in ROS, some changes in ROS scavengers and kidney toxicity [317]. One other study in the liver used 1800 MHz demonstrated changes in ROS, ROS scavengers and tissue toxicity [318].

Two studies looked at ovaries, one using 900 MHz [319] and one using 2450 MHz [320], saw changes in ROS and tissue toxicity but no changes in ROS scavengers. **Saygin et al. (2018)** [320] also looked at uterus and fallopian tubes and saw no significant changes in any oxidative stress markers.

Two studies in SD rats examined oxidative stress in the heart using laboratory-generated 900 MHz signals. One study, using in-utero exposure, saw clear increases in ROS and decreases in ROS scavengers with tissue toxicity and apoptosis [321]. The other study, using young rats, saw increased ROS, increased apoptosis, but no changes in ROS scavengers or in tissue toxicity [322].

Two studies in SD rats examined oxidative stress in the spinal cord using laboratory-generated 900 MHz signals with almost identical protocols. Both studies saw clear increases in ROS and weak or non-significant changes in ROS scavengers with tissue toxicity and apoptosis [323, 324]. One study using laboratory-generated RF looked at the sciatic nerve and saw changes in ROS and ROS scavengers, apoptosis and tissue toxicity [325].

Single studies evaluated the ear (increased ROS, no other changes) [326], pancreas (ROS, ROS scavengers and tissue changes) [327], spleen and thymus (ROS, ROS scavengers and tissue changes) [328] and eyes (ROS, ROS scavengers) [305].

#### 6.2.3 3.3 Other Rat Strains

Three studies examined RF oxidative stress in Fischer rats. One study used laboratory-generated signals at 900, 1800 and 2450 MHz and saw changes in ROS and ROS scavengers, DNA damage and inflammation in the brain [329]. A second study evaluated blood using a 900 MHz signal and saw changes in ROS and ROS scavengers in blood and changes in learning and spatial memory [330]. The final study used 900 and 1800 MHz signals and recorded changes in ROS, ROS scavengers, and tissue changes in the brain with associated learning and spatial memory deficits [331].

Two studies listed their rats as albino; these could have been Wistar rats. One study evaluated serum exposed to a 900 MHz laboratory-derived field and saw a decrease in ROS scavengers (ROS was not evaluated) [332]. The second examined parotid glands in rats exposed to a 900 MHz cellular phone and observed an increase in ROS and a decrease in ROS scavengers with associated tissue changes [333].

The only study in Long-Evans rats used a laboratory-generated 900 MHz signal and saw changes in stress hormones in the brain but no significant changes in learning or spatial memory [334].

One study appears to have used locally-caught wild rats, exposed them to a 2100 MHz mobile phone and demonstrated an increase in creatinine kinase-MB (indicator of oxidative stress in the heart) and a decrease in cardiomyocytes [335].

Four studies failed to identify the strain of rat [336-339].

#### 6.2.3 3.4 Summary in Rats

The best-studied strains of rat are the Wistar and SD rats and these show clear indications of oxidative stress induced by RF in multiple studies in the brain and testis and some indication of oxidative stress in the heart. The SD rats also seem to have consistent evidence of oxidative stress in the liver and kidney. Other findings in female reproductive organs, spinal cord, eye and other tissues are shown in 1 or 2 studies each. In other strains of rat, the most prominent findings are in the brain where there is generally increased oxidative stress. Most of these findings are at SARs below 1 W/kg and seem to occur regardless of the frequency used.

In summary, RF can cause oxidative stress in the brain, testis, and heart of SD and Wistar rats and the liver and kidney of SD rats. Brain appears to be a target for oxidative stress in Fischer rats. There is insufficient data to support a causal linkage between RF exposure and oxidative stress in other strains of rat.

#### 6.2.3.4 Other Laboratory Species

Three studies looked at the effects of RF on oxidative stress in New Zealand White rabbits. **Guler et al. (2016)** [340] used laboratory-generated 1800 MHz signals and saw increases in brain ROS (ROS scavengers were not examined) in male rabbits exposed both in-utero and

after birth but not in females. **Guler et al. (2012)** [341] used the same laboratory set up and study design and saw changes in liver ROS and ROS scavengers and an increase in 8-OHdG in females, but no direct DNA damage. **Ogur et al. (2013)** [342] in an earlier study used the same exposure and saw increased ROS in blood for males and females with in-utero exposure and for females (not males) with exposure 1 month after birth. This same research group had done an earlier study with a similar design and saw no significant changes in blood [343].

One study examined laboratory-generated 900 MHz signals in Guinea pigs and saw a reduction in ROS scavengers in the liver but no significant change in ROS.

There is insufficient data to support a causal linkage between RF exposure and oxidative stress in laboratory species other than rats and mice.

#### 6.2.4 *In Vitro* Studies in Mammalian Cells

##### 6.2.4.1 Human Cells

###### 6.2.4.1.1 Primary Cells

*In vitro* studies in primary cells refer to the use of cells taken directly from humans, then exposed in a laboratory to RF where oxidative stress is evaluated. Three studies exposed human sperm to RF and evaluated oxidative stress. Using a 900 MHz mobile phone led to changes in ROS (ROS scavengers not examined) and DNA damage [344]. Using a laboratory-generated 1950 MHz signal resulted in no significant changes in ROS [345]. Using a 2450 MHz cellular phone resulted in clear oxidative stress with changes in both ROS and ROS scavengers [346].

Three studies used peripheral blood. Monocytes showed changes in ROS, ROS scavengers and apoptosis after being exposed to a laboratory-generated 900 MHz signal [347]. In another study, monocytes, but not lymphocytes, saw an increase in ROS (ROS scavengers not evaluated) after exposure to a laboratory derived 900 MHz signal [348]. The third study, both monocytes and lymphocytes exposed to a laboratory-derived 1800 MHz signal showed changes in ROS scavengers (ROS was not directly measured) [349]. A single study used umbilical cord blood exposed using a 900 MHz cellular phone resulting in an increase in ROS [350].

A single study used astrocytes from human brains exposed to 918 MHz RF and saw a decrease in ROS (ROS scavengers not examined) [351] (Note, this study was aimed at RF as a therapy for Alzheimer's).

Human stem cells exposed to 900, 1950 or 2535 MHz RF saw no significant changes in ROS apoptosis or DNA damage except for DNA damage that was shown at 900 MHz [352].

One study used primary cells from human skin, umbilical veins and amniotic fluid and saw no increase in ROS, saw binucleated nuclei in skin but no DNA damage via comet assay [353]

The final study of human primary cells used thyroid gland cells exposed to 900 or 895 MHz RF and saw no significant increase in oxidative stress [354].

Three (3) of these studies used SAR above 1 W/kg.

#### 6.2.4.1.2 HEK293 Embryonic Kidney Cell Line

Two studies using the same basic design of 1 hour exposure to 2450 MHz RF saw a significant change in ROS and ROS scavengers [355, 356]. The only other study used a 940 MHz signal and also resulted in significant change in ROS and ROS scavengers [357].

#### 6.2.4.1.3 HL-60 Leukemia Cell Line

Two studies, one at 900 MHz [358] and the other at 2450 MHz [359] both demonstrated increases in ROS and changes in ROS scavengers. The 900 MHz study [358] also saw damage to mitochondrial DNA. Finally, HL-60 cells exposed to 900, 1950 or 2535 MHz RF saw no significant changes in ROS or apoptosis [352]. Only 1 study used SARs above 1 W/kg.

#### 6.2.4.1.4 SH-SY5Y Human Neuroblastoma Cell Line

Two studies, one with 935 MHz [360] and the other with 1800 MHz [361], saw no changes in oxidative stress. Two studies, one with 837 and 1950 MHz [362] and the other with 1800 MHz wifi device [363], saw changes in ROS only (changes in ROS scavengers were not evaluated). Finally, two studies, one with 935 MHz [364] and the other with 1800 MHz [365], saw changes in both ROS and ROS scavengers. Five of these studies used SARs greater than 1 W/kg.

#### 6.2.4.1.5 Other Human Cell Lines

Studies in ACS cells (adipose tissue), Huh7 cells (liver), and U87 cells (glioma) all studied only ROS and demonstrated a significant increase in ROS [362, 366]. Studies in U-87 MG cells (glioma), MCF-7 cells (breast cancer), MDA-MB-231 cells (breast cancer) and HLE B3 cells (lens epithelium) studied a full spectrum of ROS and ROS scavengers and saw significant indications of oxidative stress [361, 362, 367-369]. A single study in MCF10A cells (breast) saw no increase in ROS or ROS scavengers [370].

### 6.2.4.2 Cells Derived From Mice

#### 6.2.4.2.1 Primary Cells

One study in Leydig cells saw changes in ROS and ROS scavengers after exposure to RF [371]. Another study of preantral follicles (ovaries) also saw changes in ROS and ROS scavengers after exposure to RF [372]. A study of spermatocytes saw an increase in ROS associated with an increase in DNA damage [373].

#### 6.2.4.2.2 NIH/3T3 Mouse Embryonic Fibroblast Cells

Three studies used NIH/3T3 cells. All three saw increases in ROS but did not study ROS scavengers [362, 374, 375] with two also showing an increase in apoptosis [374, 375].

#### 6.2.4.2.3 GC1 and GC2 Mouse Spermatocyte Cell Lines

Four studies evaluated the effects of RF on mouse-derived spermatocyte cell line GC1 and/or GC2. All four saw increases in ROS [373, 376-378], 2 of these showed increases in DNA damage [376, 377], 2 saw increases in 8-OHdG [373, 377] and one saw an increase in apoptosis [378].

#### 6.2.4.2.4 N9 Mouse Microglia Cells

Two studies in N9 cells saw significant changes in ROS and ROS scavengers [364, 379] and one study demonstrated an increase in NO [380].

#### 6.2.4.2.5 Other Mouse Cell Lines

One study with Neuro-2A cells (neuroblastoma) saw an increase in ROS (did not study ROS scavengers), but no significant change in DNA damage [381]. Two studies in the same laboratory evaluated RF and HT22 cells (hippocampus), neither study evaluated ROS scavengers, one saw a significant increase in ROS and a change in cell cycle [382] while the other with lower SAR values and two frequencies combined saw no significant change in ROS [383]. One study in RAW 264.7 cells (macrophage) saw an increase in ROS but did not study ROS scavengers [384]. Finally, one study using TM3 cells (leydig) saw changes in ROS and ROS scavengers but no change in apoptosis [385].

#### 6.2.4.3 Cells Derived from Rats

Two studies used rat primary cells from the brain. One saw a decrease in ROS (scavengers not evaluated) in astrocytes when exposed to 918 MHz RF and challenged with hydrogen peroxide [351]. One study of rat neonatal spinal ganglia and neurons exposed to 1800 MHz RF saw an increase in ROS but no DNA damage [386].

One additional study used PC12 cells (rat derived pheochromocytoma cell line) exposed simultaneously to 837 MHz and 1950 MHz RF saw significant increased ROS at 12 hours but not at other times in a 24-hour window.

#### 6.2.4.4 Cells Derived from Hamsters

Two studies exposed V79 cells (hamster lung cells) to 1800 MHz with one seeing increased ROS (nothing else studied) [387] and the other showing increased ROS and ROS scavenger activity [388]. A final study using CHO cells (ovaries) exposed to 900 MHz saw increased ROS (scavengers not evaluated) that remained 12 hours after exposure stopped [389].

#### 6.2.5 Summary for Oxidative Stress

Most of the in-vivo and in-vitro studies of oxidative stress saw significant increases in ROS. Most of the studies that evaluated ROS scavengers saw significant changes in these markers that is associated with oxidative stress, the tissue or cells. Nineteen (19) in-vivo studies, 18 done in rats or mice and one in rabbits, evaluated oxidative stress as well as DNA damage, about half with SARs below 1 and a mix of exposure durations and almost all of them showed an increase in DNA damage.

Although reactive oxygen species can potentially cause damage to cellular function and structure and thereby impair its functionality, their presence and production cannot be immediately considered as harmful because changes in the levels of ROS and ROS scavengers is a normal part of cellular metabolism and physiology. Thus, many of the studies in this section simply demonstrate a change and not necessarily harm. However, tissue toxicity, increased DNA damage and changes in apoptosis do indicate that the changes in ROS are sufficient to impair cellular function and damage cellular components.

Many of the studies presented in this section did address these issues. With respect to cancer, of greatest concern would be damage to DNA. Twelve (12) of these in-vivo studies showed an increase in DNA damage associated with oxidative stress [239, 244, 256, 268, 272, 275, 302, 329, 338, 390-392], seven (7) did not see a significant change in DNA damage [236, 246, 256, 337, 341, 393, 394] and one saw a significant decrease in DNA damage after 15 days of exposure and an increase after 30 days of exposure [336]. Eight (8) in-vitro studies evaluated some aspect of oxidative stress as well as DNA damage, all of them with rather short exposure periods and most with SARs greater than 1. Five (5) of these studies demonstrated increases in DNA damage [344, 346, 352, 376, 377] and three (3) saw no significant increase in DNA damage [353, 381, 386].

There is sufficient evidence in the literature to conclude that oxidative stress is a possible mechanism by which RF causes cancer in humans.

## 6.3 Genotoxicity

### 6.3.1 Introduction

Genotoxicity refers to the ability of an agent (chemical or otherwise) to damage the genetic material within a cell, thus increasing the risks for a mutation. Genotoxic agents interact with the genetic material, including DNA sequence and structure, to damage cells. DNA damage can occur in several different ways, including single- and double-strand breaks, cross-links between DNA bases and proteins, formation of micronuclei and chemical additions to the DNA.

Just because a chemical can damage DNA does not mean it will cause mutations. So, while all chemicals that cause mutations are genotoxic, all genotoxic chemicals are not necessarily mutagens. Does that mean that the genotoxicity of a chemical can be ignored if all assays used for identifying mutations in cells following exposure to a chemical are negative? The answer to that question is no and is tied to the limitations in tests for mutagenicity (the ability of a chemical to cause mutations in a cell). It is unusual to see an evaluation of the sequence of the entire genome before exposure with the same sequence after exposure to determine if the genome has been altered (mutation). There are assays that can evaluate a critical set of genes that have previously been associated with cancer outcomes (e.g. cancer oncogenes), but these are seldom applied. In general, mutagenicity tests are limited in the numbers of genes they actually screen and the manner in which these screens work.

Because screening for mutagenicity is limited in scope, any genetic damage caused by chemicals should raise concerns because of the possibility of a mutation arising from that genetic damage. In what follows, the scientific findings available for evaluating the genotoxic potential of RF will be divided into four separate sources of data based on the biological source of that data: (1) data from exposed humans, (2) data from exposed human cells in a laboratory setting, (3) data from exposed mammals (non-human), and (4) data from exposed cells of mammals (non-human) in the laboratory. These four areas are based upon the priorities one would apply to the data in terms of impacts. Seeing genotoxicity in humans is more important than seeing genotoxicity in other mammals. In addition, seeing genotoxicity in whole, living organisms (*in vivo*) carries greater weight than seeing responses in cells in the laboratory (*in vitro*). Basically, the closer the findings are to real, living human beings, the more weight they should be given.

### 6.3.2 International Agency for Research on Cancer (IARC)

The IARC reviewed the potential for carcinogenicity from RF in 2011 [35]. They evaluated the scientific literature prior to 2011 and concluded “*there was weak evidence that RF radiation is genotoxic, and no evidence for the mutagenicity of RF radiation.*” This conclusion was driven by methodological shortcomings in the studies, lack of a sham-controlled group in some studies, use of mobile phones for exposures, poor dosimetry and contradictory results. Having looked over the IARC review, I agree with their assessment of these data and will not discuss any studies prior to 2010.

### 6.3.3 *In Vivo* Studies in Mammals

#### 6.3.3.1 Humans

Several studies have addressed the presence of DNA damage directly in humans using the duration or frequency of cellular phone usage and comparing easily obtained human tissues (e.g. buccal swabs, sperm/semens, peripheral blood). **Vanishree et al. (2018)** [395] examined buccal swabs from 86 18-30 year-old cell phone users (46 M, 40 F) for micronuclei (MN). They compared low mobile phone users (<5 years and <4-5 hr/week) to high mobile phone users (>5 years and more than 10 hr/week) and saw an increase in MN in the high exposure group. They also saw an increase in MN on the side of the mouth where the mobile phone is used (ipsilateral) and in those who failed to use a headphone. **de Oliveira et al. (2017)** [396] examined buccal swabs from 30 male and 30 female 20-28 year-old cell phone users for MN. They saw no increase in MN by duration of use, frequency of use or ipsilateral vs. contralateral exposure. The categories for duration of use were unbalanced and they found no relationship with smoking (which is a known risk factor). **Gulati et al. (2016)** [397] examined buccal swabs from 116 people (68 M, 48F) residing near mobile towers (not defined but Table 1 suggests ≤400 meters) to 106 people living >800 meters from mobile towers (age range not provided). They found an increase in MN in buccal cells associated with distance to the cell tower and duration of use but saw no association with tobacco use. **Bannerjee et al. (2016)** [398] examined buccal swabs from 300 male 20-30 year-old cell phone users for MN. They compared low mobile phone users (<5 years and <3 hr/week) to high mobile phone users (>5 years and more than 10 hr/week). They saw an increase in MN in the high exposure group, an increase in MN on the ipsilateral side and in those who failed to use a headphone; they did not adjust for other risk factors. **Daroit et al. (2015)** [399] examined oral mucosa swabs from 3 different regions of the mouth of 60 people (24 M, 36 F) aged 19-33 years for MN and other genetic damage markers (broken eggs, binucleated cells, karyorrhexis). They saw increased MN on the whole mucosa and lower lip and increased binucleated cells (BN) on the border of the tongue for those using cellular phones for >60 minutes per week and increased broken eggs (BE) on the border of the tongue for those using cell phones for >8 years; all other comparisons were non-significant and no other risk factors were evaluated. **Sousa et al. (2014)** [400] examined ipsilateral-only oral mucosa cells in three groups (> 5 hr/week, >1 and ≤5 hr/week, ≤ 1 hr/week) of 15 individuals (sexes not specified) for the presence of MN, BE and degenerative nuclear anomalies (DN). They saw no changes in MN or DN but did see an increase in BE as a function of duration of usage per week (no other risk factors were examined). **Ros-Lior et al. (2012)** [401] examined buccal swabs from 50 (16 M, 34 F)

Caucasian 20-40 year-old cell phone users for MN. They compared short-term mobile phone users (<10 years) to long-term mobile phone users (>10 years). They saw no increase in MN, BN or DN in the long-term users nor did they see any relationship to ipsilateral use; they did not adjust for other risk factors and saw no relationship with smoking.

**Radwan et al. (2016)** [402] studied the effect of stress on sperm DNA damage in 286 males. They saw no indication of an increase in DNA fragmentation in sperm as a function of years of cell phone use ( $\leq 5$ ,  $> 5$  to  $\leq 10$ ,  $> 10$  years). In an earlier study from the same group using 344 men (286 in the 2016 study are included here) **Jurewicz et al. (2014)** [403] had a similar finding.

**Gulati et al. (2016)** [397] also examined peripheral blood lymphocytes (PBL) from 116 people (68 M, 48F) residing near mobile towers (not defined but Table 1 suggests  $\leq 400$  meters) to 106 people living  $> 800$  meters from mobile towers (age range not provided). They found an increase in tail moment (TM) (comet assay) associated with distance to the cell tower and duration of use but saw no association with tobacco use. **Gandhi et al. (2015)** [404] used the comet assay to evaluate DNA damage in PBL from 63 (38 M, 25 F) people with residences near (50-300 meters) a mobile phone tower and 28 controls (15 M, 13 F) with no nearby towers at home or work. All evaluations of DNA damage regarding distance to towers as well as mobile phone usage were significantly higher in the high exposure categories.

**Cam and Seyhan (2012)** [405] examined the hair roots of 8 individuals (6 women, 2 men) before and after 15 minutes exposure to a cellular phone and then 2 weeks later, before and after exposure for 30 minutes to a cellular phone. The comet assay showed a clear increase in single strand breaks after both 15 and 30 minutes of use with 30 minutes of use showing the greatest amount of damage.

#### 6.3.3.2 Mice

In the NTP Study [166] using B6C3F1 mice, after 14 weeks of exposure, **Smith-Roe et al (2020)** [168] evaluated genotoxicity in several tissues of mice included in these studies for this purpose using the alkaline comet assay (three brain regions, liver, peripheral blood) and the micronucleus assay (peripheral blood). Significant increases in DNA damage were seen in the frontal cortex of male mice (DCMA and GSM) and leukocytes of female mice (CDMA only).

**Jiang et al. (2013)** [406] exposed groups of 10 male ICR mice to 900 MHz RF, SAR 0.548 W/kg, for 4 hr/day for 7 days and examined for MN in erythrocytes and bone marrow. They saw no significant changes in MN in either tissue, however, they did not use a sham control group. **Jiang et al. (2012)** [407] exposed groups of 5 male ICR mice to 900 MHz RF, SAR 0.548 W/kg, for 4 hr/day for 1,3,5,7 or 14 days and examined for general DNA damage (comet assay) in leukocytes. They saw no significant changes for any duration of exposure, however, they also did not use a sham control.

**Chaturvedi et al. (2011)** [408] exposed groups of 5 male Parks mice to 2450 MHz, SAR 0.0356 W/kg RF for 2 hr/day for 5 days. They saw an increase in tail moment, tail DNA and tail length in brain tissue using the comet assay.

#### 6.3.3.3 Rats

In the NTP Study [166] using Sprague-Dawley rats, after 14 weeks of exposure, **Smith-Roe et al (2020)** [168] evaluated genotoxicity in several tissues of rats included in these studies for this purpose using the alkaline comet assay (three brain regions, liver, peripheral blood) and the micronucleus assay (peripheral blood). Significant increases in DNA damage were seen in the hippocampus of male rats (CDMA-only). **Usikalu et al. (2013)** [409] exposed groups of 2 male and 2 female Sprague-Dawley rats to 2450 MHz RF at SARs of 0, and 2.39 W/kg for 10 minutes and evaluated the induction of DNA damage by comet assay in the ovaries (F) and testis (M). Both tissues showed a significant increase in DNA damage as a function of exposure.

**Akdag et al. (2016)** [410] exposed groups of 8 male Wistar rats to 2450 MHz RF for 24 hr/day for 12 months at SARs of 0 or  $1.41 \cdot 10^{-4}$  W/kg. Using the comet assay, they examined DNA damage in the brain, liver, kidney and testis and only saw increased DNA damage in the testis. **Gurburz et al. (2014)** [411] exposed groups of 6 male Wistar rats to 1800 MHz, SAR 0.23 or 2100 MHz, SAR 0.23 for 1 or 2 months. They examined only the urinary bladder and saw no increases in MN. **Atli et al. (2013)** [412] exposed groups of 2-week old and 10-week old Wistar rats (sex not provided) to 900 MHz RF, SAR 0.76 (2-week old) or 0.37 (10-week old) W/kg for 2 hr/day, 45 days with and without a recovery period of 15 days. Significant DNA damage (chromosomal aberrations, MN, and polychromatic erythrocytes) in bone marrow was seen for all of the experimental groups. Using the same experimental design with 1800 MHz RF, SAR 0.37 (2-week) and 0.49 (10-week), **Sekeroglu et al. (2012)** [413] saw the same significant DNA damage. **Trosic et al. (2011)** [414] exposed groups of 9 male Wistar rats to 915 MHz RF, SAR 0.6 W/kg, for 1 hr/day, 7 d/week, 2 weeks. They saw increases in DNA damage (comet assay) in liver and kidney, but not in brain.

**Gouda et al. (2013)** [415] exposed groups of 15 male albino (probably Wistar) rats to 1800 MHz RF, SAR 0.3 W/kg, from a cellular phone for 2 h/day either continuous or discontinuous (30 min on, 30 min off) for 2, 4 or 6 weeks. Using genomic DNA from the liver, they saw a significant increase in mutations to two genes (TP53 and BRCA1) after 6 weeks of exposure in the continuous group and a significant increase in DNA fragmentation at all durations for continuous exposure.

In a series of 3 studies, **Deshmukh et al. (2013, 2015, 2016)** exposed groups of 6 male Fischer rats to 900 MHz RF, SAR  $5.95 \cdot 10^{-4}$  W/kg, 1800 MHz RF,  $5.83 \cdot 10^{-4}$  W/kg, or 2450 MHz RF,  $6.67 \cdot 10^{-4}$  W/kg, for 2 h/day, 5 d/week, 30 days [416], 90 days [417] or 180 days [418]. Increases in DNA damage in the brain in the 30-day study and hippocampus in the other two studies were seen using the comet assay.

#### 6.3.3.4 Summary for DNA Damage In-Vivo

DNA damage was seen from exposure to RF in humans (5 studies of oral mucosa cells, 2 in PBL and 1 in hair follicles), mice (2 studies) and in rats (8 studies). Four studies in humans (2 oral mucosa cells, 2 sperm cells), 2 studies in mice which failed to use sham controls, and 1 study in rats saw no increases in DNA damage. In laboratory animals, 2 studies at 900 MHz saw no DNA damage while 6 were positive, one study using 1800 and 2100 MHz RF was negative while 5 using 1800 MHz were positive and all 6 studies using 2450 MHz were positive. In humans, most studies failed to control for confounders and failed to find an

association with smoking that should have been apparent. The strongest study, using hair follicles, used the individuals as their own control and this study was positive.

#### 6.3.4 *In Vitro* Studies in Mammalian Cells

##### 6.3.4.1 *Humans*

###### 6.3.4.1.1 Primary Cells

Five studies exposed human PBL to RF. One study using laboratory-generated 900 MHz for 30 minutes with 60 minutes recovery saw no change in DNA repair [419]. One multi-laboratory study using laboratory-generated 1800 MHz RF for 28 hours saw no changes in MN, sister-chromatid exchange, chromosomal aberrations or comet assay tail moment [420]. Two studies with laboratory-generated 1950 MHz RF and 20 or 24-hr exposure with a 28-hr recovery saw no changes in micronuclei [421, 422]. One study with laboratory-generated 2450 MHz RF for 72 hr and a high SAR (10.9 W/kg) saw no change in MN or binucleated DNA [423].

Both studies using semen/sperm, one using an 850 MHz phone for 60 minutes and the other using a 900/1800 MHz phone for 1 to 5 hours saw an increased DNA fragmentation index.

The final human primary cell study using amniotic cells exposed to 900 MHz RF for 24 hours at 4 different SAR values and saw no change in aneuploidy in chromosomes 1 and 17.

###### 6.3.4.1.2 Human Cell Lines

One study using SH-SY5Y neuroblastoma cells exposed to laboratory-generated 1950 MHz RF for 20 hours saw no change in tail behavior using the comet assay [424]. In contrast, a second study using the same cell line and exposure for 16 hours saw a non-significant increased tail length in the comet assay for not only SH-SY5Y cells, but also U87, U251 and U373 glioma cells and NCH421K glioblastoma cells [425]. They also observed an increase in DNA repair but no change in double strand breaks. Another study using A172 and U251 glioblastoma cells and SH-SY5Y neuroblastoma cells using 1800 MHz for 1, 6 or 24 hours saw no increase in DNA repair [426].

Two studies used HepG2 liver cells, one at 1950 MHz for 16 hours exposure saw no changes [425] while the other using 900 or 1800 MHz RF for 1-4 hours saw morphological changes in DNA at 4 hours [427].

One study used HMy2.CIR lymphoblastoma cells exposed to laboratory-generated 1800 MHz RF for 24 hours and observed changes in DNA repair proteins [428].

A study in HL-60 leukemia cells exposed to laboratory-generated 1800 MHz RF for 24 hours saw no changes in MN or DNA damage via the comet assay [429].

One study in HaCat skin cells exposed to 900 MHz RF for 30 minutes with a 4 or 24 hour recovery saw no change in MN [430].

Two studies in human/hamster AL hybrid ovary cells exposed to 900 MHz RF for 30 minutes saw different responses; one saw aberrant spindles [431] and the other saw no changes in MN but waited at least 4 hours after exposure before evaluation [430].

### 6.3.4.2 Mouse

#### 6.3.4.2.1 Mouse Primary Cells

Three studies from the same laboratory exposed bone marrow cells extracted from bone marrow stromal cells from male Kummung mice and exposed them to 900 MHz RF. In the first study, the cells were exposed for 3 hours/day for 5 days and poly(ADP-ribose) polymerase-1 mRNA expression (*PARP-1*) was shown to be significantly elevated for 10 hours after the final exposure (this is an indication of breaks in strands of DNA) [432]. The second study exposed the cells for 4 hr/day for 5 days, allowed the cells to recover for 4 hours and then, after measuring DNA damage (comet assay,  $\gamma$ -H2AX foci) saw no differences between sham controls and the RF-exposed cells [433]. The final study exposed cells for 3 hours/day for 5 days, had a three-hour recovery then measured DNA damage (comet assay, *PARP-1*) and found a large, time-dependent change in both measures but did not provide statistical p-values [434].

Another study used oocytes and spermatozoa from B6D2F<sub>1</sub> mice, exposed for 60 minutes to 1950 MHz RF, combined to allow fertilization, and then allowed 17 to 20 hours to recover. They saw no chromosomal aberrations in the resulting one-cell embryos [435].

#### 6.3.4.2.2 Mouse Cell Lines

One study exposed GC-2 mouse spermatocyte cells to 1800 MHz RF for 24 hours at SARs of 1, 2 and 4 W/kg and saw an increase in DNA damage (comet assay, 4 W/kg) but no change in DNA double strand breaks ( $\gamma$ -H2AX foci) [436]. A second study exposed GC-2 cells to a 900 MHz cellular phone signal for 24 hours to four different modes of cell phone use and saw DNA damage (comet assay) for three of the modes [437].

One study exposed ataxia telangiectasia mutated (*Atm*<sup>-/-</sup>) and *Atm*<sup>+/+</sup> mouse embryonic fibroblast cells to 1800 MHz RF for 1 to 36 hours, SAR 4 W/kg, and saw increased DNA damage (comet assay) and DNA fragmentation in the *Atm*<sup>-/-</sup> cells at multiple times [438].

### 6.3.4.3 Rat

#### 6.3.4.3.1 Primary Cells

One study exposed astrocytes extracted from Wistar rats to 872 MHz RF, SAR 0.6 or 6 W/kg, for 24 hours and saw no significant increase in micronuclei or DNA damage (comet assay) [439].

One study exposed femur and tibia lymphocytes extracted from Sprague-Dawley rats to 900 MHz RF for 30 minutes and saw no significant increase in DNA damage (comet assay) [440].

#### 6.3.4.3.2 Rat Cell Lines

One study exposed PC12 rat pheochromocytoma cells to 1950 MHz, SAR 10 W/kg, for 24 hours and saw no significant DNA damage (comet assay) [441].

### 6.3.4.4 Hamster

#### 6.3.4.4.1 Primary Cells

There were no studies of hamster primary cells.

#### 6.3.4.4.2 Hamster Cell Lines

One study using V79 hamster lung fibroblast cells exposed to laboratory-generated 2450 MHz RF for 15 minutes saw an increase in aberrant spindles and apoptosis [442]. Another study using V79 cells exposed to 1950 MHz RF for 20 hours, SAR 0.15, 0.3, 0.6 and 1.25 W/kg, saw an increase in micronuclei at the two lowest SAR values [443].

#### 6.3.4.4 Summary for DNA Damage In-Vitro

About half of the in-vitro studies showed some form of DNA damage and about half demonstrated no significant effects. There was no pattern by cell type, species, SAR or frequency. Very few of the studies used the same cell and frequency so it is difficult to give greater weight to the positive findings or the negative findings.

#### 6.3.5 Summary for Genotoxicity

In addition to the many studies cited above and in the IARC Monograph [35], Lai (2021) [444] has compiled literature on other genetic effects (e.g. changes in gene expression) and downstream changes (e.g. cell-cycle arrest) that also point toward RF having an impact on cellular genetics and their control of cellular function.

A majority of the *in vivo* studies evaluating genotoxicity and RF, either with oxidative stress or independent of evaluating oxidative stress, showed a significant increase in DNA damage. In contrast, only about half of the *in vitro* studies of genotoxicity and RF were positive with no obvious pattern of why this might have happened.

Overall, there is sufficient evidence to suggest that genotoxicity, probably due to oxidative stress, is caused by RF and could be a mechanism by which cancer is induced by RF.

### 6.3. Summary for Mechanisms of Carcinogenicity

There is sufficient evidence to suggest that both oxidative stress and genotoxicity are caused by exposure to RF and that these mechanisms could be the reason why RF can induce cancer in humans.

There is the possibility of publication bias in this body of literature on mechanism. Publication bias occurs when studies that are positive tend to get published whereas negative studies are either never submitted for publication or they are rejected because they are negative (rejection is less of a problem since journals are now very aware of problems with publication bias). This potential problem cannot be resolved with the data in hand. There is also a possible bias in these results based upon a small collection of laboratories providing a majority of the studies; this could also create a small amount of bias in the direction of the positive results since scientists seldom pursue negative findings but will generally continue to pursue reasons for positive findings.

## 7. Summary of Bradford Hill Evaluation

***RF exposure probably causes gliomas and acoustic neuromas and, given the human, animal and experimental evidence, I assert that, to a reasonable degree of scientific certainty, the probability that RF exposure causes these cancers is high.***

Table 22 summarizes the information for each of Hill’s aspects of causality. For these data, causality is strengthened because the available epidemiological studies show a **consistent positive association** between brain tumors and RF exposure. Analyzed collectively with meta-analyses using the most reasonable combinations of studies show positive responses. And, in answer to Hill’s question, the relationship between brain tumors and RF exposure has been observed by different persons, in different places, circumstances, and times. Using meningiomas as controls in some case-control studies suggests recall bias is minimal.

Causality is strengthened for these data because **the strength of the observed associations**, when evaluated simultaneously in meta-analyses, are statistically significant and the results are unlikely to be due to chance. Even though only one of the individual studies provides odds ratios that are large and precise, the meta-analyses have objectively shown that the observed association across these studies is significant and supports a positive association between brain tumors and RF.

**Biological plausibility** is strongly supported by the animal carcinogenicity data and the mechanistic data on genotoxicity and oxidative stress. When addressing biological plausibility, the first question generally asked is “Can you show that RF causes cancers in experimental animals?” In this case, the answer to that question is clearly yes. RF can cause tumors in experimental animals with strong findings for gliomas, heart Schwannomas and adrenal pheochromocytomas in male rats and harderian gland tumors in male mice and uterine polyps in female mice. There is also some evidence supporting liver tumors and lung tumors in male and possibly female mice. Thus, it is biologically plausible that RF can cause cancer in mammals.

The next question generally asked is “Does the mechanism by which RF causes cancer in experimental animals also work in humans?” The best understood mechanism by which agents cause cancer in both humans and animals is through damaging DNA that leads to mutations in cells that then leads to uncontrolled cellular replication and eventually cancer. It is absolutely clear from the available scientific data that RF causes oxidative stress in humans and experimental mammals. This has been amply demonstrated in humans that were exposed to RF, in human cells *in vitro*, and in experimental animal models and their cells *in vitro* and *in vivo*. One possible consequence of oxidative stress is damage to DNA and potentially mutations. RF induces DNA damage as measured in multiple ways, in humans, animals and cells, providing additional support for a biological mechanism that works in humans.

Table 22: Summary conclusions for Hill’s nine aspects of epidemiological data and related science

Aspect	Conclusion	Reason
Consistency of the observed association	Strong	Multiple studies, many are positive, meta-analyses with little heterogeneity show positive findings at higher exposures, different research teams, different continents, different questionnaires, no obvious bias in case-control studies, no obvious confounding, laterality is significant
Strength of the observed association	Strong	Significant meta-analyses

Biological plausibility	Very Strong	Multiple cancers in multiple species, same tumors as humans in male rats, not due to chance, increased risk of rare tumors, convincing evidence for genotoxicity and oxidative stress
Biological gradient	Strong	Clearly seen in some case-control studies, clearly seen in the meta-analyses and meta-regressions, not seen in the cohort studies, clearly seen in animal studies
Temporal relationship of the observed association	Satisfied	Exposure clearly came before cancers
Specificity of the observed association	Strong	The only cancers linked to RF exposure are gliomas and acoustic neuromas
Coherence	Strong	Cancers seen in the rats have strong similarity to human gliomas and acoustic neuromas, laterality and brain location support coherence
Evidence from human experimentation	No data	No studies are available
Analogy	No data	No studies available in the literature

In general, there is support that a **biological gradient** exists for the epidemiological data and thus support from this aspect of the Bradford-Hill evaluation. RF mRRs increased with duration of cellular phone use and with cumulative hours of exposure when studies are combined in both meta-analyses and meta-regressions. In addition, laterality is strengthened when duration of use of a cellular phone increases. The animal studies clearly demonstrate dose-response.

The proper **temporal relationship** exists with the exposure coming before the cancers.

The human evidence is **coherent**. The cancer findings in humans agree with the cancer findings in rats. Also, studies focused on the temporal lobe appear to support this area as a target for cellular phone usage. Finally, laterality, when evaluated in meta-analyses shows that tumors are more closely associated with the predominant side of the head used by people with their cellular phones.

Glioma and acoustic neuroma are not **specific** to RF exposure; however, RF exposure is specific to these two tumors. There is no **experimental evidence** in humans and I did not find any references where researchers looked for analogous exposures with similar toxicity.

**Hill (1965)**[34] asks *“is there any other way of explaining the set of facts before us, is there any other answer equally, or more, likely than cause and effect?”* There is no better way of explaining the scientific evidence relating RF exposure to an increase in gliomas and acoustic neuromas in humans than cause and effect.

**In my opinion, RF exposure probably causes gliomas and neuromas and, given the human, animal and experimental evidence, I assert that, to a reasonable degree of scientific certainty, the probability that RF exposure causes gliomas and neuromas is high.**

## 8. References Cited

1. Portier C, Hoel D: **Optimal design of the chronic animal bioassay.** *J Toxicol Environ Health* 1983, **12**(1):1-19.
2. Portier CJ, Hoel DG: **Design of the Chronic Animal Bioassay for Goodness of Fit to Multistage Models.** *Biometrics* 1983, **39**(3):809-809.
3. Bailer AJ, Portier CJ: **Effects of treatment-induced mortality and tumor-induced mortality on tests for carcinogenicity in small samples.** *Biometrics* 1988, **44**(2):417-431.
4. Portier CJ, Bailer AJ: **Testing for increased carcinogenicity using a survival-adjusted quantal response test.** *Fundam Appl Toxicol* 1989, **12**(4):731-737.
5. Portier CJ, Hedges JC, Hoel DG: **Age-specific models of mortality and tumor onset for historical control animals in the National Toxicology Program's carcinogenicity experiments.** *Cancer Res* 1986, **46**(9):4372-4378.
6. Portier CJ, Bailer AJ: **2-Stage Models of Tumor-Incidence for Historical Control Animals in the National Toxicology Programs Carcinogenicity Experiments.** *Journal of Toxicology and Environmental Health* 1989, **27**(1):21-45.
7. Portier CJ, Edler L: **Two-stage models of carcinogenesis, classification of agents, and design of experiments.** *Fundam Appl Toxicol* 1990, **14**(3):444-460.
8. Portier CJ, Hoel DG, Kaplan NL, Kopp A: **Biologically based models for risk assessment.** *IARC scientific publications* 1990(104):20-28.
9. Kopp-Schneider A, Portier CJ: **Distinguishing between models of carcinogenesis: the role of clonal expansion.** *Fundam Appl Toxicol* 1991, **17**(3):601-613.
10. Kopp-Schneider A, Portier CJ, Rippmann F: **The application of a multistage model that incorporates DNA damage and repair to the analysis of initiation/promotion experiments.** *Math Biosci* 1991, **105**(2):139-166.
11. Kopp-Schneider A, Portier CJ, Rippmann F: **The Application of a Multistage Model That Incorporates DNA Damage and Repair to the Analysis of Initiation Promotion Experiments.** *Mathematical Biosciences* 1991, **105**(2):139-166.
12. Kopp-Schneider A, Portier CJ: **Birth and Death Differentiation Rates of Papillomas in Mouse Skin.** *Carcinogenesis* 1992, **13**(6):973-978.
13. Portier CJ, Kopp-Schneider A, Sherman CD: **Using Cell Replication Data in Mathematical-Modeling in Carcinogenesis.** *Environmental Health Perspectives* 1993, **101**:79-86.
14. Kopp-Schneider A, Portier CJ, Sherman CD: **The Exact Formula for Tumor-Incidence in the 2-Stage Model.** *Risk Analysis* 1994, **14**(6):1079-1080.
15. Portier C, Kohn M, Sherman CD, Lucier G: **Modeling the number and size of hepatic focal lesions following exposure to 2378-TCDD.** *Organohalogen Compounds* 1994, **21**:393-397.

16. Kopp-Schneider A, Portier CJ: **Carcinoma formation in NMRI mouse skin painting studies is a process suggesting greater than two stages.** *Carcinogenesis* 1995, **16**(1):53-59.
17. Portier CJ, Kopp-Schneider A, Sherman CD: **Calculating tumor incidence rates in stochastic models of carcinogenesis.** *Mathematical Biosciences* 1996, **135**(2):129-146.
18. Portier CJ, Sherman CD, Kohn M, Edler L, Kopp-Schneider A, Maronpot RM, Lucier G: **Modeling the number and size of hepatic focal lesions following exposure to 2,3,7,8-TCDD.** *Toxicology and Applied Pharmacology* 1996, **138**(1):20-30.
19. Kopp-Schneider A, Portier C, Bannasch P: **A model for hepatocarcinogenesis treating phenotypical changes in focal hepatocellular lesions as epigenetic events.** *Math Biosci* 1998, **148**(2):181-204.
20. Toyoshiba H, Sone H, Yamanaka T, Parham FM, Irwin RD, Boorman GA, Portier CJ: **Gene interaction network analysis suggests differences between high and low doses of acetaminophen.** *Toxicol Appl Pharmacol* 2006, **215**(3):306-316.
21. Gohlke JM, Portier CJ: **The forest for the trees: a systems approach to human health research.** *Environ Health Perspect* 2007, **115**(9):1261-1263.
22. Gohlke JM, Thomas R, Zhang Y, Rosenstein MC, Davis AP, Murphy C, Becker KG, Mattingly CJ, Portier CJ: **Genetic and environmental pathways to complex diseases.** *BMC Syst Biol* 2009, **3**:46.
23. Gohlke JM, Thomas R, Woodward A, Campbell-Lendrum D, Pruss-Ustun A, Hales S, Portier CJ: **Estimating the global public health implications of electricity and coal consumption.** *Environ Health Perspect* 2011, **119**(6):821-826.
24. Aylward LL, Kirman CR, Schoeny R, Portier CJ, Hays SM: **Evaluation of biomonitoring data from the CDC National Exposure Report in a risk assessment context: perspectives across chemicals.** *Environ Health Perspect* 2013, **121**(3):287-294.
25. Trong I, Portier C: **Proceedings of the Viet Nam – United States Scientific Conferences on Human Health and Environmental Effects of Agent Orange/Dioxin, Part 1 and 2.** In: *2002; Ha Noi, Vietnam*: US National Institute of Environmental Sciences and the Government of Vietnam; 2002: 1100.
26. Portier CJ, Wolfe MS (eds.): **EMF Science Review Symposium Breakout Group Report for Epidemiology Research Findings.** Research Triangle Park, North Carolina: National Institute of Environmental Health Sciences; 1998.
27. Portier CJ, Wolfe MS (eds.): **EMF Science Review Symposium Breakout Group Report for Clinical and *In Vivo* Laboratory Findings.** Research Triangle Park, North Carolina: National Institute of Environmental Health Sciences; 1998.
28. Portier CJ, Wolfe MS (eds.): **Assessment of Health Effects from Exposure to Power-Line Frequency Electric and Magnetic Fields.** Research Triangle Park, North Carolina: National Institute of Environmental Health Sciences; 1998.
29. Portier CJ, Thigpen Tart K, Carter S, Dilworth CH, Grambsch A, Gohlke JM, Hess J, Howard SN, Lubber G, Lutz JT *et al*: **A Human Health Perspective on Climate Change.**

- In. Edited by Health and Human Services NIOEHS. Research Triangle Park, NC: Environmental Health Perspectives; 2010: 72.
30. **Preamble to the IARC Monographs** [<http://monographs.iarc.fr/ENG/Preamble/CurrentPreamble.pdf>]
  31. European Chemicals Agency: **Guidance on the Application of the CLP Criteria: Guidance to Regulation (EC) No 1272/2008 on classification, labelling and packaging (CLP) of substances and mixtures**. In. Edited by European Chemicals Agency. Helsinki, Finland: European Chemicals Agency; 2015.
  32. **Preamble to the IARC Monographs** [<https://monographs.iarc.fr/wp-content/uploads/2019/01/Preamble-2019.pdf>]
  33. USEPA: **Guidelines for Carcinogen Risk Assessment**. In. Edited by US Environmental Protection Agency. Washington DC; 2005: 166.
  34. Hill AB: **The Environment and Disease: Association or Causation?** *Proc R Soc Med* 1965, **58**:295-300.
  35. Humans IWGotEoCRt: **Non-ionizing radiation, Part 2: Radiofrequency electromagnetic fields**. *IARC monographs on the evaluation of carcinogenic risks to humans / World Health Organization, International Agency for Research on Cancer* 2013, **102**(Pt 2):1-460.
  36. National Research Council. Washington (DC): National Academy of Sciences Press; 1994.
  37. National Research Council. Washington (DC): National Academies Press; 2009.
  38. **Handbook for Preparing Report on Carcinogens Monographs** [[https://ntp.niehs.nih.gov/ntp/roc/handbook/roc\\_handbook\\_508.pdf](https://ntp.niehs.nih.gov/ntp/roc/handbook/roc_handbook_508.pdf)]
  39. Smith MT, Guyton KZ, Gibbons CF, Fritz JM, Portier CJ, Rusyn I, DeMarini DM, Caldwell JC, Kavlock RJ, Lambert PF *et al*: **Key Characteristics of Carcinogens as a Basis for Organizing Data on Mechanisms of Carcinogenesis**. *Environ Health Perspect* 2016, **124**(6):713-721.
  40. Muscat JE, Malkin MG, Thompson S, Shore RE, Stellman SD, McRee D, Neugut AI, Wynder EL: **Handheld cellular telephone use and risk of brain cancer**. *JAMA* 2000, **284**(23):3001-3007.
  41. Rothman KJ, Loughlin JE, Funch DP, Dreyer NA: **Overall mortality of cellular telephone customers**. *Epidemiology* 1996, **7**(3):303-305.
  42. Rothman KJ, Chou CK, Morgan R, Balzano Q, Guy AW, Funch DP, Preston-Martin S, Mandel J, Steffens R, Carlo G: **Assessment of cellular telephone and other radio frequency exposure for epidemiologic research**. *Epidemiology* 1996, **7**(3):291-298.
  43. Funch DP, Rothman KJ, Loughlin JE, Dreyer NA: **Utility of telephone company records for epidemiologic studies of cellular telephones**. *Epidemiology* 1996, **7**(3):299-302.
  44. Inskip PD, Tarone RE, Hatch EE, Wilcosky TC, Shapiro WR, Selker RG, Fine HA, Black PM, Loeffler JS, Linet MS: **Cellular-telephone use and brain tumors**. *N Engl J Med* 2001, **344**(2):79-86.

45. Auvinen A, Hietanen M, Luukkonen R, Koskela RS: **Brain tumors and salivary gland cancers among cellular telephone users.** *Epidemiology* 2002, **13**(3):356-359.
46. Gousias K, Markou M, Voulgaris S, Goussia A, Voulgari P, Bai M, Polyzoidis K, Kyritsis A, Alamanos Y: **Descriptive epidemiology of cerebral gliomas in northwest Greece and study of potential predisposing factors, 2005-2007.** *Neuroepidemiology* 2009, **33**(2):89-95.
47. Spinelli V, Chinot O, Cabaniols C, Giorgi R, Alla P, Lehucher-Michel MP: **Occupational and environmental risk factors for brain cancer: a pilot case-control study in France.** *Presse medicale* 2010, **39**(2):e35-44.
48. Interphone Study Group: **Brain tumour risk in relation to mobile telephone use: results of the INTERPHONE international case-control study.** *Int J Epidemiol* 2010, **39**(3):675-694.
49. Christensen HC, Schuz J, Kosteljanetz M, Poulsen HS, Boice JD, Jr., McLaughlin JK, Johansen C: **Cellular telephones and risk for brain tumors: a population-based, incident case-control study.** *Neurology* 2005, **64**(7):1189-1195.
50. Christensen HC, Schuz J, Kosteljanetz M, Poulsen HS, Thomsen J, Johansen C: **Cellular telephone use and risk of acoustic neuroma.** *Am J Epidemiol* 2004, **159**(3):277-283.
51. Hartikka H, Heinavaara S, Mantyla R, Kahara V, Kurttio P, Auvinen A: **Mobile phone use and location of glioma: a case-case analysis.** *Bioelectromagnetics* 2009, **30**(3):176-182.
52. Hepworth SJ, Schoemaker MJ, Muir KR, Swerdlow AJ, van Tongeren MJ, McKinney PA: **Mobile phone use and risk of glioma in adults: case-control study.** *Bmj* 2006, **332**(7546):883-887.
53. Hours M, Bernard M, Montestrucq L, Arslan M, Bergeret A, Deltour I, Cardis E: **[Cell Phones and Risk of brain and acoustic nerve tumours: the French INTERPHONE case-control study].** *Rev Epidemiol Sante Publique* 2007, **55**(5):321-332.
54. Klæboe L, Blaasaas KG, Tynes T: **Use of mobile phones in Norway and risk of intracranial tumours.** *European journal of cancer prevention : the official journal of the European Cancer Prevention Organisation* 2007, **16**(2):158-164.
55. Lonn S, Ahlbom A, Hall P, Feychting M, Swedish Interphone Study G: **Long-term mobile phone use and brain tumor risk.** *Am J Epidemiol* 2005, **161**(6):526-535.
56. Sadetzki S, Chetrit A, Jarus-Hakak A, Cardis E, Deutch Y, Duvdevani S, Zultan A, Novikov I, Freedman L, Wolf M: **Cellular phone use and risk of benign and malignant parotid gland tumors--a nationwide case-control study.** *Am J Epidemiol* 2008, **167**(4):457-467.
57. Schlehofer B, Schläefer K, Blettner M, Berg G, Bohler E, Hettlinger I, Kunna-Grass K, Wahrendorf J, Schuz J, Interphone Study G: **Environmental risk factors for sporadic acoustic neuroma (Interphone Study Group, Germany).** *Eur J Cancer* 2007, **43**(11):1741-1747.
58. Schoemaker MJ, Swerdlow AJ, Ahlbom A, Auvinen A, Blaasaas KG, Cardis E, Christensen HC, Feychting M, Hepworth SJ, Johansen C *et al*: **Mobile phone use and**

- risk of acoustic neuroma: results of the Interphone case-control study in five North European countries.** *Br J Cancer* 2005, **93**(7):842-848.
59. Schuz J, Bohler E, Berg G, Schlehofer B, Hettinger I, Schlaefer K, Wahrendorf J, Kunna-Grass K, Blettner M: **Cellular phones, cordless phones, and the risks of glioma and meningioma (Interphone Study Group, Germany).** *Am J Epidemiol* 2006, **163**(6):512-520.
  60. Takebayashi T, Akiba S, Kikuchi Y, Taki M, Wake K, Watanabe S, Yamaguchi N: **Mobile phone use and acoustic neuroma risk in Japan.** *Occup Environ Med* 2006, **63**(12):802-807.
  61. Takebayashi T, Varsier N, Kikuchi Y, Wake K, Taki M, Watanabe S, Akiba S, Yamaguchi N: **Mobile phone use, exposure to radiofrequency electromagnetic field, and brain tumour: A case-control study.** *British Journal of Cancer* 2008, **98**(3):652-659.
  62. Schuz J, Bohler E, Schlehofer B, Berg G, Schlaefer K, Hettinger I, Kunna-Grass K, Wahrendorf J, Blettner M: **Radiofrequency electromagnetic fields emitted from base stations of DECT cordless phones and the risk of glioma and meningioma (Interphone Study Group, Germany).** *Radiat Res* 2006, **166**(1 Pt 1):116-119.
  63. Cardis E, Armstrong BK, Bowman JD, Giles GG, Hours M, Krewski D, McBride M, Parent ME, Sadetzki S, Woodward A *et al*: **Risk of brain tumours in relation to estimated RF dose from mobile phones: results from five Interphone countries.** *Occup Environ Med* 2011, **68**(9):631-640.
  64. Lahkola A, Auvinen A, Raitanen J, Schoemaker MJ, Christensen HC, Feychting M, Johansen C, Klaeboe L, Lonn S, Swerdlow AJ *et al*: **Mobile phone use and risk of glioma in 5 North European countries.** *Int J Cancer* 2007, **120**(8):1769-1775.
  65. Lahkola A, Salminen T, Raitanen J, Heinavaara S, Schoemaker MJ, Christensen HC, Feychting M, Johansen C, Klaeboe L, Lonn S *et al*: **Meningioma and mobile phone use--a collaborative case-control study in five North European countries.** *Int J Epidemiol* 2008, **37**(6):1304-1313.
  66. Turner MC, Krewski D, Armstrong BK, Chetrit A, Giles GG, Hours M, McBride ML, Parent ME, Sadetzki S, Siemiatycki J *et al*: **Allergy and brain tumors in the INTERPHONE study: pooled results from Australia, Canada, France, Israel, and New Zealand.** *Cancer Causes Control* 2013, **24**(5):949-960.
  67. Interphone Study Group: **Acoustic neuroma risk in relation to mobile telephone use: results of the INTERPHONE international case-control study.** *Cancer Epidemiol* 2011, **35**(5):453-464.
  68. Berg G, Schuz J, Samkange-Zeeb F, Blettner M: **Assessment of radiofrequency exposure from cellular telephone daily use in an epidemiological study: German Validation study of the international case-control study of cancers of the brain--INTERPHONE-Study.** *J Expo Anal Environ Epidemiol* 2005, **15**(3):217-224.
  69. Lahkola A, Salminen T, Auvinen A: **Selection bias due to differential participation in a case-control study of mobile phone use and brain tumors.** *Ann Epidemiol* 2005, **15**(5):321-325.

70. Samkange-Zeeb F, Berg G, Blettner M: **Validation of self-reported cellular phone use.** *J Expo Anal Environ Epidemiol* 2004, **14**(3):245-248.
71. Vrijheid M, Armstrong BK, Bedard D, Brown J, Deltour I, Iavarone I, Krewski D, Lagorio S, Moore S, Richardson L *et al*: **Recall bias in the assessment of exposure to mobile phones.** *Journal of exposure science & environmental epidemiology* 2009, **19**(4):369-381.
72. Vrijheid M, Deltour I, Krewski D, Sanchez M, Cardis E: **The effects of recall errors and of selection bias in epidemiologic studies of mobile phone use and cancer risk.** *Journal of exposure science & environmental epidemiology* 2006, **16**(4):371-384.
73. Vrijheid M, Mann S, Vecchia P, Wiart J, Taki M, Ardoino L, Armstrong BK, Auvinen A, Bedard D, Berg-Beckhoff G *et al*: **Determinants of mobile phone output power in a multinational study: implications for exposure assessment.** *Occup Environ Med* 2009, **66**(10):664-671.
74. Vrijheid M, Richardson L, Armstrong BK, Auvinen A, Berg G, Carroll M, Chetrit A, Deltour I, Feychting M, Giles GG *et al*: **Quantifying the impact of selection bias caused by nonparticipation in a case-control study of mobile phone use.** *Ann Epidemiol* 2009, **19**(1):33-41.
75. Vrijheid M, Cardis E, Armstrong BK, Auvinen A, Berg G, Blaasaas KG, Brown J, Carroll M, Chetrit A, Christensen HC *et al*: **Validation of short term recall of mobile phone use for the Interphone study.** *Occup Environ Med* 2006, **63**(4):237-243.
76. Grell K, Frederiksen K, Schuz J, Cardis E, Armstrong B, Siemiatycki J, Krewski DR, McBride ML, Johansen C, Auvinen A *et al*: **The Intracranial Distribution of Gliomas in Relation to Exposure From Mobile Phones: Analyses From the INTERPHONE Study.** *Am J Epidemiol* 2016, **184**(11):818-828.
77. Grell K, Diggle PJ, Frederiksen K, Schuz J, Cardis E, Andersen PK: **A three-dimensional point process model for the spatial distribution of disease occurrence in relation to an exposure source.** *Stat Med* 2015, **34**(23):3170-3180.
78. Cardis E, Deltour I, Mann S, Moissonnier M, Taki M, Varsier N, Wake K, Wiart J: **Distribution of RF energy emitted by mobile phones in anatomical structures of the brain.** *Phys Med Biol* 2008, **53**(11):2771-2783.
79. Larjavaara S, Schuz J, Swerdlow A, Feychting M, Johansen C, Lagorio S, Tynes T, Klæboe L, Tonjer SR, Blettner M *et al*: **Location of gliomas in relation to mobile telephone use: a case-case and case-specular analysis.** *Am J Epidemiol* 2011, **174**(1):2-11.
80. Hardell L, Carlberg M, Hansson Mild K: **Mobile phone use and the risk for malignant brain tumors: a case-control study on deceased cases and controls.** *Neuroepidemiology* 2010, **35**(2):109-114.
81. Hardell L, Carlberg M, Mild KH: **Case-control study of the association between the use of cellular and cordless telephones and malignant brain tumors diagnosed during 2000-2003.** *Environ Res* 2006, **100**(2):232-241.

82. Hardell L, Carlberg M, Soderqvist F, Mild KH: **Case-control study of the association between malignant brain tumours diagnosed between 2007 and 2009 and mobile and cordless phone use.** *Int J Oncol* 2013, **43**(6):1833-1845.
83. Hardell L, Hansson Mild K, Carlberg M: **Case-control study on the use of cellular and cordless phones and the risk for malignant brain tumours.** *Int J Radiat Biol* 2002, **78**(10):931-936.
84. Hardell L, Hansson Mild K, Carlberg M: **Further aspects on cellular and cordless telephones and brain tumours.** *Int J Oncol* 2003, **22**(2):399-407.
85. Hardell L, Nasman A, Pahlson A, Hallquist A, Hansson Mild K: **Use of cellular telephones and the risk for brain tumours: A case-control study.** *Int J Oncol* 1999, **15**(1):113-116.
86. Hardell L, Carlberg M, Hansson Mild K: **Pooled analysis of two case-control studies on use of cellular and cordless telephones and the risk for malignant brain tumours diagnosed in 1997-2003.** *Int Arch Occup Environ Health* 2006, **79**(8):630-639.
87. Hardell L, Carlberg M: **Use of mobile and cordless phones and survival of patients with glioma.** *Neuroepidemiology* 2013, **40**(2):101-108.
88. Hardell L, Carlberg M: **Mobile phone and cordless phone use and the risk for glioma - Analysis of pooled case-control studies in Sweden, 1997-2003 and 2007-2009.** *Pathophysiology* 2015, **22**(1):1-13.
89. Baldi I, Coureau G, Jaffre A, Gruber A, Ducamp S, Provost D, Lebailly P, Vital A, Loiseau H, Salamon R: **Occupational and residential exposure to electromagnetic fields and risk of brain tumors in adults: a case-control study in Gironde, France.** *Int J Cancer* 2011, **129**(6):1477-1484.
90. Coureau G, Bouvier G, Lebailly P, Fabbro-Peray P, Gruber A, Leffondre K, Guillamo JS, Loiseau H, Mathoulin-Pelissier S, Salamon R *et al*: **Mobile phone use and brain tumours in the CERENAT case-control study.** *Occup Environ Med* 2014, **71**(7):514-522.
91. Hardell L, Carlberg M: **Re: mobile phone use and brain tumours in the CERENAT case--control study.** *Occup Environ Med* 2015, **72**(1):79.
92. Coureau G, Leffondre K, Gruber A, Bouvier G, Baldi I: **Author's response: re 'mobile phone use and brain tumours in the CERENAT case-control study'.** *Occup Environ Med* 2015, **72**(1):79-80.
93. Yoon S, Choi JW, Lee E, An H, Choi HD, Kim N: **Mobile phone use and risk of glioma: a case-control study in Korea for 2002-2007.** *Environ Health Toxicol* 2015, **30**:e2015015.
94. Schuz J, Jacobsen R, Olsen JH, Boice JD, Jr., McLaughlin JK, Johansen C: **Cellular telephone use and cancer risk: update of a nationwide Danish cohort.** *J Natl Cancer Inst* 2006, **98**(23):1707-1713.
95. Johansen C, Boice J, Jr., McLaughlin J, Olsen J: **Cellular telephones and cancer--a nationwide cohort study in Denmark.** *J Natl Cancer Inst* 2001, **93**(3):203-207.

96. Frei P, Poulsen AH, Johansen C, Olsen JH, Steding-Jessen M, Schuz J: **Use of mobile phones and risk of brain tumours: update of Danish cohort study.** *Bmj* 2011, **343**:d6387.
97. Dalton SO, Schuz J, Johansen C, Engholm G, Kjaer SK, Steding-Jessen M, Storm HH, Olsen JH: **[Social inequality and incidence of and survival from cancer in Denmark--secondary publication].** *Ugeskr Laeger* 2010, **172**(9):691-696.
98. Schuz J, Waldemar G, Olsen JH, Johansen C: **Risks for central nervous system diseases among mobile phone subscribers: a Danish retrospective cohort study.** *PLoS One* 2009, **4**(2):e4389.
99. Schuz J, Steding-Jessen M, Hansen S, Stangerup SE, Caye-Thomasen P, Poulsen AH, Olsen JH, Johansen C: **Long-term mobile phone use and the risk of vestibular schwannoma: a Danish nationwide cohort study.** *Am J Epidemiol* 2011, **174**(4):416-422.
100. **Mobile cellular subscriptions (per 100 people) - Denmark**  
[<https://data.worldbank.org/indicator/IT.CEL.SETS.P2?locations=DK>]
101. **Population, total - Denmark**  
[<https://data.worldbank.org/indicator/SP.POP.TOTL?locations=DK>]
102. Benson VS, Pirie K, Schuz J, Reeves GK, Beral V, Green J, Million Women Study C: **Mobile phone use and risk of brain neoplasms and other cancers: prospective study.** *Int J Epidemiol* 2013, **42**(3):792-802.
103. Benson VS, Pirie K, Green J, Bull D, Casabonne D, Reeves GK, Beral V, Million Women Study C: **Hormone replacement therapy and incidence of central nervous system tumours in the Million Women Study.** *Int J Cancer* 2010, **127**(7):1692-1698.
104. Beral V, Million Women Study C: **Breast cancer and hormone-replacement therapy in the Million Women Study.** *Lancet* 2003, **362**(9382):419-427.
105. de Vocht F: **The case of acoustic neuroma: Comment on mobile phone use and risk of brain neoplasms and other cancers.** *Int J Epidemiol* 2014, **43**(1):273-274.
106. Benson VS, Pirie K, Schuz J, Reeves GK, Beral V, Green J: **Authors' response to: the case of acoustic neuroma: comment on mobile phone use and risk of brain neoplasms and other cancers.** *Int J Epidemiol* 2014, **43**(1):275.
107. **Mobile cellular subscriptions (per 100 people) - United Kingdom**  
[<https://data.worldbank.org/indicator/IT.CEL.SETS.P2?locations=GB>]
108. Elliott P, Toledano MB, Bennett J, Beale L, de Hoogh K, Best N, Briggs DJ: **Mobile phone base stations and early childhood cancers: case-control study.** *Bmj* 2010, **340**:c3077.
109. Aydin D, Feychting M, Schuz J, Tynes T, Andersen TV, Schmidt LS, Poulsen AH, Johansen C, Prochazka M, Lannering B et al: **Mobile phone use and brain tumors in children and adolescents: a multicenter case-control study.** *J Natl Cancer Inst* 2011, **103**(16):1264-1276.
110. Aydin D, Feychting M, Schuz J, Andersen TV, Poulsen AH, Prochazka M, Klæboe L, Kuehni CE, Tynes T, Roosli M: **Impact of random and systematic recall errors and**

- selection bias in case–control studies on mobile phone use and brain tumors in adolescents (CEFALO study). *Bioelectromagnetics* 2011, **32**(5):396-407.
111. Morgan LL, Herberman RB, Philips A, Lee Davis D: **Re: Mobile phone use and brain tumors in children and adolescents: a multicenter case-control study.** *J Natl Cancer Inst* 2012, **104**(8):635-637; author reply 637-638.
  112. Soderqvist F, Carlberg M, Hansson Mild K, Hardell L: **Childhood brain tumour risk and its association with wireless phones: a commentary.** *Environ Health* 2011, **10**:106.
  113. Li CY, Liu CC, Chang YH, Chou LP, Ko MC: **A population-based case-control study of radiofrequency exposure in relation to childhood neoplasm.** *The Science of the total environment* 2012, **435-436**:472-478.
  114. Feltbower RG, Fleming SJ, Picton SV, Alston RD, Morgan D, Achilles J, McKinney PA, Birch JM: **UK case control study of brain tumours in children, teenagers and young adults: a pilot study.** *BMC research notes* 2014, **7**:14.
  115. **Mobile cellular subscriptions (per 100 people) - Global**  
[<https://data.worldbank.org/indicator/IT.CEL.SV.SV.P2>]
  116. Momoli F, Siemiatycki J, McBride ML, Parent ME, Richardson L, Bedard D, Platt R, Vrijheid M, Cardis E, Krewski D: **Probabilistic Multiple-Bias Modeling Applied to the Canadian Data From the Interphone Study of Mobile Phone Use and Risk of Glioma, Meningioma, Acoustic Neuroma, and Parotid Gland Tumors.** *Am J Epidemiol* 2017, **186**(7):885-893.
  117. Hardell L, Hallquist A, Mild KH, Carlberg M, Pahlson A, Lilja A: **Cellular and cordless telephones and the risk for brain tumours.** *European journal of cancer prevention : the official journal of the European Cancer Prevention Organisation* 2002, **11**(4):377-386.
  118. Roosli M, Lagorio S, Schoemaker MJ, Schuz J, Feychting M: **Brain and Salivary Gland Tumors and Mobile Phone Use: Evaluating the Evidence from Various Epidemiological Study Designs.** *Annu Rev Public Health* 2019, **40**:221-238.
  119. Wang P, Hou C, Li Y, Zhou D: **Wireless Phone Use and Risk of Adult Glioma: Evidence from a Meta-Analysis.** *World Neurosurg* 2018, **115**:e629-e636.
  120. Yang M, Guo W, Yang C, Tang J, Huang Q, Feng S, Jiang A, Xu X, Jiang G: **Mobile phone use and glioma risk: A systematic review and meta-analysis.** *PLoS One* 2017, **12**(5):e0175136.
  121. Wang Y, Guo X: **Meta-analysis of association between mobile phone use and glioma risk.** *J Cancer Res Ther* 2016, **12**(Supplement):C298-C300.
  122. Myung SK, Ju W, McDonnell DD, Lee YJ, Kazinets G, Cheng CT, Moskowitz JM: **Mobile phone use and risk of tumors: a meta-analysis.** *Journal of clinical oncology : official journal of the American Society of Clinical Oncology* 2009, **27**(33):5565-5572.
  123. Kan P, Simonsen SE, Lyon JL, Kestle JR: **Cellular phone use and brain tumor: a meta-analysis.** *Journal of neuro-oncology* 2008, **86**(1):71-78.

124. Hardell L, Carlberg M, Soderqvist F, Hansson Mild K: **Meta-analysis of long-term mobile phone use and the association with brain tumours.** *Int J Oncol* 2008, **32**(5):1097-1103.
125. Lahkola A, Tokola K, Auvinen A: **Meta-analysis of mobile phone use and intracranial tumors.** *Scandinavian journal of work, environment & health* 2006, **32**(3):171-177.
126. Hardell L, Carlberg M, Hansson Mild K: **Pooled analysis of case-control studies on malignant brain tumours and the use of mobile and cordless phones including living and deceased subjects.** *Int J Oncol* 2011, **38**(5):1465-1474.
127. Hardell L, Carlberg M, Hansson Mild K: **Use of mobile phones and cordless phones is associated with increased risk for glioma and acoustic neuroma.** *Pathophysiology* 2013, **20**(2):85-110.
128. Hardell L, Carlberg M, Hansson Mild K: **Re-analysis of risk for glioma in relation to mobile telephone use: comparison with the results of the Interphone international case-control study.** *Int J Epidemiol* 2011, **40**(4):1126-1128.
129. Deltour I, Johansen C, Auvinen A, Feychting M, Klaeboe L, Schuz J: **Time trends in brain tumor incidence rates in Denmark, Finland, Norway, and Sweden, 1974-2003.** *J Natl Cancer Inst* 2009, **101**(24):1721-1724.
130. Lonn S, Klaeboe L, Hall P, Mathiesen T, Auvinen A, Christensen HC, Johansen C, Salminen T, Tynes T, Feychting M: **Incidence trends of adult primary intracerebral tumors in four Nordic countries.** *Int J Cancer* 2004, **108**(3):450-455.
131. Inskip PD, Hoover RN, Devesa SS: **Brain cancer incidence trends in relation to cellular telephone use in the United States.** *Neuro Oncol* 2010, **12**(11):1147-1151.
132. de Vocht F, Burstyn I, Cherrie JW: **Time trends (1998-2007) in brain cancer incidence rates in relation to mobile phone use in England.** *Bioelectromagnetics* 2011, **32**(5):334-339.
133. Ding LX, Wang YX: **Increasing incidence of brain and nervous tumours in urban Shanghai, China, 1983-2007.** *Asian Pac J Cancer Prev* 2011, **12**(12):3319-3322.
134. Aydin D, Feychting M, Schuz J, Roosli M, team Cs: **Childhood brain tumours and use of mobile phones: comparison of a case-control study with incidence data.** *Environ Health* 2012, **11**:35.
135. Deltour I, Auvinen A, Feychting M, Johansen C, Klaeboe L, Sankila R, Schuz J: **Mobile phone use and incidence of glioma in the Nordic countries 1979-2008: consistency check.** *Epidemiology* 2012, **23**(2):301-307.
136. Little MP, Rajaraman P, Curtis RE, Devesa SS, Inskip PD, Check DP, Linet MS: **Mobile phone use and glioma risk: comparison of epidemiological study results with incidence trends in the United States.** *Bmj* 2012, **344**:e1147.
137. Barchana M, Margalioth M, Liphshitz I: **Changes in brain glioma incidence and laterality correlates with use of mobile phones - a nationwide population based study in Israel.** *Asian Pac J Cancer Prev* 2012, **13**(11):5857-5863.
138. Hsu MH, Syed-Abdul S, Scholl J, Jian WS, Lee P, Iqbal U, Li YC: **The incidence rate and mortality of malignant brain tumors after 10 years of intensive cell phone use in**

- Taiwan.** *European journal of cancer prevention : the official journal of the European Cancer Prevention Organisation* 2013, **22**(6):596-598.
139. Kim SJ, Ioannides SJ, Elwood JM: **Trends in incidence of primary brain cancer in New Zealand, 1995 to 2010.** *Aust N Z J Public Health* 2015, **39**(2):148-152.
  140. Sato Y, Kiyohara K, Kojimahara N, Yamaguchi N: **Time trend in incidence of malignant neoplasms of the central nervous system in relation to mobile phone use among young people in Japan.** *Bioelectromagnetics* 2016, **37**(5):282-289.
  141. Chapman S, Azizi L, Luo Q, Sitas F: **Has the incidence of brain cancer risen in Australia since the introduction of mobile phones 29 years ago?** *Cancer Epidemiol* 2016, **42**:199-205.
  142. Dobes M, Khurana VG, Shadbolt B, Jain S, Smith SF, Smee R, Dexter M, Cook R: **Increasing incidence of glioblastoma multiforme and meningioma, and decreasing incidence of Schwannoma (2000-2008): Findings of a multicenter Australian study.** *Surg Neurol Int* 2011, **2**:176.
  143. de Vocht F: **Inferring the 1985-2014 impact of mobile phone use on selected brain cancer subtypes using Bayesian structural time series and synthetic controls.** *Environ Int* 2016, **97**:100-107.
  144. de Vocht F: **Corrigendum to "Inferring the 1985-2014 impact of mobile phone use on selected brain cancer subtypes using Bayesian structural time series and synthetic controls" [Environ. Int. (2016), 97, 100-107].** *Environ Int* 2017, **101**:201-202.
  145. de Vocht F: **Analyses of temporal and spatial patterns of glioblastoma multiforme and other brain cancer subtypes in relation to mobile phones using synthetic counterfactuals.** *Environ Res* 2019, **168**:329-335.
  146. Hardell L, Carlberg M: **Mobile phones, cordless phones and rates of brain tumors in different age groups in the Swedish National Inpatient Register and the Swedish Cancer Register during 1998-2015.** *PLoS One* 2017, **12**(10):e0185461.
  147. Hardell L, Carlberg M: **Increasing rates of brain tumours in the Swedish national inpatient register and the causes of death register.** *Int J Environ Res Public Health* 2015, **12**(4):3793-3813.
  148. Philips A, Henshaw DL, Lamburn G, O'Carroll MJ: **Brain Tumours: Rise in Glioblastoma Multiforme Incidence in England 1995-2015 Suggests an Adverse Environmental or Lifestyle Factor.** *J Environ Public Health* 2018, **2018**:7910754.
  149. Keinan-Boker L, Friedman E, Silverman BG: **Trends in the incidence of primary brain, central nervous system and intracranial tumors in Israel, 1990-2015.** *Cancer Epidemiol* 2018, **56**:6-13.
  150. Karipidis K, Elwood M, Benke G, Sanagou M, Tjong L, Croft RJ: **Mobile phone use and incidence of brain tumour histological types, grading or anatomical location: a population-based ecological study.** *BMJ Open* 2018, **8**(12):e024489.
  151. Nilsson J, Jaras J, Henriksson R, Holgersson G, Bergstrom S, Estenberg J, Augustsson T, Bergqvist M: **No Evidence for Increased Brain Tumour Incidence in the Swedish**

- National Cancer Register Between Years 1980-2012.** *Anticancer Res* 2019, **39**(2):791-796.
152. Natukka T, Raitanen J, Haapasalo H, Auvinen A: **Incidence trends of adult malignant brain tumors in Finland, 1990-2016.** *Acta Oncol* 2019, **58**(7):990-996.
  153. Muscat JE, Malkin MG, Shore RE, Thompson S, Neugut AI, Stellman SD, Bruce J: **Handheld cellular telephones and risk of acoustic neuroma.** *Neurology* 2002, **58**(8):1304-1306.
  154. Warren HG, Prevatt AA, Daly KA, Antonelli PJ: **Cellular telephone use and risk of intratemporal facial nerve tumor.** *Laryngoscope* 2003, **113**(4):663-667.
  155. Lonn S, Ahlbom A, Hall P, Feychting M: **Mobile phone use and the risk of acoustic neuroma.** *Epidemiology* 2004, **15**(6):653-659.
  156. Pettersson D, Mathiesen T, Prochazka M, Bergenheim T, Florentzson R, Harder H, Nyberg G, Siesjo P, Feychting M: **Long-term mobile phone use and acoustic neuroma risk.** *Epidemiology* 2014, **25**(2):233-241.
  157. Han YY, Berkowitz O, Talbott E, Kondziolka D, Donovan M, Lunsford LD: **Are frequent dental x-ray examinations associated with increased risk of vestibular schwannoma?** *J Neurosurg* 2012, **117** Suppl:78-83.
  158. Hardell L, Carlberg M, Hansson Mild K: **Case-control study on cellular and cordless telephones and the risk for acoustic neuroma or meningioma in patients diagnosed 2000-2003.** *Neuroepidemiology* 2005, **25**(3):120-128.
  159. Hardell L, Carlberg M, Hansson Mild K: **Pooled analysis of two case-control studies on the use of cellular and cordless telephones and the risk of benign brain tumours diagnosed during 1997-2003.** *International journal of oncology* 2006, **28**(2):509-518.
  160. Hardell L, Carlberg M, Soderqvist F, Mild KH: **Pooled analysis of case-control studies on acoustic neuroma diagnosed 1997-2003 and 2007-2009 and use of mobile and cordless phones.** *Int J Oncol* 2013, **43**(4):1036-1044.
  161. Corona AP, Ferrite S, Lopes Mda S, Rego MA: **Risk factors associated with vestibular nerve schwannomas.** *Otol Neurotol* 2012, **33**(3):459-465.
  162. Pettersson D, Bottai M, Mathiesen T, Prochazka M, Feychting M: **Validation of self-reported start year of mobile phone use in a Swedish case-control study on radiofrequency fields and acoustic neuroma risk.** *Journal of exposure science & environmental epidemiology* 2015, **25**(1):72-79.
  163. Sato Y, Akiba S, Kubo O, Yamaguchi N: **A case-case study of mobile phone use and acoustic neuroma risk in Japan.** *Bioelectromagnetics* 2011, **32**(2):85-93.
  164. Tillmann T, Ernst H, Ebert S, Kuster N, Behnke W, Rittinghausen S, Dasenbrock C: **Carcinogenicity study of GSM and DCS wireless communication signals in B6C3F1 mice.** *Bioelectromagnetics* 2007, **28**(3):173-187.
  165. Gart JJ, Chu KC, Tarone RE: **Statistical issues in interpretation of chronic bioassay tests for carcinogenicity.** *J Natl Cancer Inst* 1979, **62**(4):957-974.
  166. National Toxicology P: **Toxicology and carcinogenesis studies in B6C3F1/N mice exposed to whole-body radio frequency radiation at a frequency (1,900 MHz) and**

- modulations (GSM and CDMA) used by cell phones.** *Natl Toxicol Program Tech Rept Series* 2018(596):1-260.
167. Tarone R: **The Use of Historical Control Information in Testing for a Trend in Proportions.** *Biometrics* 1982, **38**(1):6.
  168. Smith-Roe SL, Wyde ME, Stout MD, Winters JW, Hobbs CA, Shepard KG, Green AS, Kissling GE, Shockley KR, Tice RR *et al*: **Evaluation of the genotoxicity of cell phone radiofrequency radiation in male and female rats and mice following subchronic exposure.** *Environ Mol Mutagen* 2020, **61**(2):276-290.
  169. Chou CK, Guy AW, Kunz LL, Johnson RB, Crowley JJ, Krupp JH: **Long-term, low-level microwave irradiation of rats.** *Bioelectromagnetics* 1992, **13**(6):469-496.
  170. La Regina M, Moros EG, Pickard WF, Straube WL, Baty J, Roti Roti JL: **The effect of chronic exposure to 835.62 MHz FDMA or 847.74 MHz CDMA radiofrequency radiation on the incidence of spontaneous tumors in rats.** *Radiat Res* 2003, **160**(2):143-151.
  171. Anderson LE, Sheen DM, Wilson BW, Grumbein SL, Creim JA, Sasser LB: **Two-year chronic bioassay study of rats exposed to a 1.6 GHz radiofrequency signal.** *Radiat Res* 2004, **162**(2):201-210.
  172. Haseman JK, Arnold J, Eustis SL: **Tumor incidence in Fischer 344 rats: NTP historical data.** In: *Pathology of the Fischer Rat*. Edited by Boorman GA, Eustis SL, Elwell MR, Montgomery CA, MacKenzie WF. San Diego: Academic Press; 1990.
  173. **NTP Historical Control Database 1984-1999 Fischer Rat**  
[<https://ntp.niehs.nih.gov/data/controls/index.html>]
  174. OECD: **Guidance Document 116 on the Conduct and Design of Chronic Toxicity and Carcinogenicity Studies.** In. Edited by Environment Health and Safety Publications. Paris: OECD; 2012.
  175. Smith P, Kuster N, Ebert S, Chevalier HJ: **GSM and DCS wireless communication signals: combined chronic toxicity/carcinogenicity study in the Wistar rat.** *Radiat Res* 2007, **168**(4):480-492.
  176. Bartsch H, Kupper H, Scheurlen U, Deerberg F, Seebald E, Dietz K, Mecke D, Probst H, Stehle T, Bartsch C: **Effect of chronic exposure to a GSM-like signal (mobile phone) on survival of female Sprague-Dawley rats: modulatory effects by month of birth and possibly stage of the solar cycle.** *Neuro Endocrinol Lett* 2010, **31**(4):457-473.
  177. National Toxicology P: **Toxicology and carcinogenesis studies in Hsd: Sprague Dawley sd rats exposed to whole-body radio frequency radiation at a frequency (900 MHz) and modulations (GSM and CDMA) used by cell phones.** . *Natl Toxicol Program Tech Rept Series* 2018(595):1-384.
  178. Falcioni L, Bua L, Tibaldi E, Lauriola M, De Angelis L, Gnudi F, Mandrioli D, Manservigi M, Manservigi F, Manzoli I *et al*: **Report of final results regarding brain and heart tumors in Sprague-Dawley rats exposed from prenatal life until natural death to mobile phone radiofrequency field representative of a 1.8GHz GSM base station environmental emission.** *Environ Res* 2018, **165**:496-503.

179. Repacholi MH, Basten A, Gebiski V, Noonan D, Finnie J, Harris AW: **Lymphomas in E mu-Pim1 transgenic mice exposed to pulsed 900 MHz electromagnetic fields.** *Radiat Res* 1997, **147**(5):631-640.
180. Utteridge TD, Gebiski V, Finnie JW, Vernon-Roberts B, Kuchel TR: **Long-term exposure of E-mu-Pim1 transgenic mice to 898.4 MHz microwaves does not increase lymphoma incidence.** *Radiat Res* 2002, **158**(3):357-364.
181. Oberto G, Rolfo K, Yu P, Carbonatto M, Peano S, Kuster N, Ebert S, Tofani S: **Carcinogenicity study of 217 Hz pulsed 900 MHz electromagnetic fields in Pim1 transgenic mice.** *Radiat Res* 2007, **168**(3):316-326.
182. Saran A, Pazzaglia S, Mancuso M, Rebessi S, Di Majo V, Tanori M, Lovisolo GA, Pinto R, Marino C: **Effects of exposure of newborn patched1 heterozygous mice to GSM, 900 MHz.** *Radiat Res* 2007, **168**(6):733-740.
183. Sommer AM, Streckert J, Bitz AK, Hansen VW, Lerchl A: **No effects of GSM-modulated 900 MHz electromagnetic fields on survival rate and spontaneous development of lymphoma in female AKR/J mice.** *BMC Cancer* 2004, **4**:77.
184. Sommer AM, Bitz AK, Streckert J, Hansen VW, Lerchl A: **Lymphoma development in mice chronically exposed to UMTS-modulated radiofrequency electromagnetic fields.** *Radiat Res* 2007, **168**(1):72-80.
185. Lee HJ, Jin YB, Lee JS, Choi SY, Kim TH, Pack JK, Choi HD, Kim N, Lee YS: **Lymphoma development of simultaneously combined exposure to two radiofrequency signals in AKR/J mice.** *Bioelectromagnetics* 2011, **32**(6):485-492.
186. Szmigielski S, Szudzinski A, Pietraszek A, Bielec M, Janiak M, Wrembel JK: **Accelerated development of spontaneous and benzopyrene-induced skin cancer in mice exposed to 2450-MHz microwave radiation.** *Bioelectromagnetics* 1982, **3**(2):179-191.
187. Toler JC, Shelton WW, Frei MR, Merritt JH, Stedham MA: **Long-term, low-level exposure of mice prone to mammary tumors to 435 MHz radiofrequency radiation.** *Radiat Res* 1997, **148**(3):227-234.
188. Frei MR, Berger RE, Dusch SJ, Guel V, Jauchem JR, Merritt JH, Stedham MA: **Chronic exposure of cancer-prone mice to low-level 2450 MHz radiofrequency radiation.** *Bioelectromagnetics* 1998, **19**(1):20-31.
189. Frei MR, Jauchem JR, Dusch SJ, Merritt JH, Berger RE, Stedham MA: **Chronic, low-level (1.0 W/kg) exposure of mice prone to mammary cancer to 2450 MHz microwaves.** *Radiat Res* 1998, **150**(5):568-576.
190. Jauchem JR, Ryan KL, Frei MR, Dusch SJ, Lehnert HM, Kovatch RM: **Repeated exposure of C3H/HeJ mice to ultra-wideband electromagnetic pulses: lack of effects on mammary tumors.** *Radiat Res* 2001, **155**(2):369-377.
191. Chagnaud JL, Moreau JM, Veyret B: **No effect of short-term exposure to GSM-modulated low-power microwaves on benzo(a)pyrene-induced tumours in rat.** *Int J Radiat Biol* 1999, **75**(10):1251-1256.
192. Mason PA, Walters TJ, DiGiovanni J, Beason CW, Jauchem JR, Dick EJ, Jr., Mahajan K, Dusch SJ, Shields BA, Merritt JH *et al*: **Lack of effect of 94 GHz radio frequency**

- radiation exposure in an animal model of skin carcinogenesis. *Carcinogenesis* 2001, **22**(10):1701-1708.
193. Imaida K, Kuzutani K, Wang J, Fujiwara O, Ogiso T, Kato K, Shirai T: **Lack of promotion of 7,12-dimethylbenz[a]anthracene-initiated mouse skin carcinogenesis by 1.5 GHz electromagnetic near fields.** *Carcinogenesis* 2001, **22**(11):1837-1841.
  194. Huang TQ, Lee JS, Kim TH, Pack JK, Jang JJ, Seo JS: **Effect of radiofrequency radiation exposure on mouse skin tumorigenesis initiated by 7,12-dimethylbenz[alpha]anthracene.** *Int J Radiat Biol* 2005, **81**(12):861-867.
  195. Paulraj R, Behari J: **Effects of low level microwave radiation on carcinogenesis in Swiss Albino mice.** *Mol Cell Biochem* 2011, **348**(1-2):191-197.
  196. Heikkinen P, Kosma VM, Hongisto T, Huuskonen H, Hyysalo P, Komulainen H, Kumlin T, Lahtinen T, Lang S, Puranen L *et al*: **Effects of mobile phone radiation on X-ray-induced tumorigenesis in mice.** *Radiat Res* 2001, **156**(6):775-785.
  197. Bartsch H, Bartsch C, Seebald E, Deerberg F, Dietz K, Vollrath L, Mecke D: **Chronic exposure to a GSM-like signal (mobile phone) does not stimulate the development of DMBA-induced mammary tumors in rats: results of three consecutive studies.** *Radiat Res* 2002, **157**(2):183-190.
  198. Anane R, Dulou PE, Taxile M, Geffard M, Crespeau FL, Veyret B: **Effects of GSM-900 microwaves on DMBA-induced mammary gland tumors in female Sprague-Dawley rats.** *Radiat Res* 2003, **160**(4):492-497.
  199. Yu D, Shen Y, Kuster N, Fu Y, Chiang H: **Effects of 900 MHz GSM wireless communication signals on DMBA-induced mammary tumors in rats.** *Radiat Res* 2006, **165**(2):174-180.
  200. Hruby R, Neubauer G, Kuster N, Frauscher M: **Study on potential effects of "902-MHz GSM-type Wireless Communication Signals" on DMBA-induced mammary tumours in Sprague-Dawley rats.** *Mutation research* 2008, **649**(1-2):34-44.
  201. Adey WR, Byus CV, Cain CD, Higgins RJ, Jones RA, Kean CJ, Kuster N, MacMurray A, Stagg RB, Zimmerman G *et al*: **Spontaneous and nitrosourea-induced primary tumors of the central nervous system in Fischer 344 rats chronically exposed to 836 MHz modulated microwaves.** *Radiat Res* 1999, **152**(3):293-302.
  202. Adey WR, Byus CV, Cain CD, Higgins RJ, Jones RA, Kean CJ, Kuster N, MacMurray A, Stagg RB, Zimmerman G: **Spontaneous and nitrosourea-induced primary tumors of the central nervous system in Fischer 344 rats exposed to frequency-modulated microwave fields.** *Cancer Res* 2000, **60**(7):1857-1863.
  203. Zook BC, Simmens SJ: **The effects of 860 MHz radiofrequency radiation on the induction or promotion of brain tumors and other neoplasms in rats.** *Radiat Res* 2001, **155**(4):572-583.
  204. Zook BC, Simmens SJ: **The effects of pulsed 860 MHz radiofrequency radiation on the promotion of neurogenic tumors in rats.** *Radiat Res* 2006, **165**(5):608-615.
  205. Shirai T, Kawabe M, Ichihara T, Fujiwara O, Taki M, Watanabe S, Wake K, Yamanaka Y, Imaida K, Asamoto M *et al*: **Chronic exposure to a 1.439 GHz electromagnetic**

- field used for cellular phones does not promote N-ethylnitrosourea induced central nervous system tumors in F344 rats. *Bioelectromagnetics* 2005, **26**(1):59-68.
206. Shirai T, Ichihara T, Wake K, Watanabe S, Yamanaka Y, Kawabe M, Taki M, Fujiwara O, Wang J, Takahashi S *et al*: **Lack of promoting effects of chronic exposure to 1.95-GHz W-CDMA signals for IMT-2000 cellular system on development of N-ethylnitrosourea-induced central nervous system tumors in F344 rats.** *Bioelectromagnetics* 2007, **28**(7):562-572.
207. Imaida K, Taki M, Watanabe S, Kamimura Y, Ito T, Yamaguchi T, Ito N, Shirai T: **The 1.5 GHz electromagnetic near-field used for cellular phones does not promote rat liver carcinogenesis in a medium-term liver bioassay.** *Jpn J Cancer Res* 1998, **89**(10):995-1002.
208. Imaida K, Taki M, Yamaguchi T, Ito T, Watanabe S, Wake K, Aimoto A, Kamimura Y, Ito N, Shirai T: **Lack of promoting effects of the electromagnetic near-field used for cellular phones (929.2 MHz) on rat liver carcinogenesis in a medium-term liver bioassay.** *Carcinogenesis* 1998, **19**(2):311-314.
209. Szudzinski A, Pietraszek A, Janiak M, Wrembel J, Kalczak M, Szmigielski S: **Acceleration of the development of benzopyrene-induced skin cancer in mice by microwave radiation.** *Arch Dermatol Res* 1982, **274**(3-4):303-312.
210. Wu RY, Chiang H, Shao BJ, Li NG, Fu YD: **Effects of 2.45-GHz microwave radiation and phorbol ester 12-O-tetradecanoylphorbol-13-acetate on dimethylhydrazine-induced colon cancer in mice.** *Bioelectromagnetics* 1994, **15**(6):531-538.
211. Heikkinen P, Kosma VM, Alhonen L, Huuskonen H, Komulainen H, Kumlin T, Laitinen JT, Lang S, Puranen L, Juutilainen J: **Effects of mobile phone radiation on UV-induced skin tumorigenesis in ornithine decarboxylase transgenic and non-transgenic mice.** *Int J Radiat Biol* 2003, **79**(4):221-233.
212. Heikkinen P, Ernst H, Huuskonen H, Komulainen H, Kumlin T, Maki-Paakkanen J, Puranen L, Juutilainen J: **No effects of radiofrequency radiation on 3-chloro-4-(dichloromethyl)-5-hydroxy-2(5H)-furanone-induced tumorigenesis in female Wistar rats.** *Radiat Res* 2006, **166**(2):397-408.
213. Tillmann T, Ernst H, Streckert J, Zhou Y, Taugner F, Hansen V, Dasenbrock C: **Indication of cocarcinogenic potential of chronic UMTS-modulated radiofrequency exposure in an ethylnitrosourea mouse model.** *Int J Radiat Biol* 2010, **86**(7):529-541.
214. **NTP Historical Control Database 1998-2004 B6C3F1 Mice**  
[\[https://ntp.niehs.nih.gov/ntp/historical\\_controls/ntp2000\\_2009/2009-05-14-hist-miceallroutes.pdf\]](https://ntp.niehs.nih.gov/ntp/historical_controls/ntp2000_2009/2009-05-14-hist-miceallroutes.pdf)
215. Hanahan D, Weinberg RA: **Hallmarks of cancer: the next generation.** *Cell* 2011, **144**(5):646-674.
216. Higgins JPT, Green S: **Cochrane Handbook for Systematic Reviews of Interventions.** In., 5.1.0 edn: The Cochrane Collaboration; 2011.

217. Birnbaum LS, Thayer KA, Bucher JR, Wolfe MS: **Implementing systematic review at the National Toxicology Program: status and next steps.** *Environ Health Perspect* 2013, **121**(4):A108-109.
218. Murray HE, Thayer KA: **Implementing systematic review in toxicological profiles: ATSDR and NIEHS/NTP collaboration.** *Journal of environmental health* 2014, **76**(8):34-35.
219. Rooney AA, Boyles AL, Wolfe MS, Bucher JR, Thayer KA: **Systematic review and evidence integration for literature-based environmental health science assessments.** *Environ Health Perspect* 2014, **122**(7):711-718.
220. Vandenberg LN, Agerstrand M, Beronius A, Beausoleil C, Bergman A, Bero LA, Bornehag CG, Boyer CS, Cooper GS, Cotgreave I *et al*: **A proposed framework for the systematic review and integrated assessment (SYRINA) of endocrine disrupting chemicals.** *Environmental health : a global access science source* 2016, **15**(1):74.
221. Aghadavod E, Khodadadi S, Baradaran A, Nasri P, Bahmani M, Rafieian-Kopaei M: **Role of Oxidative Stress and Inflammatory Factors in Diabetic Kidney Disease.** *Iran J Kidney Dis* 2016, **10**(6):337-343.
222. Kamceva G, Arsova-Sarafinovska Z, Ruskovska T, Zdravkovska M, Kamceva-Panova L, Stikova E: **Cigarette Smoking and Oxidative Stress in Patients with Coronary Artery Disease.** *Open Access Maced J Med Sci* 2016, **4**(4):636-640.
223. Qureshi MA, Kim YO, Schuppan D: **Hepatocellular carcinoma in nonalcoholic fatty liver disease: A link between oxidative stress and T-cell suppression.** *Hepatology* 2016, **64**(5):1794-1797.
224. Sayanthooran S, Magana-Arachchi DN, Gunerathne L, Abeysekera TD, Sooriyapathirana SS: **Upregulation of Oxidative Stress Related Genes in a Chronic Kidney Disease Attributed to Specific Geographical Locations of Sri Lanka.** *Biomed Res Int* 2016, **2016**:7546265.
225. Turkmen K: **Inflammation, oxidative stress, apoptosis, and autophagy in diabetes mellitus and diabetic kidney disease: the Four Horsemen of the Apocalypse.** *Int Urol Nephrol* 2016.
226. Vakonaki E, Tsarouhas K, Spandidos DA, Tsatsakis AM: **Complex interplay of DNA damage, DNA repair genes, and oxidative stress in coronary artery disease.** *Anatol J Cardiol* 2016, **16**(12):939.
227. Hecht F, Pessoa CF, Gentile LB, Rosenthal D, Carvalho DP, Fortunato RS: **The role of oxidative stress on breast cancer development and therapy.** *Tumour Biol* 2016, **37**(4):4281-4291.
228. Li L, Chen F: **Oxidative stress, epigenetics, and cancer stem cells in arsenic carcinogenesis and prevention.** *Curr Pharmacol Rep* 2016, **2**(2):57-63.
229. Perse M: **Oxidative stress in the pathogenesis of colorectal cancer: cause or consequence?** *Biomed Res Int* 2013, **2013**:725710.
230. Prasad S, Gupta SC, Pandey MK, Tyagi AK, Deb L: **Oxidative Stress and Cancer: Advances and Challenges.** *Oxid Med Cell Longev* 2016, **2016**:5010423.

231. Toyokuni S: **Oxidative stress as an iceberg in carcinogenesis and cancer biology.** *Arch Biochem Biophys* 2016, **595**:46-49.
232. Gulati S, Yadav A, Kumar N, Priya K, Aggarwal NK, Gupta R: **Phenotypic and genotypic characterization of antioxidant enzyme system in human population exposed to radiation from mobile towers.** *Mol Cell Biochem* 2018, **440**(1-2):1-9.
233. Zothansiam, Zosangzuali M, Lalramdinpuii M, Jagetia GC: **Impact of radiofrequency radiation on DNA damage and antioxidants in peripheral blood lymphocytes of humans residing in the vicinity of mobile phone base stations.** *Electromagn Biol Med* 2017, **36**(3):295-305.
234. Khalil AM, Abu Khadra KM, Aljaberi AM, Gagaa MH, Issa HS: **Assessment of oxidant/antioxidant status in saliva of cell phone users.** *Electromagn Biol Med* 2014, **33**(2):92-97.
235. Abu Khadra KM, Khalil AM, Abu Samak M, Aljaberi A: **Evaluation of selected biochemical parameters in the saliva of young males using mobile phones.** *Electromagn Biol Med* 2015, **34**(1):72-76.
236. Malini SS: **RESOLVING THE ENIGMA OF EFFECT OF MOBILE PHONE USAGE ON SPERMATOGENESIS IN HUMANS IN SOUTH INDIAN POPULATION.** *Asian Journal of Pharmaceutical and Clinical Research* 2017, **10**(2):233-237.
237. Khalil AM, Alshamali AM, Gagaa MH: **Detection of oxidative stress induced by mobile phone radiation in tissues of mice using 8-oxo-7, 8-dihydro-20-deoxyguanosine as a biomarker.** *World Acad Sci Eng Technol* 2011, **76**:657-622.
238. Bahreyni Toossi MH, Sadeghnia HR, Mohammad Mahdizadeh Feyzabadi M, Hosseini M, Hedayati M, Mosallanejad R, Beheshti F, Alizadeh Rahvar Z: **Exposure to mobile phone (900-1800 MHz) during pregnancy: tissue oxidative stress after childbirth.** *J Matern Fetal Neonatal Med* 2018, **31**(10):1298-1303.
239. Shahin S, Singh VP, Shukla RK, Dhawan A, Gangwar RK, Singh SP, Chaturvedi CM: **2.45 GHz microwave irradiation-induced oxidative stress affects implantation or pregnancy in mice, *Mus musculus*.** *Appl Biochem Biotechnol* 2013, **169**(5):1727-1751.
240. Shahin S, Mishra V, Singh SP, Chaturvedi CM: **2.45-GHz microwave irradiation adversely affects reproductive function in male mouse, *Mus musculus* by inducing oxidative and nitrosative stress.** *Free Radic Res* 2014, **48**(5):511-525.
241. Shahin S, Singh SP, Chaturvedi CM: **Mobile phone (1800MHz) radiation impairs female reproduction in mice, *Mus musculus*, through stress induced inhibition of ovarian and uterine activity.** *Reprod Toxicol* 2017, **73**:41-60.
242. Shahin S, Banerjee S, Swarup V, Singh SP, Chaturvedi CM: **From the Cover: 2.45-GHz Microwave Radiation Impairs Hippocampal Learning and Spatial Memory: Involvement of Local Stress Mechanism-Induced Suppression of iGluR/ERK/CREB Signaling.** *Toxicol Sci* 2018, **161**(2):349-374.
243. Shahin S, Singh SP, Chaturvedi CM: **1800 MHz mobile phone irradiation induced oxidative and nitrosative stress leads to p53 dependent Bax mediated testicular apoptosis in mice, *Mus musculus*.** *J Cell Physiol* 2018, **233**(9):7253-7267.

244. Pandey N, Giri S, Das S, Upadhaya P: **Radiofrequency radiation (900 MHz)-induced DNA damage and cell cycle arrest in testicular germ cells in swiss albino mice.** *Toxicol Ind Health* 2017, **33**(4):373-384.
245. Esmekaya MA, Tuysuz MZ, Tomruk A, Canseven AG, Yucel E, Aktuna Z, Keskil S, Seyhan N: **Effects of cell phone radiation on lipid peroxidation, glutathione and nitric oxide levels in mouse brain during epileptic seizure.** *J Chem Neuroanat* 2016, **75**(Pt B):111-115.
246. Zong C, Ji Y, He Q, Zhu S, Qin F, Tong J, Cao Y: **Adaptive response in mice exposed to 900 MHz radiofrequency fields: bleomycin-induced DNA and oxidative damage/repair.** *Int J Radiat Biol* 2015, **91**(3):270-276.
247. Ahmed NA, Radwan NM, Aboul Ezz HS, Salama NA: **The antioxidant effect of Green Tea Mega EGCG against electromagnetic radiation-induced oxidative stress in the hippocampus and striatum of rats.** *Electromagn Biol Med* 2017, **36**(1):63-73.
248. Bilgici B, Akar A, Avci B, Tuncel OK: **Effect of 900 MHz radiofrequency radiation on oxidative stress in rat brain and serum.** *Electromagn Biol Med* 2013, **32**(1):20-29.
249. Cetin H, Naziroglu M, Celik O, Yuksel M, Pastaci N, Ozkaya MO: **Liver antioxidant stores protect the brain from electromagnetic radiation (900 and 1800 MHz)-induced oxidative stress in rats during pregnancy and the development of offspring.** *J Matern Fetal Neonatal Med* 2014, **27**(18):1915-1921.
250. Ertlav K, Uslusoy F, Ataizi S, Naziroglu M: **Long term exposure to cell phone frequencies (900 and 1800 MHz) induces apoptosis, mitochondrial oxidative stress and TRPV1 channel activation in the hippocampus and dorsal root ganglion of rats.** *Metab Brain Dis* 2018, **33**(3):753-763.
251. Eser O, Songur A, Aktas C, Karavelioglu E, Caglar V, Aylak F, Ozguner F, Kanter M: **The effect of electromagnetic radiation on the rat brain: an experimental study.** *Turk Neurosurg* 2013, **23**(6):707-715.
252. Maaroufi K, Had-Aissouni L, Melon C, Sakly M, Abdelmelek H, Poucet B, Save E: **Spatial learning, monoamines and oxidative stress in rats exposed to 900 MHz electromagnetic field in combination with iron overload.** *Behav Brain Res* 2014, **258**:80-89.
253. Sharma S, Shukla S: **Effect of electromagnetic radiation on redox status, acetylcholine esterase activity and cellular damage contributing to the diminution of the brain working memory in rats.** *Journal of chemical neuroanatomy* 2020, **106**:101784.
254. Asl JF, Goudarzi M, Shoghi H: **The radio-protective effect of rosmarinic acid against mobile phone and Wi-Fi radiation-induced oxidative stress in the brains of rats.** *Pharmacological Reports : PR* 2020.
255. Kesari KK, Kumar S, Behari J: **900-MHz microwave radiation promotes oxidation in rat brain.** *Electromagn Biol Med* 2011, **30**(4):219-234.
256. Motawi TK, Darwish HA, Moustafa YM, Labib MM: **Biochemical modifications and neuronal damage in brain of young and adult rats after long-term exposure to mobile phone radiations.** *Cell Biochem Biophys* 2014, **70**(2):845-855.

257. Narayanan SN, Kumar RS, Kedage V, Nalini K, Nayak S, Bhat PG: **Evaluation of oxidant stress and antioxidant defense in discrete brain regions of rats exposed to 900 MHz radiation.** *Bratislava Medical Journal-Bratislavske Lekarske Listy* 2014, **115**(5):260-266.
258. Tan S, Wang H, Xu X, Zhao L, Zhang J, Dong J, Yao B, Wang H, Zhou H, Gao Y *et al*: **Study on dose-dependent, frequency-dependent, and accumulative effects of 1.5 GHz and 2.856 GHz microwave on cognitive functions in Wistar rats.** *Sci Rep* 2017, **7**(1):10781.
259. Avci B, Akar A, Bilgici B, Tuncel OK: **Oxidative stress induced by 1.8 GHz radio frequency electromagnetic radiation and effects of garlic extract in rats.** *Int J Radiat Biol* 2012, **88**(11):799-805.
260. Boder P, Makarova K, Zawada K, Antkowiak B, Paluch M, Sobiczewska E, Sirav B, Siwicki AK, Stankiewicz W: **The effect of 1800MHz radio-frequency radiation on NMDA receptor subunit NR1 expression and peroxidation in the rat brain in healthy and inflammatory states.** *Biomed Pharmacother* 2017, **92**:802-809.
261. Shehu A, Mohammed A, Magaji RA, Muhammad MS: **Exposure to mobile phone electromagnetic field radiation, ringtone and vibration affects anxiety-like behaviour and oxidative stress biomarkers in albino wistar rats.** *Metab Brain Dis* 2016, **31**(2):355-362.
262. Gurler HS, Bilgici B, Akar AK, Tomak L, Bedir A: **Increased DNA oxidation (8-OHdG) and protein oxidation (AOPP) by low level electromagnetic field (2.45 GHz) in rat brain and protective effect of garlic.** *Int J Radiat Biol* 2014, **90**(10):892-896.
263. Naziroglu M, Celik O, Ozgul C, Cig B, Dogan S, Bal R, Gumral N, Rodriguez AB, Pariente JA: **Melatonin modulates wireless (2.45 GHz)-induced oxidative injury through TRPM2 and voltage gated Ca(2+) channels in brain and dorsal root ganglion in rat.** *Physiol Behav* 2012, **105**(3):683-692.
264. Ait-Aissa S, de Gannes FP, Taxile M, Billaudel B, Hurtier A, Haro E, Ruffie G, Athane A, Veyret B, Lagroye I: **In situ expression of heat-shock proteins and 3-nitrotyrosine in brains of young rats exposed to a WiFi signal in utero and in early life.** *Radiat Res* 2013, **179**(6):707-716.
265. Othman H, Ammari M, Sakly M, Abdelmelek H: **Effects of prenatal exposure to WIFI signal (2.45GHz) on postnatal development and behavior in rat: Influence of maternal restraint.** *Behav Brain Res* 2017, **326**:291-302.
266. Othman H, Ammari M, Sakly M, Abdelmelek H: **Effects of repeated restraint stress and WiFi signal exposure on behavior and oxidative stress in rats.** *Metab Brain Dis* 2017, **32**(5):1459-1469.
267. Hidisoglu E, Kantar Gok D, Er H, Akpınar D, Uysal F, Akkoyunlu G, Ozen S, Agar A, Yargicoglu P: **2100-MHz electromagnetic fields have different effects on visual evoked potentials and oxidant/antioxidant status depending on exposure duration.** *Brain Res* 2016, **1635**:1-11.
268. Kesari KK, Meena R, Nirala J, Kumar J, Verma HN: **Effect of 3G cell phone exposure with computer controlled 2-D stepper motor on non-thermal activation of the**

- hsp27/p38MAPK stress pathway in rat brain. *Cell Biochem Biophys* 2014, **68**(2):347-358.**
269. Sahin D, Ozgur E, Guler G, Tomruk A, Unlu I, Sepici-Dincel A, Seyhan N: **The 2100MHz radiofrequency radiation of a 3G-mobile phone and the DNA oxidative damage in brain.** *J Chem Neuroanat* 2016, **75**(Pt B):94-98.
270. Esmekaya MA, Ozer C, Seyhan N: **900 MHz pulse-modulated radiofrequency radiation induces oxidative stress on heart, lung, testis and liver tissues.** *Gen Physiol Biophys* 2011, **30**(1):84-89.
271. Ozorak A, Naziroglu M, Celik O, Yuksel M, Ozcelik D, Ozkaya MO, Cetin H, Kahya MC, Kose SA: **Wi-Fi (2.45 GHz)- and mobile phone (900 and 1800 MHz)-induced risks on oxidative stress and elements in kidney and testis of rats during pregnancy and the development of offspring.** *Biol Trace Elem Res* 2013, **156**(1-3):221-229.
272. Shahin NN, El-Nabarawy NA, Gouda AS, Megarbane B: **The protective role of spermine against male reproductive aberrations induced by exposure to electromagnetic field - An experimental investigation in the rat.** *Toxicol Appl Pharmacol* 2019, **370**:117-130.
273. Yahyazadeh A, Altunkaynak BZ: **Protective effects of luteolin on rat testis following exposure to 900 MHz electromagnetic field.** *Biotech Histochem* 2019, **94**(4):298-307.
274. Oksay T, Naziroglu M, Dogan S, Guzel A, Gumral N, Kosar PA: **Protective effects of melatonin against oxidative injury in rat testis induced by wireless (2.45 GHz) devices.** *Andrologia* 2014, **46**(1):65-72.
275. Kesari KK, Kumar S, Behari J: **Effects of radiofrequency electromagnetic wave exposure from cellular phones on the reproductive pattern in male Wistar rats.** *Appl Biochem Biotechnol* 2011, **164**(4):546-559.
276. Sokolovic D, Djordjevic B, Kocic G, Stoimenov TJ, Stanojkovic Z, Sokolovic DM, Veljkovic A, Ristic G, Despotovic M, Milisavljevic D *et al*: **The Effects of Melatonin on Oxidative Stress Parameters and DNA Fragmentation in Testicular Tissue of Rats Exposed to Microwave Radiation.** *Adv Clin Exp Med* 2015, **24**(3):429-436.
277. Koç A, Ünal D, Çimentepe E, Bayrak Ö, Karataş Ö F, Yildirim ME, Bayrak R, Aydın M: **The effects of antioxidants on testicular apoptosis and oxidative stress produced by cell phones.** *Turk J Med Sci* 2013, **43**(1):131-137.
278. Oyewopo AO, Olaniyi SK, Oyewopo CI, Jimoh AT: **Radiofrequency electromagnetic radiation from cell phone causes defective testicular function in male Wistar rats.** *Andrologia* 2017, **49**(10).
279. Al-Damegh MA: **Rat testicular impairment induced by electromagnetic radiation from a conventional cellular telephone and the protective effects of the antioxidants vitamins C and E.** *Clinics (Sao Paulo)* 2012, **67**(7):785-792.
280. Gautam R, Singh KV, Nirala J, Murmu NN, Meena R, Rajamani P: **Oxidative stress-mediated alterations on sperm parameters in male Wistar rats exposed to 3G mobile phone radiation.** *Andrologia* 2019, **51**(3):e13201.

281. Kuzay D, Ozer C, Sirav B, Canseven AG, Seyhan N: **Oxidative effects of extremely low frequency magnetic field and radio frequency radiation on testes tissues of diabetic and healthy rats.** *Bratisl Lek Listy* 2017, **118**(5):278-282.
282. Atasoy HI, Gunal MY, Atasoy P, Elgun S, Bugdayci G: **Immunohistopathologic demonstration of deleterious effects on growing rat testes of radiofrequency waves emitted from conventional Wi-Fi devices.** *J Pediatr Urol* 2013, **9**(2):223-229.
283. Zhu W, Cui Y, Feng X, Li Y, Zhang W, Xu J, Wang H, Lv S: **The apoptotic effect and the plausible mechanism of microwave radiation on rat myocardial cells.** *Can J Physiol Pharmacol* 2016, **94**(8):849-857.
284. Gumral N, Saygin M, Asci H, Uguz AC, Celik O, Doguc DK, Savas HB, Comlekci S: **The effects of electromagnetic radiation (2450 MHz wireless devices) on the heart and blood tissue: role of melatonin.** *Bratisl Lek Listy* 2016, **117**(11):665-671.
285. Djordjevic B, Sokolovic D, Kocic G, Veljkovic A, Despotovic M, Basic J, Jevtovic-Stoimenov T, Sokolovic DM: **The effect of melatonin on the liver of rats exposed to microwave radiation.** *Bratisl Lek Listy* 2015, **116**(2):96-100.
286. Boder P, Stankiewicz W, Antkowiak B, Paluch M, Kieliszek J, Sobiech J, Niemcewicz M: **Influence of electromagnetic field (1800 MHz) on lipid peroxidation in brain, blood, liver and kidney in rats.** *Int J Occup Med Environ Health* 2015, **28**(4):751-759.
287. Chauhan P, Verma HN, Sisodia R, Kesari KK: **Microwave radiation (2.45 GHz)-induced oxidative stress: Whole-body exposure effect on histopathology of Wistar rats.** *Electromagn Biol Med* 2017, **36**(1):20-30.
288. Postaci I, Coskun O, Senol N, Aslankoc R, Comlekci S: **The physiopathological effects of quercetin on oxidative stress in radiation of 4.5 g mobile phone exposed liver tissue of rat.** *Bratisl Lek Listy* 2018, **119**(8):481-489.
289. Kuybulu AE, Oktem F, Ciris IM, Sutcu R, Ormeci AR, Comlekci S, Uz E: **Effects of long-term pre- and post-natal exposure to 2.45 GHz wireless devices on developing male rat kidney.** *Ren Fail* 2016, **38**(4):571-580.
290. Demirel S, Doganay S, Turkoz Y, Dogan Z, Turan B, Firat PG: **Effects of third generation mobile phone-emitted electromagnetic radiation on oxidative stress parameters in eye tissue and blood of rats.** *Cutan Ocul Toxicol* 2012, **31**(2):89-94.
291. Eker ED, Arslan B, Yildirim M, Akar A, Aras N: **The effect of exposure to 1800 MHz radiofrequency radiation on epidermal growth factor, caspase-3, Hsp27 and p38MAPK gene expressions in the rat eye.** *Bratisl Lek Listy* 2018, **119**(9):588-592.
292. Tok L, Naziroglu M, Dogan S, Kahya MC, Tok O: **Effects of melatonin on Wi-Fi-induced oxidative stress in lens of rats.** *Indian J Ophthalmol* 2014, **62**(1):12-15.
293. Aynali G, Naziroglu M, Celik O, Dogan M, Yariktas M, Yasan H: **Modulation of wireless (2.45 GHz)-induced oxidative toxicity in laryngotracheal mucosa of rat by melatonin.** *Eur Arch Otorhinolaryngol* 2013, **270**(5):1695-1700.
294. Sangun O, Dundar B, Darici H, Comlekci S, Doguc DK, Celik S: **The effects of long-term exposure to a 2450 MHz electromagnetic field on growth and pubertal development in female Wistar rats.** *Electromagn Biol Med* 2015, **34**(1):63-71.

295. Yuksel M, Naziroglu M, Ozkaya MO: **Long-term exposure to electromagnetic radiation from mobile phones and Wi-Fi devices decreases plasma prolactin, progesterone, and estrogen levels but increases uterine oxidative stress in pregnant rats and their offspring.** *Endocrine* 2016, **52**(2):352-362.
296. Aydin B, Akar A: **Effects of a 900-MHz electromagnetic field on oxidative stress parameters in rat lymphoid organs, polymorphonuclear leukocytes and plasma.** *Arch Med Res* 2011, **42**(4):261-267.
297. Akbari A, Jelodar G, Nazifi S: **Vitamin C protects rat cerebellum and encephalon from oxidative stress following exposure to radiofrequency wave generated by a BTS antenna model.** *Toxicol Mech Methods* 2014, **24**(5):347-352.
298. Kerimoglu G, Hanci H, Bas O, Aslan A, Erol HS, Turgut A, Kaya H, Cankaya S, Sonmez OF, Odaci E: **Pernicious effects of long-term, continuous 900-MHz electromagnetic field throughout adolescence on hippocampus morphology, biochemistry and pyramidal neuron numbers in 60-day-old Sprague Dawley male rats.** *J Chem Neuroanat* 2016, **77**:169-175.
299. Ragy MM: **Effect of exposure and withdrawal of 900-MHz-electromagnetic waves on brain, kidney and liver oxidative stress and some biochemical parameters in male rats.** *Electromagn Biol Med* 2015, **34**(4):279-284.
300. Tang J, Zhang Y, Yang L, Chen Q, Tan L, Zuo S, Feng H, Chen Z, Zhu G: **Exposure to 900 MHz electromagnetic fields activates the mcp-1/ERK pathway and causes blood-brain barrier damage and cognitive impairment in rats.** *Brain Research* 2015, **1601**:92-101.
301. Varghese R, Majumdar A, Kumar G, Shukla A: **Rats exposed to 2.45GHz of non-ionizing radiation exhibit behavioral changes with increased brain expression of apoptotic caspase 3.** *Pathophysiology* 2018, **25**(1):19-30.
302. Alkis ME, Bilgin HM, Akpolat V, Dasdag S, Yegin K, Yavas MC, Akdag MZ: **Effect of 900-, 1800-, and 2100-MHz radiofrequency radiation on DNA and oxidative stress in brain.** *Electromagn Biol Med* 2019, **38**(1):32-47.
303. Yang XS, He GL, Hao YT, Xiao Y, Chen CH, Zhang GB, Yu ZP: **Exposure to 2.45 GHz electromagnetic fields elicits an HSP-related stress response in rat hippocampus.** *Brain research bulletin* 2012, **88**(4):371-378.
304. Hanci H, Kerimoglu G, Mercantepe T, Odaci E: **Changes in testicular morphology and oxidative stress biomarkers in 60-day-old Sprague Dawley rats following exposure to continuous 900-MHz electromagnetic field for 1 h a day throughout adolescence.** *Reprod Toxicol* 2018, **81**:71-78.
305. Jelodar G, Akbari A, Nazifi S: **The prophylactic effect of vitamin C on oxidative stress indexes in rat eyes following exposure to radiofrequency wave generated by a BTS antenna model.** *Int J Radiat Biol* 2013, **89**(2):128-131.
306. Liu Q, Si T, Xu X, Liang F, Wang L, Pan S: **Electromagnetic radiation at 900 MHz induces sperm apoptosis through bcl-2, bax and caspase-3 signaling pathways in rats.** *Reprod Health* 2015, **12**(1):65.

307. Bin-Meferij MM, El-Kott AF: **The radioprotective effects of Moringa oleifera against mobile phone electromagnetic radiation-induced infertility in rats.** *Int J Clin Exp Med* 2015, **8**(8):12487-12497.
308. Oguzturk H, Beytur R, Ciftci O, Turtay MG, Samdanci E, Dilek OF: **Does 3-G mobile phone radiofrequency affect oxidative stress, sperm characteristics and testis histology?** *Fresenius Environ Bull* 2011, **20**(3):646-653.
309. Saygin M, Asci H, Ozmen O, Cankara FN, Dincoglu D, Ilhan I: **Impact of 2.45 GHz microwave radiation on the testicular inflammatory pathway biomarkers in young rats: The role of gallic acid.** *Environ Toxicol* 2016, **31**(12):1771-1784.
310. Lee HJ, Jin YB, Kim TH, Pack JK, Kim N, Choi HD, Lee JS, Lee YS: **The effects of simultaneous combined exposure to CDMA and WCDMA electromagnetic fields on rat testicular function.** *Bioelectromagnetics* 2012, **33**(4):356-364.
311. Odaci E, Unal D, Mercantepe T, Topal Z, Hanci H, Turedi S, Erol HS, Mungan S, Kaya H, Colakoglu S: **Pathological effects of prenatal exposure to a 900 MHz electromagnetic field on the 21-day-old male rat kidney.** *Biotech Histochem* 2015, **90**(2):93-101.
312. Turedi S, Kerimoglu G, Mercantepe T, Odaci E: **Biochemical and pathological changes in the male rat kidney and bladder following exposure to continuous 900-MHz electromagnetic field on postnatal days 22-59**. *Int J Radiat Biol* 2017, **93**(9):990-999.
313. Okatan DO, Okatan AE, Hanci H, Demir S, Yaman SO, Colakoglu S, Odaci E: **Effects of 900-MHz electromagnetic fields exposure throughout middle/late adolescence on the kidney morphology and biochemistry of the female rat.** *Toxicol Ind Health* 2018, **34**(10):693-702.
314. Bedir R, Tumkaya L, Mercantepe T, Yilmaz A: **Pathological Findings Observed in the Kidneys of Postnatal Male Rats Exposed to the 2100 MHz Electromagnetic Field.** *Arch Med Res* 2018, **49**(7):432-440.
315. Topal Z, Hanci H, Mercantepe T, Erol HS, Keles ON, Kaya H, Mungan S, Odaci E: **The effects of prenatal long-duration exposure to 900-MHz electromagnetic field on the 21-day-old newborn male rat liver.** *Turk J Med Sci* 2015, **45**(2):291-297.
316. Ismaili LA, Joumaa WH, Moustafa ME: **The impact of exposure of diabetic rats to 900 MHz electromagnetic radiation emitted from mobile phone antenna on hepatic oxidative stress.** *Electromagn Biol Med* 2019, **38**(4):287-296.
317. Okatan DO, Kulaber A, Kerimoglu G, Odaci E: **Altered morphology and biochemistry of the female rat liver following 900 megahertz electromagnetic field exposure during mid to late adolescence.** *Biotech Histochem* 2019, **94**(6):420-428.
318. Tumkaya L, Yilmaz A, Akyildiz K, Mercantepe T, Yazici ZA, Yilmaz H: **Prenatal Effects of a 1,800-MHz Electromagnetic Field on Rat Livers.** *Cells Tissues Organs* 2019, **207**(3-4):187-196.
319. Okatan DO, Kaya H, Aliyazicioglu Y, Demir S, Colakoglu S, Odaci E: **Continuous 900-megahertz electromagnetic field applied in middle and late-adolescence causes**

- qualitative and quantitative changes in the ovarian morphology, tissue and blood biochemistry of the rat.** *Int J Radiat Biol* 2018, **94**(2):186-198.
320. Saygin M, Ozmen O, Erol O, Ellidag HY, Ilhan I, Aslankoc R: **The impact of electromagnetic radiation (2.45 GHz, Wi-Fi) on the female reproductive system: The role of vitamin C.** *Toxicol Ind Health* 2018, **34**(9):620-630.
321. Turedi S, Hanci H, Topal Z, Unal D, Mercantepe T, Bozkurt I, Kaya H, Odaci E: **The effects of prenatal exposure to a 900-MHz electromagnetic field on the 21-day-old male rat heart.** *Electromagn Biol Med* 2015, **34**(4):390-397.
322. Kerimoglu G, Mercantepe T, Erol HS, Turgut A, Kaya H, Colakoglu S, Odaci E: **Effects of long-term exposure to 900 megahertz electromagnetic field on heart morphology and biochemistry of male adolescent rats.** *Biotech Histochem* 2016, **91**(7):445-454.
323. Ikcinci A, Mercantepe T, Unal D, Erol HS, Sahin A, Aslan A, Bas O, Erdem H, Sonmez OF, Kaya H *et al*: **Morphological and antioxidant impairments in the spinal cord of male offspring rats following exposure to a continuous 900MHz electromagnetic field during early and mid-adolescence.** *J Chem Neuroanat* 2016, **75**(Pt B):99-104.
324. Kerimoglu G, Aslan A, Bas O, Colakoglu S, Odaci E: **Adverse effects in lumbar spinal cord morphology and tissue biochemistry in Sprague Dawley male rats following exposure to a continuous 1-h a day 900-MHz electromagnetic field throughout adolescence.** *J Chem Neuroanat* 2016, **78**:125-130.
325. Kerimoglu G, Guney C, Ersoz S, Odaci E: **A histopathological and biochemical evaluation of oxidative injury in the sciatic nerves of male rats exposed to a continuous 900-megahertz electromagnetic field throughout all periods of adolescence.** *J Chem Neuroanat* 2018, **91**:1-7.
326. Yang H, Zhang Y, Wang Z, Zhong S, Hu G, Zuo W: **The Effects of Mobile Phone Radiofrequency Radiation on Cochlear Stria Marginal Cells in Sprague–Dawley Rats.** *Bioelectromagnetics* 2020, **41**(3):219-229.
327. Masoumi A, Karbalaee N, Mortazavi SMJ, Shabani M: **Radiofrequency radiation emitted from Wi-Fi (2.4 GHz) causes impaired insulin secretion and increased oxidative stress in rat pancreatic islets.** *Int J Radiat Biol* 2018, **94**(9):850-857.
328. Hanci H, Turedi S, Topal Z, Mercantepe T, Bozkurt I, Kaya H, Ersoz S, Unal B, Odaci E: **Can prenatal exposure to a 900 MHz electromagnetic field affect the morphology of the spleen and thymus, and alter biomarkers of oxidative damage in 21-day-old male rats?** *Biotech Histochem* 2015, **90**(7):535-543.
329. Megha K, Deshmukh PS, Banerjee BD, Tripathi AK, Ahmed R, Abegaonkar MP: **Low intensity microwave radiation induced oxidative stress, inflammatory response and DNA damage in rat brain.** *Neurotoxicology* 2015, **51**:158-165.
330. Deshmukh PS, Banerjee BD, Abegaonkar MP, Megha K, Ahmed RS, Tripathi AK, Mediratta PK: **Effect of low level microwave radiation exposure on cognitive function and oxidative stress in rats.** *Indian J Biochem Biophys* 2013, **50**(2):114-119.

331. Megha K, Deshmukh PS, Banerjee BD, Tripathi AK, Abegaonkar MP: **Microwave radiation induced oxidative stress, cognitive impairment and inflammation in brain of Fischer rats.** *Indian J Exp Biol* 2012, **50**(12):889-896.
332. Marzook EA, Abd El Moneim AE, Elhadary AA: **Protective role of sesame oil against mobile base station-induced oxidative stress.** *Journal of Radiation Research and Applied Sciences* 2019, **7**(1):1-6.
333. Ghoneim FM, Arafat EA: **Histological and histochemical study of the protective role of rosemary extract against harmful effect of cell phone electromagnetic radiation on the parotid glands.** *Acta Histochem* 2016, **118**(5):478-485.
334. Bouji M, Lecomte A, Gamez C, Blazy K, Villegier AS: **Impact of Cerebral Radiofrequency Exposures on Oxidative Stress and Corticosterone in a Rat Model of Alzheimer's Disease.** *J Alzheimers Dis* 2020, **73**(2):467-476.
335. Kalanjati VP, Purwantari KE, Prasetiowati L: **Aluminium foil dampened the adverse effect of 2100 MHz mobile phone-induced radiation on the blood parameters and myocardium in rats.** *Environ Sci Pollut Res Int* 2019, **26**(12):11686-11689.
336. Furtado-Filho OV, Borba JB, Dallegrave A, Pizzolato TM, Henriques JA, Moreira JC, Saffi J: **Effect of 950 MHz UHF electromagnetic radiation on biomarkers of oxidative damage, metabolism of UFA and antioxidants in the livers of young rats of different ages.** *Int J Radiat Biol* 2014, **90**(2):159-168.
337. Furtado-Filho OV, Borba JB, Maraschin T, Souza LM, Henriques JA, Moreira JC, Saffi J: **Effects of chronic exposure to 950 MHz ultra-high-frequency electromagnetic radiation on reactive oxygen species metabolism in the right and left cerebral cortex of young rats of different ages.** *Int J Radiat Biol* 2015, **91**(11):891-897.
338. Hussein S, El-Saba AA, Galal MK: **Biochemical and histological studies on adverse effects of mobile phone radiation on rat's brain.** *J Chem Neuroanat* 2016, **78**:10-19.
339. Hanci H, Odaci E, Kaya H, Aliyazicioglu Y, Turan I, Demir S, Colakoglu S: **The effect of prenatal exposure to 900-MHz electromagnetic field on the 21-old-day rat testicle.** *Reprod Toxicol* 2013, **42**:203-209.
340. Guler G, Ozgur E, Keles H, Tomruk A, Vural SA, Seyhan N: **Neurodegenerative changes and apoptosis induced by intrauterine and extrauterine exposure of radiofrequency radiation.** *J Chem Neuroanat* 2016, **75**(Pt B):128-133.
341. Guler G, Tomruk A, Ozgur E, Sahin D, Sepici A, Altan N, Seyhan N: **The effect of radiofrequency radiation on DNA and lipid damage in female and male infant rabbits.** *Int J Radiat Biol* 2012, **88**(4):367-373.
342. Ozgur E, Kismali G, Guler G, Akcay A, Ozkurt G, Sel T, Seyhan N: **Effects of prenatal and postnatal exposure to GSM-like radiofrequency on blood chemistry and oxidative stress in infant rabbits, an experimental study.** *Cell Biochem Biophys* 2013, **67**(2):743-751.
343. Kismali G, Ozgur E, Guler G, Akcay A, Sel T, Seyhan N: **The influence of 1800 MHz GSM-like signals on blood chemistry and oxidative stress in non-pregnant and pregnant rabbits.** *Int J Radiat Biol* 2012, **88**(5):414-419.

344. Vasan S, Veerachari S: **Mobile Phone Electromagnetic Waves and Its Effect on Human Ejaculated Semen: An in vitro Study.** *International Journal of Infertility & Fetal Medicine* 2012, **3**(1):15-21.
345. Nakatani-Enomoto S, Okutsu M, Suzuki S, Suganuma R, Groiss SJ, Kadowaki S, Enomoto H, Fujimori K, Ugawa Y: **Effects of 1950 MHz W-CDMA-like signal on human spermatozoa.** *Bioelectromagnetics* 2016, **37**(6):373-381.
346. Ding SS, Sun P, Zhang Z, Liu X, Tian H, Huo YW, Wang LR, Han Y, Xing JP: **Moderate Dose of Trolox Preventing the Deleterious Effects of Wi-Fi Radiation on Spermatozoa In vitro through Reduction of Oxidative Stress Damage.** *Chinese Medical Journal* 2018, **131**(4):402-412.
347. Lu YS, Huang BT, Huang YX: **Reactive oxygen species formation and apoptosis in human peripheral blood mononuclear cell induced by 900 MHz mobile phone radiation.** *Oxid Med Cell Longev* 2012, **2012**:740280.
348. Kazemi E, Mortazavi SM, Ali-Ghanbari A, Sharifzadeh S, Ranjbaran R, Mostafavi-Pour Z, Zal F, Haghani M: **Effect of 900 MHz Electromagnetic Radiation on the Induction of ROS in Human Peripheral Blood Mononuclear Cells.** *Journal of Biomedical Physics & Engineering* 2015, **5**(3):105-114.
349. Lasalvia M, Scrima R, Perna G, Piccoli C, Capitanio N, Biagi PF, Schiavulli L, Ligonzo T, Centra M, Casamassima G *et al*: **Exposure to 1.8 GHz electromagnetic fields affects morphology, DNA-related Raman spectra and mitochondrial functions in human lympho-monocytes.** *PLoS ONE* 2018, **13**(2):e0192894.
350. Durdik M, Kosik P, Markova E, Somsedikova A, Gajdosechova B, Nikitina E, Horvathova E, Kozics K, Davis D, Belyaev I: **Microwaves from mobile phone induce reactive oxygen species but not DNA damage, preleukemic fusion genes and apoptosis in hematopoietic stem/progenitor cells.** *Scientific Reports* 2019, **9**(1):16182.
351. Tsoy A, Saliev T, Abzhanova E, Turgambayeva A, Kaiyrlykzy A, Akishev M, Saparbayev S, Umbayev B, Askarova S: **The Effects of Mobile Phone Radiofrequency Electromagnetic Fields on beta-Amyloid-Induced Oxidative Stress in Human and Rat Primary Astrocytes.** *Neuroscience* 2019, **408**:46-57.
352. Glaser K, Rohland M, Kleine-Ostmann T, Schrader T, Stopper H, Hintzsche H: **Effect of Radiofrequency Radiation on Human Hematopoietic Stem Cells.** *Radiation Research* 2016, **186**(5):455-465.
353. Xu S, Chen G, Chen C, Sun C, Zhang D, Murbach M, Kuster N, Zeng Q, Xu Z: **Cell type-dependent induction of DNA damage by 1800 MHz radiofrequency electromagnetic fields does not result in significant cellular dysfunctions.** *PLoS One* 2013, **8**(1):e54906.
354. Silva V, Hilly O, Strenov Y, Tzabari C, Hauptman Y, Feinmesser R: **Effect of cell phone-like electromagnetic radiation on primary human thyroid cells.** *Int J Radiat Biol* 2016, **92**(2):107-115.
355. Ozsobaci NP, Ergun DD, Tuncdemir M, Ozcelik D: **Protective Effects of Zinc on 2.45 GHz Electromagnetic Radiation-Induced Oxidative Stress and Apoptosis in HEK293 Cells.** *Biol Trace Elem Res* 2020, **194**(2):368-378.

356. Pastaci Ozsobaci N, Duzgun Ergun D, Durmus S, Tuncdemir M, Uzun H, Gelisgen R, Ozcelik D: **Selenium supplementation ameliorates electromagnetic field-induced oxidative stress in the HEK293 cells.** *J Trace Elem Med Biol* 2018, **50**:572-579.
357. Sefidbakht Y, Moosavi-Movahedi AA, Hosseinkhani S, Khodaghali F, Torkzadeh-Mahani M, Foolad F, Faraji-Dana R: **Effects of 940 MHz EMF on bioluminescence and oxidative response of stable luciferase producing HEK cells.** *Photochemical & Photobiological Sciences : Official Journal of the European Photochemistry Association and the European Society for Photobiology* 2014, **13**(7):1082-1092.
358. Sun Y, Zong L, Gao Z, Zhu S, Tong J, Cao Y: **Mitochondrial DNA damage and oxidative damage in HL-60 cells exposed to 900MHz radiofrequency fields.** *Mutation research* 2017, **797-799**:7-14.
359. Naziroglu M, Cig B, Dogan S, Uguz AC, Dilek S, Faouzi D: **2.45-Gz wireless devices induce oxidative stress and proliferation through cytosolic Ca(2)(+) influx in human leukemia cancer cells.** *Int J Radiat Biol* 2012, **88**(6):449-456.
360. von Niederhausern N, Ducray A, Zielinski J, Murbach M, Mevissen M: **Effects of radiofrequency electromagnetic field exposure on neuronal differentiation and mitochondrial function in SH-SY5Y cells.** *Toxicology in Vitro* 2019, **61**:104609.
361. Poulletier de Gannes F, Haro E, Hurtier A, Taxile M, Ruffie G, Billaudel B, Veyret B, Lagroye I: **Effect of exposure to the edge signal on oxidative stress in brain cell models.** *Radiation Research* 2011, **175**(2):225-230.
362. Kang KA, Lee HC, Lee JJ, Hong MN, Park MJ, Lee YS, Choi HD, Kim N, Ko YG, Lee JS: **Effects of combined radiofrequency radiation exposure on levels of reactive oxygen species in neuronal cells.** *Journal of Radiation Research* 2014, **55**(2):265-276.
363. Stefi AL, Margaritis LH, Skouroliakou AS, Vassilacopoulou D: **Mobile phone electromagnetic radiation affects Amyloid Precursor Protein and alpha-synuclein metabolism in SH-SY5Y cells.** *Pathophysiology* 2019, **26**(3-4):203-212.
364. Zielinski J, Ducray AD, Moeller AM, Murbach M, Kuster N, Mevissen M: **Effects of pulse-modulated radiofrequency magnetic field (RF-EMF) exposure on apoptosis, autophagy, oxidative stress and electron chain transport function in human neuroblastoma and murine microglial cells.** *Toxicology in Vitro* 2020, **68**:104963.
365. Marjanovic Cermak AM, Pavicic I, Trosic I: **Oxidative stress response in SH-SY5Y cells exposed to short-term 1800 MHz radiofrequency radiation.** *J Environ Sci Health A Tox Hazard Subst Environ Eng* 2018, **53**(2):132-138.
366. Choi J, Min K, Jeon S, Kim N, Pack JK, Song K: **Continuous Exposure to 1.7 GHz LTE Electromagnetic Fields Increases Intracellular Reactive Oxygen Species to Decrease Human Cell Proliferation and Induce Senescence.** *Scientific Reports* 2020, **10**(1):9238.
367. Cig B, Naziroglu M: **Investigation of the effects of distance from sources on apoptosis, oxidative stress and cytosolic calcium accumulation via TRPV1 channels induced by mobile phones and Wi-Fi in breast cancer cells.** *Biochim Biophys Acta* 2015, **1848**(10 Pt B):2756-2765.

368. Kahya MC, Naziroglu M, Cig B: **Selenium reduces mobile phone (900 MHz)-induced oxidative stress, mitochondrial function, and apoptosis in breast cancer cells.** *Biological trace element research* 2014, **160**(2):285-293.
369. Ni S, Yu Y, Zhang Y, Wu W, Lai K, Yao K: **Study of oxidative stress in human lens epithelial cells exposed to 1.8 GHz radiofrequency fields.** *PLoS One* 2013, **8**(8):e72370.
370. Hong MN, Kim BC, Ko YG, Lee YS, Hong SC, Kim T, Pack JK, Choi HD, Kim N, Lee JS: **Effects of 837 and 1950 MHz radiofrequency radiation exposure alone or combined on oxidative stress in MCF10A cells.** *Bioelectromagnetics* 2012, **33**(7):604-611.
371. Qin F, Shen T, Cao H, Qian J, Zou D, Ye M, Pei H: **CeO<sub>2</sub>NPs relieve radiofrequency radiation, improve testosterone synthesis, and clock gene expression in Leydig cells by enhancing antioxidation.** *Int J Nanomedicine* 2019, **14**:4601-4611.
372. Koohestani NV, Zavareh S, Lashkarbolouki T, Azimipour F: **Exposure to cell phone induce oxidative stress in mice preantral follicles during in vitro cultivation: An experimental study.** *Int J Reprod Biomed (Yazd)* 2019, **17**(9):637-646.
373. Houston BJ, Nixon B, King BV, Aitken RJ, De Luliis GN: **Probing the Origins of 1,800 MHz Radio Frequency Electromagnetic Radiation Induced Damage in Mouse Immortalized Germ Cells and Spermatozoa in vitro.** *Frontiers in public health* 2018, **6**:270.
374. Hou Q, Wang M, Wu S, Ma X, An G, Liu H, Xie F: **Oxidative changes and apoptosis induced by 1800-MHz electromagnetic radiation in NIH/3T3 cells.** *Electromagnetic Biology and Medicine* 2015, **34**(1):85-92.
375. Xing F, Zhan Q, He Y, Cui J, He S, Wang G: **1800MHz Microwave Induces p53 and p53-Mediated Caspase-3 Activation Leading to Cell Apoptosis In Vitro.** *PLoS One* 2016, **11**(9):e0163935.
376. Li R, Ma M, Li L, Zhao L, Zhang T, Gao X, Zhang D, Zhu Y, Peng Q, Luo X *et al*: **The Protective Effect of Autophagy on DNA Damage in Mouse Spermatocyte-Derived Cells Exposed to 1800 MHz Radiofrequency Electromagnetic Fields.** *Cellular Physiology and Biochemistry* 2018, **48**(1):29-41.
377. Liu C, Duan W, Xu S, Chen C, He M, Zhang L, Yu Z, Zhou Z: **Exposure to 1800 MHz radiofrequency electromagnetic radiation induces oxidative DNA base damage in a mouse spermatocyte-derived cell line.** *Toxicology Letters* 2013, **218**(1):2-9.
378. Liu K, Zhang G, Wang Z, Liu Y, Dong J, Dong X, Liu J, Cao J, Ao L, Zhang S: **The protective effect of autophagy on mouse spermatocyte derived cells exposure to 1800MHz radiofrequency electromagnetic radiation.** *Toxicology Letters* 2014, **228**(3):216-224.
379. Lu Y, He M, Zhang Y, Xu S, Zhang L, He Y, Chen C, Liu C, Pi H, Yu Z *et al*: **Differential pro-inflammatory responses of astrocytes and microglia involve STAT3 activation in response to 1800 MHz radiofrequency fields.** *PLoS ONE* 2014, **9**(9):e108318.
380. He GL, Liu Y, Li M, Chen CH, Gao P, Yu ZP, Yang XS: **The amelioration of phagocytic ability in microglial cells by curcumin through the inhibition of EMF-induced pro-inflammatory responses.** *Journal of neuroinflammation* 2014, **11**:49.

381. Wang X, Liu C, Ma Q, Feng W, Yang L, Lu Y, Zhou Z, Yu Z, Li W, Zhang L: **8-oxoG DNA glycosylase-1 inhibition sensitizes Neuro-2a cells to oxidative DNA base damage induced by 900 MHz radiofrequency electromagnetic radiation.** *Cell Physiol Biochem* 2015, **37**(3):1075-1088.
382. Kim JY, Kim HJ, Kim N, Kwon JH, Park MJ: **Effects of radiofrequency field exposure on glutamate-induced oxidative stress in mouse hippocampal HT22 cells.** *Int J Radiat Biol* 2017, **93**(2):249-256.
383. Lee JS, Kim JY, Kim HJ, Kim JC, Lee JS, Kim N, Park MJ: **Effects of combined radiofrequency field exposure on amyloid-beta-induced cytotoxicity in HT22 mouse hippocampal neurones.** *J Radiat Res* 2016, **57**(6):620-626.
384. Lopez-Furelos A, Salas-Sanchez AA, Ares-Pena FJ, Leiro-Vidal JM, Lopez-Martin E: **Exposure to radiation from single or combined radio frequencies provokes macrophage dysfunction in the RAW 264.7 cell line.** *International Journal of Radiation Biology* 2018, **94**(6):607-618.
385. Lin YY, Wu T, Liu JY, Gao P, Li KC, Guo QY, Yuan M, Lang HY, Zeng LH, Guo GZ: **1950MHz Radio Frequency Electromagnetic Radiation Inhibits Testosterone Secretion of Mouse Leydig Cells.** *Int J Environ Res Public Health* 2017, **15**(1).
386. Zuo WQ, Hu YJ, Yang Y, Zhao XY, Zhang YY, Kong W, Kong WJ: **Sensitivity of spiral ganglion neurons to damage caused by mobile phone electromagnetic radiation will increase in lipopolysaccharide-induced inflammation in vitro model.** *J Neuroinflammation* 2015, **12**:105.
387. Marjanovic AM, Pavicic I, Trosic I: **Cell oxidation-reduction imbalance after modulated radiofrequency radiation.** *Electromagnetic Biology and Medicine* 2015, **34**(4):381-386.
388. Marjanovic Cermak AM, Pavicic I, Tariba Lovakovic B, Pizent A, Trosic I: **In vitro non-thermal oxidative stress response after 1800 MHz radiofrequency radiation.** *General physiology and biophysics* 2017, **36**(4):407-414.
389. Jooyan N, Goliaei B, Bigdeli B, Faraji-Dana R, Zamani A, Entezami M, Mortazavi SMJ: **Direct and indirect effects of exposure to 900MHz GSM radiofrequency electromagnetic fields on CHO cell line: Evidence of bystander effect by non-ionizing radiation.** *Environmental Research* 2019, **174**:176-187.
390. Meena R, Kumari K, Kumar J, Rajamani P, Verma HN, Kesari KK: **Therapeutic approaches of melatonin in microwave radiations-induced oxidative stress-mediated toxicity on male fertility pattern of Wistar rats.** *Electromagn Biol Med* 2014, **33**(2):81-91.
391. Pandey N, Giri S: **Melatonin attenuates radiofrequency radiation (900 MHz)-induced oxidative stress, DNA damage and cell cycle arrest in germ cells of male Swiss albino mice.** *Toxicol Ind Health* 2018, **34**(5):315-327.
392. Kumar S, Nirala JP, Behari J, Paulraj R: **Effect of electromagnetic irradiation produced by 3G mobile phone on male rat reproductive system in a simulated scenario.** *Indian J Exp Biol* 2014, **52**(9):890-897.

393. Jeong YJ, Son Y, Han NK, Choi HD, Paek JK, Kim N, Lee YS, Lee HJ: **Impact of Long-Term RF-EMF on Oxidative Stress and Neuroinflammation in Aging Brains of C57BL/6 Mice.** *Int J Mol Sci* 2018, **19**(7).
394. Yang SY, Zhang TX, Cui YF, Zhang C, Wang SM, Xu H, Jin W: **Effect of high power microwave on gene expressio of immune tissues in rats.** *Chinese Journal of Clinical Rehabilitation* 2006, **10**(29):132-137.
395. Vanishree M, Manvikar V, Rudraraju A, Reddy KMP, Kumar NHP, Quadri SJM: **Significance of micronuclei in buccal smears of mobile phone users: A comparative study.** *J Oral Maxillofac Pathol* 2018, **22**(3):448.
396. de Oliveira FM, Carmona AM, Ladeira C: **Is mobile phone radiation genotoxic? An analysis of micronucleus frequency in exfoliated buccal cells.** *Mutation research* 2017, **822**:41-46.
397. Gulati S, Yadav A, Kumar N, Kanupriya, Aggarwal NK, Kumar R, Gupta R: **Effect of GSTM1 and GSTT1 Polymorphisms on Genetic Damage in Humans Populations Exposed to Radiation From Mobile Towers.** *Arch Environ Contam Toxicol* 2016, **70**(3):615-625.
398. Banerjee S, Singh NN, Sreedhar G, Mukherjee S: **Analysis of the Genotoxic Effects of Mobile Phone Radiation using Buccal Micronucleus Assay: A Comparative Evaluation.** *J Clin Diagn Res* 2016, **10**(3):ZC82-85.
399. Daroit NB, Visioli F, Magnusson AS, Vieira GR, Rados PV: **Cell phone radiation effects on cytogenetic abnormalities of oral mucosal cells.** *Braz Oral Res* 2015, **29**(1):1-8.
400. Souza Lda C, Cerqueira Ede M, Meireles JR: **Assessment of nuclear abnormalities in exfoliated cells from the oral epithelium of mobile phone users.** *Electromagn Biol Med* 2014, **33**(2):98-102.
401. Ros-Llor I, Sanchez-Siles M, Camacho-Alonso F, Lopez-Jornet P: **Effect of mobile phones on micronucleus frequency in human exfoliated oral mucosal cells.** *Oral Dis* 2012, **18**(8):786-792.
402. Radwan M, Jurewicz J, Merecz-Kot D, Sobala W, Radwan P, Bochenek M, Hanke W: **Sperm DNA damage-the effect of stress and everyday life factors.** *Int J Impot Res* 2016, **28**(4):148-154.
403. Jurewicz J, Radwan M, Sobala W, Ligocka D, Radwan P, Bochenek M, Hanke W: **Lifestyle and semen quality: role of modifiable risk factors.** *Syst Biol Reprod Med* 2014, **60**(1):43-51.
404. Gandhi G, Kaur G, Nisar U: **A cross-sectional case control study on genetic damage in individuals residing in the vicinity of a mobile phone base station.** *Electromagn Biol Med* 2015, **34**(4):344-354.
405. Cam ST, Seyhan N: **Single-strand DNA breaks in human hair root cells exposed to mobile phone radiation.** *Int J Radiat Biol* 2012, **88**(5):420-424.
406. Jiang B, Zong C, Zhao H, Ji Y, Tong J, Cao Y: **Induction of adaptive response in mice exposed to 900MHz radiofrequency fields: application of micronucleus assay.** *Mutation research* 2013, **751**(2):127-129.

407. Jiang B, Nie J, Zhou Z, Zhang J, Tong J, Cao Y: **Adaptive response in mice exposed to 900 MHz radiofrequency fields: primary DNA damage.** *PLoS One* 2012, **7**(2):e32040.
408. Chaturvedi CM, Singh VP, Singh P, Basu P, Singaravel M, Shukla RK, Dhawan A, Pati AK, Gangwar RK, Singh SP: **2.45 GHz (CW) MICROWAVE IRRADIATION ALTERS CIRCADIAN ORGANIZATION, SPATIAL MEMORY, DNA STRUCTURE IN THE BRAIN CELLS AND BLOOD CELL COUNTS OF MALE MICE, MUS MUSCULUS.** *Progress In Electromagnetics Research B* 2011, **29**:23-42.
409. Usikalu, M R, Obembe, O O, Akinyemi, M L, Zhu, J: **Short-duration exposure to 2.45 GHz microwave radiation induces DNA damage in Sprague Dawley rats reproductive systems.** *African Journal of Biotechnology* 2013, **12**(2):115-122.
410. Akdag MZ, Dasdag S, Canturk F, Karabulut D, Caner Y, Adalier N: **Does prolonged radiofrequency radiation emitted from Wi-Fi devices induce DNA damage in various tissues of rats?** *J Chem Neuroanat* 2016, **75**(Pt B):116-122.
411. Gurbuz N, Sirav B, Colbay M, Yetkin I, Seyhan N: **No genotoxic effect in exfoliated bladder cells of rat under the exposure of 1800 and 2100 MHz radio frequency radiation.** *Electromagn Biol Med* 2014, **33**(4):296-301.
412. Atli Sekeroglu Z, Akar A, Sekeroglu V: **Evaluation of the cytogenotoxic damage in immature and mature rats exposed to 900 MHz radiofrequency electromagnetic fields.** *Int J Radiat Biol* 2013, **89**(11):985-992.
413. Sekeroglu V, Akar A, Sekeroglu ZA: **Cytotoxic and genotoxic effects of high-frequency electromagnetic fields (GSM 1800 MHz) on immature and mature rats.** *Ecotoxicol Environ Saf* 2012, **80**:140-144.
414. Trosic I, Pavicic I, Milkovic-Kraus S, Mladinic M, Zeljezic D: **Effect of electromagnetic radiofrequency radiation on the rats' brain, liver and kidney cells measured by comet assay.** *Coll Antropol* 2011, **35**(4):1259-1264.
415. Gouda EM, Galal MK, Abdalaziz SA: **Adverse Effect of Mobile Phone on TP53, BRCA1 Genes and DNA Fragmentation in Albino Rat Liver.** *International Journal of Genomics and Proteomics* 2013, **4**(1):84-88.
416. Deshmukh PS, Megha K, Banerjee BD, Ahmed RS, Chandna S, Abegaonkar MP, Tripathi AK: **Detection of Low Level Microwave Radiation Induced Deoxyribonucleic Acid Damage Vis-a-vis Genotoxicity in Brain of Fischer Rats.** *Toxicol Int* 2013, **20**(1):19-24.
417. Deshmukh PS, Nasare N, Megha K, Banerjee BD, Ahmed RS, Singh D, Abegaonkar MP, Tripathi AK, Mediratta PK: **Cognitive impairment and neurogenotoxic effects in rats exposed to low-intensity microwave radiation.** *Int J Toxicol* 2015, **34**(3):284-290.
418. Deshmukh PS, Megha K, Nasare N, Banerjee BD, Ahmed RS, Abegaonkar MP, Tripathi AK, Mediratta PK: **Effect of Low Level Subchronic Microwave Radiation on Rat Brain.** *Biomed Environ Sci* 2016, **29**(12):858-867.
419. Danese E, Lippi G, Buonocore R, Benati M, Bovo C, Bonaguri C, Salvagno GL, Brocco G, Roggenbuck D, Montagnana M: **Mobile phone radiofrequency exposure has no**

- effect on DNA double strand breaks (DSB) in human lymphocytes.** *Ann Transl Med* 2017, 5(13):272.
420. Waldmann P, Bohnenberger S, Greinert R, Hermann-Then B, Heselich A, Klug SJ, Koenig J, Kuhr K, Kuster N, Merker M *et al*: **Influence of GSM signals on human peripheral lymphocytes: study of genotoxicity.** *Radiat Res* 2013, **179**(2):243-253.
421. Sannino A, Zeni O, Romeo S, Massa R, Gialanella G, Grossi G, Manti L, Vijayalaxmi, Scarfi MR: **Adaptive response in human blood lymphocytes exposed to non-ionizing radiofrequency fields: resistance to ionizing radiation-induced damage.** *J Radiat Res* 2014, **55**(2):210-217.
422. Zeni O, Sannino A, Romeo S, Massa R, Sarti M, Reddy AB, Prihoda TJ, Vijayalaxmi, Scarfi MR: **Induction of an adaptive response in human blood lymphocytes exposed to radiofrequency fields: influence of the universal mobile telecommunication system (UMTS) signal and the specific absorption rate.** *Mutation research* 2012, **747**(1):29-35.
423. Vijayalaxmi, Reddy AB, McKenzie RJ, McIntosh RL, Prihoda TJ, Wood AW: **Incidence of micronuclei in human peripheral blood lymphocytes exposed to modulated and unmodulated 2450 MHz radiofrequency fields.** *Bioelectromagnetics* 2013, **34**(7):542-548.
424. Falone S, Sannino A, Romeo S, Zeni O, Santini SJ, Rispoli R, Amicarelli F, Scarfi MR: **Protective effect of 1950 MHz electromagnetic field in human neuroblastoma cells challenged with menadione.** *Sci Rep* 2018, **8**(1):13234.
425. Al-Serori H, Ferk F, Kundi M, Bileck A, Gerner C, Misik M, Nersesyan A, Waldherr M, Murbach M, Lah TT *et al*: **Mobile phone specific electromagnetic fields induce transient DNA damage and nucleotide excision repair in serum-deprived human glioblastoma cells.** *PLoS One* 2018, **13**(4):e0193677.
426. Su L, Wei X, Xu Z, Chen G: **RF-EMF exposure at 1800 MHz did not elicit DNA damage or abnormal cellular behaviors in different neurogenic cells.** *Bioelectromagnetics* 2017, **38**(3):175-185.
427. Ozgur E, Guler G, Kismali G, Seyhan N: **Mobile phone radiation alters proliferation of hepatocarcinoma cells.** *Cell Biochem Biophys* 2014, **70**(2):983-991.
428. Zhijian C, Xiaoxue L, Wei Z, Yezhen L, Jianlin L, Deqiang L, Shijie C, Lifen J, Jiliang H: **Studying the protein expression in human B lymphoblastoid cells exposed to 1.8-GHz (GSM) radiofrequency radiation (RFR) with protein microarray.** *Biochem Biophys Res Commun* 2013, **433**(1):36-39.
429. Speit G, Gminski R, Tauber R: **Genotoxic effects of exposure to radiofrequency electromagnetic fields (RF-EMF) in HL-60 cells are not reproducible.** *Mutation research* 2013, **755**(2):163-166.
430. Hintzsche H, Jastrow C, Kleine-Ostmann T, Schrader T, Stopper H: **900 MHz radiation does not induce micronucleus formation in different cell types.** *Mutagenesis* 2012, **27**(4):477-483.
431. Schrader T, Kleine-Ostmann T, Munter K, Jastrow C, Schmid E: **Spindle disturbances in human-hamster hybrid (A(L) ) cells induced by the electrical component of the**

- mobile communication frequency range signal. *Bioelectromagnetics* 2011, **32**(4):291-301.
432. He Q, Sun Y, Zong L, Tong J, Cao Y: **Induction of Poly(ADP-ribose) Polymerase in Mouse Bone Marrow Stromal Cells Exposed to 900 MHz Radiofrequency Fields: Preliminary Observations.** *Biomed Res Int* 2016, **2016**:4918691.
433. Ji Y, He Q, Sun Y, Tong J, Cao Y: **Adaptive response in mouse bone-marrow stromal cells exposed to 900-MHz radiofrequency fields: Gamma-radiation-induced DNA strand breaks and repair.** *J Toxicol Environ Health A* 2016, **79**(9-10):419-426.
434. He Q, Zong L, Sun Y, Vijayalaxmi, Prihoda TJ, Tong J, Cao Y: **Adaptive response in mouse bone marrow stromal cells exposed to 900MHz radiofrequency fields: Impact of poly (ADP-ribose) polymerase (PARP).** *Mutation research* 2017, **820**:19-25.
435. Suzuki S, Okutsu M, Suganuma R, Komiya H, Nakatani-Enomoto S, Kobayashi S, Ugawa Y, Tateno H, Fujimori K: **Influence of radiofrequency-electromagnetic waves from 3rd-generation cellular phones on fertilization and embryo development in mice.** *Bioelectromagnetics* 2017, **38**(6):466-473.
436. Duan W, Liu C, Zhang L, He M, Xu S, Chen C, Pi H, Gao P, Zhang Y, Zhong M *et al*: **Comparison of the genotoxic effects induced by 50 Hz extremely low-frequency electromagnetic fields and 1800 MHz radiofrequency electromagnetic fields in GC-2 cells.** *Radiat Res* 2015, **183**(3):305-314.
437. Liu C, Gao P, Xu SC, Wang Y, Chen CH, He MD, Yu ZP, Zhang L, Zhou Z: **Mobile phone radiation induces mode-dependent DNA damage in a mouse spermatocyte-derived cell line: a protective role of melatonin.** *Int J Radiat Biol* 2013, **89**(11):993-1001.
438. Sun C, Wei X, Fei Y, Su L, Zhao X, Chen G, Xu Z: **Mobile phone signal exposure triggers a hormesis-like effect in Atm(+/-) and Atm(-/-) mouse embryonic fibroblasts.** *Sci Rep* 2016, **6**:37423.
439. Herrala M, Mustafa E, Naarala J, Juutilainen J: **Assessment of genotoxicity and genomic instability in rat primary astrocytes exposed to 872 MHz radiofrequency radiation and chemicals.** *Int J Radiat Biol* 2018, **94**(10):883-889.
440. Kumar G, Wood AW, Anderson V, McIntosh RL, Chen YY, McKenzie RJ: **Evaluation of hematopoietic system effects after in vitro radiofrequency radiation exposure in rats.** *Int J Radiat Biol* 2011, **87**(2):231-240.
441. Zeni O, Sannino A, Sarti M, Romeo S, Massa R, Scarfi MR: **Radiofrequency radiation at 1950 MHz (UMTS) does not affect key cellular endpoints in neuron-like PC12 cells.** *Bioelectromagnetics* 2012, **33**(6):497-507.
442. Ballardini M, Tusa I, Fontana N, Monorchio A, Pelletti C, Rogovich A, Barale R, Scarpato R: **Non-thermal effects of 2.45 GHz microwaves on spindle assembly, mitotic cells and viability of Chinese hamster V-79 cells.** *Mutation research* 2011, **716**(1-2):1-9.
443. Sannino A, Zeni O, Romeo S, Massa R, Scarfi MR: **Adverse and beneficial effects in Chinese hamster lung fibroblast cells following radiofrequency exposure.** *Bioelectromagnetics* 2017, **38**(4):245-254.

444. Lai H: **Genetic effects of non-ionizing electromagnetic fields.** *Electromagn Biol Med* 2021:1-10.

## Appendix I: Current CV: Christopher J. Portier

### CURRICULUM VITAE

Christopher J. Portier, Ph.D.

---

**Personal Data:** Birth Date April 3, 1956  
Birthplace Houma, Louisiana

**Addresses:** 4224 Midvale Ave N.  
Seattle, WA  
United States  
Ph: +1-206-395-3308

Scheibenstrasse 15  
CH-3600 Thun  
Switzerland  
Ph: +41-79-605-7958

**Email:** [cportier@mac.com](mailto:cportier@mac.com)

### Education:

1981 Ph.D. (Biostatistics), University of North Carolina, Chapel Hill  
1979 M.S. (Biostatistics), University of North Carolina, Chapel Hill  
1977 B.S. (Mathematics), summa cum laude, Nicholls State University

### Employment:

2018-present **Scientific Advisor**, World Health Organization, Environment Program - Europe  
2016-present **Scientific Advisor on Pesticide Policies**, multiple European Non-Government Organizations  
2013-present **Consultant** to various governmental agencies (multiple countries) and law firms  
2013-2014 **Senior Visiting Scientist**, International Agency for Research on Cancer, Lyon, France  
2013-present **Senior Contributing Scientist**, Environmental Defense Fund, New York City, NY  
2010-2013 **Director**, National Center for Environment Health, Centers for Disease Control and Prevention, Atlanta, GA  
2010-2013 **Director**, Agency for Toxic Substances and Disease Registry, Atlanta, GA  
2009 – 2010 **Senior Advisor to the Director**, National Institute of Environmental Health Sciences and National Toxicology Program, Research Triangle Park, North Carolina.  
2009 – 2010 **Visiting Scientist**, National Research Centre for Environmental Toxicology (EnTox), Queensland, Australia  
2006 - 2009 **Associate Director**, National Institute of Environmental Health Sciences, Research Triangle Park, North Carolina.

2006 - 2009	<b>Director, Office of Risk Assessment Research</b> , National Institute of Environmental Health Sciences, Research Triangle Park, North Carolina.
1993 – 2010	<b>Head, Environmental Systems Biology</b> (originally Stochastic Modeling), Laboratory of Molecular Toxicology, National Institute of Environmental Health Sciences, Research Triangle Park, North Carolina.
2000 - 2006	<b>Associate Director, National Toxicology Program</b> , National Institute of Environmental Health Sciences, Division of Intramural Research, Research Triangle Park, North Carolina.
2000 - 2006	<b>Director, Environmental Toxicology Program</b> , National Institute of Environmental Health Sciences, Division of Intramural Research, Research Triangle Park, North Carolina.
2006-2007	<b>Scientific Advisor to the Director</b> , Public Health and the Environment Department, World Health Organization, Geneva, Switzerland (detail from NIEHS – four months)
1993 - 2005	<b>Chief, Laboratory of Computational Biology and Risk Analysis</b> (originally the Laboratory of Quantitative and Computational Biology), National Institute of Environmental Health Sciences, Division of Intramural Research, Research Triangle Park, North Carolina.
1996 - 2000	<b>Associate Director for Risk Assessment</b> , Environmental Toxicology Program National Institute of Environmental Health Sciences, Division of Intramural Research, Research Triangle Park, North Carolina.
1990 - 1993	<b>Head, Risk Methodology Section</b> , National Institute of Environmental Health Sciences, Division of Biometry and Risk Assessment, Research Triangle Park, North Carolina.
1987, 1992, 1990	<b>Guest Scientist</b> , German Cancer Research Center, Heidelberg, Germany.
1978 - 1990	<b>Principal Investigator</b> , National Institute of Environmental Health Sciences, Division of Biometry and Risk Assessment, Research Triangle Park, North Carolina.
1977	<b>Mathematician</b> , Computer Sciences Division, Oak Ridge National Laboratory, Oak Ridge, Tennessee.
1976	<b>Undergraduate Research Trainee</b> , Neutron Physics Division, Oak Ridge National Laboratory, Oak Ridge, Tennessee.

*University Affiliations:*

2014 – present	Visiting Professor, Department of Toxicogenomics, Maastricht University, The Netherlands
2013 – 2016	Honorary Professor, National Research Centre for Environmental Toxicology, University of Queensland, Brisbane, Australia
2011 – present	Adjunct Professor, Department of Environmental Health, Emory University, Atlanta, GA, USA
2009 – 2010	Visiting Professor, University of Queensland, Brisbane, Australia
1986 - 2007	Adjunct Professor of Biostatistics, University of North Carolina, School of Public Health, Chapel Hill, North Carolina.
1990-1992	Adjunct Professor of Statistics, University of Waterloo, Waterloo, Ontario, Canada

*Honors & Awards:*

- 2016 Elected Fellow, Collegium Ramazzini
- 2013 President’s Dream Green Team Award for “A Human Health Perspective on Climate

Change”

- Fellow, World Innovation Foundation, 2006
- Society of Toxicology, Risk Assessment Specialty Section, Paper of the Year, 2006
- Society of Toxicology, Risk Assessment Specialty Section, Paper of the Year, 2005
- Outstanding Risk Practitioner Award, International Society for Risk Analysis, 2000.
- Elected Fellow, International Statistical Institute, 2000.
- Outstanding Performance Award, National Institute of Environmental Health Sciences, numerous dates.
- Commendation for Sustained High Quality Work Performance, National Institute of Environmental Health Sciences, numerous dates.
- Merit Award, National Institute of Health, 1998.
- Board of Publications, Best Paper Award, Society of Toxicology, 1995.
- Distinguished Achievement Award, Section on Statistics and the Environment, American Statistical Association, 1995.
- Spiegelman Award presented by the American Public Health Association to the most outstanding public health statistician under the age of 40, 1995.
- Best-applied statistics paper, Centers for Disease Control, 1993.
- Elected Fellow, American Statistical Association, 1992.
- Elected Foreign Correspondent, Russian National Academy of Natural Sciences, 1992.
- First recipient of the James E. Grizzle Distinguished Alumnus Award, The Department of Biostatistics, The University of North Carolina, 1991.

*Professional Societies Membership:*

Society of Toxicology, American Public Health Association, International Statistics Institute, Bioelectromagnetics Society

*Editorial Activities:*

- Editor in Chief - The Open Environmental Journal (2008 to 2010)
- Associate Editor – Frontiers in Predictive Toxicity (2010 to present)
- Associate Editor - Environmental Health Perspectives (1987-2006)
- Associate Editor - Risk Analysis: An International Journal (1989-2003)
- Editorial Board – Environmental and Ecological Statistics (2004-2007)
- Associate Editor – Statistics in Medicine (1998-2002)
- Associate Editor - Biometrics (1997-99)
- Editorial Board Member/Reviewer (different dates): Biometrika, Cancer Research, Communications in Statistics, Fundamental and Applied Toxicology, Journal of Applied Toxicology, Journal of the American Statistical Association, Journal of Toxicology and Environmental Health, Science, Mathematical Biosciences, Journal of Mathematical Biology, Carcinogenesis, Science, PNAS, Toxicological Sciences and others

*Advisory & Review Committees:*

2019-present	Member, UCSF PHRE Science Response Network
2016-2020	Member, World Health Organization Regional Office Europe. Setting research priorities in environment and health
2015 – 2016	Member, Committee to Review the Draft Interagency Report on the Impacts of Climate Change on Human Health in the United States, National Research Council, National Academy of Sciences, USA
2010 – 2016	Member, Science Advisory Group on Electromagnetic Fields and Health, Netherlands Organisation for Health Research and Development

2009 – 2010 Coordinating Lead Author, Interagency Working Group on Climate Change and Health

2009 – 2013 Member, Institute of Medicine Roundtable on Environmental Health Sciences Research and Medicine

2009 – 2012 Member, National Academies of Science Roundtable on Science and Technology for Sustainability

2009 Member, WHO Advisory group on the health implications of the use of DDT to reduce risks of malaria.

2005 – 2010 Chair, Subcommittee on Toxics and Risk, President's National Council on Science and Technology

1997 - 2012 Advisor, *World Health Organization*, International Program on Chemical Safety, EMF Project.

2008 – 2010 Member, Environmental Protection Agency, Science Advisory Board

2007 – 2010 Member, International Life Sciences Institute, Health and Environmental Sciences Institute, Subcommittee on Susceptible Populations

2008 Center Review Committee, Canadian National Science and Engineering Research Council Chair in Risk Assessment

2008 Chair, International Agency for Research on Cancer Monographs Advisory Group, Lyon, France

2008 Advisory Group, Center for Environmental Oncology, University of Pittsburgh Cancer Institute

2007. Chair, WHO Workshop on Low Cost Options for Reducing Exposures to ELF-EMF, Geneva

2007. Invited Participant, International Program on Chemical Safety Workshop on Aggregate and Cumulative Risk Assessment, Washington, DC.

2006 Rapporteur, International Agency for Research on Cancer, Scientific Advisory Group to Plan Volume 100 of the IARC Monograph Series

2005 Chair, International Agency for Research on Cancer, Scientific Advisory Board on the Preamble to the Cancer Monograph Series

2005 Chair, World Health Organization Expert Panel on Health Criteria Document for Extremely Low Frequency Electric and Magnetic Fields

2003 – 2005 Co-Chair, Subcommittee on Health and Environment, President's National Council on Science and Technology

2003 Ad-Hoc member, EPA Science Advisory Board, Review of Children's Cancer Risk Assessment Supplement to Cancer Guidelines

2002 – 2006 Co-Chair, Subcommittee on Mercury, President's National Council on Science and Technology

2000 – 2007 Member, Finish Academy of Sciences Centers of Excellence Program Science Advisory Committee

2000 Reviewer, *Congressional Research Service, Library of Congress*; Research needs relevant to children's environmental health risks.

1998 - 2004 Member and Chair, *Environmental Protection Agency*, FIFRA Science Advisory Panel.

1997 - 2006 Member, National Occupational Research Agenda Team, *National Institute of Occupational Safety and Health*.

1995 - 2000 Advisor, *Australian Health Council*, Risk Assessment Methodology, Member *NHMRC* Steering Committee on Cancer Risk Assessment Guidelines.

1992 - 2000 Member, *EPA* Dioxin Reassessment Working Group.

1985 - 2007	Thesis director for graduate students, Department of Biostatistics, <i>University of North Carolina - Chapel Hill, North Carolina.</i>
1997	Advisor, <i>Netherlands National Health Council, Risk Assessment Methodology.</i>
1997	Reviewer, <i>Air Force Office of Scientific Research.</i>
1996 - 1997	Temporary Advisor, <i>World Health Organization, Expert Committee on Food Additives.</i>
1996	Advisor, <i>Environmental Protection Agency; Evaluation of the benchmark dose methodology.</i>
1996	Advisor, <i>Environmental Protection Agency; Evaluation of risks from exposure to PCBs.</i>
1996	Expert Review Committee, <i>Environmental Protection Agency; Cancer dose-response for PCB's.</i>
1995 - 1996	Member, <i>California Environmental Protection Agency; Risk Assessment Advisory Committee.</i>
1994 - 1997	Science Advisory Panel, <i>Public Broadcasting System Production "Poisons in the Womb".</i>
1991 - 1995	Ad-Hoc Member, <i>Environmental Protection Agency; Science Advisory Panel.</i>

#### Legislative Hearings:

- Glyphosate Hearing, European Parliament, Brussels, October, 2017
- Glyphosate Carcinogenicity, European Parliament, Brussels, December 2015
- Glyphosate Carcinogenicity, German Parliament, Berlin, July 2015
- Lead and Children's Health, Senate Committee on Environment and Public Works, July, 2012
- Asthma and Children's Health, Senate Committee on Environment and Public Works, May, 2012
- Contaminated Drywall, Senate Committee on Commerce, Science and Transportation, December, 2012.
- Camp Lejeune Contaminated Drinking Water, House Committee on Science and Technology, September, 2010.
- Autism and Vaccines, House Committee on Government Reform, December, 2002.

#### US Government Service Activities:

- Member, President's Task Force on Environmental Justice 2010-2013
- Member, President's Task Force on Children's Environmental Health 2009-2013
- Member, National Toxicology Program Executive Committee 2010-2013
- Financial Support and International Press Conference for research on "The Health Benefits of Tackling Climate Change" appearing as a series in *Lancet*, November 25, 2009
- Organizing Committee, White House Stakeholder briefing on Climate Change and Human Health, Old Executive Office Building, November 2009.
- Member, US Delegation, World Climate Congress, Geneva (September 2009)
- Member, US Delegation, Global Risk Communication Dialogue (2008-2009)
- Member, NIEHS Corrective Action Plan Management Committee (2008-2009)
- Primary focus, all interagency activities on hazards and risk (2006 to present)
- Co-Organizer, NIEHS/EPA Workshop on Children's Environmental Health, RTP, NC, January, (2007)
- Co-Organizer, NIEHS/NTP Workshop on the Identification of Targets for the HTS Roadmap Project (2007)
- Coordinator, NIEHS/EPA Review of the Children's Environmental Health Centers Program (2006-2007)
- Organizing Committee, Global Environmental Health Initiative, NIEHS (2006 to 2009)

- NIEHS Leadership Council (2005 to 2009)
- Organizer, formal collaborative agreements between NTP and Ramazzini Foundation (2001 to 2006)
- Organizer, formal collaborative agreements between NTP and Korean NTP (2002 to 2006)
- NIEHS Title 42 Review Committee (2003 to 2004)
- NIEHS Executive Committee and Operations Update Committee (2000 to 2005)
- NIEHS Leadership Retreats, DERT Retreats, DIR Retreats (all years since 1997)
- Presenter, NIEHS-sponsored National Academy of Sciences Committee on Emerging Issues in Environmental Health, November, 2001
- Organizer and presenter, National Toxicology Program Executive Committee Meetings (multiple dates since 2000)
- Organizer and presenter, National Toxicology Program Board of Scientific Counselors (multiple dates since 1998)
- Organizer, Joint NIEHS/US Geological Survey Interagency Program on Exposure Assessment, April 2001 to present)
- Organizer, US-Vietnam Scientific Conference on the Health and Environmental Effects of Agent Orange/Dioxin in Vietnam, March, 2002
- Organizing Committee, National Toxicology Program/EPA/FDA Scientific Conference on the Allergenicity of Genetically Modified Food, November, 2001
- NIEHS Town Hall Meeting, Los Angeles California, November, 2001
- NTP Research Directions, NAEHSC, Research Triangle Park, NC, May, 2001.
- NCI Study Section Center Presite Meeting, Seattle, Washington, January, 2001.
- Program committee member, *NIEHS Colorado State University* conference on the Application of Technology to Chemical Mixture Research, 2001.
- Coordinating Core Committee, National Center for Toxicogenomics, NIEHS, 2000 to present
- Organizer, Joint US-Vietnam Consultation on Research on Agent Orange Health Effects in Vietnam. Singapore, 2000
- *ICCVAM NICEATM*, Up-and-Down Procedure Peer Review Meeting, 2000.
- Chairman, *NIEHS* Risk Assessment Research Committee, 1995-present.
- Discussant, *NIEHS PNNL* Workshop on Human Biology Models for Environmental Health Effects, 2000.
- Risk Assessment Coordinator, *NIEHS US RAPID* Program for the Evaluation of Health Risks from Exposure to Electric and Magnetic Fields, 1996-99.
- Organizer and Chair, Four Public Comment Sessions on the report of the *NIEHS/DOE* Working Group on the Health Effects of Exposure to Electric and Magnetic Fields, 1998.
- Organizer and Co-Chair, *NIEHS/DOE* Working Group on the Health Effects of Exposure to Electric and Magnetic Fields, 1998.
- Scientific Organizing Committee, *NIEHS* Workshop on Risk Assessment Issues Associated with Endocrine Disrupting Chemicals, 1998.
- Organizer, *NIEHS/DOE* Science Research Symposium on the Health Effects of Exposure to Electric and Magnetic Fields I: Biophysical Mechanisms and *In Vitro* Experimentation, 1998.
- Organizer, *NIEHS/DOE* Science Research Symposium on the Health Effects of Exposure to Electric and Magnetic Fields II: Epidemiological Findings, 1998.
- Organizer, *NIEHS/DOE* Science Research Symposium on the Health Effects of Exposure to Electric and Magnetic Fields III: *In Vitro* and Clinical Research Findings, 1998.
- Head, Toxicokinetics Faculty, *NIEHS*, 1994-97.
- Coordinator/Director, *NIEHS/ATSDR* Interagency Course on Mechanistic Modeling in Environmental Risk Assessment, 1996.
- Organizer, *NIEHS EPA* Workshop on Research Priorities for New Risk Assessment Guidelines,

- 1996.
- Co-Organizer, *National Institute of Statistical Sciences, NIEHS EPA Workshop on Mechanistic Modeling in Risk Assessment*, 1995.
  - Scientific Coordinator and Mission Director, *NIEHS "Mission to Vietnam"* to assess the potential for scientific collaboration on the impact of Agent Orange on the Vietnamese Population, 1995.
  - Chairman, *NIEHS Computer Science Focus Group*, 1995.
  - Discussant, *National Toxicology Program Workshop on Mechanistic Modeling in Toxicology, NIEHS*, 1995.
  - Discussant, *National Toxicology Program Workshop on Mechanisms of Carcinogenesis, NIEHS*, 1995.
  - Co-Organizer, *International Conference on The Role of Cell Proliferation in Carcinogenesis*, co-sponsored by *NIEHS, The Chemical Industry Institute of Toxicology, The International Life Sciences Institute* and *The American Industrial Health Council*, 1992.
  - Organizer and Director, *Scientific Basis of Animal Carcinogenicity Testing*, Moscow, Russia, co-sponsored by the *International Agency for Research on Cancer, NIEHS, Health and Welfare Canada* and *The All-Union Cancer Research Center*, 1991.
  - Chairman, *Computer Technology Advisory Forum, NIEHS*, 1989.
  - Organizer and Director, *Design and Analysis of Long-Term Animal Carcinogenicity Experiments*, Lyon, France, co-sponsored by the *International Agency for Research on Cancer* and the *NIEHS*, 1988.

#### *Non-Governmental (US) Activities:*

- Member, *NRC Committee to review the Draft Interagency Report on the Impacts of Climate Change on Human Health in the United States*, Washington, DC, 2015
- Expert Scientist, *International Agency for Research on Cancer Monograph Meeting on Some Organophosphate Pesticides and Herbicides*, Lyon, France, March, 2015
- Overall Chair, *International Agency for Research on Cancer Monograph Meeting on Diesel and Gasoline Engine Exhausts and related compounds*, Lyon, France, June, 2012
- Advisor to Wellcome Trust at "International Research Futures Symposium on Global Change, Economic Sustainability, and Human Health", London, England, March, 2012.
- Expert Panel Member for review of *Hollings Marine Laboratory*, National Oceanographic and Atmospheric Agency, Charleston, USA, February, 2012.
- Chair, *Mechanism Subgroup, International Agency for Research on Cancer Monograph Meeting on Radiofrequency Electric and Magnetic Fields*, Lyon, France, May, 2011
- Advisor, *Greek Ministry Health, Working group on hexavalent chromium in the environment*, January, 2011
- Member, *WHO Consultation on Human Health Risks from DDT*, Geneva, Switzerland, November, 2010
- Associate Editor, *Frontiers in Predictive Toxicity*, 2010 – 2011
- Scientific Advisor, *Health Investigation Levels Workshop*, Canberra, Australia, January, 2010
- Chair, *IARC Working Group, IARC Monograph 100-G*, Lyon, France, October, 2009
- Scientific Organizing Committee, *VII World Congress on Alternatives and Animal Use in Life Sciences*, Rome, Italy, September, 2009
- Chair, *Research Directions Working Group, World Health Organization Consultation on Global Research on Climate Change and Health*, October, 2008.
- Editor-in-Chief, *The Open Environment Journal*, May 2008-August, 2010
- Member, *EPA Science Advisory Board*, July, 2008-present
- Working Group Member, *IARC Monograph 98 - Fire-fighting, Painting and Shift-work*, Lyon, France, November, 2007

- Chair, WHO Extremely Low Frequency Magnetic and Electric Fields Workshop on Intervention Strategies, June, 2007
- Special Advisor to the Director, Program on Public Health and the Environment, WHO, Geneva, May-July, 2007
- Member, International Life Sciences Institute Working Group on Susceptible Populations, March, 2007 – present
- Special Advisor to the Director, Program on Public Health and the Environment, WHO, Geneva, November, 2006-January, 2007
- Breakout Group Chair, International Workshop on Uncertainty and Variability in PBPK Modeling, RTP, NC USA, October, 2006
- Member, Health Effects Sciences Institute Committee on Sensitive Subpopulations and Groups, Washington, DC, 2006 to present
- Rapporteur, Steering Committee for developing the 100<sup>th</sup> Monograph of the International Agency for Research on Cancer, Lyon, France, September, 2006
- Co-Organizer, parallel workshops on the advancement of PBPK modeling in risk assessment, Research Triangle Park, November, 2006, Corfu, Greece, April, 2007.
- Organizer, Alternative Models in Developmental Neurotoxicity, Alexandria, Virginia, March, 2006.
- Organizer, NTP High Throughput Screening Workshop, Washington, DC, December, 2005
- Organizer, ISRTP Meeting on Alternative Methods in Toxicology, Baltimore, Maryland, November, 2005
- Organizer, NTP 25<sup>th</sup> Anniversary Meeting, Washington, DC, May, 2005
- Organizer, IPCS/WHO Workgroup on Dose-Response Modeling, Geneva, Switzerland, September, 2004
- Organizer, Consultation on harmonization of toxicological research between the NTP, Ramazzini Foundation and the European Union, European Congress of Toxicology, Florence, Italy, September, 2003.
- Member, WHO Workgroup on the epidemiology of cellular phone toxicity, Tskuba, Japan, September, 2003.
- Program Committee, 12<sup>th</sup> International Conference on Global Warming, Boston, Massachusetts, May 2003
- Program Committee, International Conference on Cancer Risk Assessment, Athens, Greece, August, 2003
- Chair, WHO Public Consultation on Risk Communication, Luxembourg, February, 2003.
- Chair, WHO Committee on Establishing a Plan for Implementation of the Precautionary Principle in Risk Management, Luxembourg, February, 2003.
- Presenter (on behalf of US Government), National Academy of Sciences Panel on the Use of Third Party Toxicity Research with Human Research Participants, December, 2002
- Member, US Science Delegation, United Nations Environmental Program Consultation on Organic Mercury, September, 2002
- Science Panel Member, IARC Carcinogenicity Review of ELF-EMF, Lyon, France, June, 2001.
- Reviewer, Finish Ministry of Health Centers of Excellence Program, Helsinki, April, 2001.
- EPA dioxin reassessment peer review workshop and public comment session, Washington, DC, 2000.
- Organizer: Dioxin Dose-Response Working Group Meeting, Fort Collins, Colorado, February, 2000.
- Chair, Spiegelman Award Committee, *American Public Health Association*, 1998.
- Chair, *Bioelectromagnetics Society* Symposium on the use of Transgenic Animals in Evaluating Health Risks from Exposure to Cellular Phones, St. Petersburg, Florida, 1998.

- Member, *World Health Organization* International Program on Chemical Safety, Workshop on Issues in Cancer Risk Assessment, 1998.
- Advisor, *Joint Committee on Food Additives, World Health Organization Food and Agriculture Organization*. Evaluation of certain food additives and contaminants
- Member, US Government Methylene Chloride Risk Characterization Science Committee, 1996-1998.
- Scientific Organizing Committee, *Colorado State University* Workshop on Biomedical Advances on Chemical Mixtures, 1997.
- *National Academy of Sciences*, Institute of Medicine, Committee on Funding Future Agent Orange Research in Vietnam, 1996.
- Discussant, Workshop on the role of Endocrine Disruptors in Human Health, 1995.
- Advisor to *Australian Health Council* on Risk Assessment Methodology, Member *NHMRC* Steering Committee on Cancer Risk Assessment Guidelines
- Participant, International Program on Chemical Safety of the *World Health Organization* Workshop on Chemical Risk Assessment, London, England, 1995.
- Participant, *IARC* Workshop on Receptor-Mediated Carcinogenesis, Lyon, France, 1994.
- Co-Organizer, Symposium on Quantitative Risk Assessment, *German Cancer Research Center*, Heidelberg, Germany, 1993.
- Participant, *IARC* Monograph on Risk Assessment Methodology, *International Agency for Research on Cancer*, Lyon, France, 1993.
- Thesis advisor for graduate student, *University of Waterloo*, Waterloo, Ontario, Canada, 1991-93.
- Co-Organizer, *Russian Academy of Sciences* Informatics and Cybernetics Research Award, 1992.
- Official Observer, *IARC* Monograph on the Biological Effects of Ultraviolet Radiation, *International Agency for Research on Cancer*, Lyon, France, 1992.
- Member, *International Life Sciences Institute*, Dose-Response Working Group, 1991.
- Participant in Banbury Conference on Human Health Risks from Exposures to Dioxins, Banbury Conference Center, Cold Spring Harbor, New York, 1990.
- Co-Chairman, Session on Biostatistical Developments in Cancer Research, *15th International Cancer Congress*, Hamburg, Germany, 1990.
- Participant in *Environmental Protection Agency* Workshop on Risk Assessment Guidelines, Virginia Beach, Virginia, 1989.

Direction of Ph.D. Theses:

- A Bailer. *The effects of treatment lethality on tests of carcinogenicity*. Department of Biostatistics, University of North Carolina, Chapel Hill, North Carolina, 1986.
- P Williams. *Estimating tumor incidence rates using the method of moments and maximum likelihood estimation combined*. Department of Biostatistics, University of North Carolina, Chapel Hill, North Carolina, 1989.
- G Carr. *The analysis of data on adverse reactions to chemicals in developmental toxicology*. Department of Biostatistics, University of North Carolina, Chapel Hill, North Carolina, 1989.
- S Liu. *Estimating parameters in a two-stage model of carcinogenesis using information on enzyme-altered foci from initiation-promotion experiments*. Department of Biostatistics, University of North Carolina, Chapel Hill, North Carolina, 1993.
- CD Sherman. *Multipath multistage models of carcinogenesis*. Department of Statistics and Actuarial Sciences, University of Waterloo, Waterloo, Ontario, Canada, 1994.
- C Lyles. *Cell labeling data: Models and parameter estimation*. Department of Biostatistics, University of North Carolina, Chapel Hill, North Carolina, 1995.
- F Yc. *The equal slopes test for benchmark doses*. Department of Biostatistics, University of North Carolina, Chapel Hill, North Carolina, 2001
- S Whitaker. *Development of a biologically-based mathematical model of fetal development*. Department of Mathematics, North Carolina State University, Raleigh, North Carolina, 2000.

R Helms. *Homeostatic feedback control of growth on multistage cancer models*. Department of Biostatistics, University of North Carolina, Chapel Hill, North Carolina, 2001.

*Journal Articles: (peer reviewed)*

1. Portier CJ: A comprehensive analysis of the animal carcinogenicity data for glyphosate from chronic exposure rodent carcinogenicity studies. *Environmental Health* 2020, 19(1):18.
2. Robinson, C., Portier, C., Čavoški, A., Mesnage, R., Roger, A., Clausen, P., Whaley, P., Muilerman, H., Lyssimachou, A.: **Achieving a High Level of Protection from Pesticides in Europe: Problems with the Current Risk Assessment Procedure and Solutions**, *European Journal of Risk Regulation* 2020, 1-31
3. Krewski D, Rice JM, Bird M, Milton B, Collins B, Lajoie P, Billard M, Grosse Y, Coglianò VJ, Caldwell JC *et al*: **Concordance between sites of tumor development in humans and in experimental animals for 111 agents that are carcinogenic to humans**. *Journal of toxicology and environmental health Part B, Critical reviews* 2019, **22**(7-8):203-236.
4. Alexceff, S. E., A. Roy, J. Shan, X. Liu, K. Messier, J. S. Apte, C. Portier, S. Sidney and S. K. Van Den Eeden (2018). "High-resolution mapping of traffic related air pollution with Google street view cars and incidence of cardiovascular events within neighborhoods in Oakland, CA." *Environ Health* **17**(1): 17-38.
5. Messier, K. P., Chambliss, S. E., Choi, J. J., Roy, A., Marshall, J. D., Brauer, M., Szpiro, A. A., Portier, C. J., Lunden, M. M., Kerckhoffs, J., Vermeulen, R. C. H., Hamburg, S. P., Apte, J. S., Mapping Air Pollution with Google Streetview Cars: Efficient Approaches with Mobile Monitoring and Land Use Regression, *Environmental Science and Technology*, October, 2018
6. Espín-Pérez, A., Portier, C. J., Chadeau-Hyam, M., van Veldhoven, K., Kleinjans, J., de Kok, T., Comparison of statistical methods and the use of quality control samples for batch effect correction in human transcriptome data, *PLOS One* 13(8), 2018
7. Apte, JS, Messier, KP, Gani, S, Brauer, M, Kirchstetter, TW, Lunden, MM, Marshall, JD, Portier, CJ, Vermeulen, RCH, Hamburg, S., High-Resolution Air Pollution Mapping with Google Streetview Cars: Exploiting Big Data, *Environmental Science and Technology* 2017, **51** (12) 6999-7008
8. Sand S, Parham F, Portier CJ, Tice RR, Krewski D. Comparison of Points of Departure for Health Risk Assessment Based on High-Throughput Screening Data. *Environ Health Perspect* (2017) **125** (4) 623-633 . doi: 10.1289/EHP408. PubMed PMID: 27384688.
9. Cote I, Andersen ME, Ankley GT, Barone S, Birnbaum LS, Bockelheide K, et al. The Next Generation of Risk Assessment Multi-Year Study-Highlights of Findings, Applications to Risk Assessment, and Future Directions. *Environ Health Perspect* (2016) **124**(11):1671-82. doi: 10.1289/EHP233. PubMed PMID: 27091369; PubMed Central PMCID: PMC5089888.

10. Parham F, Portier CJ, Chang X, Mevissen M. The Use of Signal-Transduction and Metabolic Pathways to Predict Human Disease Targets from Electric and Magnetic Fields Using in vitro Data in Human Cell Lines. *Frontiers in public health* (2016) **4**:193. doi: 10.3389/fpubh.2016.00193. PubMed PMID: 27656641; PubMed Central PMCID: PMC5013261.
11. Portier CJ, Armstrong BK, Baguley BC, Baur X, Belyaev I, Belle R, et al. Differences in the carcinogenic evaluation of glyphosate between the International Agency for Research on Cancer (IARC) and the European Food Safety Authority (EFSA). *Journal of epidemiology and community health* (2016) **70**(8):741-5. doi: 10.1136/jech-2015-207005. PubMed PMID: 26941213; PubMed Central PMCID: PMC4975799.
12. Scinicariello F, Portier C. A simple procedure for estimating pseudo risk ratios from exposure to non-carcinogenic chemical mixtures. *Archives of toxicology* (2016) **90**(3):513-23. doi: 10.1007/s00204-015-1467-z. PubMed PMID: 25667015.
13. Smith MT, Guyton KZ, Gibbons CF, Fritz JM, Portier CJ, Rusyn I, et al. Key Characteristics of Carcinogens as a Basis for Organizing Data on Mechanisms of Carcinogenesis. *Environ Health Perspect* (2016) **124**(6):713-21. doi: 10.1289/ehp.1509912. PubMed PMID: 26600562; PubMed Central PMCID: PMC4892922.
14. McPartland, J., Dantzker, H.C., Portier, C. J. Building a robust 21st century chemical testing program at the U.S. Environmental Protection Agency: recommendations for strengthening scientific engagement. *Environ Health Perspect* 2015. **123** (1); p. 1-5.
15. Smith, M.T., Gibbons, C.F., Fritz, J.M., Rusyn, I., Lambert, P., Kavlock, R., Hecht, S.S., Bucher, J., Caldwell, J.C., Demarini, D., Coglianò, V., Portier, C., Paan, R., Straif, K., Guyton, K.Z., Key Characteristics of Carcinogens and an Approach to using Mechanistic Data in their Classification. *Environ Health Perspect* 2015 (in press)
16. Thomas, R., Thomas, R.S., Auerbach, S. S., Portier, C. J., Biological networks for predicting chemical hepatocarcinogenicity using gene expression data from treated mice and relevance across human and rat species. *PLoS One*. 2013. **8**(5): p. e63308.
17. Scinicariello, F., Buser, M.C., Mevissen, M., Portier, C.J., Blood lead level association with lower body weight in NHANES 1999-2006. *Toxicol Appl Pharmacol*. 2013. **273**(3): p. 516-23.
18. Thomas R, Portier CJ., Gene Expression Networks. *Methods Mol Biol*. 2013;**930**:165-78.
19. Aylward LL, Kirman CR, Schoeny R, Portier CJ, Hays SM., Evaluation of Biomonitoring Data from the CDC National Exposure Report in a Risk Assessment Context: Perspectives across Chemicals. *Environ Health Perspect*. 2012 **121** (3)
20. Sand, S., Portier, C.J., Krewski, D. A Signal-to-noise crossover dose as the point of departure for risk assessment. *Environmental Health Perspectives*. 119(12):1766-74, 2011
21. Gohlke, J.M., Thomas, R., Woodward, A., Campbell-Lundrum, D., Pruss-Ustun, A., Hales, S., Portier, C.J. Estimating the global public health implications of electricity and coal consumption. *Environmental Health Perspectives* 2011 **119** (6): 821-6

22. McHale CM, Zhang L, Lan Q, Vermeulen R, Li G, Hubbard AE, Porter KE, Thomas R, Portier CJ, Shen M, Rappaport SM, Yin S, Smith MT, Rothman N. Global gene expression profiling of a population exposed to a range of benzene levels. *Environ Health Perspect.* 2011 May;119(5):628-34.
23. Prause AS, Guionaud CT, Stoffel MH, Portier CJ, Mevissen M. Expression and function of 5-hydroxytryptamine 4 receptors in smooth muscle preparations from the duodenum, ileum, and pelvic flexure of horses without gastrointestinal tract disease. *Am J Vet Res.* 2010 Dec;71(12):1432-42.
24. Luke, N.S., DeVito, M.J., Portier, C.J., El-Masri, H.A., Employing a mechanistic model for the MAPK pathway to examine the impact of cellular all-or-none behavior on overall tissue response, *Dose-Response* 2010 8(3): 347-67.
25. Crump, KS, Chen, C., Chiu, W.A., Louis, T.A., Portier, C. J., Subramaniam, R.P., Wgite, P.D., What role for biologically-based Dose-Response Models in Estimating Low-Dose Risk. *Env. Health Persp.* 2010 118(5):585-8
26. Parham F, Austin C, Southall N, Huang R, Tice R, Portier C. Dose-Response modeling of High-Throughput Screening Data. *J Biomol Screen.* 2009 **14**(10), 1216-27
27. Hines RN, Sargent D, Autrup H, Birnbaum LS, Brent RL, Doerrer NG, Cohen Hubal EA, Juberg DR, Laurent C, Luebke R., Olejniczak K, Portier CJ, Slikker W. Approaches for assessing risks to sensitive populations: lessons learned from evaluating risks in the pediatric population. *Tox. Sci.* 2010 **113** (4), 4-26.
28. Portier, C. Toxicological decision making on hazards and risks – status quo and the way forward: current concepts and schemes of science-driven decision making – an overview. *Human and Experimental Toxicology* 2009 **28**(2-3), 123-125
29. Prause, A.S., Stoffel, M.H., Portier, C.J., Mevissen, M., Expression and function of 5-HT7 receptors in smooth muscle preparation from equine duodenum, ileum, and pelvic flexure, *Research in Veterinary Science* 2009 **87**(2), 292-299
30. Boyd, W.A., Smith, M. V., Kissling, G. E., Rice, J., R., Snyder, D. W., Portier, C. J., Freedman, J. H. Application of a Mathematical Model to Describe the Effects of Chlorpyrifos on *Caenorhabditis elegans* Development, *PLoS ONE* 2009 **4**(9): e7024. doi:10.1371/journal.pone.0007024
31. Smith MV, Boyd WA, Kissling GE, Rice JR, Snyder DW, et al. A Discrete Time Model for the Analysis of Medium-Throughput *C. elegans* Growth Data. *PLoS ONE* 2009 **4**(9): e7018. doi:10.1371/journal.pone.0007018
32. Gohlke, J. M., Stockton, P.S., Sieber, S., Foley, J., Portier, C. J. AhR-mediated gene expression in the developing mouse telencephalon. *Reproductive Toxicology* 2009 **28** (3)
33. Thomas, R., Gohlke, J., Parham, F., Smith, M., Portier, C. (2009) Choosing the right path: enhancement of biologically-relevant sets of genes or proteins using pathway structure. *Genome Biology* 2009 **10**(4), R44.
34. Julia M Gohlke, Reuben Thomas, Yonqing Zhang, Michael C Rosenstein, Allan P Davis, Cynthia Murphy, Carolyn J Mattingly, Kevin G Becker, Christopher J Portier, Genetic and Environmental Pathways to Complex Disease. *BMC Systems Biology* 2009 May 5, 3:46.

35. Schmitz, A., Portier, C. J., Thurmann, W., Theurillat, R., Mevissen, M. Stereoselective biotransformation of ketamine in equine liver and lung microsomes. *J. Vet. Pharm. And Therapeutics* 2008 **31** (5): 446-455
36. Xia, M; Huang, R; Witt, KL; Southall, N; Fostel, J; Cho, MH; Jadhav, A; Smith, CS; Inglese, J; Portier, CJ; Tice, RR; Austin, CP Compound cytotoxicity profiling using quantitative high-throughput screening. *Env. Health Perspectives* 2008 **116** (3): 284-291
37. Gohlke, J. M., Armant, O., Parham, F., M., Smith, M., V., Zimmer, C., Castro, D., S., Nguyen, L., Parker, J., S., Gradwohl, G., Guillemot, F., Portier, C. J. Characterization of proneural gene regulatory network during mouse telencephalon development., *BMC Biology* 2008 **6** (15)
38. Subramaniam, R. P., Chen, C., Crump, K. S., Devoney, D., Fox, J. F., Portier, C. J., Schlosser, P. M., Thompson, C. M., White, P. Uncertainties in Biologically-based modeling of formaldehyde-induced respiratory cancer risk: Identification of key issues. *Risk Analysis* 2008 **28**(4): 907-923
39. Buehler, M., Steiner, A., Meylan, M., Portier, C. J., and Mevissen, M., In vitro effects of bethanechol on smooth muscle preparations obtained from abomasal fundus, corpus and antrum of dairy cows. *Research in Vet. Sci.* **2008 84** (3), 444-451
40. Barton, H.A., W.A. Chiu, R.W. Setzer, M.E. Andersen, A.J. Bailer, F.Y. Bois, R.S. Dewoskin, S. Hays, G. Johanson, N. Jones, G. Loizou, R.C. Macphail, C.J. Portier, M. Spendiff, and Y.M. Tan, Characterizing Uncertainty and Variability in Physiologically-based Pharmacokinetic (PBPK) Models: State of the Science and Needs for Research and Implementation. *Toxicol Sciences* 2007 **99** (2) 395-402.
41. Pfeiffer, J.B., M. Mevissen, A. Steiner, C.J. Portier, and M. Meylan, In vitro effects of bethanechol on specimens of intestinal smooth muscle obtained from the duodenum and jejunum of healthy dairy cows. *Am J Vet Res*, 2007. **68**(3): p. 313-22.
42. Smith, M., Miller, C., Kohn, M., Walker, N.J., Portier, C. J., Absolute estimation of initial concentrations of amplicon in a real-time PT-PCR process, *BMC Bioinformatics* 2007 **8**(1), 409
43. Toyoshiba, H., Sone, H., Yamanaka, T., Parham, F., Irwin, R., Boorman, G., and Portier, C. Gene network analysis suggests differences between high and low doses of acetaminophen. *Toxicology and Applied Pharmacology* 2006 **215** (3), 306-316
44. Knobloch M, Portier CJ, Levionnois OL, Theurillat R, Thormann W, Spadavecchia C, Mevissen M. Antinociceptive effects, metabolism and disposition of ketamine in ponies under target-controlled drug infusion. *Toxicology And Applied Pharmacology* 2005 **216** (3): 373-386
45. Portier, C.J., H. Toyoshiba, H. Sone, F. Parham, R.D. Irwin, and G.A. Boorman, Comparative analysis of gene networks at multiple doses and time points in livers of rats exposed to acetaminophen. *Altex* 2005 **23 Suppl**: p. 380-4.
46. Resnik, D.B. and C. Portier, Pesticide testing on human subjects: weighing benefits and risks. *Environ Health Perspect.* 2005 **113**(7): p. 813-7.

47. Walker, N. J., Crockett, P. W., Nyska, A., Brix, A. E., Jokinen, M. P., Sells, D. M., Hailey, J. R., Easterling, M., Haseman, J. K., Yin, M., Wyde, M. E., Bucher, J. R., and Portier, C. J. Dose-additive carcinogenicity of a defined mixture of "dioxin-like compounds". *Environ Health Perspect* 2005 113, 43-48.<sup>5</sup>
48. Boorman, G. A., Irwin, R. D., Vallant, M. K., Gerken, D. K., Lobenhofer, E. K., Hejtmancik, M. R., Hurban, P., Brys, A. M., Travlos, G. S., Parker, J. S., and Portier, C. J. Variation in the hepatic gene expression in individual male Fischer rats. *Toxicol Pathol* 2005 33, 102-110.
49. Toyoshiba, H., Walker, N.J., Bailer, A.J. and Portier, C.J. Evaluation of toxic equivalency factors for induction of cytochromes P450 CYP1A1 and CYP1A2 enzyme activity by dioxin-like compounds. *Toxicol Appl Pharmacol* 2004 194, 156-68.
50. Trachsel, D., Tschudi, P., Portier, C.J., Kuhn, M., Thormann, W., Scholtysik, G. and Mevissen, M. Pharmacokinetics and pharmacodynamic effects of amiodarone in plasma of ponies after single intravenous administration. *Toxicology and Applied Pharmacology* 2004, 195, 113-125.
51. Bucher, J. R., and Portier, C. Human carcinogenic risk evaluation, Part V: The national toxicology program vision for assessing the human carcinogenic hazard of chemicals. *Toxicol Sci* 2004 82, 363-366.
52. Germolec, D. R., Kashon, M., Nyska, A., Kuper, C. F., Portier, C., Kommineni, C., Johnson, K. A., and Luster, M. I. The accuracy of extended histopathology to detect immunotoxic chemicals. *Toxicol Sci* 2004 82, 504-514.
53. Germolec, D. R., Nyska, A., Kashon, M., Kuper, C. F., Portier, C., Kommineni, C., Johnson, K. A., and Luster, M. I. Extended histopathology in immunotoxicity testing: interlaboratory validation studies. *Toxicol Sci* 2004 78, 107-115.
54. Shelby, M., Portier, C., Goldman, L., Moore, J., Iannucci, A., Jahnke, G., and Donkin, S., NTP-CERHR expert panel report on the reproductive and developmental toxicity of methanol. *Reprod Toxicol* 2004 18, 303-390.
55. Smith, M. V., Nyska, A., and Portier, C. Application of a statistical dynamic model investigating the short-term cellular kinetics induced by riddelliine, a hepatic endothelial carcinogen. *Toxicol Sci* 2004 80, 258-267.
56. Toyoshiba, H., Yamanaka, T., Sone, H., Parham, F. M., Walker, N. J., Martinez, J., and Portier, C. J. Gene interaction network suggests dioxin induces a significant linkage between aryl hydrocarbon receptor and retinoic acid receptor beta. *Environ Health Perspect* 2004 112, 1217-1224.<sup>6</sup>
57. Yamanaka, T., Toyoshiba, H., Sone, H., Parham, F. M., and Portier, C. J. The TAO-Gen algorithm for identifying gene interaction networks with application to SOS repair in *E. coli*. *Environ Health Perspect* 2004 112, 1614-1621

---

<sup>5</sup> Awarded outstanding published paper in 2005 by the Risk Assessment Specialty Section of the Society of Toxicology

<sup>6</sup> Awarded outstanding published paper in 2004 by the Risk Assessment Specialty Section of the Society of Toxicology

58. Schecter, A., Lucier, G. W., Cunningham, M. L., Abdo, K. M., Blumenthal, G., Silver, A. G., Melnick, R., Portier, C., Barr, D. B., Barr, J. R., Stanfill, S. B., Patterson, D. G., Jr., Needham, L. L., Stopford, W., Masten, S., Mignogna, J., and Tung, K. C. Human consumption of methyleugenol and its elimination from serum. *Environ Health Perspect* 2004 112, 678-680.
59. Bucher, J and Portier, C. Human carcinogenic risk evaluation, Part V: The national toxicology program vision for assessing the human carcinogenic hazard of chemicals. *Toxicol Sci.* 2004 Dec;82(2):363-6
60. Chhabra, R.S., Bucher, J.R., Wolfe, M. and Portier, C. Toxicity characterization of environmental chemicals by the US National Toxicology Program: an overview. *Int J Hyg Environ Health*, 2003 206, 437-45.
61. Germolec, D.R., Nyska, A., Kashon, M., Kuper, C.F., Portier, C., Kommineni, C., Johnson, K.A. and Luster, M.I. Extended Histopathology in Immunotoxicity Testing: Interlaboratory Validation Studies. *Toxicol Sci.* 2003 78(1) 107-115
62. Koken, P.J., Piver, W.T., Ye, F., Elixhauser, A., Olsen, L.M. and Portier, C.J. Temperature, air pollution, and hospitalization for cardiovascular diseases among elderly people in Denver. *Environ Health Perspect* 2003 111, 1312-7.
63. Whitaker, S.Y., Tran, H.T. and Portier, C.J. (2003) Development of a biologically-based controlled growth and differentiation model for developmental toxicology. *J Math Biol*, 46, 1-16.
64. Hauser, D.S., Mevissen, M., Weiss, R., Portier, C.J., Scholtysik, G., Studer, U.E. and Danuser, H. Effects of ketanserin and DOI on spontaneous and 5-HT-evoked peristalsis of the pig ureter in vivo. *Br J Pharmacol* 2002 135, 1026-32.
65. Kim, A.H., Kohn, M.C., Portier, C.J. and Walker, N.J. Impact of physiologically based pharmacokinetic modeling on benchmark dose calculations for TCDD-induced biochemical responses. *Regul Toxicol Pharmacol* 2002 36, 287-96.
66. Kohn, M.C., Melnick, R.L., Ye, F. and Portier, C.J. Pharmacokinetics of sodium nitrite-induced methemoglobinemia in the rat. *Drug Metab Dispos* 2002, 30, 676-83.
67. Koo, J.W., Parham, F., Kohn, M.C., Masten, S.A., Brock, J.W., Needham, L.L. and Portier, C.J. The association between biomarker-based exposure estimates for phthalates and demographic factors in a human reference population. *Environ Health Perspect* 2002, 110, 405-10.
68. Parham, F.M., Matthews, H.B. and Portier, C.J. A physiologically based pharmacokinetic model of p,p'-dichlorodiphenylsulfone. *Toxicol Appl Pharmacol* 2002, 181, 153-63.
69. Portier, C.J. Endocrine dismodulation and cancer. *Neuroendocrinol Lett* 2002, 23 Suppl 2, 43-7.
70. Simmons, P.T. and Portier, C.J. Toxicogenomics: the new frontier in risk analysis. *Carcinogenesis* 2002, 23, 903-5.
71. Weiss, R., Mevissen, M., Hauser, D.S., Scholtysik, G., Portier, C.J., Walter, B., Studer, U.E. and Danuser, H. Inhibition of human and pig ureter motility in vitro and in vivo by the K(+) channel openers PKF 217-744b and nicorandil. *J Pharmacol Exp Ther* 2002, 302, 651-8.

72. Whitaker, S.Y. and Portier, C. A controlled growth and differentiation model for non-monotonic dose-response. *Human and Ecological Risk Assessment* 2002, 8, 1739-1755.
73. Kohn, M.C., Walker, N.J., Kim, A.H. and Portier, C.J. Physiological modeling of a proposed mechanism of enzyme induction by TCDD. *Toxicology* 2001, 162, 193-208.
74. Mevissen, M., Denac, H., Schaad, A., Portier, C.J. and Scholtysik, G. Identification of a cardiac sodium channel insensitive to synthetic modulators. *J Cardiovasc Pharmacol Ther* 2001, 6, 201-12.
75. Portier, C.J. Linking toxicology and epidemiology: the role of mechanistic modeling. *Stat Med* 2001, 20, 1387-93.
76. Willems, B.A., Melnick, R.L., Kohn, M.C. and Portier, C.J. A physiologically based pharmacokinetic model for inhalation and intravenous administration of naphthalene in rats and mice. *Toxicol Appl Pharmacol* 2001, 176, 81-91.
77. Ye, F., Piver, W.T., Ando, M. and Portier, C.J. Effects of temperature and air pollutants on cardiovascular and respiratory diseases for males and females older than 65 years of age in Tokyo, July and August 1980-1995. *Environ Health Perspect* 2001, 109, 355-9.
78. Halmes, N.C., Roberts, S.M., Tolson, J.K. and Portier, C.J. Reevaluating cancer risk estimates for short-term exposure scenarios. *Toxicol Sci* 2000, 58, 32-42.
79. Kohn, M.C., Parham, F., Masten, S.A., Portier, C.J., Shelby, M.D., Brock, J.W. and Needham, L.L. Human exposure estimates for phthalates. *Environ Health Perspect* 2000, 108, A440-2.
80. Portier, C.J. and Parham, F.M. Comments on a biochemical model of cyclophosphamide hematotoxicity. *J Toxicol Environ Health A* 2000, 61, 525-8.
81. Portier, C., Sherman, C.D. and Kopp-Schneider, A. Multistage, stochastic models of the cancer process: a general theory for calculating tumor incidence. *Stochastic Environmental Research and Risk Assessment* 2000, 14, 173-179.
82. Sherman, C.D. and Portier, C.J. Calculation of the cumulative distribution function of the time to a small observable tumor. *Bull Math Biol* 2000, 62, 229-40.
83. Smith, M.V. and Portier, C.J. Incorporating observability thresholds of tumors into the two-stage carcinogenesis model. *Math Biosci* 2000, 163, 75-89.
84. Tritscher, A.M., Mahler, J., Portier, C.J., Lucier, G.W. and Walker, N.J. Induction of lung lesions in female rats following chronic exposure to 2,3,7,8-tetrachlorodibenzo-p-dioxin. *Toxicol Pathol* 2000, 28, 761-9.
85. Walker, N.J., Tritscher, A.M., Sills, R.C., Lucier, G.W. and Portier, C.J. Hepatocarcinogenesis in female Sprague-Dawley rats following discontinuous treatment with 2,3,7,8-tetrachlorodibenzo-p-dioxin. *Toxicol Sci* 2000, 54, 330-7.
86. Andersen, M.E., Conolly, R.B., Faustman, E.M., Kavlock, R.J., Portier, C.J., Sheehan, D.M., Wier, P.J. and Ziese, L. Quantitative mechanistically based dose-response modeling with endocrine-active compounds. *Environ Health Perspect* 1999, 107 Suppl 4, 631-8.
87. el Masri, H. and Portier, C. Replication potential of cells via the protein kinase C-MAPK pathway: application of a mathematical model. *Bulletin of Mathematical Biology* 1999, 61, 379-398.

88. El-Masri, H.A., Bell, D.A. and Portier, C.J. Effects of glutathione transferase theta polymorphism on the risk estimates of dichloromethane to humans. *Toxicol Appl Pharmacol* 1999, *158*, 221-30.
89. Melnick, R.L., Sills, R.C., Portier, C.J., Roycroft, J.H., Chou, B.J., Grumbein, S.L. and Miller, R.A. Multiple organ carcinogenicity of inhaled chloroprene (2-chloro-1,3-butadiene) in F344/N rats and B6C3F1 mice and comparison of dose-response with 1,3-butadiene in mice. *Carcinogenesis* 1999, *20*, 867-78.
90. Piver, W.T., Ando, M., Ye, F. and Portier, C.J. Temperature and air pollution as risk factors for heat stroke in Tokyo, July and August 1980-1995. *Environ Health Perspect* 1999, *107*, 911-6.
91. Walker, N.J., Portier, C.J., Lax, S.F., Crofts, F.G., Li, Y., Lucier, G.W. and Sutter, T.R. Characterization of the dose-response of CYP1B1, CYP1A1, and CYP1A2 in the liver of female Sprague-Dawley rats following chronic exposure to 2,3,7,8-tetrachlorodibenzo-p-dioxin. *Toxicol Appl Pharmacol* 1999, *154*, 279-86.
92. El-Masri, H.A. and Portier, C.J. Physiologically based pharmacokinetics model of primidone and its metabolites phenobarbital and phenylethylmalonamide in humans, rats, and mice. *Drug Metab Dispos* 1998, *26*, 585-94.
93. Jung, D., Berg, P.A., Edler, L., Ehrenthal, W., Fenner, D., Flesch-Janys, D., Huber, C., Klein, R., Koitka, C., Lucier, G., Manz, A., Muttray, A., Needham, L., Papke, O., Pietsch, M., Portier, C., Patterson, D., Prellwitz, W., Rose, D.M., Thews, A. and Konietzko, J. Immunologic findings in workers formerly exposed to 2,3,7,8-tetrachlorodibenzo-p-dioxin and its congeners. *Environ Health Perspect* 1998, *106 Suppl 2*, 689-95.
94. Kopp-Schneider, A., Portier, C. and Bannasch, P. A model for hepatocarcinogenesis treating phenotypical changes in focal hepatocellular lesions as epigenetic events. *Math Biosci* 1998, *148*, 181-204.
95. Murrell, J.A., Portier, C.J. and Morris, R.W. Characterizing dose-response: I: Critical assessment of the benchmark dose concept. *Risk Anal* 1998, *18*, 13-26.
96. Parham, F.M. and Portier, C.J. Using structural information to create physiologically based pharmacokinetic models for all polychlorinated biphenyls. II. Rates of metabolism. *Toxicol Appl Pharmacol* 1998, *151*, 110-6.
97. Portier, C.J. and Bell, D.A. Genetic susceptibility: significance in risk assessment. *Toxicol Lett* 1998, *102-103*, 185-9.
98. Portier, C.J. and Ye, F. U-shaped dose-response curves for carcinogens. *Hum Exp Toxicol* 1998, *17*, 705-7; discussion 716-8.
99. Sherman, C.D. and Portier, C.J. Eyes closed: simple, intuitive, statistically sound, and efficient methods for estimating parameters of clonal growth cancer models. *Risk Anal* 1998, *18*, 529-34.
100. Blumenthal, G.M., Kohn, M.C. and Portier, C.J. A mathematical model of production, distribution, and metabolism of melatonin in mammalian systems. *Toxicol Appl Pharmacol* 1997, *147*, 83-92.

101. Gaido, K.W., Leonard, L.S., Lovell, S., Gould, J.C., Babai, D., Portier, C.J. and McDonnell, D.P. Evaluation of chemicals with endocrine modulating activity in a yeast-based steroid hormone receptor gene transcription assay. *Toxicol Appl Pharmacol* 1997, *143*, 205-12.
102. Parham, F.M., Kohn, M.C., Matthews, H.B., DeRosa, C. and Portier, C.J. Using structural information to create physiologically based pharmacokinetic models for all polychlorinated biphenyls. *Toxicol Appl Pharmacol* 1997, *144*, 340-7.
103. Portier, C. and el Masri, H. Statistical research needs in mechanistic modeling for carcinogenic risk assessment. *Stat Methods Med Res* 1997, *6*, 305-15.
104. Sherman, C.D. and Portier, C.J. The two-stage model of carcinogenesis: overcoming the nonidentifiability dilemma. *Risk Anal* 1997, *17*, 367-74.
105. Buchanan, J.R. and Portier, C.J. The use of data on biologically reactive intermediates in risk assessment. *Adv Exp Med Biol* 1996, *387*, 429-37.
106. Bucher, J.R., Portier, C.J., Goodman, J.I., Faustman, E.M. and Lucier, G.W. Workshop overview. National Toxicology Program Studies: principles of dose selection and applications to mechanistic based risk assessment. *Fundam Appl Toxicol* 1996, *31*, 1-8.
107. Kohn, M.C., Sewall, C.H., Lucier, G.W. and Portier, C.J. A mechanistic model of effects of dioxin on thyroid hormones in the rat. *Toxicol Appl Pharmacol* 1996, *136*, 29-48.
108. Luster, M.I., Portier, C., Simeonova, P. and Munson, A.E. Relationship between chemical-induced immunotoxicity and carcinogenesis. *Drug Information Journal* 1996, *30*, 281-286.
109. Melnick, R.L., Kohn, M.C. and Portier, C.J. Implications for risk assessment of suggested nongenotoxic mechanisms of chemical carcinogenesis. *Environ Health Perspect* 1996, *104 Suppl 1*, 123-34.
110. Portier, C.J. and Lyles, C.M. Practicing safe modeling: GLP for biologically based mechanistic models. *Environ Health Perspect* 1996, *104*, 806.
111. Portier, C.J., Kopp-Schneider, A. and Sherman, C.D. Calculating tumor incidence rates in stochastic models of carcinogenesis. *Math Biosci* 1996, *135*, 129-46.
112. Portier, C.J., Sherman, C.D., Kohn, M., Edler, L., Kopp-Schneider, A., Maronpot, R.M. and Lucier, G. Modeling the number and size of hepatic focal lesions following exposure to 2,3,7,8-TCDD. *Toxicol Appl Pharmacol* 1996, *138*, 20-30.
113. Sherman, C.D. and Portier, C.J. Stochastic simulation of a multistage model of carcinogenesis. *Math Biosci* 1996, *134*, 35-50.
114. Kopp-Schneider, A. and Portier, C.J. Carcinoma formation in NMRI mouse skin painting studies is a process suggesting greater than two stages. *Carcinogenesis* 1995, *16*, 53-9.
115. Sherman, C.D. and Portier, C.J. Quantitative analysis of multiple phenotype enzyme-altered foci in rat hepatocarcinogenesis experiments: the multipath/multistage model. *Carcinogenesis* 1995, *16*, 2499-506.
116. Hoel, D.G. and Portier, C.J. Nonlinearity of dose-response functions for carcinogenicity. *Environ Health Perspect* 1994, *102 Suppl 1*, 109-13.

117. Kohn, M.C., Lucier, G.W. and Portier, C.J. The importance of biological realism in dioxin risk assessment models. *Risk Anal* 1994, *14*, 993-1000.
118. Kohn, M.C. and Portier, C.J. A model of effects of TCDD on expression of rat liver proteins. *Prog Clin Biol Res* 1994, *387*, 211-22.
119. Kopp-Schneider, A., Portier, C.J. and Sherman, C.D. The exact formula for tumor incidence in the two-stage model. *Risk Anal* 1994, *14*, 1079-80.
120. Kopp-Schneider, A. and Portier, C.J. A stem cell model for carcinogenesis. *Math Biosci* 1994, *120*, 211-32.
121. Luster, M.I., Portier, C., Pait, D.G. and Germolec, D.R. Use of animal studies in risk assessment for immunotoxicology. *Toxicology* 1994, *92*, 229-43.<sup>7</sup>
122. Portier, C.J., Lucier, G.W. and Edler, L. Risk from low-dose exposures. *Science* 1994, *266*, 1141-2.
123. Portier, C.J. Biostatistical issues in the design and analysis of animal carcinogenicity experiments. *Environ Health Perspect* 1994, *102 Suppl 1*, 5-8.
124. Sherman, C.D., Portier, C.J. and Kopp-Schneider, A. Multistage models of carcinogenesis: an approximation for the size and number distribution of late-stage clones. *Risk Anal* 1994, *14*, 1039-48.
125. Bailer, A.J. and Portier, C.J. An index of tumorigenic potency. *Biometrics* 1993, *49*, 357-65.
126. Carr, G.J. and Portier, C.J. An evaluation of some methods for fitting dose-response models to quantal-response developmental toxicology data. *Biometrics* 1993, *49*, 779-91.
127. Fenger, J., Forslund, J., Grandjean, P., Gron, P., Jensen, F., Keiding, L., Kjaergaard, S., Larsen, J., Lewtas, J., Lynge, E., Moller, H., Moller, L., Nielsen, P., Ostensfeldt, N., Pilsgaard, H., Portier, C., Poulsen, E., Rastogi, S., Skov, T., Thomsen, A., Ulbak, K. and Osterlind, A. Report: The Working Group On Cancer And The Non-Occupational Environment. *Pharmacology and Toxicology* 1993, *72*, 167-171.
128. Kohn, M.C. and Portier, C.J. Effects of the mechanism of receptor-mediated gene expression on the shape of the dose-response curve. *Risk Anal* 1993, *13*, 565-72.
129. Kohn, M.C., Lucier, G.W., Clark, G.C., Sewall, C., Tritscher, A.M. and Portier, C.J. A mechanistic model of effects of dioxin on gene expression in the rat liver. *Toxicol Appl Pharmacol* 1993, *120*, 138-54.
130. Lucier, G.W., Portier, C.J. and Gallo, M.A. Receptor mechanisms and dose-response models for the effects of dioxins. *Environ Health Perspect* 1993, *101*, 36-44.
131. Luster, M.I., Portier, C., Pait, D.G., Rosenthal, G.J., Germolec, D.R., Corsini, E., Blaylock, B.L., Pollock, P., Kouchi, Y., Craig, W. and et al. Risk assessment in immunotoxicology. II. Relationships between immune and host resistance tests. *Fundam Appl Toxicol* 1993, *21*, 71-82.
132. Maronpot, R.R., Foley, J.F., Takahashi, K., Goldsworthy, T., Clark, G., Tritscher, A., Portier, C. and Lucier, G. Dose response for TCDD promotion of hepatocarcinogenesis in rats initiated with DEN: histologic, biochemical, and cell proliferation endpoints. *Environ Health Perspect* 1993, *101*, 634-42.

---

<sup>7</sup> Board of Publications, Best Paper Award, Society of Toxicology, 1995

133. Meier, K.L., Bailer, A.J. and Portier, C.J. A measure of tumorigenic potency incorporating dose-response shape. *Biometrics* 1993, 49, 917-26.
134. Melnick, R.L., Huff, J., Barrett, J.C., Maronpot, R.R., Lucier, G. and Portier, C.J. Cell proliferation and chemical carcinogenesis: symposium overview. *Environ Health Perspect* 1993, 101 Suppl 5, 3-7.
135. Melnick, R.L., Huff, J., Barrett, J.C., Maronpot, R.R., Lucier, G. and Portier, C.J. Cell proliferation and chemical carcinogenesis: a symposium overview. *Mol Carcinog* 1993, 7, 135-8.
136. Portier, C.J., Kopp-Schneider, A. and Sherman, C.D. Using cell replication data in mathematical modeling in carcinogenesis. *Environ Health Perspect* 1993, 101 Suppl 5, 79-86.
137. Portier, C., Tritscher, A., Kohn, M., Sewall, C., Clark, G., Edler, L., Hoel, D. and Lucier, G. Ligand/receptor binding for 2,3,7,8-TCDD: implications for risk assessment. *Fundam Appl Toxicol* 1993, 20, 48-56.<sup>8</sup>
138. Kopp-Schneider, A. and Portier, C.J. Birth and death/differentiation rates of papillomas in mouse skin. *Carcinogenesis* 1992, 13, 973-8.
139. Luster, M.I., Pait, D.G., Portier, C., Rosenthal, G.J., Germolec, D.R., Comment, C.E., Munson, A.E., White, K. and Pollock, P. Qualitative and quantitative experimental models to aid in risk assessment for immunotoxicology. *Toxicol Lett* 1992, 64-65 Spec No, 71-8.
140. Luster, M.I., Portier, C., Pait, D.G., White, K.L., Jr., Gennings, C., Munson, A.E. and Rosenthal, G.J. Risk assessment in immunotoxicology. I. Sensitivity and predictability of immune tests. *Fundam Appl Toxicol* 1992, 18, 200-10.
141. Piegorsch, W.W., Carr, G.J., Portier, C.J. and Hoel, D.G. Concordance of carcinogenic response between rodent species: potency dependence and potential underestimation. *Risk Anal* 1992, 12, 115-21.
142. Tritscher, A.M., Goldstein, J.A., Portier, C.J., McCoy, Z., Clark, G.C. and Lucier, G.W. Dose-response relationships for chronic exposure to 2,3,7,8-tetrachlorodibenzo-p-dioxin in a rat tumor promotion model: quantification and immunolocalization of CYP1A1 and CYP1A2 in the liver. *Cancer Res* 1992, 52, 3436-42.
143. Williams, P. and Portier, C. Analytic expressions for maximum likelihood estimators in a non-parametric model of tumor incidence and death. *Communications in Statistics - Theory and Methods* 1992, 21, 711-732.
144. Williams, P. and Portier, C. Explicit solutions for constrained maximum likelihood estimators in survival/sacrifice experiments. *Biometrika* 1992, 74, 717-729.
145. Carr, G.J. and Portier, C.J. An evaluation of the Rai and Van Ryzin dose-response model in teratology. *Risk Anal* 1991, 11, 111-20.
146. Kopp-Schneider, A. and Portier, C.J. Distinguishing between models of carcinogenesis: the role of clonal expansion. *Fundam Appl Toxicol* 1991, 17, 601-13.
147. Kopp-Schneider, A., Portier, C.J. and Rippmann, F. The application of a multistage model that incorporates DNA damage and repair to the analysis of initiation/promotion experiments. *Math Biosci* 1991, 105, 139-66.

---

<sup>8</sup> Best-applied statistics paper, Centers for Disease Control, 1993.

148. Portier, C.J. and Kopp-Schneider, A. A multistage model of carcinogenesis incorporating DNA damage and repair. *Risk Anal* 1991, *11*, 535-43.
149. Rogan, W.J., Blanton, P.J., Portier, C.J. and Stallard, E. Should the presence of carcinogens in breast milk discourage breast feeding? *Regul Toxicol Pharmacol* 1991, *13*, 228-40.
150. Bailer, A.J. and Portier, C.J. A note on fitting one-compartment models: non-linear least squares versus linear least squares using transformed data. *J Appl Toxicol* 1990, *10*, 303-6.
151. Portier, C.J. and Edler, L. Two-stage models of carcinogenesis, classification of agents, and design of experiments. *Fundam Appl Toxicol* 1990, *14*, 444-60.
152. Kopp, A. and Portier, C.J. A note on approximating the cumulative distribution function of the time to tumor onset in multistage models. *Biometrics* 1989, *45*, 1259-63.
153. Portier, C.J. and Kaplan, N.L. Variability of safe dose estimates when using complicated models of the carcinogenic process. A case study: methylene chloride. *Fundam Appl Toxicol* 1989, *13*, 533-44.
154. Portier, C.J. and Bailer, A.J. Testing for increased carcinogenicity using a survival-adjusted quantal response test. *Fundam Appl Toxicol* 1989, *12*, 731-7.
155. Portier, C.J. and Bailer, A.J. Two-stage models of tumor incidence for historical control animals in the National Toxicology Program's carcinogenicity experiments. *J Toxicol Environ Health* 1989, *27*, 21-45.
156. Bailer, A.J. and Portier, C.J. Effects of treatment-induced mortality and tumor-induced mortality on tests for carcinogenicity in small samples. *Biometrics* 1988, *44*, 417-31.
157. Bailer, A.J. and Portier, C.J. An illustration of dangers of ignoring survival differences in carcinogenic data. *J Appl Toxicol* 1988, *8*, 185-9.
158. Portier, C.J. Species correlation of chemical carcinogens. *Risk Anal* 1988, *8*, 551-3.
159. Portier, C. Life table analysis of carcinogenicity experiments. *Journal of the American College of Toxicology* 1988, *7*, 575-582.
160. Portier, C.J. Statistical properties of a two-stage model of carcinogenesis. *Environ Health Perspect* 1987, *76*, 125-31.
161. Portier, C.J. and Hoel, D.G. Issues concerning the estimation of the TD50. *Risk Anal* 1987, *7*, 437-47.
162. Portier, C.J. and Bailer, A.J. Simulating failure times when the event of interest is unobservable with emphasis on animal carcinogenicity studies. *Comput Biomed Res* 1987, *20*, 458-66.
163. Portier, C.J. and Dinse, G.E. Semiparametric analysis of tumor incidence rates in survival/sacrifice experiments. *Biometrics* 1987, *43*, 107-14.
164. Kupper, L.L., Portier, C., Hogan, M.D. and Yamamoto, E. The impact of litter effects on dose-response modeling in teratology. *Biometrics* 1986, *42*, 85-98.
165. Portier, C.J., Hedges, J.C. and Hoel, D.G. Age-specific models of mortality and tumor onset for historical control animals in the National Toxicology Program's carcinogenicity experiments. *Cancer Res* 1986, *46*, 4372-8.

166. Portier, C.J. Type 1 error and power of the linear trend test in proportions under the National Toxicology Program's modified pathology protocol. *Fundam Appl Toxicol* 1986, 6, 515-9.
167. Portier, C. Estimating the tumor onset distribution in animal carcinogenesis experiments. *Biometrika* 1986, 3, 371-378.
168. Portier, C.J. and Hoel, D.G. Design of animal carcinogenicity studies for goodness-of-fit of multistage models. *Fundam Appl Toxicol* 1984, 4, 949-59.
169. Krewski, D., Crump, K.S., Farmer, J., Gaylor, D.W., Howe, R., Portier, C., Salsburg, D., Sielken, R.L. and Van Ryzin, J. A comparison of statistical methods for low dose extrapolation utilizing time-to-tumor data. *Fundam Appl Toxicol* 1983, 3, 140-60.
170. Portier, C. and Hoel, D. Low-dose-rate extrapolation using the multistage model. *Biometrics* 1983, 39, 897-906.
171. Portier, C. and Hoel, D. Optimal design of the chronic animal bioassay. *J Toxicol Environ Health* 1983, 12, 1-19.
- 172.

*Journal Articles: (reviews and other)*

173. Portier, C. J., Clausing, P., **Re:** Tarazona et al. (2017): Glyphosate toxicity and carcinogenicity: a review of the scientific basis of the European Union assessment and its differences with IARC. *Arch. Toxicol.* 2017, **91** (9) 3195-3197
174. McPartland, J., Dantzker, H.C., Portier, C.J., Elucidating environmental dimensions of neurological disorders and disease: Understanding new tools from federal chemical testing programs, *Sci Total Env* 2017 **593-594** pages 634-640
175. McPartland, J., Dantzker, H.C., Portier, C.J., Building a robust 21st century chemical testing program at the U.S. Environmental Protection Agency: recommendations for strengthening scientific engagement, *Environ Health Perspect*, 2015. **123**(1): 1-5.
176. Portier, C.J., L.R. Goldman, and B.D. Goldstein, Inconclusive findings: now you see them, now you don't! *Environ Health Perspect*, 2014. 122(2): p. A36.
177. Portier, C., ATSDR in the 21<sup>st</sup> Century. *Journal of Environmental Health*, 74(7):30-1, 2011
178. Portier, C. *Comprehensive Public Health*. *Public Health Rep.* 2011 126: Supp 1:3-6
179. Glass, R., Rosenthal, J., Jessup, C., Birnbaum, L., Portier, C. Tackling the research challenges of health and climate change. *Env. Health Perspectives* 2009 117 (12), A534-A535
180. Woodruff TJ, Zeise L, Axelrad DA, Guyton KZ, Janssen S, Miller M, et al. 2008. Meeting report: moving upstream-evaluating adverse upstream end points for improved risk assessment and decision-making. *Environmental health perspectives* 116(11): 1568-1575.
181. Portier, C. Risk Factors for Childhood Leukemia: Discussion and Summary, *Radiation Protection Dosimetry* 2009 **132**(2), 273-274.
182. Gohlke, J. M., Hrynkow, S. H., Portier, C. J., Health, Economy and Environment: Sustainable Energy Choices for a Nation, *Environmental Health Perspectives* 2008 **116** (6), 236-237

183. Kissling, G. E., C. J. Portier and J. Huff. MtBE and cancer in animals: Statistical issues with poly-3 survival adjustments for lifetime studies. *Regul Toxicol Pharmacol*. 2008 **50** (3) 428-429
184. Barton, H. A., W. A. Chiu, R. Woodrow Setzer, M. E. Andersen, A. J. Bailer, F. Y. Bois, R. S. Dewoskin, S. Hays, G. Johanson, N. Jones, G. Loizou, R. C. Macphail, C. J. Portier, M. Spendiff and Y. M. Tan. Characterizing uncertainty and variability in physiologically based pharmacokinetic models: state of the science and needs for research and implementation. *Toxicol Sci* 2007 **99**(2): 395-402.
185. Gohlke, J. M. and C. J. Portier (2007). The forest for the trees: a systems approach to human health research. *Environ Health Perspect* 2007 **115**(9): 1261-3.
186. Portier, C. J., Suk, W. A., Schwartz, D. A., Filling the translation-policy gap. *Env. Health Perspectives* 2007 **115** (3) A125
187. Becker, R. A., C. J. Borgert, S. Webb, J. Ansell, S. Amundson, C. J. Portier, A. Goldberg, L. H. Bruner, A. Rowan, R. D. Curren and W. T. Stott (2006). "Report of an ISRTP workshop: progress and barriers to incorporating alternative toxicological methods in the U.S." *Regul Toxicol Pharmacol* 2006 **46**(1): 18-22.
188. TCDD in female Sprague-Dawley rats. *Organohalogen Compounds* 1996, **29**, 222-227.
189. Portier, C.J. and D.A. Schwartz, The NIEHS and the National Toxicology Program: an integrated scientific vision. *Environ Health Perspect*, 2005. **113**(7): p. A440.
190. Portier, C. J., and Schwartz, D. A.. The NIEHS and the National Toxicology Program: an integrated scientific vision. *Environ Health Perspect* 2005, **113**, A440.
191. Portier, C.J., Suk, W. A. and D.A. Schwartz, Filling the translation-policy gap. *Environ Health Perspect*, 2005. **115**(3): p. A125.
192. Portier, C.J. Comments on the International Symposium on Light, Endocrine Systems and Cancer. *Neuroendocrinol Lett* 2002, **23 Suppl 2**, 79-81.
193. Portier, C. Chipping away at environmental health risk assessment. *Risk Policy Report* 2001, **8**, 37-38.
194. Portier, C. Decisions about environmental health risks: What are the key questions and how does this apply to melatonin? *Zentralblatt fur Arbeitsmedizin* 2000, **50**, 312-314.
195. Portier, C. Risk ranges for various endpoints following exposure to 2,3,7,8-TCDD. *Food Addit Contam* 2000, **17**, 335-46.
196. Brooks, E., Kohn, M., Van Birgelen, A., Lucier, G. and Portier, C. Stochastic models for papilloma formation following exposure to TCDD. *Organohalogen Compounds* 1999, **41**, 522-524.
197. Portier, C., Edler, L., Jung, D., Needham, L., Masten, S.A., Parham, F. and Lucier, G. Half-lives and body burdens for dioxin and dioxin-like compounds in humans estimated from an occupational cohort in Germany. *Organohalogen Compounds* 1999, **42**, 129-138.
198. Tritscher, A., Mahler, J., Portier, C., Lucier, G. and Walker, N.J. TCDD-induced lesions in rat lung following oral exposure. *Organohalogen Compounds* 1999, **42**, 285-288.
199. Lucier, G. and Portier, C. New Cancer Risk Guidelines. *Issues in Science and Technology* 1996, **13**, 10.

200. Portier, C. and Kohn, M. A biologically-based model for the carcinogenic effects of 2378-  
2378-
201. Portier, C., Lucier, G. and Damstra, T. Dioxin research in Vietnam. *Science* 1995, 270,  
901.
202. Portier, C. and Kohn, M. Receptor Mediated Carcinogenesis and Dioxin.  
*Organohalogen Compounds* 1995, 26, 267-272.
203. Edler, L. and Portier, C. Pharmacokinetic modeling of receptor/enzyme pathways for  
the action of dioxins. *Chemosphere* 1994, 25, 239-242.
204. Edler, L., Portier, C. and Kopp-Schneider, A. Zur existenz von schwellenwerten:  
Wissenschaftliche methode oder statistisches artefakt in der risikoabschätzung. *Zbl  
Arbeitsmed* 1994, 44, 16-21.
205. Kohn, M., Lucier, G., Sewall, C. and Portier, C. Physiological modeling of the effects  
of TCDD on thyroid hormones in the rat. *Organohalogen Compounds* 1994, 21, 223-  
226.
206. Portier, C., Kohn, M., Sherman, C.D. and Lucier, G. Modeling the number and size of  
hepatic focal lesions following exposure to 2378-TCDD. *Organohalogen Compounds*  
1994, 21, 393-397.
207. Sherman, C.D. and Portier, C. The multipath/multistage model. *Informatik und  
Epidemiologie in Medizin und Biologie* 1994, 25, 250-254.
208. Sherman, C.D. and Portier, C. The multipath/multistage model of carcinogenesis.  
*Organohalogen Compounds* 1994, 21, 451-455.
209. Portier, C.J. Mechanistic modeling and risk assessment. *Pharmacol Toxicol* 1993, 72  
*Suppl 1*, 28-32.
210. Portier, C. Quantitative Risk Assessment. *ChemTech* 1990, 20, 484-487.

*Books/Book Chapters:*

211. International Agency for Research on Cancer. Shiftwork, painting and firefighting (C.  
Portier member). *IARC Monographs on the Evaluation of Carcinogenic Risks to  
Humans Volume 98*. 2008 In press.
212. Trong, L., Portier, C.J. (eds) Proceedings of the Viet Nam – United States Scientific  
Conferences on Human Health and Environmental Effects of Agent Orange/Dioxin,  
Part 1: Environmental Effects - 3-6 March, 2002. Ha Noi, Vietnam, 2004.
213. Trong, L., Portier, C.J. (eds) Proceedings of the Viet Nam – United States Scientific  
Conferences on Human Health and Environmental Effects of Agent Orange/Dioxin,  
Part 2: Human Health Effects - 3-6 March, 2002. Ha Noi, Vietnam, 2004.
214. DeVito, M., Kim, A.H., Walker, N., Parham, F. and Portier, C. Dose-response  
modeling for 2,3,7,8-tetrachlorodibenzo-p-dioxin. *Dioxins and Health*; 2 ed.; John  
Wiley and Sons: Hoboken, NJ, 2003.
215. World Health Organization. Establishing a dialogue on Risks from Electromagnetic  
Fields. Sahl, J., Bonner, P., Kemp, R., Kheifets, L., Repacholi, M., van Deventer, E.,  
Vogel, E. and Portier, C. WHO Press, 2002

216. International Agency for Research on Cancer. Non-ionizing Radiation, Part 1: Static and Extremely Low Frequency Electric and Magnetic Fields (C. Portier member). I *IARC Monographs on the Evaluation of Carcinogenic Risks to Humans Volume 80*. 19-26 2002
217. Wolfe, M., Boorman, G., Olden, K. and Portier, C. Power-Line Frequency Electric and Magnetic Fields: Effects on Human Health. *Human Health Risks from Exposure to ELF-EMF*; ICNIRP: Maastricht, The Netherlands, 2000.
218. Joint Committee on Food Additives (including C. Portier), World Health Organization/Food and Agriculture Organization. (1999) Evaluation of certain food additives and contaminants: Forty-ninth report of the Joint FAO/WHO Expert Committee on Food Additives. JCAFA. WHO Technical Series #884. 1999.
219. Piver, W., Ando, M., Ye, F. and Portier, C. Temperature and air pollution as risk factors for cerebral vascular diseases in Tokyo, July-August 1980-1995. Proceedings of the 10-th Global Warming International Conference 1999
220. National Institute of Environmental Health Sciences. NIEHS Report on Health Effects from Exposure to Power-Line Frequency Electric and Magnetic Fields. Portier, C.J., Wolfe, M.S., Boorman, G.A., Bernheim, N.J., Galvin, M.J., Newton, S.A., Parham, F.M. and Olden, K.O. NIEHS 1999
221. Willems, B., Portier, C. and Lucier, G. Mechanism-based carcinogenic risk assessment of estrogens and estrogen-like compounds. *The Handbook of Environmental Chemistry*; Springer-Verlag: Heidelberg, 1999.
222. Portier, C.J. and Germolec, D. Immunotoxicology. *Encyclopedia of Biostatistics*; Wiley and Sons: Sussex, England, 1998 pp 1998-2000.
223. Portier, C.J. and Wolfe, M.S. Risk Communication: The focus in the NIEHS RAPID Program's review of EMF health Hazards. *Risk Perception, Risk Communication and Its Application to EMF Exposure*; ICNIRP: Oberschleisheim, Germany, 1998; pp 295-302.
224. Portier, C.J. Risk ranges for various endpoints following exposure to 2,3,7,8-TCDD. *WHO-ECEH/IPCS Assessment of health risk of dioxins*; International Program for Chemical Safety: Geneva, 1998; pp 23.
225. Portier, C.J. and Wolfe, M.S. (Eds.) EMF Science Review Symposium Breakout Group Report for Epidemiology Research Findings; National Institute of Environmental Health Sciences: Research Triangle Park, North Carolina, 1998.
226. Portier, C.J. and Wolfe, M.S. (Eds.) EMF Science Review Symposium Breakout Group Report for Clinical and *In Vivo* Laboratory Findings. In *NIH Publication Number 98-4400*; National Institute of Environmental Health Sciences: Research Triangle Park, North Carolina, 1998.
227. Portier, C.J. and Wolfe, M.S. (Eds.) Assessment of Health Effects from Exposure to Power-Line Frequency Electric and Magnetic Fields. In *NIH Publication Number 98-3981*; National Institute of Environmental Health Sciences: Research Triangle Park, North Carolina, 1998; pp 508.
228. Sherman, C.D. and Portier, C.J. Multistage carcinogenesis Models. *Encyclopedia of Biostatistics*; Wiley and Sons: Sussex, England, 1998; pp 2808-2814.

229. Portier, C.J. and Wolfe, M.S. Linking science to decisions: A strategy for electric and magnetic fields. *Biological Effects of ELF Electric and Magnetic Fields*; ICNIRP: Oberschleisheim, Germany, 1997; pp 211-218.
230. Portier, C.J. and Wolfe, M.S. (Eds.) EMF Science Review Symposium Breakout Group Report for Theoretical Mechanisms and *In Vitro* Research Findings; National Institute of Environmental Health Sciences: Research Triangle Park, North Carolina, 1997.
231. Buchanan, R. and Portier, C.J. The use of data on biologically reactive intermediates in risk assessment. *Biological reactive intermediates V : basic mechanistic research in toxicology and human risk assessment*; 387 ed.; Plenum Press: New York, 1995.
232. Luster, M., Portier, C., Pait, D.G., Rosenthal, G. and Germolec, D. Immunotoxicology and Risk Assessment. *Methods in immunotoxicology*; Wiley-Liss: New York, 1995; pp 51-68.
233. Portier, C.J. Quantitative models for cancer dose-response relationships: parameter estimation. *Low-Dose Extrapolation of Cancer Risks*; ILSI Press: Washington, 1995; pp 123-134.
234. Bailer, A.J. and Portier, C.J. Modeling risks from water contaminants: the application of concentration-response models. *Water contamination and health : integration of exposure assessment, toxicology, and risk assessment*; Dekker: New York, 1994; pp 447-466.
235. Kohn, M.C. and Portier, C.P. A model of the effects of TCDD on expression of rat liver proteins. *Receptor Mediated Biological Processes: Implications for Evaluating Carcinogens*; Wiley-Liss: New York, 1994; pp 211-222.
236. Kohn, M.C., Lucier, G.W. and Portier, C.J. Receptor mechanisms and dioxin risk assessment. *Hazardous Waste and Public Health: International Congress on the Health Effects of Hazardous Waste*; Princeton Press: Princeton, NJ, 1994; pp 421-432.
237. Portier, C.J. and Sherman, C.D. The potential effects of chemical mixtures on the carcinogenic process within the context of the mathematical multistage model. *Risk Assessment of Chemical Mixtures: Biological and Toxicological Issues*; Academic Press: New York, 1994; pp 665-686.
238. Portier, C.J., Hoel, D.G., Kaplan, N.L. and Kopp, A. Biologically based models for risk assessment. *IARC Sci Publ* 1990, 20-8.
239. Portier, C. Quantitative risk assessment. *Carcinogenicity and Pesticides*; American Chemical Society: New York, 1989; pp 164-174.
240. Portier, C. Design of long-term animal carcinogenicity experiments: dose allocation, animal allocation and sacrifice times. *Statistical Methods in Toxicological Research*; Gordan and Brecch: New York, 1989; pp 455-467.
241. Portier, C. Utilizing biologically-based models to estimate carcinogenic risk. *Scientific Issues in Quantitative Cancer Risk Assessment*; Birkhauser: New York, 1989; pp 252-266.
242. Kaplan, N., Hogan, M., Portier, C. and Hoel, D. An evaluation of the safety factor approach in risk assessment. *Developmental Toxicology: Mechanisms and Risk*; Cold Spring Harbor Laboratory: New York, 1988.

243. Portier, C. (1985) Optimal dose/animal allocation for terminal sacrifice studies. Proceedings of the ASA Conference on Long-Term Animal Carcinogenicity Studies, 42-50.
244. Portier, C.J., Hoel, D. and VanRyzin, J. Statistical analysis of the carcinogenesis bioassay data relating to the risks from exposure to 2,3,7,8-tetrachlorodibenzo-p-dioxin. *Public Health Risks of the Dioxins*; W. Kaufmann: Los Altos, 1984; pp 99-120.

*Reports and Agency Publications:*

245. A Human Health Perspective on Climate Change, Report of the Interagency Working Group on Climate Change and Health (C. Portier, chair), NIEHS, RTP, NC 2009
246. IARC Report of the Advisory Working Group for Priorities for future IARC Monographs, International Agency for Research on Cancer, Lyon, France 2008
247. NIEHS Centers for Children's Environmental Health and Disease Prevention Research Program, Review Panel Report, NIEHS, RTP, NC 2007
248. WHO Environmental Health Criteria Monograph 238; Extremely Low Frequency Electric and Magnetic Fields, (C. Portier, Chair), pp 543, World Health Organization Press, Geneva, Switzerland – 2007 ISBN 978 92 4 157238 5
249. IARC Report of the Advisory Group to Plan Monograph Volume 100: A Review of Human Carcinogens, International Agency for Research on Cancer, Lyon, France 2006
250. NIEHS Report of the Expert Panel to the NIEHS on Thimerosal Exposure in Pediatric Vaccines: Feasibility of Studies Using the Vaccine Safety Datalink, NIEHS, RTP, NC 2006
251. IARC Report of the Advisory Group to Revise the IARC Monographs Preamble International Agency for Research on Cancer, Lyon, France 2005
252. NTP. A National Toxicology Program for the 21st Century: A roadmap to achieve the NTP vision, pp. 4. National Toxicology Program/National Institute of Environmental Health Sciences, Research Triangle Park, NC. 2004
253. NTP. NTP Current directions and evolving strategies, pp. 4. National Toxicology Program/National Institute of Environmental Health Sciences, Research Triangle Park, NC. 2004
254. NTP. NTP Vision: Toxicology in the 21st Century: The role of the National Toxicology Program, pp. 4. National Toxicology Program/National Institute of Environmental Health Sciences, Research Triangle Park, NC 2003
255. USEPA Science Advisory Panel (C. Portier, Chair). Review of Characterization of Epidemiology Data Relating to Prostate Cancer and Exposure to Atrazine. FIFRA-SAP. US EPA Science Advisory Panel Report No. 2003-02.
256. USEPA Science Advisory Panel (C. Portier, Member). Review of Proposed Science Policy: PPAR-alpha Agonist-Mediated Hepatocarcinogenesis in Rodents and Relevance to Human Health Risk Assessments. FIFRA-SAP. US EPA Science Advisory Panel Report No. 2003-04.
257. USEPA Science Advisory Panel (C. Portier, Chair). Review of Physiologically-Based Pharmacokinetic/Pharmacodynamic Modeling: Preliminary Evaluation and Case Study for the N-Methyl Carbamate Pesticides: A Consultation. FIFRA-SAP. US EPA Science Advisory Panel Report No. 2003-05.

258. USEPA Science Advisory Panel (C. Portier, Member). Comments on Determination of the Appropriate FQPA Safety Factor(s) in the Organophosphorous Pesticide Cumulative Risk Assessment: Susceptibility and Sensitivity to the Common Mechanism, Acetylcholinesterase Inhibition. FIFRA-SAP. US EPA Science Advisory Panel Report No. 2002-03.
259. USEPA Science Advisory Panel (C. Portier, Member). Review Of Draft Termite Bait Product Performance Testing Guideline. FIFRA-SAP. US EPA Science Advisory Panel Report No. 2002-04.
260. USEPA Science Advisory Panel (C. Portier, Chair). Review of Corn Rootworm Plant-Incorporated Protectant Insect Resistance Management and Non-Target Insect Issues. FIFRA-SAP. US EPA Science Advisory Panel Report No. 2002-05.
261. USEPA Science Advisory Panel (C. Portier, Member). Review of Stochastic Human Exposure and Dose Simulation Model (SHEDS). FIFRA-SAP. US EPA Science Advisory Panel Report No. 2002-06.
262. IARC Monographs Volume 80 (C. Portier member) Non-Ionizing Radiation, Part 1: Static and Extremely Low-Frequency (ELF) Electric and Magnetic Fields, 429 pages; 2002
263. USEPA Science Advisory Panel (C. Portier, Member). Comments on Methods to Conduct a Preliminary Cumulative Risk Assessment for Organophosphorous Chemicals. FIFRA-SAP. US EPA Science Advisory Panel Report No. 2002-01.
264. USEPA Science Advisory Panel (C. Portier, Chair). BT Plant-Pesticides Risk and Benefit Assessment. FIFRA-SAP. US EPA Science Advisory Panel Report No. 2001-07.
265. USEPA Science Advisory Panel (C. Portier, Chair). Review of dietary exposure evaluation model (DEEM) and MaxLIP pesticide residue decomposing procedures and software. FIFRA-SAP. Science Advisory Panel Report No. 2001-01B.
266. USEPA Science Advisory Panel (C. Portier, Chair). Review of dietary exposure evaluation model (DEEM). FIFRA-SAP. EPA Science Advisory Panel Report No. 2001-01C.
267. USEPA Science Advisory Panel (C. Portier, Chair). Consultation on Development and Use of Distributions of Pesticide Concentrations in Drinking Water for FQPA Assessments. FIFRA-SAP. EPA Science Advisory Panel Report No. 2001-01D.
268. USEPA Science Advisory Panel (C. Portier, Member). Review of Probabilistic models and methodologies: Advancing the ecological risk assessment process in the EPA Office of Pesticides Programs. FIFRA-SAP. US EPA Science Advisory Panel Report No. 2001-06.
269. USEPA Science Advisory Panel (C. Portier, Chair). Review of Common Mechanism of Action of Thiocarbamates and Dithiocarbamates. FIFRA-SAP. US EPA Science Advisory Panel Report No. 2001-12.
270. USEPA Science Advisory Panel (C. Portier, Member). Review of Preliminary Cumulative Hazard and Dose-Response Assessment for Organophosphorus Pesticides: Determination of Relative Potency and Points of Departure for Cholinesterase Inhibition. FIFRA-SAP. US EPA Science Advisory Panel Report No. 2001-11.

271. USEPA Science Advisory Panel (C. Portier, Chair). Review of Characterization and Non-Target Organism Data Requirements for Protein Plant-Pesticides and- Cumulative Risk Assessment Methodology Issues of Pesticide Substances that Have a Common Mechanism of Toxicity. FIFRA-SAP. EPA-SAP-99-06. February 4, 2000.
272. USEPA Science Advisory Panel (C Portier, Member). Review of food allergenicity of CRY9C Endotoxin and other non-digestible proteins. FIFRA-SAP. USEPA Science Advisory Panel Report 2000-01A.
273. USEPA Science Advisory Panel (C Portier, Member). Review of atrazine: hazard and dose-response assessment and characterization. FIFRA-SAP. USEPA Science Advisory Panel Report No. 2000-05.
274. USEPA Science Advisory Panel (C. Portier, Chairman). Review of Statistical Methods for Use of Composite Data in Acute Dietary Exposure Assessment. FIFRA-SAP. EPA-SAP 99-03B. May 26, 1999.
275. USEPA Science Advisory Panel (C. Portier, Chairman). Review of the Use of Watershed-derived Percent Crop Areas as a Refinement Tool in FQPA Drinking Water Exposure Assessments for Tolerance Reassurance. FIFRA-SAP. EPA-SAP 99-03C. May 26, 1999.
276. USEPA Science Advisory Panel (C. Portier, Member). *Review of Burkholderia cepacia*: Risk Assessment of a Biopesticide with Affinities to a Human Opportunistic Pathogen. FIFRA-SAP. EPA-SAP-99-04. September 30, 1999.
277. USEPA Science Advisory Panel (C. Portier, Member). A Consultation on Protocol Design to Assess Acute Neurotoxicity Studies following Oral Administration of Pesticides. FIFRA-SAP. EPA-SAP-99-04B. September 30, 1999.
278. USEPA Science Advisory Panel (C. Portier, Member). Review of Higher Tier Ecological Risk Assessment for Chlorfenapyr. FIFRA-SAP. EPA-SAP-99-04C. September 30, 1999.
279. USEPA Science Advisory Panel (C. Portier, Chair). Review of -Spray Drift: Review of Proposed Pesticide Deposition Curves to Adjacent Areas. FIFRA-SAP. EPA-SAP-99-04C. September 30, 1999.
280. USEPA Science Advisory Panel (C. Portier, Member). Review of Guidance Document for Small Scale Prospective Ground Water Monitoring Studies. FIFRA-SAP. SAP98-01. November 19, 1998.
281. USEPA Science Advisory Panel (C. Portier, Member). Review of Common Mechanism of Action of Organophosphates. FIFRA-SAP. March, 1998.
282. USEPA Science Advisory Panel (C. Portier, Member). Review of Suggested Probabilistic Risk Assessment Methodology for Evaluating Pesticides That Exhibit a Common Mechanism of Action. FIFRA-SAP. March, 1998.
283. USEPA Science Advisory Panel (C. Portier, Member). Review of the Use of FQPA 10x Safety Factor to Address Special Sensitivity of Infants and Children to Pesticides. FIFRA-SAP. March, 1998.
284. USEPA Science Advisory Panel (C. Portier, Member). Review of Post Application Exposure Guidelines: Series 875-Group B. FIFRA-SAP. March, 1998.

285. USEPA Science Advisory Panel (C. Portier, Member). Review of Proposed Methods for Basin-scale Estimation of Pesticide Concentrations in Flowing Water and Reservoirs for Tolerance Reassessment. FIFRA-SAP. July, 1998.
286. USEPA Science Advisory Panel (C. Portier, Member). Review of Linear Low Dose Extrapolation for Cancer Risk Decisions: Sources of Uncertainty and How They Affect the Precision of Risk Estimates. FIFRA-SAP. July, 1998.
287. USEPA Science Advisory Panel (C. Portier, Member). Review of DDVP (Dichlorvos) Risk Issues. FIFRA-SAP. July, 1998.
288. USEPA Science Advisory Panel (C. Portier, Member). Review of FQPA 10x Safety Factor: Status Report. FIFRA-SAP. July, 1998.
289. USEPA Science Advisory Panel (C. Portier, Member). Review of Chlorothalonil: Mechanism for the Formation of Renal and Forestomach Tumors. FIFRA-SAP. July, 1998.
290. Environmental Protection Agency. Dose Response Modeling for TCDD. Lucier, G., Gallo, M., Portier, C.J., Bayard, S., Cooper, K., Georgopolous, P., McGrath, L., Andersen, M., DeVito, M., White, P., Kedderis, L., Mills, J. and Silbergeld, E. EPA/600/BP-92/001b 1994
291. Portier, C.J. Optimal bioassay design under the Armitage-Doll multistage model of carcinogenesis. PhD, University of North Carolina. 1981.

## Appendix II: Previous Cases Resulting in Depositions and Court Appearances

Glyphosate multidistrict litigation under Judge Vince Chhabria. MDL 2741, Case 3:16-md-02741-VC, US District Court, Northern District of California

Edwin Hardeman (plaintiff) v. Monsanto Company (defendant), MDL 2741, Case 3:16-cv-00525-VC, US District Court, Northern District of California

Edwin Hardeman (plaintiff) v. Monsanto Company (defendant), MDL 2741, Case 3:16-cv-00525-VC, US District Court, Northern District of California

Alva and Alberta Pilliod (plaintiffs) v. Monsanto Company (defendant), Alameda County Superior Court, Case A158228

Walter Winston et al. (plaintiffs) v. Monsanto Company (defendant), Circuit Court of the City of St. Louis, State of Missouri, Case No. 1822-CC00515

Depositions from Winston v. Monsanto were also to be used for the following

- Bellah v. Monsanto, Lake C., CA
- Caballero v. Monsanto, Alameda County, CA
- Bargas v. Monsanto, Alameda County, CA
- Wade v. Monsanto, St. Louis City, MO
- Stevick v. Monsanto, San Francisco, CA

Seitz v. Monsanto, St. Louis City, MO  
Kane v. Monsanto, St. Louis City, MO  
Bogner v. Monsanto, St. Louis County, MO  
Neal v. Monsanto, St. Louis CCity, MO

### Appendix III: Compensation

Billing is at \$500.00 per hour in 30-minute increments for all activities including depositions and trial testimony with the exception of travel time which will be billed at \$200.00 per hour with a maximum of 8 hours per day. Reasonable expenses incurred including transportation costs, hotels and meals will be reimbursed.

### Certification

I hereby certify that this report is a complete and accurate statement of all of my opinions, and the basis and reasons for them, to which I will testify under oath.



3/1/2021

---

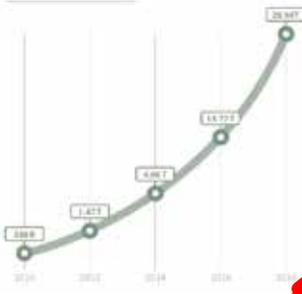
Christopher J. Portier

---

Date

# SLT WIRELESS BROADBAND AND YOU

## REPORTED WIRELESS DATA TRAFFIC (MEGABYTES)



## October 2019

- **Wireless data use almost doubles in just one year.** Wireless data puts the internet in the palm of our hand and allows us to access nearly anything or anyone on the go, and its tremendous value to consumers shows no signs of slowing.
- This year, we saw mobile data grow by **12.89 trillion MBs** to a **total of 28.58 trillion.**
- That's an over **82 percent increase** in the last year alone and is more data than was used in the first six and a half years of this decade—combined.
- In fact, data use is up over **73x since 2010.**

### Reference point 1. CTIA 2019 Annual Survey

<https://www.ctia.org/news/2019-annual-survey-highlights/>

## NEED MORE TOWERS



Less Towers = Longer Wait

- Using phones in areas of good reception decreases exposure as it allows the phone to transmit at reduced power." More towers mean better coverage and hence less EMF radiation exposure from mobile phones.

### Reference 3. FCC Factsheet No 193. Reviewed October 2014

## STUDIED FOR SAFETY

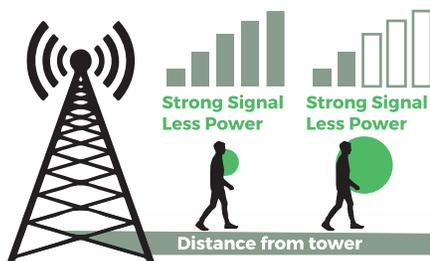


- A fast wireless network is a critical resource for our citizens, and failing to provide them is no different than failing to provide clean drinking water, natural gas, sewage service, or electricity.
- When a disaster occurs, many people need to know about it. An increasingly large segment of the population now uses mobile devices instead of landlines. Receiving an alert on mobile devices is vital for emergency preparedness.

### Reference 2. Wireless Emergency Alerts Report by the Department of Homeland Security

[www.dhs.gov/sites/default/files/publications/Wireless\\_Emergency%20Alerts%20Mobile%20Penetration%20Strategy.pdf](http://www.dhs.gov/sites/default/files/publications/Wireless_Emergency%20Alerts%20Mobile%20Penetration%20Strategy.pdf)

## THIS IS NOT A 5G TOWER



- A 5G tower is different than a 4G tower both physically and functionally: more 5G towers are needed to cover the same amount of space, they're much smaller, and they transmit data on an entirely different part of the radio spectrum.

### Reference 4. 5G Cell Towers: Why You See Them and How They Work

<https://www.lifewire.com/5g-cell-towers-4584192>

## Do Cell Towers Lower Property Values?



- The distance from a wireless facility has no apparent impact on the value or sale price of a home. The relationship between the list and sale price remained the same no matter how close the property was to the wireless facility.

### Reference 5. Joint Ventures Wireless Communications Initiative Study Wireless Facilities Impact on Property Values November 2012

<https://jointventure.org/images/stories/pdf/WirelessFacilitiesImpactOnPropertyValues.pdf>

## Are Cellphone Towers Dangerous?

Research by organizations such as the National Institute for Occupational Safety and Health, The Environmental Protection Agency (EPA), FCC and others have found **RF energy within the regulated levels are not harmful to humans.**

Radiofrequency (RF) waves, a form of energy, is released when a mobile device (phone, tablet or laptop) connects with a cell tower.

Different devices create different frequencies on the Electro Magnetic Spectrum. Some frequencies are harmful to humans while others are not.

For instance, the frequencies that cell phones and gamma rays are on the radioactive range of the electromagnetic spectrum, and can cause harmful damage to the chemical bonds in our DNA.

**RF energy from cell towers and mobile devices is “non-ionizing,” similar to radio and television waves.**

Tall cell towers keep RF energy high above the ground. **At ground level, RF energy from towers is thousands of times less than the FCC safe exposure limits.** Other antennas, such as those used for radio and television broadcast transmissions, use power levels that are generally much higher than those used for cellular antennas.”

## DEFINITIONS & REFERENCES

**Mobile Broadband** – The use of high speed internet via mobile devices (smart phone, tablet or laptop) that utilizes frequencies on the electro magnetic spectrum.

**Electro Magnetic Spectrum** – The range of frequencies that emit electro magnetic energy. The lower end of the spectrum has low frequencies and longer waves of energy, while the higher end has high frequencies and shorter waves.

**Electro Magnetic Energy** – Any energy emitted or absorbed by charged particles traveling through space, anything from visible light to nuclear reactions.

**Ionizing and Non-ionizing Energy** – Ionizing energy is energy on the high end of the spectrum that is harmful to human DNA. Energies that are on the low end of the spectrum are considered non-ionizing energy and are not harmful to humans.

**Radio Frequency (RF) Energy** - The range of frequencies on the non-ionizing end of the electro magnetic spectrum used for telecommunication devices such as mobile phones, laptops, radios and television.

## What the Experts Say...

A systematic review of existing academic studies on the potential health risks of RF emissions found that the majority of research on the subject currently indicates no ill-health related to RF energy exposure.

The World Health Organization (WHO) has classified RF energy as “possibly carcinogenic to humans.” WHO also states that in the last twenty years “no adverse health effects have been established as being caused by mobile phone use.”

The American Cancer Society, the International Agency for Research on Cancer and the National Toxicology Program claim that cell towers are unlikely to cause cancer.

Research is ongoing. There is consensus that additional research is warranted to address gaps in knowledge, such as the effects of cell phone use over the long-term and on pediatric populations.



World Health Organization, Electromagnetic fields and public health: mobile Phones, WHO Fact Sheet #102, June, 2011

FCC Radio Frequency Safety <http://transition.fcc.gov/oet/rfsafety/rf-faqs.html>

The American Cancer Society, <http://www.cancer.gov/cancer-causes/othercarcinogens/athome/cellphone-towers>

Martin Rööfli et al., “Systematic Review on the Health Effects of Exposure to Radiofrequency Electromagnetic Fields from Mobile Phone Base Stations,” Bulletin of the World Health Organization, November 1, 2010): 887–896F.

The Food and Drug Administration, <http://www.fda.gov/Radiation-EmittingProducts/RadiationEmittingProductsandProcedures/HomeBusinessandEntertainment/CellPhones/ucm116335.htm>

CTIA 2019 Annual Survey, <https://www.ctia.org/news/2019-annual-survey-highlights/>

Wireless Emergency Alerts Report by the Department of Homeland Security, [www.dhs.gov/sites/default/files/publications/Wireless%20Emergency%20Alerts%20Mobile%20Penetration%20Strategy.pdf](http://www.dhs.gov/sites/default/files/publications/Wireless%20Emergency%20Alerts%20Mobile%20Penetration%20Strategy.pdf)

WHO Factsheet No 193. Reviewed October 2014

5G Cell Towers: Why You See Them and How They Work, <https://www.lifewire.com/5g-cell-towers-4584192>

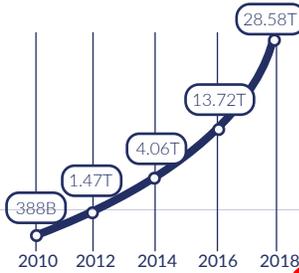
Joint Ventures Wireless Communications Initiative Study Wireless Facilities Impact on Property Values. November 2012 <https://jointventure.org/images/stories/pdf/WirelessFacilitiesImpactOnPropertyValues.pdf>



TAHOE PROSPERITY CENTER

# TAHOE WIRELESS BROADBAND AND YOU

## REPORTED WIRELESS DATA TRAFFIC (MEGABYTES)



- **Wireless data use almost doubles in just one year.** Wireless data puts the internet in the palm of our hand and allows us to access nearly anything or anyone on the go, and its tremendous value to consumers shows no signs of slowing.
- This year, we saw mobile data grow by **12.89 trillion MBs** to a **total of 28.58 trillion.**
- That's an **82 percent increase** in the last year alone and is more data than was used in the first six and a half years of this decade combined.
- In fact, data use is up over **73 times since 2010.**<sup>1</sup>

## NEED MORE TOWERS



Less Towers = Longer Wait

## STUDIED FOR SAFETY

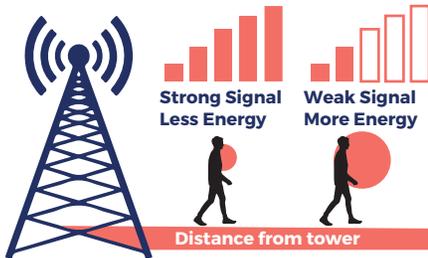


Regulated Levels Are Safe to Humans



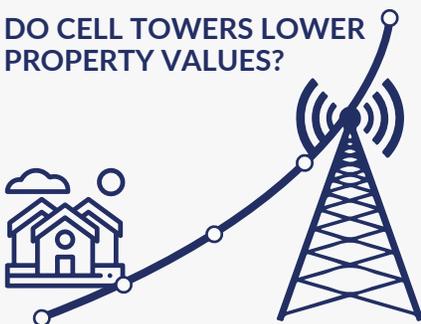
- **A fast wireless network is a critical resource for our community,** and failing to provide fast wireless networks is no different than failing to provide clean drinking water, natural gas, sewage service, or electricity.<sup>3</sup>
- When a disaster occurs people need to know about it. An increasingly large segment of the population use mobile devices instead of landlines. **Receiving a report on mobile devices is vital for emergency preparedness.**
- The World Health Organization (WHO) has classified radio frequency energy as “possibly carcinogenic to humans.” **WHO also states that in the last twenty years no adverse health effects have been established as being caused by mobile phone use.**<sup>2</sup>
- The American Cancer Society, the International Agency for Research on Cancer and the National Toxicology Program claim that **cell towers are unlikely to cause cancer.**

## CELL TOWER SIGNAL



- **Using phones in areas of good reception decreases exposure as it allows the phone to transmit at reduced power.** More towers mean better coverage and hence less electromagnetic field radiation exposure from mobile phones.<sup>2</sup>

## DO CELL TOWERS LOWER PROPERTY VALUES?



- The distance from a wireless facility has no apparent impact on the value or sale price of a home. The relationship between **the list and sale price remained the same no matter how close the property was to the wireless facility.**<sup>5</sup>

## ARE CELLPHONE TOWERS DANGEROUS?

Research by organizations such as the National Institute for Occupational Safety and Health, the environmental Protection Agency (EPA), FCC and others have found **radio frequency energy within the regulated levels are not harmful to humans.**

Radio frequency waves, a form of energy, is released when a mobile device (phone, tablet or laptop) connects with a cell tower.

Different devices create different frequencies on the electromagnetic spectrum. **Some frequencies are harmful to humans while others are not.**

For instance, the frequencies that carry x-rays and gamma rays are on the radioactive end of the electromagnetic spectrum, and can cause harmful damage to the chemical bonds in our DNA.

Radio frequency energy from cell towers and mobile devices is "non-ionizing," similar to radio and television waves.

All cell towers keep radio frequency energy high above the ground. **At ground level, radio frequency energy from towers is thousands of times less than the FCC safe exposure limits.** Other antennas, such as those used for radio and television broadcast transmissions, use power levels that are generally much higher than those used for cellular antennas.<sup>6</sup>

## DEFINITIONS

**Mobile Broadband** – The use of high speed internet via mobile devices (smart phone, tablet or laptop) that utilizes frequencies on the electro magnetic spectrum.

**Electromagnetic Spectrum** – The range of frequencies that emit electromagnetic energy. The lower end of the spectrum has low frequencies and longer waves of energy, while the higher end has high frequencies and shorter waves.

**Electromagnetic Energy** – Any energy emitted or absorbed by charged particles traveling through space, anything from visible light to nuclear reactions.

**Ionizing and Non-ionizing Energy** – Ionizing energy is energy on the high end of the spectrum that is harmful to human DNA. Energies that are on the low end of the spectrum are considered non-ionizing energy and are not harmful to humans.

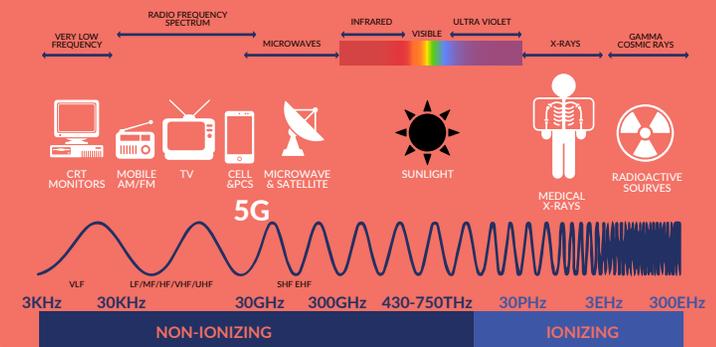
**Radio Frequency Energy** - The range of frequencies on the non-ionizing end of the electromagnetic spectrum used for telecommunications devices such as mobile phones, laptops, radios and television.

## WHAT THE EXPERTS SAY...

A systematic review of existing academic studies on the potential health risks of radio frequency emissions found that **the majority of research on the subject currently indicates no ill-health related to radio frequency energy exposure.**<sup>7</sup>

Research is ongoing. There is consensus that additional research is warranted to address gaps in knowledge, such as the effects of cell phone use over the long-term and on pediatric populations.<sup>8</sup>

### THE ELECTROMAGNETIC SPECTRUM



The electromagnetic spectrum. CNET

## REFERENCES

1. CNET 2019 Annual Survey, [www.cnet.com/news/2019-annual-survey-highlights](http://www.cnet.com/news/2019-annual-survey-highlights)
2. World Health Organization, Electromagnetic fields and public health: mobile Phone. WHO Fact Sheet #193. June, 2011. Reviewed October 2014
3. Wireless Emergency Alerts report by the Department of Homeland Security, [www.dhs.gov/sites/default/files/publications/Wireless%20Emergency%20Alerts%20Mobile%20Communication%20Strategy.pdf](http://www.dhs.gov/sites/default/files/publications/Wireless%20Emergency%20Alerts%20Mobile%20Communication%20Strategy.pdf)
4. The American Cancer Society, [www.cancer.org/cancer/cell-phones/othercarcinogens/athome/cellular-phone-towers](http://www.cancer.org/cancer/cell-phones/othercarcinogens/athome/cellular-phone-towers)
5. Joint Ventures Wireless Communications Initiative Study - Wireless Facilities Impact on Property Values, November 2012 [www.jointventure.org/images/stories/pdf/WirelessFacilitiesImpactOnPropertyValues.pdf](http://www.jointventure.org/images/stories/pdf/WirelessFacilitiesImpactOnPropertyValues.pdf)
6. FCC Radio Frequency Safety, [www.transition.fcc.gov/oet/rfsafety/rf-faqs.html](http://www.transition.fcc.gov/oet/rfsafety/rf-faqs.html)
7. Martin Röösli et al., "Systematic Review on the Health Effects of Exposure to Radiofrequency Electromagnetic Fields from Mobile Phone Base Stations," Bulletin of the World Health Organization 88, no. 12 (December 1, 2010): 887–896F.
8. The Food and Drug Administration, [www.fda.gov/Radiation-EmittingProducts/RadiationEmittingProductsandProcedures/HomeBusinessandEntertainment/CellPhones/ucm116335.htm](http://www.fda.gov/Radiation-EmittingProducts/RadiationEmittingProductsandProcedures/HomeBusinessandEntertainment/CellPhones/ucm116335.htm)



TAHOE PROSPERITY CENTER

[tahoeprosperity.org](http://tahoeprosperity.org)



TAHOE PROSPERITY CENTER  
tahoeprosperity.org

October 14, 2019

Dear Mayor and City Council Members,

As you know, the Lake Tahoe Basin Prosperity Plan, completed in 2010, created the Tahoe Prosperity Center and was focused on ways to improve the local community and economy. The top two issues in the original Lake Tahoe Basin Prosperity Plan that would improve prosperity in our community were:

1. Certainty in the marketplace and
2. Broadband and cell phone connectivity.

You have an opportunity to do both in the case of the cell tower located at 1360 Ski Run Boulevard and begin that process of improving prosperity. As stated in our previous email of August 5, 2019 the Tahoe Prosperity Center is very concerned about the public safety ramifications (and negative consequences) of reversing the approval of a previously approved cell tower that is desperately needed.

LIE

We are also concerned about the misinformation being shared about potential negative impacts from cell towers and about the process they believe you should follow as you make a determination. We address each of those below using the -quoted language- of those who have not been named, but list themselves as "Concerned Citizens of South Lake Tahoe" as they have been emailing me.

- 1) **"We already get good coverage here."** Public safety is our number one priority. Provider maps have two primary levels of service and while coverage maps do show much of this region as "covered" that is simply one level of service. My house in Meyers is "covered" on both the Verizon and AT&T maps. However, I have to stand in my driveway to get one bar of service, and generally only mid-week on clear, sunny days. I am not able to use my phone inside my house or even outside on my back deck, so I have a land-line. In the Ski Run area, you can stand out on the sidewalk and probably get a bar or two of service, but in-building service is not consistent in much of the area this new cell tower will serve. Having service both inside and outside of buildings is needed for emergencies.

LIE

- 2) **"You are complicit in 'harming our children'."** As you will recall from the expert scientific testimony on April 2, 2019, there are no negative long-term health impacts related to cell towers and the radio frequencies they utilize. The American Cancer Society, World Health Organization and the Federal Drug Administration concur. Most of us drink coffee every day. Coffee and cell phones/towers are both listed as a "possible" 2b carcinogen according to the International Agency for Research on Cancer (IARC). In addition, baby monitors, WiFi routers and other electronic devices in our homes use the same radiofrequency waves. We are not suggesting banning coffee, baby monitors or Wifi, yet this group asks you to ban cell towers.

LIE

F.U.

- 3) **"Just put them on public lands."** Some have suggested that cell towers can "easily be relocated to public lands" in the Tahoe Basin. That is simply untrue. Our Connected Tahoe project mapped all of the public land in the Tahoe Basin and the towers that are able to be placed on those lands have been evaluated. The few sites identified for public lands are moving forward through the normal permit processes, but one of those has been in process for nine years! Yes – nine years of permitting. Our evaluation found only a handful of sites determined as viable on public lands. We recognized that private property, such as the land at 1360 Ski Run Boulevard is a better solution for improving public safety and cell service and it will be co-located with multiple carriers.

LIE

Heavenly  
DAS  
used  
ski resort  
special  
use permit

4) **“This is not proper planning.”** It has also been suggested that these sites are being proposed without thoughtful consideration and that providers should give up their “master plans” publicly. Not only does this fly in the face of “business competitive advantage”, it is also factually incorrect. Tahoe Prosperity Center did map proposed cell tower sites in the region and this location is a priority site. Additionally, a significant amount of research, engineering, design and thought goes into the siting of a proposed cell tower. Each one of these proposed towers goes through an extensive (some might say exhaustive) permit process with the local jurisdiction, the Tahoe Regional Planning Agency. The Planning Commission did its thorough review and approved this site and we believe you should uphold their decision. The TRPA will also do its thorough review.

LIE: pretextual

5) **“Just create a new ordinance for cell towers.”** Regarding certainty in the marketplace, Tahoe is already well-known as a place that is challenging to do business. We struggle with bringing private investment to our region due to the level of uncertainty in the market – whether for a cell tower, a housing development or retail and business opportunities. This exact site was approved six years ago for a mono-pine for AT&T, who chose to re-direct their investment into CAF-II (Connect America Funding) instead. Their approved permit expired as they redirected into CAF-II. It should be noted, there was no opposition by neighbors to the exact same location at that time. Verizon decided to apply for the same site and the City’s Planning Commission unanimously approved it. While we agree a clear and concise City telecommunications policy makes sense, changing the rules halfway through a permit does not. We applaud the effort of the City Manager to try and find a suitable alternate location for this tower as a win-win solution, however, that effort is costing Verizon and the City - in terms of staff time, re-design studies and engineering. It should be fully accounted for and factored into any future permitting costs to Verizon.

BINGO

6) **“We don’t really need another tower.”** Another critical issue is capacity. Without adding some large co-located towers such as this one, along with small cell towers on utility or light poles, we run the risk of not being able to send texts or make calls in an emergency situation. Given our heavy population increases during holiday periods, as well as our winter and summer visitation seasons, we must add cell service capacity in order to serve both our residents who live here full-time, and our visitors when our population swells. We can see up to 250,000 visitors in busy holiday weekends, so both large and small cell towers are needed to cover that many people. You simply cannot protect the community with the existing cell tower infrastructure. Even your Police and Fire departments rely on cell phones to communicate – something that could greatly impact their ability to respond in emergencies without improved coverage.

LIE

BINGO

LIE

I would argue that those who oppose this cell tower would still like to see improved cell service, but just not in “their neighborhood.” As stated earlier, these towers can’t simply be located on US Forest Service (USFS) public lands. USFS lands have already been evaluated and the minimum number of sites that were determined feasible are moving forward, but those few sites will not be enough to improve coverage for all our residents, businesses and visitors in the community.

We hope that you support the City Planning Commission and the previous approval of the cell tower at 1360 Ski Run Blvd – for the safety of all the residents of the City of South Lake Tahoe.

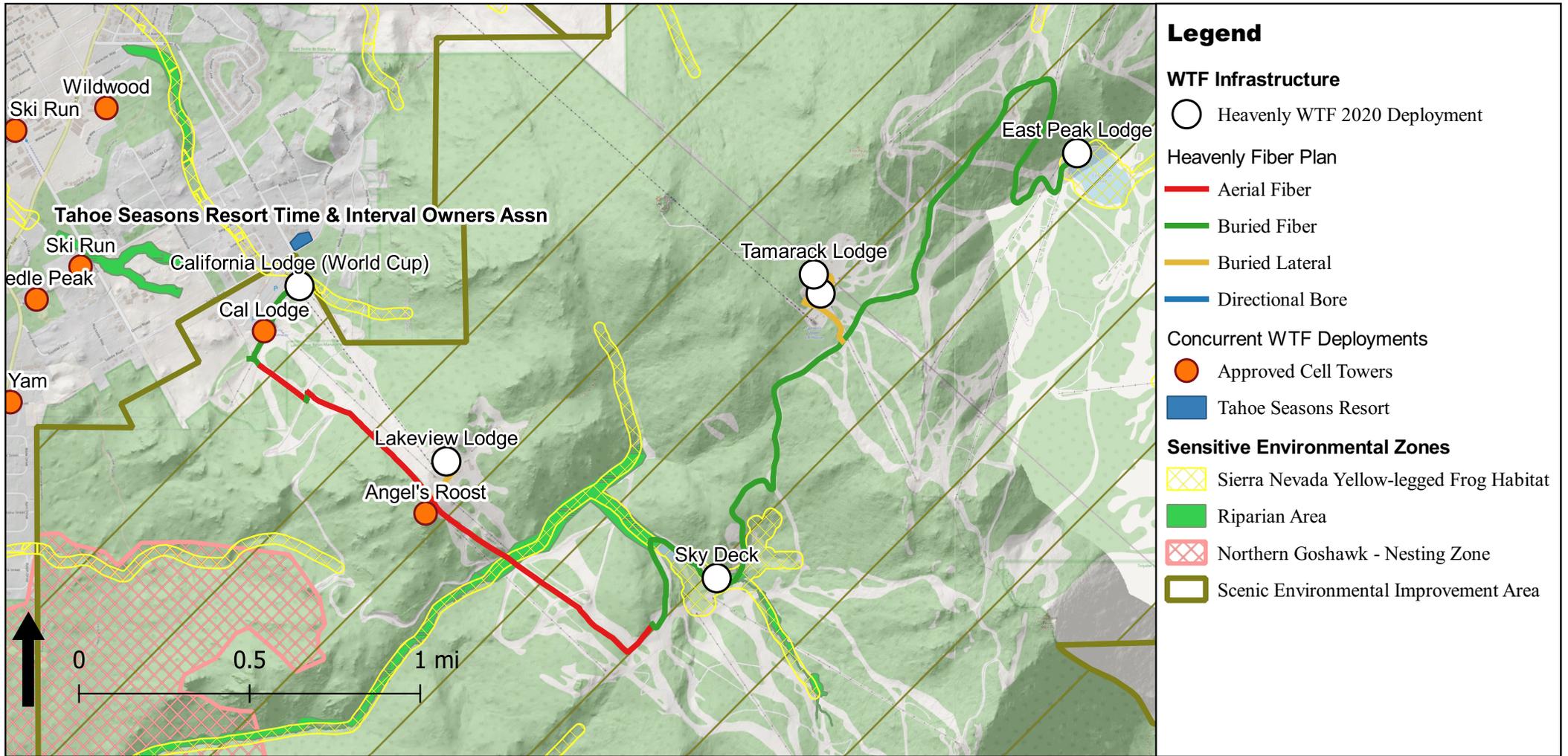
Thank you,



Heidi Hill Drum  
CEO, Tahoe Prosperity Center

Lie

# Verizon's Unfair and Deceptive Business Practice: Concurrent National Forest Deployment



## Unfair and Deceptive Business Practices

Verizon and the Tahoe Prosperity Center deceived City of South Lake Tahoe elected leaders, officials, staff, and residents into believing it was too burdensome to deploy towers in the adjacent National Forest lands, while simultaneously using existing special use permits to "fast-track" cell tower deployments and side-step non-discretionary environmental review of which they had feigned as onerous. This constitutes Unfair and Deceptive Business Practices.

(See Business & Professions Code §§ 17200 *et seq.*):

# In Real Time

## WHO DOES THAT?

Just when you thought we'd heard the last from former city manager Nancy Kerry, she has resurfaced with a \$50,000 liability claim against the city for comments posted on Facebook by Councilman Cody Bass that she claims have caused her "emotional distress."

She further proposed a public apology from the city and Councilman Bass in order to clear her sullied name. She set a deadline for the apology or an additional \$5,000 would be added to the original claim.

Bass considers his comments regarding her receiving \$300,000 after she was terminated, for what could possibly have been criminal behavior, to be valid. Will the Nancy Kerry soap-opera ever end?

## NEXT:

We have a planning commissioner (Diana Madson) who has misplaced her ethical guidebook. A series of emails and text messages reveal her efforts to influence the city council concerning the approval of a 12-story cell tower on Ski Run Blvd. She refers to residents who opposed the cell tower as "crazies" and their well-documented health and property-value-loss objections as

"junk science."

Heidi Hill Drum from the Prosperity Center and Jenna Palacio were party to this orchestrated campaign. Further, Ms. Madson provided scripted material for Cory Rich and Chris McNamara to read at the hearing.

Ms. Madson's removal from the planning commission should be certain and swift. Meanwhile, Jenna Palacio announced her departure from the planning commission citing work obligations related to the pandemic.

## AND:

Mayor Jason Collin has become the subject of a smear campaign organized by a group of self-appointed "business leaders" who believe that his statements to the media about the governor-imposed travel restrictions have destroyed our local economy.

It would be fair to acknowledge that the economic fallout from the pandemic is worldwide and not unique to Tahoe. These "leaders" have created a fake news blog to disparage the mayor along with a GoFundMe account that falsely claims to be raising money to "Help Move Mayor Jason Collin out of Tahoe." Jason told KCRA News that



## Keeping It Real

by  
Peggy  
Bourland

he does not plan to run for reelection.

## BETTER GOVERNMENT

At a time when we are witnessing pervasive civil unrest and demonstrations reminiscent of the turbulent 1960s, a level of dissatisfaction with previous and current local government decisions has been revived.

By promoting vacation rental businesses in neighborhoods, parking meters all over town and cell towers in residential areas, city government and city council members create distrust by their constituents. When the people stand up *en masse* to be heard, elected officials need to be paying attention. Another task force or paid consultant just delays the inevitable public outrage.

Proper management and good governance should be looking ahead

to avoid these kinds of controversies and the civil discord that often follows when informed/organized citizens rise up and demand better.

## FULL COURT PRESS

The citizen's vacation rental initiative (Measure T) passed in November of 2018. Designed to phase out VHRs in residential zones over three years, our "hoods" have already begun to feel more like real neighborhoods and less like motel districts. Michelle Benedict, Kathy Jo Liebhardt and others sought to overturn Measure T by filing a lawsuit against the city to challenge the measure, saying it was, among other things, unconstitutional.

When the lawsuit was first filed, "armchair attorney" Steve Teshara stated, "*Tahoe Chamber leaders have reviewed the initial legal complaint filed by the South Lake Tahoe Property Owners Group and we agree that Measure T is unconstitutional and unenforceable.*"

On June 1, 2020 the EDC Superior Court Judge Dylan Sullivan made a detailed and unambiguous tentative ruling that denied the plaintiff's claim that Measure T is unconstitutional.

The judge denied and eliminated

the following claims from the case: whether Measure T interferes with vested rights; whether it exceeds the initiative power; whether the occupancy limits are unconstitutional; whether the permanent resident exception is unconstitutional; and whether Measure T is vague and ambiguous.

City Attorneys Heather Stroud and Beverly Roxas competently defended the voter initiative and prevailed with one claim still to be decided.

The ruling by the court confirms that VHR owners do not have a vested right to convert residential housing to commercial uses. It is a zoning issue that has been upheld in courts throughout California. People deserve to live without the disruption of tourism invading residential neighborhoods.

Even the TRPA in their recent housing report now identifies Measure T as part of the solution to Tahoe's housing crisis.

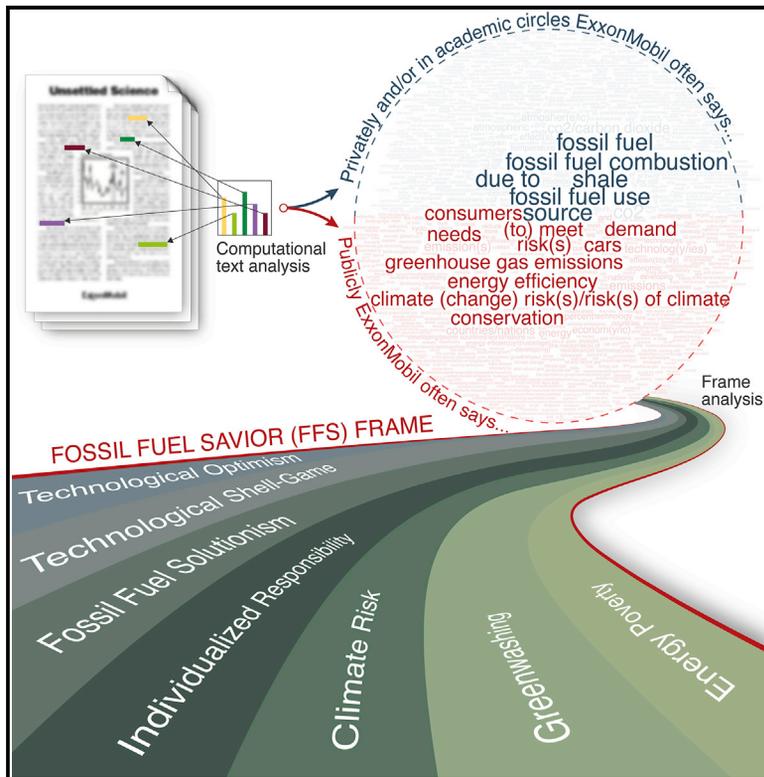
## REPEAT:

Wash your hands, wear a mask in public and support local businesses.

To be continued...

# Rhetoric and frame analysis of ExxonMobil's climate change communications

## Graphical abstract



## Authors

Geoffrey Supran, Naomi Oreskes

## Correspondence

gjsupran@fas.harvard.edu

## In brief

This is the first computational assessment of how ExxonMobil has used language to subtly yet systematically frame public discourse about climate change. We show that ExxonMobil uses rhetoric mimicking the tobacco industry to downplay the reality and seriousness of climate change, to present fossil fuel dominance as reasonable and inevitable, and to shift responsibility for climate change away from itself and onto consumers. Our work is relevant to lawsuits, policy proposals, and grassroots activism seeking to hold fossil fuel companies accountable for deceptive marketing.

## Highlights

- ExxonMobil's public climate change messaging mimics tobacco industry propaganda
- Rhetoric of climate “risk” downplays the reality and seriousness of climate change
- Rhetoric of consumer “demand” (versus fossil fuel supply) individualizes responsibility
- Fossil Fuel Savior frame uses “risk” and “demand” to justify fossil fuels, blame customers



## Article

# Rhetoric and frame analysis of ExxonMobil's climate change communications

Geoffrey Supran<sup>1,2,\*</sup> and Naomi Oreskes<sup>1</sup><sup>1</sup>Department of the History of Science, Harvard University, Cambridge, MA 02138, USA<sup>2</sup>Lead contact\*Correspondence: [gjsupran@fas.harvard.edu](mailto:gjsupran@fas.harvard.edu)<https://doi.org/10.1016/j.oneear.2021.04.014>

**SCIENCE FOR SOCIETY** A dominant public narrative about climate change is that “we are all to blame.” Another is that society must inevitably rely on fossil fuels for the foreseeable future. How did these become conventional wisdom? We show that one source of these arguments is fossil fuel industry propaganda. ExxonMobil advertisements worked to shift responsibility for global warming away from the fossil fuel industry and onto consumers. They also said that climate change was a “risk,” rather than a reality, that renewable energy is unreliable, and that the fossil fuel industry offered meaningful leadership on climate change. We show that much of this rhetoric is similar to that used by the tobacco industry. Our research suggests warning signs that the fossil fuel industry is using the subtle micro-politics of language to downplay its role in the climate crisis and to continue to undermine climate litigation, regulation, and activism.

## SUMMARY

This paper investigates how ExxonMobil uses rhetoric and framing to shape public discourse on climate change. We present an algorithmic corpus comparison and machine-learning topic model of 180 ExxonMobil climate change communications, including peer-reviewed publications, internal company documents, and advertorials in *The New York Times*. We also investigate advertorials using inductive frame analysis. We find that the company has publicly overemphasized some terms and topics while avoiding others. Most notably, they have used rhetoric of climate “risk” and consumer energy “demand” to construct a “Fossil Fuel Savior” (FFS) frame that downplays the reality and seriousness of climate change, normalizes fossil fuel lock-in, and individualizes responsibility. These patterns mimic the tobacco industry’s documented strategy of shifting responsibility away from corporations—which knowingly sold a deadly product while denying its harms—and onto consumers. This historical parallel foreshadows the fossil fuel industry’s use of demand-as-blame arguments to oppose litigation, regulation, and activism.

## INTRODUCTION

In previous work, we have shown that Exxon, Mobil, and ExxonMobil Corp misled the public about anthropogenic global warming (AGW) by contributing to climate science through academic and internal research, while promoting doubt about it in advertorials and other propaganda.<sup>1–3</sup> (We refer to Exxon Corporation as Exxon, Mobil Oil Corporation as Mobil, ExxonMobil Corporation as ExxonMobil Corp, and generically refer to all three as Exxon-Mobil.) We have also observed that, starting in the mid-2000s, ExxonMobil’s statements of explicit doubt about climate science and its implications (for example, that “there does not appear to be a consensus among scientists about the effect of fossil fuel use on climate”<sup>4</sup>) gave way to implicit acknowledgments couched in ambiguous statements about climate “risk” (such as discussion of lower-carbon fuels for “addressing the risks

posed by rising greenhouse gas emissions,”<sup>5</sup> without mention of AGW). This invites research as to how, beyond outright disinformation, ExxonMobil may have employed rhetoric and framing to construct misleading public narratives about AGW. Here, we take up this question.

“Framing” is a term of art in communications science that refers to how an issue is portrayed and understood.<sup>6–9</sup> Frames construct meaning by selecting “some aspects of a perceived reality” and making them “more salient in a communicating text, in such a way as to promote a particular problem definition, causal interpretation, moral evaluation, and/or treatment recommendation.”<sup>10</sup> (Here and throughout, we strictly refer to “emphasis frames” rather than “equivalency frames.”)<sup>11</sup> Analyzing which frames are present and absent in public discourse helps to reveal how actors have tried to shape policy debates by setting agendas and legitimating certain participants



and responses, while discouraging or precluding others.<sup>12–15</sup> Framing of responsibility, for example, can determine whether society calls upon individuals, industry, or government to take action.<sup>16</sup>

One of the fossil fuel industry's primary AGW frames has been scientific uncertainty.<sup>17</sup> Researchers have documented in detail industry's over-emphasis of uncertainty to deny climate science and delay action.<sup>1,2,17–25</sup> Subtler forms of rhetoric and framing, which dominate today's AGW discourse, are only just beginning to receive similar attention.<sup>7,26–29</sup> Fossil fuel interests have spent billions of dollars on AGW public affairs, yet their role in perpetuating these narratives is underexplored.<sup>30,31</sup>

In this paper, we analyze how ExxonMobil has publicly constructed AGW frames by selectively emphasizing some terms and topics while avoiding others. Our analysis compares the terms and topics between ExxonMobil's different AGW communications, including peer-reviewed publications, internal documents, and paid, editorial-style advertisements—known as advertorials—published on the Op-Ed page of *The New York Times* (NYT). We also identify frames in the latter. These well-defined, longitudinal corpora are conducive to a rigorous case study of fossil fuel industry messaging on AGW.

Our study offers the first computational assessment of how ExxonMobil has used language to frame public discourse about AGW. By bringing to bear the mixed-methods of computational linguistics and inductive frame analysis, our results add to (1) analyses of ExxonMobil's public affairs practices,<sup>32–44</sup> (2) qualitative accounts of the company's AGW communications,<sup>23,45–49</sup> and (3) the application of discourse and (algorithmic) content analysis to AGW communications by ExxonMobil and the wider climate countermovement.<sup>1,2,17–19,26,27,29,50–57</sup> A “distant”—that is, quantitative, statistical, and macroscopic—reading of ExxonMobil's AGW communications offers three practical advantages.<sup>58</sup> First, it complements the qualitative and/or manual methodologies previously applied to the AGW communications of ExxonMobil and other fossil fuel interests, and corroborates our prior work, which used manual coding to demonstrate systematic discrepancies between ExxonMobil's private and public AGW communications.<sup>1,2</sup> Second, automated methods of textual analysis allow detection of broad, sometimes subtle, patterns of language that would otherwise be unattainable. Third, by using existing corpora to establish the application of computational techniques to the analysis of AGW discourse, we help demonstrate the efficacy of these approaches, which researchers will be able to use to analyze the large numbers of documents that lawsuits against fossil fuel companies are anticipated to generate.

Our analysis is the first computational study illustrating how the fossil fuel industry has encouraged and embodied AGW narratives fixated on individual responsibility. Our findings corroborate the insights of qualitative discourse analyses about the role of fossil fuel interests, and add to what Kent<sup>59</sup> has called an “under-theorised” understanding “of why contemporary interest focuses on individual responsibility for climate change.”<sup>26,51</sup> In so doing, this work helps to decrypt the fossil fuel industry's playbook of climate delay framings, illuminating how sense-making schema conveyed by subtle yet systematic deployments of language may have “penetrated public

discourse to become naturalized as common sense or unfortunate realities.”<sup>13,26</sup> Although misleading frames that deceive the public may be defended on First Amendment grounds, the history of tobacco litigation shows that a misleading framework may also be held in some circumstances to be part of a pattern of fraudulent activities. Our work may, therefore, be relevant to ongoing lawsuits against ExxonMobil alleging “deceptive marketing” and “greenwashing,” as well as to calls for policymakers to ban fossil fuel industry advertisements or require that they come with tobacco-style warning labels.<sup>60–65</sup> Our research also adds to an expanding scholarly and journalistic AGW literature—spanning emissions accounting and extreme weather attribution,<sup>66,67</sup> supply-side policy analysis,<sup>68–70</sup> decarbonization theory,<sup>71,72</sup> the history of climate denial, lobbying, and propaganda by fossil fuel interests;<sup>73–83</sup> ethical philosophy;<sup>84,85</sup> and climate litigation<sup>86,87</sup>—challenging the zeitgeist of individualized responsibility. Finally, this study contributes to broader literatures on discourse and content analysis;<sup>88–91</sup> corporate issue management and advocacy marketing;<sup>56,92–96</sup> and the cross-pollination of corporate strategies of public affairs, litigation, and deceit.<sup>13,86,97–100</sup>

We adopt a mixed-method, computational approach to rhetorical frame analysis of 180 ExxonMobil documents previously compiled for manual content analysis<sup>1,2</sup>: 32 internal company documents (1977–2002; from ExxonMobil Corp,<sup>101</sup> *InsideClimate News*,<sup>102</sup> and Climate Investigations Center),<sup>103</sup> 72 peer-reviewed publications (1982–2014; from ExxonMobil Corp),<sup>104</sup> and 76 advertorials in the NYT expressing any positions on AGW (real and human caused, serious, or solvable) (1972–2009; from PolluterWatch and ProQuest).<sup>105,106</sup> To our knowledge, these constitute all publicly available internal and peer-reviewed ExxonMobil documents concerning AGW, including those made available by the company. They also include all discovered ExxonMobil advertorials in the NYT taking any positions on AGW. These corpora thus offer bound sets reflecting ExxonMobil's internal, academic, and public AGW communications, respectively.

Following text pre-processing and vectorization into document-term matrices, we first use frequency score (FS) and Dunning log-likelihood (LL) ratio corpus comparison algorithms to identify statistically distinctive keywords (“divergent terms”) that help locate rhetorical frames.<sup>107–110</sup> The FS indicates how often a given term appears in corpus A versus corpus B (accounting for corpus sizes), and ranges from 0 (only in corpus A) to 1 (only in corpus B). The LL ratio ( $G^2$ ) indicates the statistical significance of the relative frequencies of a given term between corpora A and B, and ranges from large and negative (term is disproportionately common in corpus A) to large and positive (disproportionately common in corpus B). Second, we complement this approach with latent Dirichlet allocation (LDA) topic modeling to identify statistically distinctive, thematically connected texts and vocabularies (“divergent topics”), which are commonly equated to either frames or frame elements.<sup>111–115</sup> Third, we integrate these quantitative tools into an inductive, qualitative approach to constructing frames as “frame packages” in advertorials.<sup>17,116–118</sup> In the **discussion**, we examine the congruence of our findings with the tobacco industry's rhetorical strategies in public relations and litigation.<sup>13,109,119,120</sup>

**Table 1. Rhetorical tropes and taboos: Highly divergent terms in (left) ExxonMobil Corp advertorials versus (right) Mobil advertorials, by LL ratio ( $G^2$ ) and FS**

	ExxonMobil Corp advertorials often say:				Mobil advertorials often say:				
	ExxonMobil Corp	Mobil	$G^2$	FS	ExxonMobil Corp	Mobil	$G^2$	FS	
*energy*	279	99	110.51	0.76	*nations*	4	79	-74.90	0.05
challenge(s)	52	4	54.33	0.94	plan	0	21	-26.84	0.00
(to) meet	51	14	26.70	0.80	senate	0	16	-20.45	0.00
demand	32	8	18.22	0.82	treaty	0	14	-17.89	0.00
use	60	27	16.78	0.71	in kyoto	0	13	-16.61	0.00
needs	27	9	11.53	0.77	the us [United States]	18	51	-12.99	0.28
*risk(s)*	46	3	50.30	0.94	*co2/carbon dioxide*	33	105	-31.90	0.26
climate (change) risk(s)/risk(s) of climate	26	0	39.02	1.00	emission(s)	97	197	-24.48	0.35
longterm	37	3	38.05	0.93	greenhouse gases	8	39	-18.96	0.19
*research*	75	21	38.53	0.80	effect	1	18	-16.67	0.06
gcep [Global Climate and Energy Project]	17	0	25.51	1.00	global warming	2	21	-16.25	0.10
technologies	55	18	24.00	0.77	evs [electric vehicles]	0	12	-15.34	0.00
solar	24	3	21.02	0.90					
stanford	14	0	21.01	1.00					
policies	27	5	19.17	0.86					
wind	18	3	13.62	0.87					

Terms that appear to be thematically related have been grouped (asterisked, high-scoring terms identify each group). ExxonMobil Corp advertorials often say terms (“tropes”) with large positive  $G^2$  scores and rarely say terms (“taboos”) with FS scores near 0. Mobil advertorials often say terms with large negative  $G^2$  scores and rarely say terms with FS scores near 1. p values < 0.001 for all  $G^2$  and FS scores.

## RESULTS

In the section entitled “[divergent terms and topics](#),” we compare divergent terms and topics between pairs of document categories. In “[rhetorical frames](#),” we summarize the findings of frame package analysis of advertorials: three dominant frames communicated by 11 constituent discourses. Other sections then focus on two of these complementary discourses, “[discourse of climate risk](#)” and “[discourse of individualized responsibility](#),” and analyze how they work alongside other discourses to construct one specific frame, Fossil Fuel Savior (FFS) (“[FFS frame](#)”).

### Divergent terms and topics

Table 1 presents a selection of highly divergent terms in ExxonMobil Corp advertorials versus Mobil advertorials, as identified by LL and FS. Likewise, Tables 2 and 3 compare highly divergent terms between all advertorials (Mobil plus ExxonMobil Corp) and, respectively, Exxon internal documents (Table 2) and Exxon/ExxonMobil Corp peer-reviewed publications (Table 3). In all three tables, the highest  $|G^2|$ -scoring terms, marked with asterisks, are suggestive of distinctive themes around which we group other relevant terms. These themes closely resemble the divergent topics shown in Table 4, which emerge from LL analysis of our LDA topic model solutions in all advertorials (top half of Table 4) and in combined internal and peer-reviewed documents (bottom half). The top 20 words associated with each topic are listed, together with assigned topic labels.

### Mobil versus ExxonMobil Corp advertorials

We have previously shown that both Mobil and ExxonMobil Corp advertorials often promoted doubt about climate science.<sup>1,2</sup> Terms conveying explicit doubt are therefore common to both corpora, and so do not appear in Table 1 (for examples, see S2.1, [supplemental information](#)). This undercuts ExxonMobil Corp’s suggestion that only Mobil, not ExxonMobil Corp, promoted doubt.<sup>2,3</sup> Both did. Moreover, when Exxon and Mobil merged in 1999, ExxonMobil Corp inherited legal and moral responsibility for both parent companies.

Comparison of advertorials over time can nevertheless be insightful in revealing other rhetorical trends. In this regard, Mobil and ExxonMobil Corp advertorial corpora serve as well-defined longitudinal proxies.

Table 1 shows, for example, that earlier, Mobil advertorials disproportionately contested climate science head-on, discussing emission(s) of CO<sub>2</sub>/carbon dioxide and the global warming effect (terms exhibiting statistically significant divergence are underlined throughout). Mobil advertorials also notably engaged in climate policy debates concerning the role of the US (and Senate) compared with other nations as part of the Kyoto treaty plan. By contrast, ExxonMobil Corp advertorials no longer referred to “global warming”: the term became taboo (FS = 0.10). Relative usage of “climate change” versus “global warming” went from 3-to-1 pre-merger to 34-to-1 post merger. Indeed, ExxonMobil Corp mostly sidestepped detailed discussions about climate science, acknowledging only the long-term risks of climate change before reframing it as a challenge to meet the public’s energy demand and needs. ExxonMobil

**Table 2. Rhetorical tropes and taboos: Highly divergent terms in (left) advertorials versus (right) internal documents, by LL ratio ( $G^2$ ) and FS**

	Advertorials often say:				Internal documents often say:				
	Advertorials	Internal	$G^2$	FS	Advertorials	Internal	$G^2$	FS	
*emission(s)*	294	97	293.80	0.86	*co2/carbon dioxide*	138	1,053	-291.63	0.21
risk(s)	49	7	72.48	0.93	atmosher(e/ic)	36	458	-187.01	0.14
greenhouse gas emissions	42	7	58.90	0.92	fossil fuel	9	144	-66.26	0.11
climate (change) risk(s)/risk(s) of climate	26	0	57.89	1.00	ppm [parts per million]	0	78	-62.12	0.00
climate change	124	103	45.39	0.71	co2 concentration	1	61	-40.57	0.03
dont [don't]	24	2	40.93	0.96	fossil fuel combustion	1	48	-30.69	0.04
know	32	8	37.59	0.89	co2 increase	0	28	-22.30	0.00
longterm	40	17	33.14	0.83	source	6	39	-9.08	0.24
doom(sday/sdayers)/apocalypse/hype/scare	11	0	24.49	1.00	*effect(s)*	27	359	-150.31	0.13
debate	26	12	20.05	0.82	temperature	15	270	-130.89	0.10
(un)know(/n/ing/ledge)	57	66	9.63	0.64	doubling	2	83	-51.60	0.05
*energy*	378	222	227.73	0.78	greenhouse effect	10	119	-46.69	0.15
(to) meet	65	2	128.34	0.99	ocean	15	135	-43.38	0.19
challenge(s)	56	5	94.08	0.96	due to	5	89	-42.94	0.10
energy efficiency	30	1	58.76	0.98	ph [pH]	0	44	-35.04	0.00
electricity	29	1	56.60	0.98	radiation	1	44	-27.68	0.04
consumers	21	0	46.76	1.00	co2 greenhouse	0	33	-26.28	0.00
oil and natural gas	18	0	40.08	1.00	sea	6	65	-23.99	0.16
energy use	23	4	31.75	0.92	global temperature	0	30	-23.89	0.00
demand	40	21	27.24	0.80	2050	0	30	-23.89	0.00
needs	36	22	20.69	0.77	temperature increase	3	50	-23.44	0.11
for generations/foreseeable future/several decades/decades to come/next 25 years	12	3	14.10	0.89	polar	1	28	-15.83	0.07
*countries/nations*	157	17	251.77	0.95	*program*	12	195	-90.37	0.11
developing/poorer countries/world/nations	53	3	97.01	0.97	natuna [Natuna Island, Indonesia]	0	67	-53.36	0.00
kyoto	59	7	92.31	0.95	doe [Department of Energy]	0	38	-30.26	0.00
targets	26	4	37.52	0.93	tanker	1	35	-20.96	0.06
*econom(y/ic)*	148	22	216.08	0.93	*model(s)*	30	309	-110.12	0.17
economic growth/impact	29	2	51.34	0.97	figure	0	112	-89.19	0.00
prosperity	15	0	33.40	1.00	rate	2	122	-81.13	0.03
jobs	13	0	28.95	1.00	data	10	98	-33.68	0.17
prices	12	0	26.72	1.00	vugraph	0	41	-32.65	0.00
cost	33	17	22.92	0.80	scenario	1	42	-26.17	0.05
tax	15	2	22.68	0.94					
living standard(s)/standard(s) of living/quality of life	10	0	22.27	1.00					
*steps*	36	1	71.76	0.99					
reduce emissions	23	0	51.21	1.00					
voluntary	18	0	40.08	1.00					
wise(r)/prudent/reasonable/responsible/sound(er)	39	21	25.87	0.79					
*technology(ies)*	198	40	257.20	0.91					
vehicles	33	0	73.48	1.00					

(Continued on next page)

**Table 2. Continued**

	Advertorials often say:				Internal documents often say:			
	Advertorials	Internal	G <sup>2</sup>	FS	Advertorials	Internal	G <sup>2</sup>	FS
natural gas	48	18	43.87	0.85				
trees	24	2	40.93	0.96				
invest(ing/ment(s))	27	4	39.46	0.93				
gcep [Global Climate and Energy Project]	17	0	37.85	1.00				
evs [electric vehicles]	16	0	35.63	1.00				
gasoline	20	2	32.72	0.95				
innovat(e/ion(s))	17	1	30.93	0.97				
solutions	26	7	29.36	0.88				
renewables	13	0	28.95	1.00				
wind	21	5	25.29	0.90				

Terms that appear to be thematically related have been grouped (asterisked, high-scoring terms identify each group). Advertorials often say terms (“tropes”) with large positive G<sup>2</sup> scores and rarely say terms (“taboos”) with FS scores near 0. Internal documents often say terms with large negative G<sup>2</sup> scores and rarely say terms with FS scores near 1. p values < 0.001 for all G<sup>2</sup> and FS scores.

Corp advertorials emphasized the need for more climate and energy technologies research, such as the company’s sponsorship of the GCEP (Global Climate and Energy Project) at Stanford University. Current solar and wind technologies were presented as inadequate.

#### Advertorials versus internal documents

Comparing divergent terms in all advertorials against those in internal documents, a combination of the above advertorial themes emerges (Tables 2 and 4). Numerous Mobil and Exxon-Mobil Corp advertorials promoted explicit doubt about whether AGW is real and human caused. They emphasized debate and focused on what scientists “do and don’t know” [Climate science uncertainty] (topic labels from Table 4 are indicated in bracketed italics throughout). This eventually gave way to rhetoric about potential long-term risks of AGW (after several years of overlap in ~2000–2005 and 2007), juxtaposed against the challenge to meet demand [Energy/emissions challenge]. The energy use and needs of consumers, such as electricity and oil and natural gas, are presented as necessitating greater energy efficiency and new technologies [Energy/emissions challenge; Vehicles]. The public is told about how ExxonMobil Corp is partnering with GCEP at Stanford to develop solutions such as more efficient gasoline vehicles and “clean...natural gas” [Vehicles; Energy technologies]. ExxonMobil Corp touts its efforts to plant trees, but renewables such as wind and electric vehicles/EVs are given short shrift [Conservation; Energy technologies]. Algorithmic analysis also documents Mobil’s public rhetoric on the Kyoto Protocol: targets that exempt developing countries threaten American jobs, prosperity, and economic growth; instead, governments and industry should pursue market-based, voluntary steps to reduce emissions [Climate policy].

Compared with Mobil advertorials, which promoted debate about climate science, and ExxonMobil Corp advertorials, which did the same or ignored it, Exxon’s internal conversations focused on it. Internal documents are notable for their detailed articulation of the causes and consequences of AGW. The source of the observed CO<sub>2</sub> increase in the atmosphere was

fossil fuel combustion [AGW science/projections]. Effects of the resulting greenhouse effect would include a global temperature increase. Internal discussions adopted a rigor absent from the company’s public communications, including reference to climate models, scenarios, and rates of change [Climate modeling]. One scenario they examined—the doubling of atmospheric CO<sub>2</sub> concentration by 2050—threatened melting of the polar icecaps, a decrease in ocean pH, and rising sea levels [AGW science/projections]. ExxonMobil advertorials disputed or remained silent about not just this early knowledge of climate science and its implications but also Exxon’s “CO<sub>2</sub> program” that helped acquire and apply that knowledge [AGW science/projections]. Internal memos report that this program included measuring CO<sub>2</sub> with a tanker, monitoring DOE (US Department of Energy) climate science, and evaluating the CO<sub>2</sub> emissions from their natural gas project in Natuna, Indonesia [Climate research programs].

#### Advertorials versus peer-reviewed publications

Table 3 compares divergent terms in all advertorials against those in peer-reviewed publications. Advertorials are distinguished by the same rhetorical themes as in “advertorials versus internal documents”; indeed, the contrast against academic articles is more pronounced. Independently and collectively, Mobil and ExxonMobil Corp advertorials offset the risks of manmade climate change by also promoting debate about complex science [Climate science uncertainty]. Advertorials are again seen to frame AGW as a challenge to meet the needs of consumers for more energy from fossil fuels, while seeking to allay concerns by publicizing the promise of advanced technology innovation (including cogeneration) [Energy/emissions challenge; Energy technologies]. In comparison with peer-reviewed papers, advertorials stand out for their emphasis of corporate environmental programs to reduce emissions through energy efficiency and conservation [Conservation].

While advertorials talk about the scientific process—research, science, and the extent of scientists’ knowledge are disproportionately discussed—peer-reviewed publications

**Table 3. Rhetorical tropes and taboos: Highly divergent terms in (left) advertorials versus (right) peer-reviewed documents, by LL ratio (G<sup>2</sup>) and FS**

	Advertorials often say:				Peer-reviewed documents often say:				
	Advertorials	Peer reviewed	G <sup>2</sup>	FS	Advertorials	Peer reviewed	G <sup>2</sup>	FS	
*energy*	378	1,777	500.41	0.82	et al	0	4,001	-372.50	0.00
(to) meet	65	98	191.64	0.93	model	5	3,000	-236.23	0.03
challenge(s)	56	100	151.75	0.92	figure	0	1,475	-137.32	0.00
needs	36	71	92.45	0.91	table	1	909	-75.18	0.02
more energy	21	12	87.65	0.97	rate	2	823	-60.90	0.05
consumers	21	33	60.70	0.93	estimates	5	978	-59.17	0.10
energy use	23	83	39.00	0.85	observed	1	715	-57.60	0.03
energy efficiency	30	152	36.65	0.81	scenario	1	562	-43.84	0.04
for generations/foreseeable future/several decades/decades to come/next 25 years	12	28	27.91	0.90	noise	0	311	-28.95	0.00
fossil fuels	24	149	22.89	0.77	projections	0	273	-25.42	0.00
gasoline	20	117	20.61	0.78	ipcc [Intergovernmental Panel on Climate Change]	4	505	-25.00	0.14
demand	40	422	14.35	0.67	error	1	317	-22.17	0.06
*research*	96	209	232.87	0.91	*co2*	69	5,161	-172.61	0.22
science	61	74	198.02	0.95	ocean	15	2,412	-134.77	0.12
scientists	39	25	157.74	0.97	transport	0	825	-76.81	0.00
dont [don't]	24	0	148.34	1.00	carbon cycle	0	462	-43.01	0.00
greenhouse gas emissions	42	60	126.97	0.94	ghg [greenhouse gas]	0	446	-41.52	0.00
carbon dioxide	69	227	126.15	0.86	ppm [parts per million]	0	397	-36.96	0.00
know	32	25	121.96	0.96	atmospheric co2	1	480	-36.52	0.04
climate (change) risk(s)/risk(s) of climate	26	10	119.09	0.98	ch4	0	272	-25.32	0.00
debate	26	30	86.15	0.95	gt [gigaton]	0	243	-22.62	0.00
manmade	15	2	80.58	0.99	*temperature*	15	1,836	-89.31	0.15
climate change	124	1,122	63.41	0.70	anthropogenic	0	609	-56.70	0.00
(un)know(/n/ing/ledge)	57	330	59.52	0.78	effect(s)	27	1,727	-48.70	0.25
risk(s)	49	261	56.56	0.80	due to	5	731	-39.08	0.13
longterm	40	282	31.82	0.75	radiative forcing	0	338	-31.47	0.00
gap(s)	11	39	18.93	0.86	climate sensitivity	0	219	-20.39	0.00
better science/understanding	6	10	16.85	0.93	temperature change	0	198	-18.43	0.00
complex	14	120	7.97	0.71	*mitigation*	4	880	-55.49	0.09
*technolog(y/ies)*	198	1,016	238.49	0.80	injection	0	443	-41.24	0.00
gcep [Global Climate and Energy Project]	17	1	97.44	1.00	ccs [carbon capture and storage]	0	374	-34.82	0.00
promise	20	12	82.39	0.97	dissolution	0	270	-25.14	0.00
evs [electric vehicles]	16	11	63.42	0.97	alkalinity	0	260	-24.21	0.00
trees	24	48	61.15	0.91	caco3	0	251	-23.37	0.00
cars	24	59	54.00	0.90	budget	0	180	-16.76	0.00
solutions	26	78	51.00	0.87	cement	1	237	-15.31	0.08
nuclear	26	82	49.12	0.87					
renewables	13	18	39.86	0.94					
wind	21	82	33.25	0.84					
cogeneration	12	26	29.19	0.91					
innovat(e/ion(s))	17	93	19.02	0.79					
invest(ing/ment(s))	27	243	13.96	0.70					

(Continued on next page)

**Table 3. Continued**

	Advertorials often say:				Peer-reviewed documents often say:			
	Advertorials	Peer reviewed	G <sup>2</sup>	FS	Advertorials	Peer reviewed	G <sup>2</sup>	FS
*steps*	36	36	126.05	0.95				
programs	28	14	120.90	0.98				
reduce emissions	23	25	78.03	0.95				
wise(r)/prudent/reasonable/ responsible/sound(er)	39	119	75.54	0.87				
environmental	56	384	46.45	0.75				
conservation	15	66	21.23	0.83				
*nations*	83	110	259.48	0.94				
kyoto	59	182	113.35	0.87				
governments	36	62	99.41	0.92				
senate	16	0	98.89	1.00				
developing/poorer countries/ world/nations	53	196	88.01	0.85				
*econom(y/ic)*	148	714	190.67	0.81				
prosperity	15	1	85.32	1.00				
economic growth/impact	29	74	63.68	0.89				
living standard(s)/standard(s) of living/quality of life	10	0	61.81	1.00				
voluntary	18	32	48.89	0.92				
jobs	13	11	48.27	0.96				

Terms that appear to be thematically related have been grouped (asterisked, high-scoring terms identify each group). Advertorials often say terms (“tropes”) with large positive G<sup>2</sup> scores and rarely say terms (“taboos”) with FS scores near 0. Peer-reviewed documents often say terms with large negative G<sup>2</sup> scores and rarely say terms with FS scores near 1. p values < 0.001 for all G<sup>2</sup> and FS scores.

actually engage in it. As expected, academic articles—even more so than internal documents—are distinguished by their articulation of AGW science. Observed atmospheric CO<sub>2</sub> concentrations are reported in ppm (parts per million), anthropogenic temperature change due to radiative forcing by GHG (greenhouse gases) such as CO<sub>2</sub> and CH<sub>4</sub> is acknowledged, and AGW model projections are run for different scenarios based on climate sensitivity [AGW science/projections]. The academic language of estimates and noise and references to the IPCC (Intergovernmental Panel on Climate Change) are commonplace [Climate modeling]. While advertorials offer unfocused representations of technologies such as renewables, nuclear, and EVs as variously promising, hypothetical, or insufficient, Exxon/ExxonMobil Corp supported peer-reviewed studies that squarely centered AGW mitigation around approaches consistent with continued reliance on fossil fuels: CCS (carbon capture and storage); and the injection of CO<sub>2</sub> into oceans through dissolution of minerals such as CaCO<sub>3</sub> to increase alkalinity [CO<sub>2</sub> disposal/storage; Carbon cycles]. As a recent literature review observed, the “use of enhanced ocean alkalinity for C storage was first proposed by [chief Exxon climate scientist Haroon] Kheshgi.”<sup>122</sup>

Like internal documents, peer-reviewed publications attribute GHG emissions and/or AGW to fossil fuels significantly more often than advertorials (p < 0.01–0.03). Common terms include fossil fuel emissions, fossil fuel CO<sub>2</sub>, and fossil fuel combustion [AGW science/projections] (see Table 5).

### Rhetorical frames

Frame package analysis leads us to identify three dominant frames in ExxonMobil’s advertorials, which we name (1) Scientific Uncertainty, (2) Socioeconomic Threat, and (3) Fossil Fuel Savior (FFS) (for details, see S4, supplemental information). The Scientific Uncertainty frame presents AGW as unproven and advocates additional climate science research. The Socioeconomic Threat frame argues that binding climate policies (such as the Kyoto Protocol) are alarmist and threaten prosperity, urging voluntary measures instead. The FFS frame describes AGW as the inevitable (and implicitly acceptable) risk of meeting consumer energy demand with fossil fuels for the foreseeable future, and presents technological innovation as the long-term solution.

These frames are constructed of reasoning and framing devices variously communicated by the 11 discourses listed in Figure 1. Figure 1 is a Venn diagram representing the chain of logic (i.e., reasoning devices) of each frame as defined by Entman:<sup>10</sup> problem, cause, moral evaluation, and solution (as indicated, these reasoning devices are the logical bases challenged by denials that AGW is real, human caused, serious, and solvable, respectively).<sup>10</sup> Discourses are manifest in one or more framing devices (e.g., lexical choices, catchphrases, depictions), and their positions in Figure 1 depict their contributions to the reasoning devices of each frame (definitions and examples of each frame’s reasoning and framing devices are provided in S4 and S5, supplemental information). For example, discourses of Technological

**Table 4. Topical tropes: Highly divergent topics in (top) advertorials versus (bottom) internal and peer-reviewed documents, by LL ratio ( $G^2$ ) of topics identified by LDA topic modeling**

Category	Topic labels	$G^2$	Top terms
Advertorials	energy/emissions challenge	10,271.93	*energy, *technolog(y/ies), *emission(s), *efficien(t/tly/cy), *world, *global, <u>fuel(s)</u> , *improv(e/es/ed/ing/ements), *develop(ing), *environment(/al/ally), *econom(y/ic), *need(s), *challenge(s), *percent, *demand, *risk(s), *gas, *reduce, *invest(ing/ment/ments), <u>future</u> , [*meet, *longterm]
	climate policy	6,045.82	*countries/nations, *kyoto, *emission(s), *econom(y/ic), *protocol, *targets, *gases, *agree(ment)/consensus, *industrialized, *administration, <u>reduction</u> , *participat(e/tion/ing), *senate, *plan, <u>measures</u> , *governments, *developed, *develop(ing), *public, *treaty [*jobs/*employment, <u>cost(/s/ly/liar/liest)</u> , *bind(ing), <u>lifestyle(s)</u> , *voluntary]
	vehicles	1,992.81	*vehicles, *evs/electric vehicles, <u>vehicle</u> , *gasoline, *cars, <u>diesel</u> , *citizenship, *math, <u>corporate</u> , *engine, *performance, *road, *engines, *social, car, *science, *education, <u>balancing</u> , dieselpowered, spills
	energy technologies	1,627.41	<u>nuclear</u> , *power, solar/photovoltaic(s), *oil, *renewable(s), <u>trillion</u> , <u>natural</u> , cell, brooklyn, reserves, <u>barrels</u> , turbine, *wind, generate, *gas, petroleum, fine, hydropower, inexhaustible, vote [ <u>offshore</u> , onshore, ethanol, biofuels]
	conservation	304.39	*tree(s), forest(s), *plant(/ing), *helped, buildings, lands, sequestration, star, *protect(/ion/ing), acres, eco(logical/system), enhance, conservancy, epas [EPA's], habitat, planted, threat, *conservation, agricultural, carefully [diversity, eagle, indigenous, preservation, restoring, wildlife]
	climate science uncertainty	201.47	<u>climate</u> , <u>change</u> , <u>research</u> , <u>scientific</u> , <u>science</u> , <u>human</u> , uncertain(ly/ties), ( <u>un</u> )*know(/n/ing/ledge), national, *scientists, <u>earths</u> , predict, *debate, underst(and/anding/ood), variability, weather, <u>impacts</u> , <u>consequences</u> , ability, <u>development</u> [ <u>program(s)</u> , *policy, compl(ex/exity/icated), *universit(y/ies)]
Internal and peer reviewed	AGW science/projections	-4,554.30	*co2/carbon dioxide, atmospher(e/ic), *effect(s), <u>fossil</u> , *temperature, fuel(s), *concentration, <u>increase</u> , *concentrations, carbon, *rate, global, *ocean, *ppm, <u>average</u> , level, *due, *oceans, combust(ion)/burn(ing), *biosphere [*scenarios, impact]
	climate modeling	-3,897.21	*model(s), <u>results</u> , <u>forc(e/ed/ing)</u> , climate, *data, *estimates, <u>response</u> , <u>variability</u> , *temperature, *shown, *flux, <u>anthropogenic</u> , <u>range</u> , *projections, emission(s), <u>detection</u> , <u>parameter</u> , *estimated, <u>studies</u> , <u>based</u>
	CO <sub>2</sub> disposal/storage	-2,668.42	*co2/carbon dioxide, *ph [pH], *figure, <u>time</u> , *seawater, *depth, <u>km</u> , *vertical, <u>retention</u> , *model(s), seafloor, <u>sparger</u> , <u>degassing</u> , diffusive, <u>natuna</u> , <u>release</u> , flow, *mixed, *surface, <u>fraction</u> [*injection]
	mitigation assessments	-1,917.80	*transport, <u>mitigation</u> , price, cost(/s/ly/liar/liest), <u>biomass</u> , <u>waste</u> , *al [et al.], infrastructure, china, <u>usa</u> , wastewater, reduction, potentially, forestry, losses, sector, availability, capture, <u>direct</u> , sectors
	climate research programs	-1,259.86	<u>dr</u> [Dr.], <u>program(s)</u> , <u>exxon</u> , <u>tanker</u> , <u>ere</u> [Exxon Research and Engineering Company], <u>phase</u> , federal, fund(/ed/ing), plan, division, <u>weinberg</u> [Harold Weinberg], additional, mass, academy, interface, underway, wines, organization, <u>shaw</u> [Henry Shaw], engineering [ <u>committee</u> , funds, scoping]
	carbon cycles	-1,215.66	*al [et al.], *ocean, <u>deep</u> , carbon, broecker [Wallace Broecker], upwelling, bbsr [Bermuda Biological Station for Research], <u>stocks</u> , <u>uptake</u> , <u>land</u> , <u>gt</u> [gigaton], vegetation, bermuda, landuse, <u>cycles</u> , jain [Atul Jain], station, transient, <u>biospheric</u> , <u>column</u> [dissolved, *water, <u>inventory</u> ]
	oil and gas production	-1,034.26	*ccs [carbon capture and storage], hs [HS], gas, acid, <u>cement</u> , n2 [N <sub>2</sub> ], processing, date, <u>natuna</u> [Natuna Island, Indonesia], park, project, earliest, eor [enhanced oil recovery], field, oil, mw [megawatt], recovery, describes, liquid, substantial [pipeline]

For each emergent topic, a topic label and its corresponding top 20 terms are listed (additional informative terms are in brackets at the end of each list). Top 20 terms are ordered according to the relevance metric proposed by Sievert and Shirley,<sup>121</sup> which accounts for both per-term ( $w$ )-per-topic ( $k$ ) probabilities ( $\varphi_{w,k}$ ) and the marginal probability of each term in the corpus ( $\rho_w$ ). We indicate divergent terms, as identified earlier by  $G^2$  and FS, between advertorials versus (italics) internal documents, (underlining) peer-reviewed publications, and (asterisks) internal and peer-reviewed documents.  $p$  values < 0.001 for all  $G^2$  and FS scores.

Shell Game, which, as Schneider et al.<sup>27</sup> define them, use “misdirection that relies on strategic ambiguity about the feasibility, costs, and successful implementation of technologies,” serve to downplay the need for public and political concern by trivializing the seriousness and solvability of AGW. Technological Shell

Game discourse is therefore placed in the overlapping areas of Moral evaluation (“Serious”) and Solutions (“Solvable”) in Figure 1.

The frame of Scientific Uncertainty—and its underlying taxonomy of explicit doubt about climate science and its

**Table 5. Rhetoric of individualized responsibility: Highly divergent terms in (top) advertorials and (bottom) internal and/or peer-reviewed documents, by LL ratio ( $G^2$ ) and FS**

	Advertorials	Internal	Peer reviewed	$G^2$ (Int./P.r.)	FS (Int./P.r.)	Example
Advertorials often say:						
(to) meet	65	2	98	128.34/191.64	0.99/0.93	" <u>To meet</u> this demand, while addressing the risks posed by rising greenhouse gas emissions, we'll need to call upon broad mix of energy sources." <sup>5</sup>
vehicles	33	0	240	73.48/25.02	1/0.74	"[T]he cars and trucks we drive aren't just <u>vehicles</u> , they're opportunities to solve the world's energy and environmental challenges." <sup>123</sup>
greenhouse gas emissions	42	7	60	58.9/126.97	0.92/0.94	"We're supporting research and technology efforts, curtailing our own <u>greenhouse gas emissions</u> and helping customers scale back their emissions of carbon dioxide." <sup>124</sup>
energy efficiency	30	1	152	58.76/36.65	0.98/0.81	"We have invested \$1.5 billion since 2004 in activities to increase <u>energy efficiency</u> and reduce greenhouse gas emissions. We are on track to improve energy efficiency in our worldwide refining and chemical operations." <sup>125,126</sup>
cars	24	0	59	53.44/54	1/0.9	"By enabling <u>cars</u> and trucks to travel farther on a gallon of fuel, drivers not only spend less money per mile, they also emit less carbon dioxide (CO <sub>2</sub> ) per mile." <sup>127</sup>
reduce emissions	23	0	25	51.21/78.03	1/0.95	"During the fact-finding period, governments should encourage and promote voluntary actions by industry and citizens that <u>reduce emissions</u> and use energy wisely. Governments can do much to raise public awareness of the importance of energy conservation." <sup>128</sup>
consumers	21	0	33	46.76/60.7	1/0.93	"We also are developing new vehicle technologies that can help <u>consumers</u> use energy more efficiently." <sup>125,126</sup>
world	91	64	338	43.45/150.55	0.74/0.85	"By 2030, experts predict that the <u>world</u> will require about 60 percent more energy than in 2000 .... As a result, greenhouse gas emissions are predicted to increase too." <sup>129</sup>
developing countries	27	3	162	43/26.94	0.95/0.78	Through 2030, " <u>developing countries</u> ... will rely on relatively carbon-intensive fuels like coal to meet their needs." <sup>5</sup>
transportation	23	2	121	38.87/26.93	0.96/0.8	"Ongoing advances in vehicle and fuel technology will be critical to meeting global demand for <u>transportation</u> fuels. They will also help address the risk posed by rising greenhouse-gas emissions." <sup>123</sup>
energy use	23	4	83	31.75/39	0.92/0.85	"Central to any future policy should be the understanding that man-made greenhouse gas emissions arise from essential <u>energy use</u> in the everyday activities of people, governments and businesses." <sup>130</sup>
people	30	11	61	27.87/75.73	0.85/0.91	"Thus, we're pleased to extend our support of ... American Forests ... whose 'Global Releaf 2000' program is mobilizing <u>people</u> around the world to plant and care for trees." <sup>131</sup>
demand	40	21	422	27.24/14.35	0.8/0.67	"[I]n the electric power sector, growing <u>demand</u> will boost CO <sub>2</sub> emissions." <sup>132</sup>

(Continued on next page)

Table 5. Continued

	Advertorials	Internal	Peer reviewed	G <sup>2</sup> (Int./P.r.)	FS (Int./P.r.)	Example
needs	36	22	71	20.69/92.45	0.77/0.91	"[F]ossil fuels must be relied upon to meet society's immediate and near-term <u>needs</u> ." <sup>133</sup>
conservation	15	5	66	14.89/21.23	0.86/0.83	"Prudent measures such as <u>conservation</u> and investment in energy-efficient technology make sense, but embarking on regulatory [climate/energy] policies that may prove wasteful or counterproductive does not." <sup>134</sup>
energy demand	15	14	59	4.38**/23.59	0.69**/0.84	"[I]ncreasing prosperity in the developing world [is] the main driver of greater <u>energy demand</u> (and consequently rising CO <sub>2</sub> emissions) over the coming decades." <sup>135</sup>
Internal and/or peer-reviewed documents often say:						
fossil fuel	9	144	359	-66.26/-4.48**	0.11/0.34***	"Release of this amount of CO <sub>2</sub> to the atmosphere raises concern with respect to its effect on the CO <sub>2</sub> greenhouse problem. Global <u>fossil fuel</u> emissions of CO <sub>2</sub> currently amount to about 1.8 × 10 <sup>10</sup> metric tons per year." <sup>136</sup> "Arrhenius put forth the idea that CO <sub>2</sub> from <u>fossil fuel</u> burning could ... warm the Earth. ... fossil fuel greenhouse warming ... fossil fuel greenhouse effect ..." <sup>137</sup>
natuna	0	67	NA	-53.36/NA	0/NA	"This would make <u>Natuna</u> the world's largest point source emitter of CO <sub>2</sub> and raises concern for the possible incremental impact of <u>Natuna</u> on the CO <sub>2</sub> greenhouse problem." <sup>136</sup>
due to	5	89	731	-42.94/-39.08	0.1/0.13	"The CO <sub>2</sub> concentration in the atmosphere has increased .... The most widely held theory is that: the increase is <u>due to</u> fossil fuel combustion." <sup>138</sup> "About three-quarters of the anthropogenic emissions of CO <sub>2</sub> to the atmosphere during the past 20 years is <u>due to</u> fossil fuel burning." <sup>139</sup>
fossil fuel combustion	1	48	NA	-30.69/NA	0.04/NA	"[T]here is the potential for our [climate] research to attract the attention of the popular news media because of the connection between Exxon's major business and the role of <u>fossil fuel combustion</u> in contributing to the increase of atmospheric CO <sub>2</sub> ." <sup>140</sup>
shale	1	41	NA	-25.43/NA	0.05/NA	"The quantity of CO <sub>2</sub> emitted by various fuels is shown in Table 1 .... They show the high CO <sub>2</sub> /energy ratio for coal and shale ... ["Shale oil"] is not predicted to be a major future energy source due to ... rather large amounts of CO <sub>2</sub> emitted per unit energy generated (see Table 1)." <sup>138</sup>
ccs	0	NA	374	NA/-34.82	NA/0	" <u>CCS</u> includes applying technologies that capture the CO <sub>2</sub> whether generated by combustion of carbon-based fuels or by the separation of CO <sub>2</sub> from natural gas with a high CO <sub>2</sub> concentration." <sup>141</sup>
source	6	39	322	-9.08*/-7.16**	0.24*/0.28**	"[F]ossil fuel combustion is the only readily identifiable <u>source</u> [of CO <sub>2</sub> ] which is (1) growing at the same rate, (2) large enough to account for the observed increases ..." <sup>142</sup>

(Continued on next page)

**Table 5. Continued**

	Advertorials	Internal	Peer reviewed	G <sup>2</sup> (Int./P.r.)	FS (Int./P.r.)	Example
fossil fuel use	0	13	NA	−10.35*/NA	0**/NA	Table 1 presents "coal combustion" and "natural gas combustion" as the "source[s]" of CO <sub>2</sub> , CH <sub>4</sub> , and SO <sub>2</sub> <sup>143</sup>
fossil fuel CO <sub>2</sub>	0	NA	64	NA/−5.96**	NA/0***	"[F]or scenarios with higher fossil fuel use (hence, higher carbon dioxide emissions ..." <sup>139</sup>
fossil fuel emissions	0	NA	54	NA/−5.03**	NA/0***	"This long tail on the fossil fuel CO <sub>2</sub> forcing of climate may well be more significant to the future glacial/interglacial timescale evolution of Earth's climate." <sup>144</sup> "We use our Integrated Science Model to ... estimate the time variation fossil fuel emissions of CO <sub>2</sub> ... required to match the [IPCC] concentration stabilization scenarios." <sup>145</sup>

Divergent terms in advertorials are identified by frame package analysis as framing devices of individualized responsibility discourse. Example quotations illustrate how advertorials use divergent terms to disproportionately present: (1) consumer demand for energy as the cause of—and culpable for—fossil fuel use, greenhouse gas emissions, and/or AGW; and (2) individual/demand-side actions as accountable for mitigating AGW. By contrast, divergent terms in internal and/or peer-reviewed documents often articulate the causality and culpability of fossil fuel combustion. p values < 0.001 for all G<sup>2</sup> and FS scores except: \* <0.005; \*\* <0.05; \*\*\* ≥0.05. NA, not available.

implications—has previously received detailed scrutiny and is here discussed further only in S4.1, [supplemental information](#).<sup>1,2,17–24</sup> By contrast, frames of Socioeconomic Threat and FFS—and the subtler discourses of delay that underpin them—are underexplored.<sup>17,26–28</sup> For further discussion of the Socioeconomic Threat frame, see S4.2, [supplemental information](#). In the remainder of this paper, we focus on the role of two specific, complementary discourses, Climate Risk and Individualized Responsibility, in constructing the FFS frame. As [Figure 1](#) suggests, these discourses serve as rhetorical gateways connecting the problem and cause of the FFS frame to its moral evaluation and solution.

### Discourse of climate risk

We have previously noted that, accompanying the emergence in the mid-2000s of implicit acknowledgments by some ExxonMobil Corp advertorials that AGW is real and human caused, there appeared to be a rhetorical framework focused on risk.<sup>2</sup> Algorithmic analyses here demonstrate that this was part of a wider trend in which, following the merger of Exxon and Mobil at the end of 1999, "risk" was incorporated into advertorials communicating explicit doubt. Specifically, LL and FS results in [Table 1](#) show that "risk(s)" is among the terms that most statistically distinguish Mobil advertorials from ExxonMobil Corp advertorials. Within all advertorials published prior to the merger and expressing any positions on AGW (as real and human caused, serious, or solvable), "risk(s)" appears three times, only once in reference to the risk(s) of AGW or greenhouse gases. By contrast, from 2000 onwards, such "risk(s)" are cited 46 times: an average of once per advertorial; 10 times higher than an average NYT article.<sup>146</sup> Permutations include "risk," "risks," "potential risks," "long-term risk," "long-term risks," "legitimate long-term risk," "legitimate long-term risks," and "potential long-term risks."

In 2000, for instance, ExxonMobil Corp's first post-merger advertorial in our corpus promoted "scientific uncertainty" that

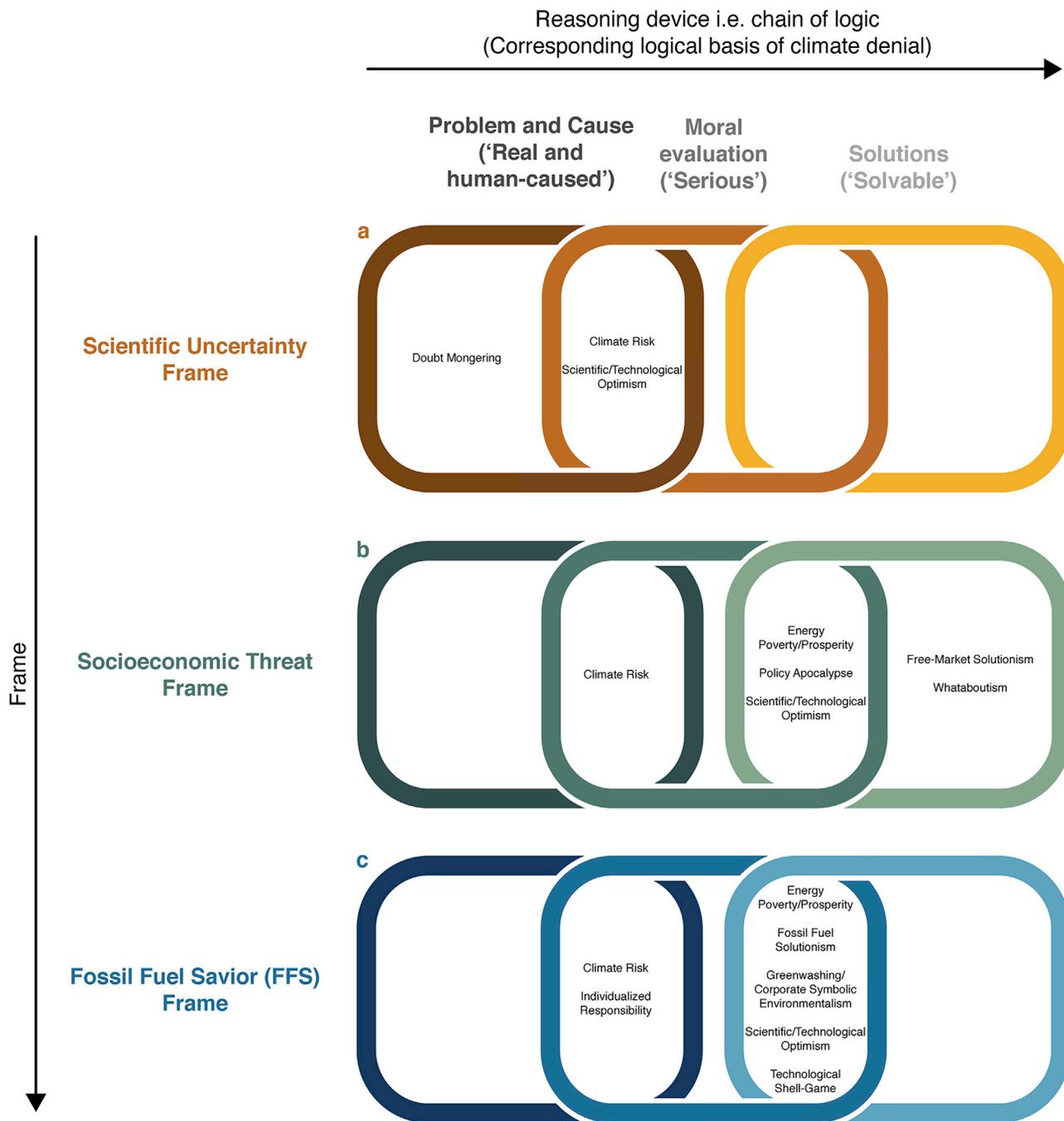
AGW is real, human caused, serious, and solvable, acknowledging only that it "may pose a legitimate long-term risk, and that more needs to be learned about it."<sup>147</sup> By the time the company took out its last advertorial expressing a position on AGW in 2009, its tune had changed but "risk" rhetoric remained. The advertorial was entitled, "Tackling climate risks with technology," followed by the subtitle, "Support for oil and natural gas innovation can reduce emissions."<sup>148</sup>

The function of "risk" rhetoric in moderating the conveyed status of AGW or greenhouse gases is unambiguous. First, "risks" is among the top terms characterizing the LDA-generated topic of *Energy/Emissions Challenge*, which is the primary topic that introduces readers to AGW (and compares it with energy demand; see "discourse of individualized responsibility") ([Table 4](#)). Second, "climate (change) risk(s)/risk(s) of climate" is, like "risk(s)" itself, a statistically distinctive term of ExxonMobil Corp advertorials versus Mobil advertorials, internal documents, and peer-reviewed publications ([Tables 1, 2, and 3](#)). Indeed, automated collocation analysis reveals that the highest scoring collocate of "climate change" and "global warming" in ExxonMobil Corp advertorials is "risk(s)." By contrast, in Mobil advertorials, it is "science" (followed by "gases" and "debate") ([Table S18](#)).

### Discourse of individualized responsibility

[Table 5](#) (top half) collates terms that are (1) identified by frame package analysis as framing devices communicating Individualized Responsibility in advertorials, and (2) highly divergent between all advertorials and internal and/or peer-reviewed documents according to LL and FS analyses. Two patterns emerge.

First, we observe that advertorials disproportionately employ terms that present consumer demand for energy (rather than corporate supply of oil, coal, and gas) as the cause of fossil fuel production, greenhouse gas emissions, and/or AGW. A characteristic example of this "(energy) demand" rhetoric is a 2008 ExxonMobil Corp advertorial stating: "By 2030, global



**Figure 1. Typology of discourses of climate denial and delay**

Using frame package analysis, we identify three dominant frames in ExxonMobil’s advertorials: (a, top) Scientific Uncertainty; (b, middle) Socioeconomic Threat; and (c, bottom) Fossil Fuel Savior (FFS). For each frame, a Venn diagram is presented corresponding to the reasoning devices (i.e., chains of logic) defined by Entman:<sup>10</sup> (left) problem and cause; (middle) moral evaluation; and (right) solution (as indicated, these reasoning devices are the logical bases challenged by denials that AGW is real, human caused, serious, and solvable, respectively). Each reasoning device is communicated by one or more of the 11 discourses of climate denial and delay listed within each chain of logic. Although not shown, these discourses are manifest in one or more framing devices (e.g., lexical choices, catchphrases, depictions), as identified in S4, [supplemental information](#). As an example, discourses of Technological Shell Game, which, as Schneider et al.<sup>27</sup> define them, use “misdirection that relies on strategic ambiguity about the feasibility, costs, and successful implementation of technologies,” serve to downplay the need for public and political concern by trivializing the seriousness and solvability of AGW. Technological Shell Game discourse is therefore placed in the overlapping areas of Moral evaluation (“Serious”) and Solutions (“Solvable”) in the diagram. For definitions and examples of all reasoning devices, framing devices, and discourses, see S4 and S5, [supplemental information](#).

energy demand will be about 30 percent higher than it is today ... oil and natural gas will be called upon to meet ... the world's energy requirements."<sup>149</sup> Another, in 2007, says that "increasing prosperity in the developing world [will be] the main driver of greater energy demand (and consequently rising CO<sub>2</sub> emissions)."<sup>135</sup> A 1999 Mobil advertorial is even blunter: "[G]rowing demand will boost CO<sub>2</sub> emissions."<sup>132</sup> In other words, they present growing energy demand as inevitable, and imply that it can only be met with fossil fuels.

Synonyms for "(energy) demand" include "needs" ("fossil fuels must be relied upon to meet society's immediate and near-term needs") and "energy use" ("man-made greenhouse gas emissions arise from essential energy use in the everyday activities of people, governments and businesses"). Fossil fuels are either presented as passively responding "to meet this demand" of consumers, developing countries, and the world; or they are left out of the equation entirely: "[A]s populations and economies have grown, energy use has increased, and so have greenhouse gas emissions."<sup>150</sup>

Second, we observe that, to the extent that advertorials admit the need for AGW mitigation, they disproportionately introduce terms conveying individual and/or demand-side actions as the appropriate response. Even while promoting explicit doubt about the reality of AGW, advertorials focus on downstream energy efficiency and greenhouse gas emissions, rather than upstream supply of fossil fuels, as the appropriate target of mitigation efforts. "During the [climate science] fact-finding period," a 1997 advertorial states, "governments should encourage and promote voluntary actions by industry and citizens that reduce emissions and use energy wisely. Governments can do much to raise public awareness of the importance of energy conservation."<sup>128</sup> Twelve years later, advertorials continued to equate the "global environmental challenge" with "curbing greenhouse gas emissions," but not with constraining fossil fuel supply.<sup>151</sup> As one 2000 advertorial put it: "Prudent measures such as conservation and investment in energy-efficient technology make sense, but embarking on regulatory [energy] policies that may prove wasteful or counter-productive does not."<sup>134</sup>

Advertorials repeatedly highlighted ways the public could, as one in 1998 put it, "show a little voluntary 'can do.'"<sup>152</sup> A 2008 advertorial suggested that the "cars and trucks we drive aren't just vehicles, they're opportunities to solve the world's energy and environmental challenges."<sup>123</sup> A 2007 advertorial offered readers "simple steps to consider": "Be smart about electricity use"; "Heat and cool your home efficiently"; "Improve your gas mileage"; "Check your home's greenhouse gas emissions" using an online calculator.<sup>153</sup> Mobil and ExxonMobil Corp presented themselves as facilitating, and participating in, such demand-side AGW mitigation. A 1997 advertorial laid the groundwork: "We're supporting research and technology efforts, curtailing our own greenhouse gas emissions and helping customers scale back their emissions of carbon dioxide."<sup>124</sup> In 1999, Mobil announced that "we're pleased to extend our support of ... American Forests ... whose 'Global Releaf 2000' program is mobilizing people around the world to plant and care for trees."<sup>131</sup> This narrative was echoed by advertorials a decade later: "By enabling cars and trucks to travel farther on a gallon of fuel, drivers...emit less carbon dioxide (CO<sub>2</sub>) per mile," said

a 2008 advertorial.<sup>127</sup> "We also are developing new vehicle technologies that can help consumers use energy more efficiently," said two more the following year.<sup>125,126</sup>

By contrast, Exxon and ExxonMobil Corp's internal and/or academic communications recognized AGW and/or greenhouse gases as also an upstream problem caused by fossil fuel supply and burning (see also S2.2, supplemental information). "[Fossil fuel combustion is the only readily identifiable source [of CO<sub>2</sub> consistent with the rate and scale of] observed increases..." observed Exxon scientist James Black<sup>142</sup> in a 1978 presentation to the Exxon Corporation Management Committee. Other internal (1979) and peer-reviewed (2001) documents likewise attributed CO<sub>2</sub> accumulation in the atmosphere as "due to fossil fuel burning" and "fossil fuel combustion."<sup>138,139</sup> A 1984 internal report and a 1994 academic article spoke of "fossil fuel emissions of CO<sub>2</sub>," while a 1998 paper referred to "fossil fuel CO<sub>2</sub> forcing of climate."<sup>136,144,145</sup> A 1982 internal memo went further, acknowledging "the connection between Exxon's major business and the role of fossil fuel combustion in contributing to the increase of atmospheric CO<sub>2</sub>."<sup>140</sup> The 1979 and 1984 internal documents discuss the CO<sub>2</sub> emissions of specific fossil fuel sources such as shale oil and Exxon's natural gas reservoir off Natuna Island in Indonesia.<sup>136,138</sup>

In sum, ExxonMobil's advertorials statistically overuse terms that reduce AGW to a downstream problem caused by consumer energy demand, to be solved primarily by energy efficiency to reduce greenhouse gas emissions. In contrast, their private and academic documents disproportionately recognize that AGW is an upstream problem caused by fossil fuel supply.

As we show in S6.2, supplemental information, this statistical dichotomy extends throughout all of ExxonMobil Corp's flagship reports concerning AGW spanning 2002–2019 compared with the firm's internal and academic publications.

### FFS frame

In addition to Climate Risk and Individualized Responsibility, the FFS frame comprises the five other discourses shown in Figure 1 and defined in S5, supplemental information. Together, they establish the frame's chain of logic (i.e., reasoning devices, see Table S4).

First, as shown in the previous two sections, discourses of Climate Risk and Individualized Responsibility present AGW as the inevitable "risk" of meeting consumer energy demand.

In response to this problem definition and causal attribution, discourses of Scientific/Technological Optimism (which gives primacy to scientific or technological breakthroughs as the solutions to AGW) and Greenwashing/Corporate Symbolic Environmentalism (which is when companies make changes for environmental reasons that, in the case of greenwashing, are merely and deliberately symbolic) lend what Plec and Pettenger<sup>52</sup> (2012) call "an aura of scientific and technical authority," which "resigns us to putting our faith in the power of industry, technology, and science" (see also Schneider et al.<sup>26</sup>). "[W]e believe that technology provides the key avenue to solutions that manage long-term risk and preserve prosperity," says the voice of reason presented by a 2002 advertorial entitled "A responsible path forward on climate." "[This] will almost certainly require decades."<sup>154</sup> ExxonMobil asserts its leadership in this challenge with advertorials citing "our industry-leading investments in research and

development,”<sup>149</sup> such as “supporting climate-related research efforts at major universities, including Stanford and MIT.”<sup>155</sup> Visual images such as graphs, charts, and science iconography reinforce this impression.

This technocratic authority helps legitimize accompanying discourses of Fossil Fuel Solutionism and Technological Shell Game, which join the dots between energy demand and continued reliance on fossil fuels. An example of Fossil Fuel Solutionism (which presents fossil fuels and their industry as an essential and inevitable part of the solution to AGW) is a 2007 advertorial that unequivocally depicts the future: “Coal, oil, and natural gas will remain indispensable to meeting total projected energy demand growth” through 2030.<sup>156</sup> “Oil and gas will be essential to meeting demand,” reiterates another in 2008.<sup>5</sup> “Meeting this growing long-term demand requires that we develop all economic sources of energy – oil, natural gas, coal, nuclear and alternatives,” says a third in 2009.<sup>151</sup>

The non-fossil fuel alternatives are then dismissed by Technological Shell Game discourse promoting doubt and confusion about AGW’s technological solvability, such as three advertorials in 2005 depicting, again unequivocally, how “Wind and solar ... meet about 1% of total world demand by 2030.”<sup>157–159</sup> Another, 3 years later, updates the figure to “only 2 percent” (including bio-fuels).<sup>5</sup> ExxonMobil also takes aim at clean energy subsidies and renewable energy’s “highly variable output” and “enormous land-use requirements.”<sup>133,154,160</sup> Meanwhile, the three 2005 advertorials, and another in 2009, falsely promote natural gas as “clean-burning” and “clean,” respectively.<sup>157–159</sup>

In a 2009 advertorial, ExxonMobil acknowledges that there is “a dual challenge” to “provide energy” and “protect the environment” (notably, they say that this challenge concerns energy rather than fossil fuels, and that it applies to “all of us”).<sup>150</sup> But then they tip the scales by pitting concrete, unequivocal benefits (“[Energy] lights our homes. Fuels our transportation. Powers our industries. ... [D]riv[es] our economy and rais[es] living standards”) against amorphous, uncertain costs (the “risks of climate change”). Two 2007 advertorials similarly compare “economic growth and human development” against undefined “risks of climate change.”<sup>161,162</sup>

In cases such as these, discourses of Energy Poverty/Prosperity and Policy Apocalypse (which respectively articulate social justices of energy access and alleged socioeconomic tolls of decarbonization—the latter strictly assigned to the socioeconomic threat frame), contrasted against that of Climate Risk, work to affirm the moral evaluation of the FFS frame that fossil fuel lock-in is righteous and reasonable.

## DISCUSSION

The patterns observed in “results” are similar to those documented in the tobacco industry. In “risk rhetoric facilitates ExxonMobil’s have-it-both-ways position on AGW” and “energy demand rhetoric individualizes AGW responsibility,” we discuss the strategic functions of AGW “risk” rhetoric and individualized responsibility framings, respectively, in comparison with the history of the tobacco industry. “Energy demand rhetoric individualizes AGW responsibility” distinguishes how consumer energy demand is presented in public (“demand as fossil fuel lock-in in public relations”) versus in legal defense (“demand as blame

in litigation”). “Historical contexts, ramifications, and trajectories of ExxonMobil’s communication tactics” explores the historical contexts, ramifications, and trajectories of ExxonMobil’s “risk” rhetoric (“risk”) and individualized responsibility framings (“individualized responsibility”).

### Risk rhetoric facilitates ExxonMobil’s have-it-both-ways position on AGW

Our identification of ExxonMobil’s discursive shift to “risk” rhetoric (see “discourse of climate risk”) is broadly consistent with independent findings. Jaworska<sup>51</sup> observes the emergence of “risk” as one of the most frequent collocations of “climate change” in the late 2000s within the corporate social responsibility reports of the world’s major oil corporations, including ExxonMobil. Grantham and Vieira,<sup>44</sup> examining “welcome letters” from ExxonMobil’s CEO in the company’s Corporate Citizenship Reports, note that “risk” is one of the most influential words coinciding with emphasis on the “planet.” Schlichting<sup>17</sup> concludes that, over the course of the 2000s, industry actors increasingly adopted the framing that “climate change [might be/is] a risk.”

ExxonMobil’s rhetorical pattern of stressing “risk” is consistent with the company’s effort in the mid-2000s, chronicled by journalist Steve Coll,<sup>48</sup> “to reposition ExxonMobil’s arguments about warming to more fully account for consensus scientific opinion, without admitting that any of the corporation’s previous positions had been mistaken, for that might open a door to lawsuits.”

This approach resembles the tobacco industry’s well-documented response to the scientific consensus on the harms of tobacco use, described by historian Allen Brandt<sup>163</sup> as a “shift” in focus from scientific “uncertainty” to “(alleged) risks” of smoking (see also Proctor<sup>164,165</sup>). This scientific hedging strategy was made explicit in a 1996 Reynolds training manual instructing new employees to tell reporters that smoking was “a risk factor” but “not a proven cause.”<sup>165</sup> In 1998, for example, Philip Morris’s CEO Geoffrey Bible conceded a “possible risk” but not a “proven cause,” the distinction being in what historian Robert Proctor<sup>165</sup> calls “a kind of legal having-it-both-ways: an admission strong enough to ward off accusations of having failed to warn, yet weak enough to exculpate from charges of having marketed a deadly product.” This carefully parsed conclusion became the industry’s new official position.<sup>163</sup>

“Risk” facilitates ExxonMobil’s have-it-both-ways position on AGW. It is a “‘good’ candidate to serve various rhetorical purposes,” Jaworska<sup>51</sup> notes, because it “opens up many semantic slots.” Fillmore and Atkins<sup>166</sup> work on the conceptual meaning of risk, for example, shows that “risk” has two dominant sub-frames, “Chance” and “Harm,” and many optional valence description categories. “Chance” is defined as “uncertainty about the future,” such that risk rhetoric (1) implies inherent uncertainty and (2) is subject to temporal discounting heuristics.<sup>167–169</sup> “The essence of risk is not that it is happening, but that it *might* be happening.”<sup>170,171</sup>

“Risk” is never clearly or consistently defined by ExxonMobil. The presence and absence of risk’s various sub-frames introduce so-called strategic ambiguity—and therefore flexibility—in contemporaneous and retrospective interpretations of what ExxonMobil wants us to see as a “risk” rather than a “reality.”<sup>27,172</sup> For instance, does the “Chance” sub-frame of “risk”—and

therefore the implication of uncertainty—apply to whether AGW is happening, human caused, serious, or solvable? Sub-frames of Harm, Actor, Victim, and Valued Object are also rarely articulated: who assumes the risk(s) of AGW: the public, the company, its shareholders, or others? What might be the consequences, and when? In contrast, the “Gain,” “Beneficiary,” and “Motivation” sub-frames of risk taking, manifest in discourse of Policy Apocalypse, are stated explicitly, as discussed in [“demand as fossil fuel lock-in in public relations.”](#)

Like its weaponized rhetorical cousins—such as “uncertainty,” “sound science,” and “more research” and the hedging words “may,” “potential,” etc.—“risk” has the strategic advantage of not necessarily implying intent to deny or delay, because it is coopted from common academic, regulatory, journalistic, and colloquial parlance (S1.4.2, Supran and Oreskes<sup>1</sup>).<sup>15,146,167,173,174</sup> It can be used correctly (for example, to refer to expected *future* damages and stranded fossil fuel assets—a risk that we have previously shown ExxonMobil was publicly silent about) or incorrectly (for example, to describe AGW and past/present climatic changes such as sea level rise as risks rather than realities).<sup>1</sup>

ExxonMobil employs almost identical “risk” language in advertorials promoting explicit doubt about AGW as in those that implicitly acknowledge it. For example, they refer to “the risk of global warming” in 1989 (accompanied by explicit doubt); the “risk(s)” “that climate changes may pose” in 2000 (alongside explicit doubt); and “the risks of climate change” in 2009 (which, in the absence of doubt, is coded as an implicit acknowledgment).<sup>150,175,176</sup> This is not limited to advertorials (for wide-ranging examples, see table 3 of Supran and Oreskes<sup>2</sup>). In ExxonMobil Corp’s 2005 *Corporate Citizenship Report*, for instance, which extensively questions whether AGW is human caused and serious, a member of the public asks: “Why won’t ExxonMobil recognize that climate change is *real* ...?” The company replies: “ExxonMobil recognizes the *risk* of climate change and its *potential* impact” (emphases added).<sup>177</sup> By shifting the conversation from the semantics of reality to the semantics of risk, they inject uncertainty into the AGW narrative, even while superficially appearing not to.

### Energy demand rhetoric individualizes AGW responsibility

Two dimensions of issue responsibility are commonly identified in communications and psychological research: causality and treatment.<sup>16,178</sup> Causality responsibility addresses the source of a problem—who or what causes it. Treatment responsibility identifies who or what has the power to alleviate the problem, and should be held responsible for doing so. Studies of responsibility framing and attribution theory argue that attribution of these responsibilities broadly takes two conflicting forms: individual versus social.<sup>16,179,180</sup> Expressing our findings in [“discourse of individualized responsibility”](#) through this analytical lens, ExxonMobil’s public advertorials are biased toward individualist framings of both causality and treatment responsibilities for AGW as compared with their private and academic representations.

Jaworska<sup>51</sup> has observed similar appeals to energy demand as the driving force behind greenhouse gas emissions in the corporate citizenship reports of ExxonMobil Corp and other fos-

sil fuel companies, noting that they are “an example of differentiation, which shifts the responsibility to other constituencies.” Princen et al.<sup>72</sup> similarly argue that a focus on carbon and greenhouse gases—and away from fossil fuels—is reductionist. “This chemical framing,” they note, “implies that the problem arises after a chemical transformation, after fuels are burned. It effectively absolves of responsibility all those who organize to extract, process, and distribute...So constructed...the burden of harm and responsibility for amelioration falls on governments and consumers rather than extractors.”

“The most effective propaganda,” Parenti<sup>181</sup> contends, “is that which relies on framing rather than on falsehood.” As with the language of risk, a rhetorical power of narratives that individualize responsibility is that they do not require the statement of outright falsehoods. After all, consumer demand is one valid and universally recognized aspect of the AGW problem and its solution, and not all advertorials entirely disregard the role of fossil fuels. On balance, however, the disproportionate public fixation of ExxonMobil, a supplier company, on demand-side causation and accountability (as shown in [“discourse of individualized responsibility”](#)) fulfills the fundamental function of emphasis frames to “call attention to some aspects of reality while obscuring other elements.”<sup>10</sup> It is in this selection process that the individualized responsibility framing device creates a false dichotomy, leading readers toward AGW problem definitions, evaluations, and solutions skewed toward consumer demand and away from industry supply.<sup>11,16,178</sup>

ExxonMobil’s framing is reminiscent of the tobacco industry’s effort “to diminish its own responsibility (and culpability) by casting itself as a kind of neutral innocent, buffeted by the forces of consumer demand.”<sup>165</sup> It is widely recognized that the tobacco industry used, and continues to use, narrative frames of personal responsibility—often marketed as “freedom of choice”—to combat public criticism, influence policy debates, and defend against litigation and regulation.<sup>13,100,119,164,182–184</sup> Friedman et al.<sup>13</sup> recently demonstrated that tobacco companies use “freedom of choice” to imply two distinct concepts: liberty and blame. In their public relations messaging, industry asserts smokers’ rights as individuals who are at liberty to smoke. In the context of litigation, industry asserts that those who choose to smoke are solely to blame for their injuries.

In the following two subsections, we further explore the congruence between ExxonMobil’s public responsibility framing and these tobacco tactics ([“demand as fossil fuel lock-in in public relations”](#); [“demand as fossil fuel lock-in in public relations”](#)). We discuss how this Individualized Responsibility discourse is rationalized and reinforced by the semantic duality of “risk.”

### [Demand as fossil fuel lock-in in public relations](#)

In [“FFS frame,”](#) we showed that ExxonMobil’s FFS frame insists—typically as self-fulfilling fact rather than opinion—upon society’s inevitable and indefinite reliance on fossil fuels. Rather than asserting that demand is a personal choice and liberty, ExxonMobil’s public “(energy) demand” rhetoric inverts the tobacco industry’s “freedom of choice” messaging. Liberty becomes lock-in.

Within this frame, discourses of Energy Poverty/Prosperity and Policy Apocalypse contrast against that of Climate Risk ([“FFS frame”](#)). The role of “risk” rhetoric here is to downplay the downside, namely AGW, of this alleged dichotomy: fossil

fuels are essential, whereas the potential effects—indeed realities—of AGW are uncertain.<sup>26</sup> Such assertions, St. John III<sup>35</sup> notes, extend Mobil's messaging in its "Observations" columns "about what constitutes reasonable risk." Observations were "pithy, easy-to-read" advertorials that Mobil ran in Sunday newspaper supplements between 1975 and 1980.<sup>35,185</sup> In a 1980 "Observations" column, for example, Mobil lamented that "the country seems to be afflicted with the Chicken Little Syndrome" of "cry[ing] that 'The sky is falling!'"<sup>186</sup> "Hardly a day passes," they said, without "fresh perils" like "harmful rain" or "cancerous sunshine." But a "risk-free society" through government regulation is impossible, the advertorial reasoned, because "everything people do everyday involves a slight measure of risk" (emphasis in original). The company concluded with the warning that to "avoid risk, fight change" may be a short-term solution, "but for the long pull, it's a way to certain stagnation." Tobacco industry apologists made the same arguments, calling it "the menace of daily life."<sup>187</sup>

To the extent that advertorials concede AGW may be a problem, the "risk" angle helps frame AGW as unpredictable, positioning the oil industry "not as a contributor but as a victim" alongside consumers.<sup>51</sup> As a 2009 advertorial put it, "[we'll need] a global approach to managing the risks of climate change. Everyone has a role to play – industry, governments, individuals."<sup>150</sup> This complemented Mobil's broader use of advertorials to rhetorically reframe itself as what Kerr<sup>42</sup> terms a "corporate citizen." "A citizen of many lands" is how Mobil described itself in a 1999 advertorial.<sup>131</sup> "Climate change: we're all in this together," another was titled in 1996.<sup>188</sup> With this narrative of an "empathetic fellow traveler," St. John III<sup>35</sup> argues, "Mobil offers up the reasonable, risk-taking corporate persona who is willing to take the initiative to provide a beneficial product to all Americans...[B]y appealing to Americans' penchant for valorizing the self-starting individual, such a message of energy harvesting as never being 100% safe could well explain how a significant amount of Americans today do not see fossil fuel-induced climate change as a significant risk."<sup>35</sup>

ExxonMobil's advertorials say almost nothing about the seriousness of AGW.<sup>1,2</sup> Nor do they mention the concepts of carbon budgets and stranded fossil fuel assets, which are part of the argument for the fundamental incompatibility of unrestricted fossil fuel supply with climate mitigation.

Overall, the didactic framing of demand as fossil fuel lock-in communicates what Plec and Pettenger<sup>52</sup> describe as "a rhetoric of resignation, naturalizing consumption of resources and teaching us to put our trust in industry solutions to energy problems." Or as Schneider et al.<sup>27</sup> and Cahill<sup>26</sup> put it, quoting the neoliberal bromide: "There is no alternative" to the *status quo*.

### **Demand as blame in litigation**

Although the tobacco industry sells "freedom of choice" as *liberty* in public relations, in litigation they equate it with *blame* toward individuals who exercised their choice to smoke.<sup>13,164,183,184</sup> Climate litigation is nascent, yet the fossil fuel industry has already successfully repackaged demand as *lock-in* to instead impute *blame* on customers for being individually responsible.

In 2018, arguing in defense of five oil companies (including ExxonMobil Corp) against a lawsuit brought by California cities seeking climate damages, Chevron lawyer Theodore Broutrous

Jr. offered his interpretation of the IPCC's latest report: "I think the IPCC does not say it's the production and extraction of oil that is driving these emissions. It's the energy use. It's economic activity that creates demand for energy." "It's the way people are living their lives."<sup>189</sup> The judge's dismissal of the case accepted this framing: "[W]ould it really be fair to now ignore our own responsibility in the use of fossil fuels and place the blame for global warming on those who supplied what we demanded?"<sup>190</sup>

Even if plaintiffs prove their case, fossil fuel companies can invoke "affirmative defenses"—as tobacco companies often have—such as "common knowledge" and "assumption of the risk."<sup>164,183</sup> These respectively argue (1) "that the plaintiff had engaged in an activity [such as smoking] that involved obvious or widely known risks," and (2) "that the plaintiff knew about and voluntarily undertook the risk."<sup>13</sup> As Brandt<sup>163</sup> explains it, "If there was a risk, even though 'unproven,' it nonetheless must be the smoker's risk, since the smoker had been fully informed of the 'controversy.' The industry had secured the best of both worlds."

By way of the FFS frame, ExxonMobil appears to have constructed an ability to do the same. On the one hand, "risk" rhetoric is weak enough to allow the company to maintain a position on climate science that is ambiguous, flexible, and unalarming ("risk rhetoric facilitates ExxonMobil's have-it-both-ways position on AGW"). On the other, it is strong enough—and prominent enough, in NYT advertorials and elsewhere—that ExxonMobil may claim that the public has been well informed about AGW. This duality has been a cornerstone of the tobacco industry's legal position on the "risks" of smoking: "Everyone knew but no one had proof."<sup>163,164</sup> Akin to early, tepidly worded warning labels on cigarette packages, ExxonMobil's advertorials in America's newspaper of record help establish this claim, sometimes explicitly: "*Most people acknowledge* that human-induced climate change is a long-term *risk*," a 2001 advertorial states<sup>13,130</sup> (emphases added). "The *risk* of climate change and its *potential* impacts on society and the ecosystem are *widely recognized*," says another the following year.<sup>191</sup> As Baker<sup>192</sup> has pointed out about the socialization of risk, "a transfer of risk is also a transfer of responsibility .... [R]isk creates responsibility."

The fossil fuel industry's use of demand-as-blame framing is not limited to its legal defenses. As Schneider et al.<sup>27</sup> describe, fossil fuel interests have likewise sought to delegitimize AGW activism, such as the fossil fuel divestment movement, by deploying a rhetorical "hypocrite's trap [that] performs the disciplinary work of individualizing responsibility" (see also Ayling<sup>193</sup>).

### **Historical contexts, ramifications, and trajectories of ExxonMobil's communication tactics**

ExxonMobil's selective use of rhetoric and discourse to frame AGW epitomizes the first "general principle" of effective public affairs according to Herbert Schertz,<sup>185</sup> Mobil Oil's Vice President of Public Affairs (1969–1988) and the pioneer of their advertorials: "Grab the good words – and the good concepts – for yourself."<sup>185</sup> "[B]e sensitive to semantic infiltration, the process whereby language does the dirty work of politics...Be sensitive to these word choices, and be competitive in how you use them. Your objective is to wrap yourself in the good phrases while sticking your opponents with the bad ones."

## Risk

ExxonMobil Corp's systematic introduction of "risk" rhetoric into its doubt-mongering advertorials coincided with the 1999 merger of Exxon and Mobil, suggestive of a strategic shift in public relations.

A second shift, in the mid-2000s, from explicit doubt to implicit acknowledgment confused by "risk" rhetoric, coincides with what one ExxonMobil Corp manager saw as "an effort by [then CEO Rex] Tillerson to carefully reset the corporation's profile on climate positions so that it would be more sustainable and less exposed."<sup>48</sup>

To this day, ExxonMobil Corp's (also Chevron's and ConocoPhillips') refrain on AGW, and the primary basis on which the company is now widely perceived to accept basic climate science, is that it is a "risk."<sup>26,194,195</sup> Across all of ExxonMobil Corp's flagship reports concerning AGW, by far the highest scoring collocates of "climate change" and "global warming" is "risk(s)" (S6.1, [supplemental information](#)). Compared with internal and peer-reviewed documents, terms in flagship reports invoking "risks of climate change" are highly divergent (S6.1). As with advertorials, none say that climate change is real and human caused.

## Individualized responsibility

The findings in the [results](#) section lead us to conclude that ExxonMobil advertorials used frames of individualized responsibility and the rhetoric of "risk" to construct what St. John III<sup>35</sup> calls a "sense-making corporate persona" that appealed to the enduring principles of "rugged individualism" and self-reliance that pervade US culture and ideology.<sup>35,196–201</sup> Their public affairs campaign coincided with solidifying, intertwined notions of distributed risks and individualized responsibility in western public policy debates since the 1970s, which have been driven by the global embrace of neoliberalism and globalization<sup>27,197,202,203</sup> and encouraged by reductive, episodic news framings<sup>16,179</sup> (and which are conceptualized by social theories<sup>59,204,205</sup> such as Beck et al.'s "risk society,"<sup>170,206,207</sup> Douglas et al.'s "risk culture,"<sup>208</sup> and Foucault et al.'s "governmentality").<sup>209,210</sup> ExxonMobil tapped into this trend toward the individualization of social risks, and brought it to bear on AGW.<sup>59,208,211</sup>

ExxonMobil is part of a lineage of industrial producers of harmful commodities that have used personal responsibility framings to disavow themselves.<sup>212–214</sup> Among them: tobacco companies;<sup>13,119,120</sup> the National Association of Manufacturers;<sup>215</sup> plastics producers (including Exxon, Mobil, and ExxonMobil Corp), packaging and beverage manufacturers, and waste companies;<sup>197,216–222</sup> and purveyors of sugar-sweetened beverages and junk food,<sup>98,99,214</sup> leaded products,<sup>223,224</sup> motor vehicles,<sup>94,225</sup> alcohol,<sup>12,226</sup> electronic gambling,<sup>227</sup> and firearms.<sup>228</sup>

Among, in particular, the public AGW communications of major fossil fuel companies, individualized responsibility framings—and the accompanying narrative of fossil fuel lock-in—have become seemingly ubiquitous.<sup>26,51</sup> The very notion of a personal "carbon footprint," for example, was first popularized in 2004–2006 by oil firm BP as part of its \$100+ million per year "beyond petroleum" US media campaign.<sup>229–235</sup> Discourse analysis of this campaign led Doyle<sup>236</sup> to conclude that "BP places responsibility for combatting climate change upon the individual consumer." Smerecnik and Renegar<sup>57</sup> have shown that subsequent BP branding activities similarly "plac[e] participatory emphasis

on consumer conservation behavior as opposed to corporate responsibility." This industry framing continues to dominate today.<sup>26,81</sup> In 2019, for instance, BP launched a new "Know your Carbon Footprint" publicity campaign.<sup>237</sup> In 2020, the CEO of Total said that "Change will not come from changing the source of supply. You have to reduce demand."<sup>238</sup> Until 2020, all major oil and gas companies disregarded or disavowed accountability for all Scope 3 greenhouse gas emissions resulting from the use of their products. ExxonMobil Corp, Chevron, and ConocoPhillips continue to do so.<sup>239</sup>

The result is that fossil fuel industry discourse on AGW appears to have encouraged and embodied what Maniates<sup>197</sup> describes as "an accelerating individualization of responsibility" that "is narrowing, in dangerous ways, our 'environmental imagination'" by "ask [ing] that individuals imagine themselves as consumers first and citizens second."<sup>197,26,27,52,56</sup> This depoliticized "capitalistic agency," Smerecnik and Renegar<sup>57</sup> argue, works to "prohibit fundamental social change that would disrupt the fossil fuel industry."<sup>57,59</sup> Experimental evidence appears to support this conclusion. Palm et al.,<sup>240</sup> for example, observe that messages framed in terms of individual behavior not only "decreased individuals' willingness to take personal actions" but also "decreased willingness to [take collective action such as to] support pro-climate candidates, reduced belief in the accelerated speed of climate change, and decreased trust in climate scientists." Illustrations of how narratives of individualized responsibility have protected fossil fuel interests from climate action are widespread. One is Yale University's 2014 refusal to divest from fossil fuel companies, which was "predicated on the idea that consumption of fossil fuels, not production, is the root of the climate change problem."<sup>241</sup> Another is the Republican Party's 2020 legislative agenda on AGW, whose premise was that "fossil fuels aren't the enemy. It's emissions."<sup>242,243</sup> A third is that the Paris Agreement "is silent on the topic of fossil fuels."<sup>68</sup>

## Summary and conclusion

Available documents show that, during the mid-2000s, ExxonMobil's public AGW communications shifted from explicit doubt (a Scientific Uncertainty frame) to implicit acknowledgment couched in discourses conveying two frames: a Socioeconomic Threat frame, and a Fossil Fuel Savior (FFS) frame. According to the FFS frame:

- (1) Everything about AGW is uncertain: a "risk," as contrasted with a reality.
- (2) Fossil fuel companies are passive suppliers responding to consumer energy demand.
- (3) Continued fossil fuel dominance is (1) inevitable, given the insufficiency of low-carbon technologies; and (2) reasonable and responsible, because fossil fuels lead to profound, explicit benefits and only ambiguous, uncertain climate "risk(s)."
- (4) Customers are to blame for demanding fossil fuels, whose "risk(s)" were common knowledge. Customers knowingly chose to value the benefits of fossil fuels above their risks.

Ignored and obscured by these perspectives are fossil fuel interests' pervasive marketing, disinformation campaigns, and lobbying against climate and clean energy policies, all of which

have served to establish and reinforce infrastructural, institutional, and behavioral carbon lock-ins, thereby undercutting consumer choice and agency.<sup>244,245</sup>

Propaganda tactics of the fossil fuel industry such as these have received less scrutiny than those of their tobacco counterparts. Further attention is needed, because although individualized narratives of risk, responsibility, and the like are less blatant than outright climate science denial, such “discursive grooming” is now pervasive in structuring the agenda of scholars, policymakers, and the public.<sup>59,68,69,197,246</sup>

## EXPERIMENTAL PROCEDURES

### Resource availability

#### Lead contact

Further information and reasonable requests for resources by qualified researchers should be directed to and will be fulfilled by the lead contact, Geoffrey Supran ([gjsupran@fas.harvard.edu](mailto:gjsupran@fas.harvard.edu)).

#### Materials availability

This study did not generate new unique materials.

#### Data and code availability

Raw data (original PDF internal documents, peer-reviewed publications, and advertorials) for this study cannot be reproduced due to copyright restrictions. However, a catalog of all 180 analyzed documents, and links to public archives containing these data, are provided in S7, [supplemental information](#). Additionally, raw searchable .txt versions of all documents, as well as post-processed flattened text and document term matrices, are deposited on Harvard Dataverse: <https://doi.org/10.7910/DVN/XXQUKJ>. The datasets and code generated during this study are provided in the same repository. Access will be granted upon reasonable request by qualified researchers.

### Corpora

For detailed descriptions of how we previously compiled the 180 ExxonMobil documents analyzed in this study, see Supran and Oreskes.<sup>1,2</sup> For a catalog of all 180 documents, and links to their public archives, see S7, [supplemental information](#). In summary, the 32 internal company documents (1977–2002) were collated from public archives provided by ExxonMobil Corp,<sup>101</sup> *InsideClimate News*,<sup>102</sup> and Climate Investigations Center.<sup>103</sup> The 72 peer-reviewed publications (1982–2014) were obtained by identifying all peer-reviewed documents among ExxonMobil Corp’s lists of Contributed Publications, except for three articles discovered independently during our research. All 72 publications were (co-)authored by at least one ExxonMobil employee.<sup>104</sup> The 76 advertorials (1972–2009) expressing any positions on AGW (real and human caused, serious, or solvable) were identified by manual content analysis of 1,448 ExxonMobil advertorials (1924–2013) collated from PolluterWatch and ProQuest archives.<sup>105,106</sup>

### Pre-processing

To enable computational analysis, scanned documents were converted to searchable text files using optical character recognition. Text was stripped of formatting details and punctuation, tokenized, and lowercased (for details, see S1.1, [supplemental information](#)). This yielded internal, peer-reviewed, and advertorial corpora comprising 69,802 words, 716,477 words, and 34,141 words (16,121 in Mobil advertorials and 18,020 in ExxonMobil Corp advertorials), respectively.

For divergent term (topic) analysis, we added (substituted) several synthetic tokens that combine: terms of identical cognate form (e.g., “effect” and “effects” became “effect(s)”; and terms judged by the authors to be near-synonyms (e.g., “co2” and “carbon dioxide” became “co2/carbon dioxide”; “countries” and “nations” became “countries/nations”)—for all synthetic tokens, see `vectorize.R` script.<sup>109,247</sup> Document collections were transformed into document-term matrices comprising all: 1- to 5-grams (unique, contiguous word strings of 1–5 tokens in length) for divergent term analysis; and 1-grams for divergent topic analysis.<sup>248</sup>

### Divergent term analysis (FS and LL ratio)

Internal, peer-reviewed, and advertorial corpora were compared pairwise to identify rhetorical distinctiveness (or divergence) between the terms communicated in each text. (We combine all (Mobil plus ExxonMobil Corp) advertorials before comparing them against internal and peer-reviewed documents from Exxon and Exxon/ExxonMobil Corp, respectively. This simplifies the presentation of results without substantively affecting our findings.) To capture different forms of divergence, we applied two algorithms: FS and Dunning LL ratio ( $G^2$ ) score.<sup>108–110</sup> FS and LL are established, complementary tools for word frequency analysis in computational linguistics and digital humanities.<sup>110,249,250</sup>

The FS indicates how often a given term appears in one corpus versus another. The score ranges from 0 (when only corpus A features the term) to 1 (when only corpus B includes the term). To account for the difference in word counts between corpora, we normalized scores by using relative frequencies. For example, a score of 0.8 means that 80% of all normalized instances of a term appear in corpus B. As Risi and Proctor observe, “FSs are useful for identifying taboos: terms generally avoided by one side or the other.”<sup>109</sup>

FSs produce immediately interpretable results, yet their reliance on multiplicative ratios—versus additive differences—tends to over-represent rare words.<sup>108</sup> To identify subtle patterns that might otherwise escape notice, we also use the LL ( $G^2$ ) statistic proposed by Dunning (1993), which is a parametric analysis that primarily identifies “surprising,” additively over-represented words, while also giving some weight to multiplication.<sup>108,110,251</sup> Large  $[G^2]$  scores indicate terms that have statistically significant relative frequency differences between two corpora. LLs are therefore useful for identifying tropes: terms used disproportionately by one side.

### Divergent topic analysis (LDA)

In the field of automated text summarization, divergent terms identified by LL are referred to as “topic signatures.”<sup>249,252</sup> In order to identify the topics represented by such terms, and to better understand the roles these terms play in framing each topic, we also examine the documents using topic modeling with LDA.<sup>111</sup> LDA is a computational, unsupervised machine-learning algorithm for discovering hidden thematic structure in collections of texts.<sup>253</sup> *A priori* coding schemes are not supplied. Rather, ‘topics’ (clusters of words associated with a single theme) emerge inductively based on patterns of co-occurrence of words in a corpus.

We are specifically interested in identifying the topical distinctiveness (or divergence) between document categories. In the main text, we compare topics between ( $\alpha$ ) all advertorials and ( $\beta$ ) combined internal and peer-reviewed documents.

To do so, we first model the distribution of topics over all document categories, by inputting to LDA an aggregated corpus comprising all advertorials, internal documents, and peer-reviewed publications (for details of LDA model selection, topic validation, and labeling, see section S1.2, [supplemental information](#)). Once topic-word distributions are obtained, we then take an approach analogous to that for finding divergent terms above, noting that just as LL ratios of term frequencies identify divergent terms, LL ratios of topic weights identify divergent topics. We compute LL ratios of topic weights by constructing document-topic matrices for each of sub-corpora  $\alpha$  and  $\beta$ .

Although they are run independently, analyses of divergent terms (by FS and LL) and topics (by LL of LDA) are complementary. The former identifies the distinctive usage of individual n-grams by one corpus versus another. The latter helps contextualize the thematic role that these words together play in communicating and framing topics.

### Frame package analysis

Van Gorp<sup>117</sup> argues that the “strongly abstract nature of frames implies that quantitative research methods should be combined with the interpretative prospects of qualitative methods.” To this end, we use the distinctive terms and topics identified using computational techniques to then inform an inductive, qualitative approach to constructing frames as frame packages in advertorials. Van Gorp<sup>117</sup> defines frame packages as an integrated structure of framing devices (manifest textual elements that function as indicators of a frame) and reasoning devices (logical chains of causal reasoning), and proposes Strauss and Corbin’s<sup>254</sup> three-step coding scheme for identifying frame packages and

assembling them into a so-called “frame matrix.”<sup>6,10,17,116–118,254</sup> We adopt this approach.

### Open coding

The first step is to compile what Van Gorp<sup>116</sup> calls an “inventory of empirical indicators that may contribute to the readers’ interpretation of the text,” comprising feasible framing or reasoning devices identified in each document. We used FS, LL, and LDA to systematize this process of locating frames and detecting how they are shaped by lexical composition (for details, see S1.3, [supplemental information](#)). We further investigated these discursive constructs by performing collocation searches.<sup>51</sup> The logDice statistic was computed to measure collocational association because it permits meaningful comparison of different sized corpora.<sup>255,256</sup>

### Axial coding

The second step is to arrange coded devices along “axes of meaning” by comparing and contrasting open-coding results between documents and then reducing the results to broader meanings or dimensions.<sup>113,116</sup> We do so with reference to an inventory of discourses that we assembled based on a literature review of past studies of AGW communications by fossil fuel interests (see S3, [supplemental information](#)).<sup>116</sup>

### Selective coding

The last step is to enter axial codes into a “frame matrix” that summarizes the framing and reasoning devices of each frame package.<sup>116</sup>

## SUPPLEMENTAL INFORMATION

Supplemental information can be found online at <https://doi.org/10.1016/j.oneear.2021.04.014>.

## ACKNOWLEDGMENTS

This research was supported by Harvard University Faculty Development Funds and by the Rockefeller Family Fund. The authors thank Stephan Risi (Stanford University), Richard A. Daynard (Northeastern University), and Viktoria Cologna (ETH Zürich) for helpful discussions; Ploy Achakulwisut (Stockholm Environment Institute) for helpful discussions and assistance with inter-coder reliability testing; and three anonymous peer reviewers. G.S. dedicates this publication to the life and memory of his father, Lyle David Supran.

## AUTHOR CONTRIBUTIONS

Conceptualization, G.S.; methodology, G.S.; validation, G.S. and N.O.; formal analysis, G.S.; investigation, G.S.; writing – original draft, G.S.; writing – review & editing, G.S. and N.O.; visualization, G.S.; supervision, G.S. and N.O.; funding acquisition, G.S. and N.O.

## DECLARATION OF INTERESTS

The authors have received speaking and writing fees (and N.O. has received book royalties) for communicating their research, which includes but is not limited to the topics addressed in this paper. The authors have no other relevant financial ties and declare no competing interests.

## INCLUSION AND DIVERSITY

While citing references scientifically relevant for this work, we also actively worked to promote gender balance in our reference list.

Received: October 14, 2020

Revised: March 2, 2021

Accepted: April 22, 2021

Published: May 13, 2021

## REFERENCES

- Supran, G., and Oreskes, N. (2017). Assessing ExxonMobil’s climate change communications (1977–2014). *Environ. Res. Lett.* *12*, 084019.
- Supran, G., and Oreskes, N. (2020). Addendum to “Assessing ExxonMobil’s climate change communications (1977–2014).” *Environ. Res. Lett.* *15*, 119401.
- Supran, G., and Oreskes, N. (2020). Reply to comment on ‘Assessing ExxonMobil’s climate change communications (1977–2014).’ *Environ. Res. Lett.* *15*, 118002.
- ExxonMobil. (2000). Global Climate Change – A Better Path Forward. <https://perma.cc/PJ4Q-WG32>.
- ExxonMobil. (2008). The Fuels of the Future (Advertorial) (The New York Times).
- Gamson, W.A., and Modigliani, A. (1989). Media discourse and public opinion on nuclear power: a constructionist approach. *Am. J. Sociol.* *95*, 1–37.
- Nisbet, M.C. (2020). Framing the debates over climate change and poverty. In *Doing News Framing Analysis: Empirical and Theoretical Perspectives*, J.A. Kuypers and P. D’Angelo, eds. (Taylor & Francis Group), pp. 43–82.
- Bateson, G. (1955). A theory of play and fantasy. *Psychiatr. Res. Rep. Am. Psychiatr. Assoc.* *2*, 39–51.
- Goffman, E. (1974). *Frame Analysis: An Essay on the Organization of Experience* (Harvard University Press).
- Entman, R.M. (1993). Framing: toward clarification of a fractured paradigm. *J. Commun.* *43*, 51–58.
- Cacciatore, M.A., Scheufele, D.A., and Iyengar, S. (2016). The end of framing as we know it...and the future of media effects. *Mass Commun. Soc.* *19*, 7–23.
- Hawkins, B., and Holden, C. (2013). Framing the alcohol policy debate: industry actors and the regulation of the UK beverage alcohol market. *Crit. Policy Stud.* *7*, 53–71.
- Friedman, L.C., Cheyne, A., Givelber, D., Gottlieb, M.A., and Daynard, R.A. (2015). Tobacco industry use of personal responsibility rhetoric in public relations and litigation: Disguising freedom to blame as freedom of choice. *Am. J. Public Health* *105*, 250.
- Hilgartner, S., and Bosk, C.L. (1988). The rise and fall of social problems: a public arenas model. *Am. J. Sociol.* *94*, 53–78.
- Wynne, B. (2010). Strange weather, again: climate science as political art. *Theory, Cult. Soc.* *27*, 289–305.
- Iyengar, S. (1989). How citizens think about national issues: a matter of responsibility. *Am. J. Pol. Sci.* *33*, 878–900.
- Schlichting, I. (2013). Strategic framing of climate change by industry actors: a meta-analysis. *Environ. Commun.* *7*, 493–511.
- Farrell, J. (2015). Network structure and influence of the climate change counter-movement. *Nat. Clim. Chang.* *6*, 370–374.
- Boussalis, C., and Coan, T.G. (2016). Text-mining the signals of climate change doubt. *Glob. Environ. Chang.* *36*, 89–100.
- Dunlap, R.E., and McCright, A.M. (2011). Organized climate change denial. In *The Oxford Handbook of Climate Change and Society*, J.S. Dryzek, R.B. Norgaard, and D. Schlosberg, eds. (Oxford University Press), pp. 144–160.
- Oreskes, N., and Conway, E.M. (2010). *Merchants of Doubt: How a Handful of Scientists Obscured the Truth on Issues from Tobacco Smoke to Global Warming* (Bloomsbury Press).
- Gelbspan, R. (1997). *The Heat Is on* (Addison-Wesley Publishing).
- Union of Concerned Scientists. (2007). *Smoke, Mirrors & Hot Air - How ExxonMobil Uses Big Tobacco’s Tactics to Manufacture Uncertainty on Climate Science*. <https://perma.cc/64RJ-8SBZ>.
- Michaels, D. (2008). *Doubt Is Their Product* (Oxford University Press).
- SkepticalScience.com. Climate Myths Sorted by Taxonomy. <https://perma.cc/7LAF-MHEX>.

26. Cahill, S. (2017). *Imagining Alternatives in the Emerald City: The Climate Change Discourse of Transnational Fossil Fuel Corporations* (University of Victoria).
27. Schneider, J., Schwarze, S., Bsumek, P.K., and Peeples, J. (2016). *Under Pressure - Coal Industry Rhetoric and Neoliberalism* (Palgrave Macmillan UK).
28. Lamb, W.F., Mattioli, G., Levi, S., Roberts, J.T., Minx, J.C., Müller-hansen, F., Capstick, S., Creutzig, F., Culhane, T., and Steinberger, J.K. (2020). Discourses of climate delay. *Glob. Sustain.* 3, 1–5.
29. Coan, T.G., Boussalis, C., Cook, J., and Nanko, M.O. (2021). Computer-assisted detection and classification of misinformation about climate change. Working Paper. <https://doi.org/10.31235/osf.io/crxfm>.
30. Climate Investigations Center (2019). *Trade Associations and the Public Relations Industry*. <https://perma.cc/PN3M-P4FU>.
31. Brulle, R.J., Aronczyk, M., and Carmichael, J. (2020). Corporate promotion and climate change: an analysis of key variables affecting advertising spending by major oil corporations, 1986 – 2015. *Clim. Change* 159, 87–101.
32. Brown, C., Waltzer, H., and Waltzer, M.B. (2001). Daring to Be heard: advertorials by organized interests on the op-ed page of the New York times, 1985-1998. *Polit. Commun.* 18, 23–50.
33. Brown, C., and Waltzer, H. (2005). Every Thursday: advertorials by Mobil oil on the op-ed page of the New York times. *Public Relat. Rev.* 31, 197–208.
34. St. John, B., III (2014). The “creative confrontation” of Herbert Schmertz: public relations sense making and the corporate persona. *Public Relat. Rev.* 40, 772–779.
35. St. John, B., III (2014). Conveying the sense-making corporate persona: the Mobil Oil “Observations” columns, 1975–1980. *Public Relat. Rev.* 40, 692–699.
36. Crable, R.E., and Vibbert, S.L. (1983). Mobil's epideictic advocacy: “Observations” of Prometheus-bound. *Commun. Monogr.* 50, 380–394.
37. Murphree, V., and Aucoin, J. (2010). The energy crisis and the media: Mobil oil corporation's debate with the media 1973–1983. *Am. J.* 27, 7–30.
38. Smith, G.L., and Heath, R.L. (1990). Moral appeals in Mobil Oil's op-ed campaign. *Public Relat. Rev.* XVI, 48–54.
39. Heath, R.L., and Nelson, R.A. (1986). *Issues Management: Corporate Public Policymaking in an Information Society* (SAGE).
40. Kerr, R.L. (2005). *Rights of Corporate Speech: Mobil Oil and the Legal Development of the Voice of Big Business* (LFB Scholarly Publishing LLC).
41. Cooper, C.A., and Nownes, A.J. (2004). Money well spent? An experimental investigation of the effects of advertorials on citizen opinion. *Am. Polit. Res.* 32, 546–569.
42. Kerr, R.L. (2004). Creating the corporate citizen: Mobil Oil's editorial-advocacy campaign in the New York times to advance the right and practice of corporate political speech, 1970–80. *Am. J. Public Health* 21, 39–62.
43. Anderson, J.W. (1984). *A Quantitative and Qualitative Analysis of Mobil's Advocacy Advertising in the New York Times* (Pennsylvania State University).
44. Grantham, S., and Vieira, E.T., Jr. (2018). Exxonmobil's social responsibility messaging - 2002-2013 CEO letters. *Appl. Environ. Educ. Commun.* 17, 266–279.
45. Jerving, S., Jennings, K., Hirsh, M.M., and Rust, S. (2015). What Exxon Knew about the Earth's Melting Arctic (Los Angeles Times). <https://perma.cc/NA86-5PWH>.
46. Banerjee, N., Song, L., Hasemyer, D., and Cushman, J.H., Jr. (2015). Exxon: the road not taken (InsideClimate News). <https://perma.cc/ACY4-8NW5>.
47. Achakulwisut, P., Scandella, B., Supran, G., and Voss, B. (2016). Ending ExxonMobil Sponsorship of the American Geophysical Union - How ExxonMobil's Past and Present Climate Misinformation Violates the AGU's Organizational Support Policy and Scientific Integrity. <https://perma.cc/PBN7-V59J>.
48. Coll, S. (2012). *Private Empire: ExxonMobil and American Power* (Penguin Books).
49. Rowlands, I.H. (2000). Beauty and the beast? BP's and Exxon's positions on global climate change. *Environ. Plan. C Gov. Policy* 18, 339–354.
50. Farrell, J. (2015). Corporate funding and ideological polarization about climate change. *Proc. Natl. Acad. Sci. U S A* 113, 92–97.
51. Jaworska, S. (2018). Change but no climate change: discourses of climate change in corporate social responsibility reporting in the oil industry. *Int. J. Bus. Commun.* 55, 194–219.
52. Plec, E., and Pettenger, M. (2012). Greenwashing consumption: the didactic framing of ExxonMobil's energy solutions. *Environ. Commun.* 6, 459–476.
53. Vang, P. (2014). *Good Guys: A Cultural Semiotic Study of the Print Advertising of the Oil Industry (1900-2000)* (Linköping University).
54. McCright, A.M., and Dunlap, R.E. (2000). Challenging global warming as a social problem: an analysis of the conservative movement's counter-claims. *Soc. Probl.* 47, 499–522.
55. Nelson, D. (2019). *Framing the Carbon Tax in Australia: An Investigation of Frame Sponsorship and Organisational Influence behind Media Agendas* (University of Technology Sydney).
56. Livesey, S.M. (2002). Global warming wars: rhetorical and discourse analytic approaches to Exxonmobil's corporate public discourse. *J. Bus. Commun.* 39, 117–148.
57. Smerecnik, K.R., and Renegar, V.R. (2010). Capitalistic agency: the rhetoric of BP's Helios power campaign. *Environ. Commun.* ISSN 4, 152–171.
58. Underwood, T. (2017). A genealogy of distant reading. *Digit. Humanit. Q.* 11, 1–12.
59. Kent, J. (2009). Individualized responsibility and climate change: 'if climate protection becomes everyone's responsibility, does it end up being no-one's?'. *Cosmop. Civ. Soc. J.* 7, 132–149.
60. State of Minnesota v. (2020). *American Petroleum Institute (62-CV-20-3837)*. <https://perma.cc/5FWM-6ZWU>.
61. District of Columbia v. (2020). *ExxonMobil corporation (1:20-CV-01932)*. <https://perma.cc/ENQ9-M9V9>.
62. Commonwealth of Massachusetts v. (2019). *Exxon Mobil corporation (1984CV03333)*. <https://perma.cc/6ZN2-JTMG>.
63. State of Delaware v. (2020). *BP America Inc (N20C-09-097)*. <https://perma.cc/3AG9-5495>.
64. State of Connecticut v. (2020). *ExxonMobil Corporation (3:20-cv-01555)*. <https://perma.cc/S5LM-T2GB>.
65. Carrington, D. (2021). “A Great Deception”: Oil Giants Taken to Task over “Greenwash” Ads (The Guardian), <https://perma.cc/6HND-LS3V>.
66. Ekwurzel, B., Boneham, J., Dalton, M.W., Heede, R., Mera, R.J., Allen, M.R., and Frumhoff, P.C. (2017). The rise in global atmospheric CO<sub>2</sub>, surface temperature, and sea level from emissions traced to major carbon producers. *Clim. Change* 144, 579–590.
67. Heede, R. (2014). Tracing anthropogenic carbon dioxide and methane emissions to fossil fuel and cement producers, 1854-2010. *Clim. Change* 122, 229–241.
68. Piggot, G., Erickson, P., van Asselt, H., and Lazarus, M. (2018). Swimming upstream: addressing fossil fuel supply under the UNFCCC. *Clim. Policy* 18, 1189–1202.
69. Green, F., and Denniss, R. (2018). Cutting with both arms of the scissors: the economic and political case for restrictive supply-side climate policies. *Clim. Change* 150, 73–87.
70. Stockholm Environment Institute, International Institute for Sustainable Development, Overseas Development Institute, Climate Analytics, Centre for International Climate and Environmental Research, and Programme, U.E. (2019). *The Production Gap: The Discrepancy between*

- Countries' Planned Fossil Fuel Production and Global Production Levels Consistent with Limiting Warming to 1.5°C or 2°C. <https://perma.cc/C6WU-LYPT>.
71. Turnheim, B., and Geels, F.W. (2012). Regime destabilisation as the flipside of energy transitions: lessons from the history of the British coal industry (1913-1997). *Energy Policy* 50, 35–49.
  72. T. Princen, J.P. Manno, and P.L. Martin, eds. (2015). *Ending the Fossil Fuel Era* (MIT Press).
  73. Monbiot, G. (2019). The big polluters' masterstroke was to blame the climate crisis on you and me (The Guardian). <https://perma.cc/X4DP-YFLF>.
  74. Mann, M.E. (2019). Lifestyle Changes Aren't Enough to Save the Planet. Here's what Could (TIME). <https://perma.cc/JRS7-DVVE>.
  75. Grover, S. (2019). In Defense of Eco-Hypocrisy (Noteworthy). <https://perma.cc/3N2Y-GWJW>.
  76. Heglar, M.A. (2018). The Big Lie We're Told about Climate Change Is that It's Our Own Fault (Vox.com). <https://perma.cc/Y3AC-B97T>.
  77. Frumhoff, P.C., Heede, R., and Oreskes, N. (2015). The climate responsibilities of industrial carbon producers. *Clim. Change* 132, 157–171.
  78. Franta, B. (2018). Early oil industry knowledge of CO<sub>2</sub> and global warming. *Nat. Clim. Chang.* 8, 1024–1025.
  79. Atkin, E. (2019). Introducing: The Fossil Fuel Ad Anthology (HEATED Newsletter). <https://perma.cc/8DR7-7P9C>.
  80. Westervelt, A. (2018). Drilled: A True Crime Podcast about Climate Change. <https://perma.cc/JD2B-553V>.
  81. (2019). Big Oil's Real Agenda on Climate Change - How the Oil Majors Have Spent \$1bn since Paris on Narrative Capture and Lobbying on Climate (Influence Map). <https://perma.cc/BG6R-RWT9>.
  82. Brulle, R.J. (2018). The climate lobby: a sectoral analysis of lobbying spending on climate change in the USA, 2000 to 2016. *Clim. Change* 149, 289–303.
  83. Grasso, M. (2019). Oily politics: a critical assessment of the oil and gas industry's contribution to climate change. *Energy Res. Soc. Sci.* 50, 106–115.
  84. Shue, H. (2017). Responsible for what? Carbon producer CO<sub>2</sub> contributions and the energy transition. *Clim. Change* 144, 591–596.
  85. Grasso, M. (2020). Towards a broader climate ethics: confronting the oil industry with morally relevant facts. *Energy Res. Soc. Sci.* 62, 101383.
  86. Olszynski, M., Mascher, S., and Doelle, M. (2017). From smokes to smokestacks: lessons from tobacco for the future of climate change liability. *Georg. Environ. L. Rev.* 30, 1–45.
  87. Muffett, C., and Feit, S. (2017). Smoke and Fumes - the Legal and Evidentiary Basis for Holding Big Oil Accountable for the Climate Crisis (Center for International Environmental Law). <https://perma.cc/UT88-STQJ>.
  88. Callaghan, M.W., Minx, J.C., and Forster, P.M. (2020). A topography of climate change research. *Nat. Clim. Chang.* 10, 118–123.
  89. Elgesem, D., Steskal, L., Diakopoulos, N., Elgesem, D., Steskal, L., and Diakopoulos, N. (2015). Structure and content of the discourse on climate change in the Blogosphere: the big picture. *Environ. Commun.* 9, 169–188.
  90. O'Neill, S., Williams, H.T.P., Kurz, T., Wiersma, B., and Boykoff, M. (2015). Dominant frames in legacy and social media coverage of the IPCC Fifth Assessment Report. *Nat. Clim. Chang.* 5, 380–385.
  91. Metag, J. (2016). Content analysis methods for assessing climate change communication and media portrayals. In *Oxford Encyclopedia of Climate Change Communication*, M. Nisbet, S. Ho, E. Markowitz, S. O'Neill, M.S. Schäfer, and J. Thaker, eds. (Oxford University Press), pp. 1–34.
  92. Miller Gaither, B., and Gaither, T.K. (2016). Marketplace advocacy by the U.S. Fossil fuel industries: Issues of representation and environmental discourse. *Mass Commun. Soc.* 19, 585–603.
  93. Gaither, B.M., and Sinclair, J. (2018). Environmental marketplace advocacy: Influences and implications of U.S. Public response. *J. Mass Commun. Q.* 95, 169–191.
  94. Aronczyk, M. (2018). Public relations, issue management, and the transformation of American environmentalism, 1948 – 1992. *Enterp. Soc.* 19, 836–863.
  95. Robinson, M.L. (2014). *Marketing Big Oil - Brand Lessons from the World's Largest Companies* (Palgrave Macmillan).
  96. Cho, C.H., Laine, M., Roberts, R.W., and Rodrigue, M. (2018). The frontstage and Backstage of corporate sustainability reporting: evidence from the Arctic National Wildlife Refuge Bill. *J. Bus. Ethics* 152, 865–886.
  97. Sanchez, L., Gerasimchuk, I., and Beagley, J. (2019). Burning Problems, Inspiring Solutions: Sharing Lessons on Action against Tobacco and Fossil Fuels (International Institute for Sustainable Development, NCD Alliance). <https://perma.cc/4Z83-YEMA>.
  98. Dorfman, L., Cheyne, A., Friedman, L.C., Wadud, A., and Gottlieb, M. (2012). Soda and tobacco industry corporate social responsibility campaigns: how do they compare? *Plos Med.* 9, e1001241.
  99. Brownell, K.D., and Haven, N. (2009). The perils of ignoring history: big tobacco played dirty and millions died. How similar is big food? *Milbank Q.* 87, 259–294.
  100. Chaiton, M., Ferrence, R., and Legresley, E. (2006). Perceptions of industry responsibility and tobacco control policy by US tobacco company executives in trial testimony. *Tob. Control* 15, iv98–iv106.
  101. ExxonMobil Corp. Supporting Materials. <https://perma.cc/D862-KB2N>.
  102. ICN Documents (Exxon: The Road Not Taken). InsideClimate News. <https://perma.cc/KCG8-M9ZM>.
  103. Climate Investigations Center. Climate Files. [www.climatefiles.com](http://www.climatefiles.com).
  104. ExxonMobil Corp. (2015). ExxonMobil Contributed Publications. <https://perma.cc/3QEV-KLFP>.
  105. PolluterWatch Exxon and Mobil Ads. <https://perma.cc/8XHW-5GZE>.
  106. ProQuest ProQuest Historical Newspapers Database. <https://search.proquest.com/>.
  107. Touri, M., and Koteyko, N. (2015). Using corpus linguistic software in the extraction of news frames: towards a dynamic process of frame analysis in journalistic texts. *Int. J. Soc. Res. Methodol.* 18, 599–614.
  108. Schmidt, B. (2011). Comparing Corporates by Word Use (Sapping Atten). <https://perma.cc/S4EJ-7NRJ>.
  109. Risi, S., and Proctor, R.N. (2020). Big tobacco focuses on the facts to hide the truth: an algorithmic exploration of courtroom tropes and taboos. *Tob. Control* 29, e41–e49.
  110. Dunning, T. (1993). Accurate methods for the statistics of surprise and coincidence. *Comput. Linguist.* 19, 61–74.
  111. Blei, D.M., Ng, A.Y., and Jordan, M.I. (2003). Latent Dirichlet allocation. *Journal of Machine Learning Research* 3, 993–1022.
  112. Walter, D., and Ophir, Y. (2019). News frame analysis: an inductive mixed-method computational approach. *Commun. Methods Meas.* 13, 248–266.
  113. Klebanov, B.B., Diermeier, D., Beigman, E., and Diermeier, D. (2008). Automatic annotation of semantic fields for political science research. *J. Inf. Technol. Polit.* 5, 95–120.
  114. Greussing, E., and Boomgaarden, H.G. (2017). Shifting the refugee narrative? An automated frame analysis of Europe's 2015 refugee crisis. *J. Ethn. Migr. Stud.* 43, 1749–1774.
  115. Jacobi, C., Atteveldt, W. Van, and Welbers, K. (2016). Quantitative analysis of large amounts of journalistic texts using topic modelling. *Digit. J.* 4, 89–106.
  116. Gorp, B. Van (2009). Strategies to take subjectivity out of framing analysis. In *Doing News Framing Analysis: Empirical and Theoretical Perspectives*, J.A. Kuypers and P. D'Angelo, eds. (Routledge), pp. 84–109.
  117. Gorp, B. Van (2007). The constructionist approach to framing: bringing culture back in. *J. Commun.* 57, 60–78.

118. Gorp, B. Van, and Verduyck, T. (2012). Frames and counter-frames giving meaning to dementia: a framing analysis of media content. *Soc. Sci. Med.* 74, 1274–1281.
119. Mejia, P., and Dorfman, L. (2014). The origins of personal responsibility rhetoric in news coverage of the tobacco industry. *Am. J. Public Health* 104, 1048–1051.
120. Dorfman, L., Cheyne, A., Gottlieb, M.A., Mejia, P., Nixon, L., Friedman, L.C., and Daynard, R.A. (2014). Cigarettes become a dangerous product: tobacco in the rearview mirror, 1952 – 1965. *Am. J. Public Health* 104, 37–46.
121. Sievert, C., and Shirley, K.E. (2014). LDAvis: a method for visualizing and interpreting topics. In *Proceedings of the Workshop on Interactive Language Learning, Visualization, and Interfaces*, Jason Chuang, Spence Green, Marti Hearst, Jeffrey Heer, and Philipp Koehn, eds. (Association for Computational Linguistics), pp. 63–70.
122. Renforth, P., and Henderson, G. (2017). Assessing ocean alkalinity for carbon sequestration. *Rev. Geophys.* 55, 636–674.
123. ExxonMobil. (2008). *Vehicles of Change* (Advertorial) (The New York Times).
124. ExxonMobil. (1997). *Climate Change: A Prudent Approach* (Advertorial) (The New York Times).
125. ExxonMobil. (2009). *Citizenship for the Long Term* (Advertorial, 22 May 2009) (The New York Times).
126. ExxonMobil. (2009). *Citizenship for the Long Term* (Advertorial, 29 June 2009) (The New York Times).
127. ExxonMobil. (2008). *Energy Efficiency – One Quart at a Time* (Advertorial) (The New York Times).
128. Mobil. (1997). *Climate Change: A Degree of Uncertainty* (Advertorial) (The New York Times).
129. ExxonMobil. (2006). *Changing the Game* (Advertorial) (The New York Times).
130. ExxonMobil. (2001). *To a Sounder Climate Policy* (Advertorial) (The New York Times).
131. Mobil. (1999). *Helping Earth Breathe Easier* (Advertorial) (The New York Times).
132. Mobil. (1999). *Lessons Learned* (Advertorial) (The New York Times).
133. ExxonMobil. (2001). *Renewable Energy: Tomorrow's Promise* (Advertorial) (The New York Times).
134. ExxonMobil. (2000). *Facts and Fundamentals* (Advertorial) (The New York Times).
135. ExxonMobil. (2007). *Addressing the Risks of Climate Change* (Advertorial) (The New York Times).
136. Flannery, B.P., Callegari, A.J., Nair, B., and Roberge, W.G. (1984). *The Fate of CO<sub>2</sub> from the Natuna Gas Project if Disposed by Subsea Sparging* (Internal Document).
137. Hoffert, M.I., Caldeira, K., Benford, G., Criswell, D.R., Green, C., Herzog, H., Jain, A.K., Kheshgi, H.S., Lackner, K.S., Lewis, J.S., et al. (2002). Advanced technology paths to global climate stability: energy for a greenhouse planet. *Science* 298, 981–988.
138. Mastracchio, R.L. (1979). *Controlling Atmospheric CO<sub>2</sub>* (Internal Document).
139. Albritton, D.L., Allen, M.R., Alfons, P.M., Baede, J.A., Church, U.C., Xiaosu, D., Yihui, D., Ehalt, D.H., Folland, C.K., Giorgi, F., et al. (2001). *Climate Change 2001: The Scientific Basis, Summary for Policymakers. Contribution of Working Group I to the Third Assessment Report of the Intergovernmental Panel on Climate Change.*
140. Cohen, R.W., and Levine, D.G. (1982). *Untitled (Consensus on CO<sub>2</sub> Letter)* (Internal Document).
141. Burgers, W.F.J., Northrop, P.S., Kheshgi, H.S., and Valencia, J.A. (2011). Worldwide development potential for sour gas. *Energy Proced.* 4, 2178–2184.
142. Black, J. (1978). *The Greenhouse Effect* (Internal Document).
143. Hayhoe, K., Kheshgi, H.S., Jain, A.K., and Wuebbles, D.J. (2002). Substitution of natural gas for coal: climatic effects of utility sector emissions. *Clim. Change* 54, 107–139.
144. Archer, D., Kheshgi, H., and Maier-reimer, E. (1998). Dynamics of fossil fuel CO<sub>2</sub> neutralization by marine CaCO<sub>3</sub>. *Glob. Biogeochem. Cycles* 12, 259–276.
145. Jain, A.K., Kheshgi, H.S., and Wuebbles, D.J. (1994). *Integrated science model for assessment of climate change. In 87th Annual Meeting and Exhibition of the Air and Waste Management Association (94-TP59.08).*
146. Zinn, J.O., and McDonald, D. (2018). *Risk in the New York Times (1987–2014) - A Corpus-Based Exploration of Sociological Theories* (Palgrave Macmillan).
147. ExxonMobil. (2000). *Do No Harm* (Advertorial) (The New York Times).
148. ExxonMobil. (2009). *Tackling Climate Risks with Technology* (Advertorial) (The New York Times).
149. ExxonMobil. (2008). *Next-generation Energy* (Advertorial) (The New York Times).
150. ExxonMobil. (2009). *Provide Energy. Protect the Environment. A Dual Challenge for All of Us.* (Advertorial) (The New York Times).
151. ExxonMobil. (2009). *Many Parts Working Together - the Only Way to Solve the World's Energy Challenges* (Advertorial) (The New York Times).
152. Mobil. (1998). *Voluntary "Can Do"* (Advertorial) (The New York Times).
153. ExxonMobil. (2007). *Saving Energy and Reducing Greenhouse Gas Emissions* (Advertorial) (The New York Times).
154. ExxonMobil. (2002). *A Responsible Path Forward on Climate* (Advertorial) (The New York Times).
155. ExxonMobil. (2004). *Directions for Climate Research* (Advertorial) (The New York Times).
156. ExxonMobil. (2007). *Answering Energy Questions* (Advertorial) (The New York Times).
157. ExxonMobil. (2005). *More Energy and Lower Emissions?* (Advertorial, 14 June 2005) (The New York Times).
158. ExxonMobil. (2005). *More Energy and Lower Emissions?* (Advertorial, 7 July 2005) (The New York Times).
159. ExxonMobil. (2005). *More Energy and Lower Emissions?* (Advertorial, 11 May 2005) (The New York Times).
160. ExxonMobil. (2001). *Renewable Energy: Today's Basics* (Advertorial) (The New York Times).
161. ExxonMobil. (2007). *Let's Talk about Climate Change* (Advertorial, 14 February 2007) (The New York Times).
162. ExxonMobil. (2007). *Let's Talk about Climate Change* (Advertorial, 16 February 2007) (The New York Times).
163. Brandt, A. (2007). *The Cigarette Century: The Rise, Fall, and Deadly Persistence of the Product that Defined America* (Basic Books).
164. Proctor, R.N. (2006). "Everyone knew but no one had proof": tobacco industry use of medical history expertise in US courts, 1990–2002. *Tob. Control* 15, 117–125.
165. Proctor, R.N. (2011). *Golden Holocaust - Origins of the Cigarette Catastrophe and the Case for Abolition* (University of California Press).
166. Fillmore, C.J., and Atkins, B.T. (1992). *Towards a frame-based lexicon: the semantics of RISK and its neighbors. In Frames, Fields and Contrasts: New Essays in Semantic and Lexicon Organization*, Adrienne Lehrer, Eva Feder Kittay, and Richard Lehrer, eds. (Routledge), pp. 75–102.
167. Zinn, J.O. (2010). *Risk as discourse: Interdisciplinary perspectives. Critical Approaches to Discourse Analysis Across Disciplines* 4, 106–124.
168. Weber, E.U. (2006). Experience-based and description-based perceptions of long-term risk: why global warming does not scare us (yet). *Clim. Change* 77, 103–120.
169. Aven, T., and Renn, O. (2009). On risk defined as an event where the outcome is uncertain. *J. Risk Res.* 12, 1–11.

170. Mythen, G. (2004). *Ulrich Beck: A Critical Introduction to the Risk Society* (Pluto Press).
171. Barbara, A., and van Loon, J. (2000). Introduction: Repositioning risk; the challenge for social theory. In *The Risk Society and Beyond: Critical Issues for Social Theory*, B. Adam, U. Beck, and J. van Loon, eds. (SAGE Publications), pp. 1–32.
172. Eisenberg, E.M. (1984). Ambiguity as strategy in organizational communication. *Commun. Monogr.* 51, 227–242.
173. Painter, J. (2013). *Climate Change in the Media: Reporting Risk and Uncertainty* (I.B. Tauris).
174. Daniel, K.D., Litterman, R.B., and Wagner, G. (2019). Declining CO<sub>2</sub> price paths. *Proc. Natl. Acad. Sci. U S A* 116, 20886–20891.
175. Mobil. (1989). *People Who Live in greenhouses...* (Advertorial) (The New York Times).
176. ExxonMobil. (2000). *Unsettled Science* (Advertorial) (The New York Times).
177. ExxonMobil (2005). *2005 Corporate Citizenship Report*.
178. Kim, B.S., Carvalho, I., and Davis, A.G. (2010). Talking about poverty: news framing of who is responsible for causing and fixing the problem. *J. Mass Commun. Q.* 87, 563–581.
179. Kim, S.-H. (2015). Who is responsible for a social problem? News framing and attribution of responsibility. *J. Mass Commun. Q.* 92, 554–558.
180. Weiner, B. (1995). *Judgments of Responsibility: A Foundation for a Theory of Social Conduct* (Guilford Press).
181. Parenti, M. (1986). *Inventing Reality: The Politics of Mass Media* (St. Martin's Press).
182. Brandt, A.M. (2012). Inventing conflicts of interest: a history of tobacco industry tactics. *Am. J. Public Health* 102, 63–71.
183. Daynard, R.A., and Gottlieb, M. (2000).  *Casting Blame on the Tobacco Victim: Impact on Assumption of the Risk and Related Defenses in the United States Tobacco Litigation* (Norwegian Ministry of Health and Care Services). <https://perma.cc/3HHT-45AA>.
184. Chapman, S. (2002). Blaming tobacco's victims. *Tob. Control* 11, 167–168.
185. Schmertz, H. (1986). *Good-bye to the Low Profile - the Art of Creative Confrontation* (Little, Brown and Company).
186. Mobil (1980). *Beware! Beware!* ("Observations" Advertorial). *Parade*, the New York Sunday News, and Other Sunday Supplements.
187. Feinstein, A.R. (1988). Scientific standards in epidemiologic studies of the menace of daily life. *Science* 242, 1257–1263.
188. Mobil. (1996). *Climate Change: We're All in This Together* (Advertorial) (The New York Times).
189. (2018). *City of oakland v. BPP L.C. (18-16663) Transcript of proceedings, 21 march 2018*. <https://perma.cc/EJ4Y-HDQV>.
190. Alsup, W. (2018). *Order Granting Motion to Dismiss Amended Complaints, US District Court for the Northern District of California* (Judge William Alsup). <https://perma.cc/F9BJ-5CX6>.
191. ExxonMobil. (2002). *Managing Greenhouse Gas Emissions* (Advertorial) (The New York Times).
192. Baker, T. (2002). Risk, insurance, and the social construction of responsibility. In *Embracing Risk - The Changing Culture of Insurance and Responsibility*, T. Baker and J. Simon, eds. (University of Chicago Press), pp. 33–51.
193. Ayling, J. (2017). A contest for legitimacy: the divestment movement and the fossil fuel. *L. Policy* 39, 349–371.
194. Chevron. (2019). *Climate Change*. chevron.com. <https://perma.cc/6H9L-N4GE>.
195. ConocoPhillips. (2019). *Managing Climate-Related Risks*. conocophillips.com. <https://perma.cc/QSP7-DEVF>.
196. St. John, B., III (2017). *Public Relations and the Corporate Persona: The Rise of the Affinitive Organization* (Routledge).
197. Maniates, M.F. (2001). Individualization: plant a tree, buy a bike, save the world? *Glob. Environ. Polit.* 1, 31.
198. de Tocqueville, A. (2000). *Democracy in America* (University of Chicago Press), H.C. Mansfield and D. Winthrop, translators.
199. Glendon, M.A. (1993). *Rights Talk: The Impoverishment of Political Discourse* (Free Press).
200. Lipset, S.M. (1996). *American Exceptionalism: A Double-Edged Sword* (W. W. Norton & Compan).
201. Brulle, R.J. (2020). Denialism: organized opposition to climate change action in the United States. In *Handbook of Environmental Policy*, D. Konisky, ed. (Edward Elgar Publishing), pp. 328–341.
202. Harvey, D. (2006). Neo-liberalism as creative destruction. *Geogr. Ann. Ser. B, Hum. Geogr.* 88, 145–158.
203. Hacker, J.S. (2006). *The Great Risk Shift: The Assault on American Jobs, Families, Health Care, and Retirement - and How You Can Fight Back* (Oxford University Press).
204. Lupton, D. (2013). *Risk* (Routledge).
205. Bialostok, S. (2015). Risk theory and education: policy and practice. *Policy Futur. Educ.* 13, 561–576.
206. Beck, U. (1992). *Risk Society: Towards a New Modernity*. Mark Ritter (Translation) (SAGE).
207. Giddens, A. (1999). Risk and responsibility. *Mod. L. Rev.* 62, 1–10.
208. Douglas, M., and Wildavsky, A. (1983). *Risk and Culture: An Essay on the Selection of Technological and Environmental Dangers* (University of California Press).
209. Foucault, M. (1991). Governmentality. In *The Foucault Effect: Studies in Governmentality*, G. Burchell, C. Gordon, and P. Miller, eds. (Harvester Wheatsheaf), pp. 87–104.
210. Rose, N., O'Malley, P., and Valverde, M. (2006). Governmentality. *Annu. Rev. L. Soc. Sci.* 2, 83–104.
211. Beck, U., and Beck-Gernsheim, E. (2002). *Individualization: Institutionalized Individualism and its Social and Political Consequences* (SAGE Publications Ltd.).
212. R.N. Proctor, and L. Schiebinger, eds. (2008). *Agnology - The Making and Unmaking of Ignorance* (Stanford University Press).
213. Proctor, R.N. (1995). *Cancer Wars: How Politics Shapes what We Know and Don't Know about Cancer* (Basic Books).
214. Michaels, D. (2020). *The Triumph of Doubt* (Oxford University Press).
215. St. John, B., III (2014). *The National Association of Manufacturers' community relations short film Your Town: Parable, propaganda, and big individualism*. *J. Public Relations Res.* 26, 103–116.
216. Dunaway, F. (2015). *Seeing Green: The Use and Abuse of American Environmental Images* (University of Chicago Press).
217. Beder, S. (2002). *Global Spin: The Corporate Assault on Environmentalism* (Chelsea Green Publishing Company).
218. Rogers, H. (2013). *Gone Tomorrow: The Hidden Life of Garbage* (The New Press).
219. Melillo, W. (2013). *How McGruff and the Crying Indian Changed America: A History of Iconic Ad* (Smithsonian Books).
220. Lerner, S. (2019). *Waste Only - How the Plastics Industry Is Fighting to Keep Polluting the World* (The Intercept). <https://perma.cc/T6PE-S8K6>.
221. Buranyi, S. (2018). *The Plastic Backlash: What's behind Our Sudden Rage - and Will it Make a Difference?* (The Guardian). <https://perma.cc/43U7-DKZM>.
222. Sullivan, L. (2020). *Plastic Wars: Industry Spent Millions Selling Recycling - to Sell More Plastic* (NPR/Frontline). <https://perma.cc/52E5-4V7S>.
223. Markowitz, G., and Rosner, D. (2002). *Deceit and Denial: The Deadly Politics of Industrial Pollution*. (University of California Press).
224. Markowitz, G., and Rosner, D. (2013). *Lead Wars: The Politics of Science and the Fate of America's Children* (University of California Press).
225. Hathaway, T. (2018). *Corporate power beyond the political arena: the case of the 'big three' and CAFE standards*. *Bus. Polit.* 20, 1–37.

226. Jahiel, R.I., and Babor, T.F. (2007). Industrial epidemics, public health advocacy and the alcohol industry: lessons from other fields. *Addiction* 102, 1335–1339.
227. Schüll, N.D. (2012). *Addiction by Design: Machine Gambling in Las Vegas* (Princeton University Press).
228. Hemenway, D. (2006). *Private Guns, Public Health* (University of Michigan Press).
229. Safire, W. (2008). On Language: Footprint. *The New York Times*. <https://perma.cc/U8QC-RR22>.
230. Solman, G. (2008). BP: Coloring Public Opinion? *Adweek*. <https://perma.cc/DF67-UCXG>.
231. BP. (2006). Carbon Footprint Calculator. *bp.com*. <https://perma.cc/3W2X-B9R8>.
232. BP. (2005). What on Earth Is a Carbon Footprint? (Advertisement) (*The New York Times*).
233. BP. (2005). Reduce Your Carbon Footprint. But First, Find Out what it is. (Advertisement) (*The New York Times*).
234. Miller, D. (2005). Ogilvy & Mather: BP Corporate Portfolio. <https://perma.cc/X8CG-87DN>.
235. BP television advertisement (2003). What size is your carbon footprint?. <https://perma.cc/8GHT-6TC6>.
236. Doyle, J. (2011). Where has all the oil gone? BP branding and the discursive elimination of climate change risk. In *Culture, Environment and Eco-Politics*, N. Heffernan and D.A. Wragg, eds. (Cambridge Scholars Publishing), pp. 200–225.
237. BP StudioSix (2020). Know Your Carbon Footprint. <https://perma.cc/P7PH-QLLN>.
238. Atkin, E. (2020). A line-by-line response to Fred Hiatt's pro-oil, anti-Sanders climate op-ed. *HEATED* newsletter. <https://perma.cc/6UP6-MDMN>.
239. Coffin, M. (2020). Absolute Impact: Why Oil Majors' Climate Ambitions Fall Short of Paris Limits (Carbon Tracker Initiative). <https://perma.cc/5UMM-A889>.
240. Palm, R., Bolsen, T., and Kingsland, J.T. (2020). 'Don't tell me what to do': Resistance to climate change messages suggesting behavior changes. *Weather Clim. Soc.* 1–29.
241. Climate change (Yale University Investments Office) (accessed 10 September 2020) (<https://perma.cc/9UVU-YK8F>).
242. Siegel, J. (2020). How House Republicans Won over Conservatives to Gain Consensus on a Climate Agenda (*Washington Examiner*). <https://perma.cc/376Z-5N87>.
243. Roberts, D. (2020). New Conservative Climate Plans Are Neither Conservative Nor Climate Plans (*Vox.com*). <https://perma.cc/WG43-8GU5>.
244. Seto, K.C., Davis, S.J., Mitchell, R.B., Stokes, E.C., Unruh, G., and Urge-Vorsatz, D. (2016). Carbon lock-in: Types, causes, and policy implications. *Annu. Rev. Environ. Resour.* 41, 425–452.
245. Erickson, P., van Asselt, H., Koplrow, D., Lazarus, M., Newell, P., Oreskes, N., and Supran, G. (2020). Why fossil fuel producer subsidies matter. *Nature* 578, E1–E4.
246. Lazarus, M., and van Asselt, H. (2018). Climatic change fossil fuel supply and climate policy: Exploring the road less taken. *Clim. Change*.
247. Inkpen, D., and Hirst, G. (2006). Building and using a lexical knowledge base of near-synonym differences. *Comput. Linguist.* 32, 223–262.
248. Denny, M.J., and Spirling, A. (2018). Text preprocessing for unsupervised learning: Why it matters, when it misleads, and what to do about it. *Polit. Anal.* 26, 168–189.
249. Aggarwal, C.C. (2012). In *Mining Text Data*, C. Zhai, ed. (Springer).
250. Kilgariff, A. (2001). Comparing corpora. *Int. J. Corpus Linguist.* 6, 97–133.
251. Rayson, P., and Garside, R. (2000). Comparing corpora using frequency profiling. In *Proceedings of the Workshop on Comparing Corpora*, Adam Kilgariff and Tony Berber Sardinha, eds. (Association for Computational Linguistics), pp. 1–6.
252. Lin, C., and Hovy, E. (2000). The Automated Acquisition of Topic Signatures for Text Summarization. In *Proceedings of the 18th Conference on Computational linguistics*, pp. 495–501.
253. Maier, D., Waldherr, A., Miltner, P., Wiedemann, G., Niekler, A., Keinert, A., Pfetsch, B., Heyer, G., Reber, U., Häussler, T., et al. (2018). Applying LDA topic modeling in communication research: toward a valid and reliable methodology. *Commun. Methods Meas.* 12, 93–118.
254. Strauss, A.L., and Corbin, J. (1990). *Basics of Qualitative Research: Grounded Theory Procedures and Techniques* (SAGE).
255. Gablasova, D., Brezina, V., and McEnery, T. (2017). Collocations in corpus-based language learning research: identifying, comparing, and interpreting the evidence. *Lang. Learn.* 67, 155–179.
256. Rychlý, P. (2008). A lexicographer-friendly association score. In *Proceedings of Recent Advances in Slavonic Natural Language Processing (RASLAN)*, pp. 6–9.

**One Earth, Volume 4**

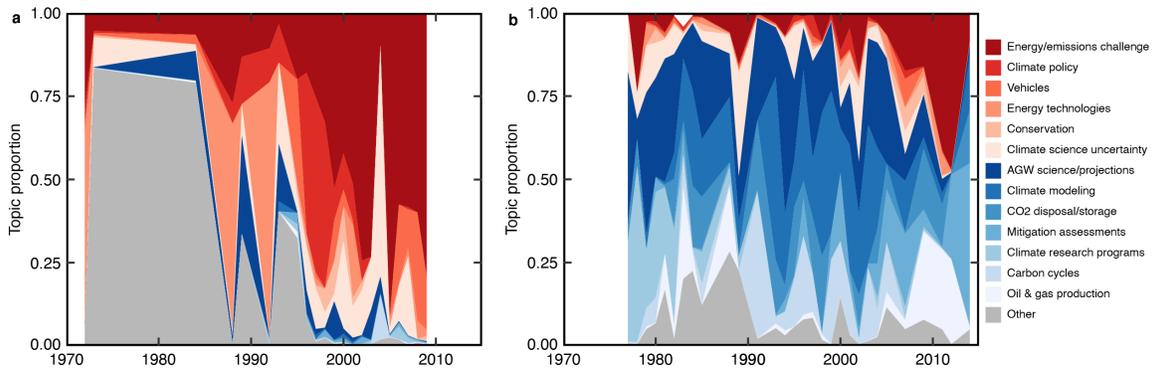
**Supplemental information**

**Rhetoric and frame analysis of ExxonMobil's  
climate change communications**

**Geoffrey Supran and Naomi Oreskes**

# SUPPLEMENTAL INFORMATION

## SUPPLEMENTAL FIGURES



**Figure S1.** Topic prevalence over time in (a) advertorials and (b) internal and peer-reviewed publications. Topic proportions are calculated as the normalized sum of LDA per-document ( $d$ )-per-topic ( $k$ ) probabilities ( $\theta_{d,k}$ ) of all documents published each year. Note that, as documented in table S20, our corpus comprises only three advertorials published prior to 1988.

## SUPPLEMENTAL TABLES

**Table S1.** Fossil fuel industry AGW discourses, based on a meta-analysis of existing academic literature. In some cases, discourses from individual studies straddle two or more discourses in our classification.

Author	Rowlands (2000) <sup>38</sup>	Livesey (2002) <sup>39</sup>	Smerecnik & Renegar (2010) <sup>40</sup>	Doyle (2011) <sup>41</sup>	Plec & Pettenger (2012) <sup>42</sup>	Schlichting (2013) <sup>29</sup>
Corpus	Exxon and BP's public statements on AGW	Four advertorials in The New York Times	BP's "Helios Power" campaign	BP advertising campaigns	ExxonMobil's "Energy Solutions" TV advertisements	38 studies on industry actors' AGW communications
Time period	Unspecified (~1997-2000)	March-April 2000	2007	2005-06	2009, 2011	1990-2010
Focus	Positions of Exxon and BP Amoco on AGW	How ExxonMobil's public discourses construct social "reality"	Discourses in green marketing	Discursive strategies to create an environmental brand image	(Didactic) frames and discourses in green marketing	Strategic frames of industry actors
Analytic method	Review (not specifically defined)	Rhetorical and discourse analyses	Rhetorical analysis	Discourse analysis	Frame analysis	Frame meta-analysis
<b>Discourses</b>						
Climate Risk				"Risk" rhetoric channelling Beck's risk society. AGW as a future event rather than a present reality.		"Climate change might be/is a risk"
Doubt Mongering	Scientific uncertainty	Demonize most climate scientists		Scientific doubt-mongering		"Scientific uncertainty"
Free-Market Solutionism	Support "voluntary market-driven efforts"	Primacy of "the market", private sector, and economists.	Individual, capitalistic agency	Late capitalism economic discourse: global capitalism equals expanding global environmental good.		
Individualized Responsibility		Governments sidelined, regulatory controls rejected  Re-constitute citizen as consumer ExxonMobil as responsible citizen ExxonMobil as vulnerable human entity in complex natural scene		Citizen as consumer. Blame on consumers for not buying BP's ostensibly environmentally friendly products. Responsibility for combating AGW placed on individual consumer.		"Industry is responsible for the climate. Consumers must also take responsibility".
Energy Poverty/Prosperity		"Lifestyle" protection		Prioritization of human needs and economic growth over the environment		
Energy Utopia			"Utopian fantasy world where fossil fuel-based transportation and a clean environment are harmoniously united"			
Fossil Fuel Solutionism		Responsible corporate actor pursuing "prudent", rationalist approach		BP as solution, rather than contributor, to AGW. Environmental leadership: Highlighting progressive and green values and investments.		"Fossil energy sources can be used sustainably"
Greenwashing/Corporate Symbolic Environmentalism	Reducing scope-1 GHG emissions				Alternative energy leader/expert solving environmental problems; Green energy	"Industrial leadership": "Corporate achievements in climate protection"; Green "visionaries"
Scientific/Technological Optimism	Support "continued research"	Scientific powerhouse and technological leader. Entrepreneurship and technology will provide solutions.		Technology as the solution	Technocratism: Scientific & technological solutions; authoritarian values	"Technological innovations are the solution"
Policy Apocalypse	Socioeconomic harm of "premature" climate policies (e.g. Kyoto Protocol)					"Socioeconomic consequences"
Technological Shell-Game				"Clean" natural gas presented as equivalent to renewable energy		
Whataboutism	Developing countries must participate in climate policies					
Other						

**Table S1, continued.**

Author	Robinson (2014) <sup>43</sup>	Gaither & Gaither (2016) <sup>44</sup>	Schneider <i>et al.</i> (2016) <sup>45</sup>	Cahill (2017) <sup>46</sup>	Ayling (2017) <sup>47</sup>
Corpus	Marketing campaigns of oil majors	Advertisements on APCCCE (coal) and API (petroleum) trade group websites	Five US coal industry corporate advocacy campaign case studies	Corporate websites, blogs, and social media channels of five oil and gas majors	Australian coal industry (Minerals Council of Australia) statements
Time period	N/A (Case studies span ~1998-present)	Spring 2014	N/A (Case studies span ~2008-present)	2016	2013-16
Focus	Brand lessons from oil industry image marketing campaign case studies	Discourses in trade group marketplace advocacy	Rhetorical strategies of US coal industry	Discourse and framing by oil and gas companies	Coal industry discourse in response to fossil fuel divestment activism
Analytic method	Review (not specifically defined)	Circuit of culture discourse analysis	Critical approaches from environmental communication, rhetoric, cultural studies	Critical discourse analysis	Content analysis
<b>Discourses</b>					
Climate Risk				"Risk management lens that downplays the material impact of climate change while foregrounding the economic impacts of mitigation"	
Doubt Mongering			"Corporate ventriloquism": "corporations transmit messages through other entities, usually of their own making, in order to construct and animate an alternative ethos, voice, or identity that advances their interests".		
Free-Market Solutionism				"Free markets = fair and efficient solutions"	
Individualized Responsibility	Employees as global citizenry present corporations as citizens		"Hypocrite's trap": "set of interrelated arguments that attempts to disarm critics of industries...based on the critics' own consumption of or reliance on those goods".	"The world needs more energy (increasing energy demand inevitable)". "Corporations as citizens vs Citizens as consumers".	Divestment activists are "hypocritical"
Energy Poverty/Prosperity			"Energy utopia": "particular energy source as the key to providing a "good life" that transcends the conflicts of environment, justice, and politics".	"Supplying energy is a humanitarian project".	"Contribution to the Australian community through exports, wages, jobs, investment, taxes, and royalties, as well as its provision of reliable and affordable electricity for Australian households and businesses".  "Concern for the overseas poor".
Energy Utopia					
Fossil Fuel Solutionism				"Fossil fuels must continue to play an integral role in the global economy for the foreseeable future".	Coal is "essential to Australia's past and future development"
Greenwashing/Corporate Symbolic Environmentalism	Green rebranding: showcase investments in clean energy, climate research; conservation grants; scope-1 GHG emissions reductions			"Increasing efficiency and innovating new technologies". "Scientific knowledge and technical expertise".	"Support for indigenous youth through employment opportunities"
Scientific/Technological Optimism					Innovation: "progress is being made on carbon capture and storage (CCS) and new-generation technologies"
Policy Apocalypse		Industry supporter (America's everyman/everywoman) adversely impacted by environmental regulations. Industry as paternal caretaker for American citizens, under threat by regulation.	"Industrial apocalyptic": "imminent demise of a particular industry, economic, or political system and the catastrophic ramifications associated with that loss".		"Lack of support [for industry] will result in job losses, higher electricity bills, and loss of government revenues"
Technological Shell-Game	Natural gas as "climate-friendly"		"Technological shell game": "misdirection that relies on strategic ambiguity about the feasibility, costs, and successful implementation of technologies in order to deflect attention from environmental pollution and health concerns".	"Renewable energy is expensive and unreliable". "Natural gas is the new coal".	Australian coal "is the cleanest coal in the world"
Whataboutism					
Other					

**Table S1, continued.**

Author	Scanlan (2017) <sup>48</sup>	Grantham & Vieira Jr. (2018) <sup>49</sup>	Jaworska (2018) <sup>25</sup>	Lamb <i>et al.</i> (2020) <sup>50</sup>
Corpus	Oil and gas industry advertisements	12 CEO/President welcome letters	Corporate social responsibility and environmental reports of major oil companies	N/A (Theorized taxonomy of discourses of climate delay)
Time period	2000-15	2002 to 2013	2000-13	N/A
Focus	Frames in industry rhetoric on fracking	ExxonMobil's social responsibility communication	Discourses in corporate social responsibility	Discourses of climate delay
Analytic method	Content analysis	Text network analysis	Corpus-linguistic and discourse analyses	Expert elicitation
<b>Discourses</b>				
Climate Risk		"Planet" theme introduces keyword of "risk"	Industry as victim of unpredictable climate "risk"	
Doubt Mongering			Scientific doubt-mongering	
Free-Market Solutionism				"No sticks, just carrots": "we should only pursue voluntary policies ('carrots'), in particular those that expand consumer choices"
Individualized Responsibility			Differentiation: shifting responsibility to other stakeholders (consumers, governments)	"Individualism": "redirects climate action from systemic solutions to individual actions"
Energy Poverty/Prosperity	Natural gas offers "economic development and jobs"; "energy independence and security"		Downplay AGW urgency by foregrounding the economy and energy demand	"Appeal to social justice": "moves social impacts to the forefront of policy discussions, framing a transition to renewable energy as burdensome and costly to society"
Energy Utopia				
Fossil Fuel Solutionism			Non-radical changes proposed	"Fossil fuel solutionism": "the fossil fuel industry is "part of the solution to the scourge of climate change""
Greenwashing/Corporate Symbolic Environmentalism			Industry as technological leader of breakthrough solutions. Enthusiasm for breakthrough technological solutions.	"All talk, little action": "points to recent advances in lowering emissions or in setting ambitious climate targets, thus downplaying the need for more stringent or new types of additional action"
Scientific/Technological Optimism	"Faith in science and American ingenuity"			"Technological optimism": "technological progress will rapidly bring about emissions reductions in the future"
Policy Apocalypse				"Appeal to well-being": "climate policy threatens fundamental livelihoods and living standards"
Technological Shell-Game	Natural gas offers "environmental protection and sustainability"			
Whataboutism				"Whataboutism": "Actors [point to] their own small contribution to global emissions"
Other				"Free rider" excuse: "others will actively take advantage of those who lead on climate change mitigation".  "Policy perfectionism": "argues for disproportional caution in setting ambitious levels of climate policy in order not to lose public support".  "Change is impossible": "Reifies the current state of things and denies the ability of societies to organize large socio-economic transformations".  "Doomism": "any actions we take are too little, too late. Catastrophic climate change is already locked-in"

## **S1. SUPPLEMENTAL EXPERIMENTAL PROCEDURES**

### **S1.1. Corpora**

The 180 ExxonMobil documents analyzed in this study were previously compiled in refs. <sup>1,2</sup>. One 1989 advertisement, however, was here omitted because, as noted in ref. <sup>2</sup>, it is not in fact an advertorial, but an advertisement in *The New York Times Magazine* that may or may not have actually included Exxon among its industry sponsors<sup>3</sup>.

Unlike advertorials in the *NYT*, peer-reviewed publications disclosed by ExxonMobil Corp, and internal documents recovered to date, all three of which are bound sets, ‘non-peer-reviewed’ documents analyzed in our original study are virtually limitless in potential number and scope and so are excluded in this study. Indeed, as noted in ref. <sup>1</sup>, there are countless additional climate change communications from ExxonMobil that could be included in future work, including as yet undiscovered internal documents, advertorials and advertisements published in outlets beyond the *NYT*, and non-peer-reviewed materials such as speech transcripts, television advertisements, social media posts, patent documents, shareholder reports, and third-party communications (for example, from lobbyists, think-tanks, and politicians funded by ExxonMobil). These documents are potentially important, but are not the focus of the present study.

See section S6, however, for algorithmic analysis of all ExxonMobil Corp flagship reports concerning AGW.

### **S1.2. Pre-processing**

To enable computational analysis in *R*, scanned documents were converted to searchable text files using *Readiris Corporate 17* optical character recognition (OCR) software<sup>4,5</sup>. We then used regular expression search algorithms and manual cleaning to strip out formatting details such as boilerplate archive timestamps and copyright statements; column breaks and whitespaces; author, journal, and publisher information; publication dates; and page numbers. Bibliographies, contents pages, disclosure and acknowledgment statements, appendices, and forewords (unless written by ExxonMobil representatives) were also removed from internal and peer-reviewed documents. In the case of advertorials, company logos and graphics (except for pullout quotations) were removed. Spellcheck was used to identify and correct common OCR-generated errors.

We did not use a stemmer or lemmatiser to reduce related words to their base forms, but we added several synthetic tokens that combine terms of similar cognate form (e.g. “co2” and “carbon dioxide” became “co2/carbon dioxide”; “effect” and “effects” became “effect(s)”).

For divergent term analysis (section 2.3), stopwords were not removed. For divergent topic analysis (section 2.4), stopwords were removed, after which word counts of internal and peer-reviewed corpora were respectively scaled down – by randomly sampling the same fraction of words from each document of each corpus – to match one another and to collectively match the word count of advertorials.

Only terms appearing at least 10 times in a corpus were included in document-term matrices.

### **S1.3 Topic Modeling**

#### *S1.3.1 Model selection*

LDA topic modeling is performed using the *R* ‘topicmodels’ package by Grün and Hornik (2011)<sup>6,7</sup>. The units of analysis were individual words. These words were itemized for LDA into ‘documents’ (as defined by Maier *et al.* (2018)) comprising the original 180 articles<sup>8</sup>. As prescribed by Maier *et al.*, hyperparameter  $\alpha$  {0.001, 0.005, 0.01, 0.05, 0.1, 0.5, 1} was optimized

by maximizing *intrinsic topic coherence* (as defined by Mimno *et al.* (2011)) for fixed  $\beta = 1/K$  and for a range of  $K$  values  $\{10, 11, \dots, 30\}$ <sup>8-10</sup>. For each value of  $K$ , models corresponding to the two top-scoring  $\alpha$  values were retained. The most appropriate model was then selected based on intersubjective qualitative author judgment, using what Maier *et al.* (2018) term a *substantive search in coherence-optimized candidates*<sup>8</sup>. This involved assessing the interpretability and relative efficacy of the optimized models for each value of  $K$  (and two  $\alpha$  values) in terms of (a) per-term-per-topic probability distributions ( $\phi_{w,k}$ ) and (b) reordered lists of the top words assigned to each topic using Sievert and Shirley (2014)'s *relevance* metric<sup>11</sup>. Models with  $K < 15$  led topics to blur together, while  $K > 20$  yielded diminishing returns due to excessive granularity. Final parameters based on this recursive process were  $K = 16$ ,  $\alpha = 0.1$ ,  $\beta = 1/K = 10$ .

### SI.3.2 Topic validation and labeling

The semantics of each topic solution were examined on the basis of (a) authors' expert knowledge about climate (denial) communications and familiarity with the documents; (b) four metrics proposed by Maier *et al.* (2018): (i) *Rank-1*, which counts how many times each topic is the most prevalent in a document; (ii) *intrinsic coherence* of individual topics<sup>10</sup>; (iii) *relevance* (with weighting  $\lambda=0.6$ ), which accounts for both per-term-per-topic probabilities ( $\phi_{w,k}$ ) and the marginal probability of each term in the corpus ( $p_w$ )<sup>11</sup>; and (iv) *concentration* (Hirschman-Herfindahl Index), which measures the extent to which topics are spread across documents<sup>8</sup>; and (c) LL ratio, which, as previously introduced, quantifies the distinctiveness of topics in one sub-corpus versus the other. Accordingly, three “junk” topics were excluded owing to semantically incoherent word lists, and/or low *Rank-1*, and/or low *coherence*, and/or low LL ratio, and/or high *concentration*.

Remaining topics were validated by intra-topic and inter-topic semantic validity. To evaluate the former, for each topic, we read all documents with relatively large per-document ( $d$ )-per-topic ( $k$ ) probabilities  $\theta_{d,k} > 0.2$ , with particular attention to terms with high *relevance* scores and that are most exclusive to that topic<sup>8,12</sup>. The guiding questions in our readings were: (i) Is the topic semantically coherent – communicating a substantive theme consistent with the qualitative meaning of the texts?; and (ii) What label should be given to the topic to describe the theme most comprehensively? Table 4 in the main text presents these validated, manually labeled topics. (Note that due to the relatively small corpora under investigation, and, accordingly, a relatively small number of topics emergent from our LDA model, semantic validation based on algorithmic clustering of topics into higher-order themes is not applicable here<sup>8,9,13</sup>.)

Finally, following Boussalis and Coan (2016), we evaluated inter-topic semantic validity by comparing LDA model topic assignments against those identified by manual content analysis of a random sample of 72 documents (40% of all documents)<sup>9</sup>.

In the pilot phase of human coding, two coders – one author and a research assistant – independently coded 10 randomly selected documents. This involved assigning each document a primary topic of either: one of the 13 topics in table 4; or “other” if none of those LDA model-derived topics meaningfully captured the main theme. The coders then compared and discussed their coding choices. The coders then independently coded an additional 10 randomly selected documents and again reviewed their assignments. Finally, the coders independently coded another 36 randomly selected documents (20% of all documents); the results of this sample were used to calculate intercoder reliability in terms of percentage agreement (81%) and Krippendorff's  $\alpha$  coefficient (0.79) using ReCal2 online software<sup>14-16</sup>. Through “negotiated agreement” of discrepancies between coders, intercoder agreement was also calculated (89%;  $\alpha = 0.88$ )<sup>17</sup>.

Having achieved satisfactory intercoder reliability and agreement, one of the coders proceeded to code an additional 36 randomly selected documents. These results, combined with those of the previous 36 coded documents, yielded a sample of 72 randomly coded documents (40% of all documents, including: 47% of internal documents; 44% of peer-reviewed publications; and 33% of advertorials). This sample was then compared against our LDA model's assignments. We find the microaveraged precision and recall for primary topic classifications to be 0.59 and 0.60, respectively. Although these values are lower than common cutoffs of 0.7 to 0.8, they are comparable to those reported by Boussalis and Coan (2016) and are considerably better than rolling a 13-sided dice<sup>9,18</sup>.

Moreover, as Boussalis and Coan (2016) note, “assessing a topic model using only the primary topic offers a conservative estimate of performance. Several distinct themes often contribute to a document's composition and deciding which is ‘primary’ is often quite difficult for both human and machine. Indeed, allowing documents to be composed of multiple topics...is one of the major advantages of using the LDA”<sup>9</sup>. When we account for the two most probable topics identified by our LDA model, the proportion of documents correctly recalled rises to 0.74.

Figure S1 displays the relative prevalence of LDA-generated topics over time in (a) advertorials and (b) internal and peer-reviewed publications. Although, as noted in section S1.4.1, these trends fall short of a comprehensive longitudinal frame analysis and as such should be interpreted with caution, the topic proportions in fig. S1a nevertheless suggest some initial insights. We see, for example, that the topics of ‘Climate science uncertainty’ and ‘AGW science/projections’ are interwoven throughout both Mobil's advertorials in the 1990s and ExxonMobil Corp's advertorials in the 2000s. We also observe the strong emergence of the ‘Climate policy’ topic in the run up to and wake of the 1997 UN climate negotiations in Kyoto. Even more apparent is an ever-growing dominance of the ‘Energy/emissions challenge’ topic throughout the 2000s.

These trends are broadly consistent with (i) our observations during ‘frame package’ open-coding of shifts in the relative prevalence of ExxonMobil's public framing devices over time; (ii) our past codings of ExxonMobil's public positions on climate change over time (Supran and Oreskes (2017, 2020))<sup>1,2</sup>; and (iii) Schlichting (2013)'s observations of industry actors' shifting climate change “master frames” over time<sup>29</sup>. The trends we observe paint an overall picture of coevolving topics – and, by approximate extension, frames – whose center of mass has gradually shifted away from explicit attacks on science (represented by our Scientific Uncertainty frame) and towards subtler narratives about energy and emissions (represented by our Socioeconomic Threat and Fossil Fuel Savior frames).

### *S1.3.3 Log-likelihood ratios of topic weights*

We compute LL ratios of topic weights by constructing document-topic matrices for each of sub-corpora  $\alpha$  and  $\beta$ . In these matrices, we include only those topics whose weights correspond to  $\geq 1\%$  of each sub-corpus's total word count.

## **S1.4 ‘Frame package’ analysis**

As noted above, due to the relatively small corpora under investigation, a relatively small number of interpretable topics emerge from our LDA model. We therefore adopt a qualitative approach to inductive frame analysis rather than, for instance, algorithmically clustering topics into frames.

The units of analysis in our frame package analysis were individual advertorials. The unit of observation was the advertorial corpus.

#### *SI.4.1 Open-coding*

We conducted open-coding using *NVivo* digital annotation software, and used corpus linguistic tools to systematize the process in two ways<sup>19</sup>.

First, we used FS and LL as statistical methods for extracting central meanings and locating potential frames; and ran concordance searches to automatically collect text extracts for frame analysis<sup>20,21</sup>. Although we analyzed each advertorial in its entirety, this approach helped us identify the loci for frames. Touri and Koteyko (2015) have previously demonstrated the efficacy of combining LL analysis with the frame package approach in this way<sup>22</sup>. Indeed, this was a mutually reinforcing process in that open-coding aided contextual interpretation of how divergent terms identified by FS and LL analysis construct meanings.

Second, and in parallel, we used LDA analysis to extract topics that may generally be regarded as “frame elements [or] a full frame package, or...a combination of the two” (Walter and Ophir (2019))<sup>12</sup>. Just as divergent terms from FS and LL help extract central meanings and locate potential frames, “regularities of [word] co-occurrence” in topic models, write Klebanov *et al.* (2008), “are considered in some linguistic theories as the major building block for characterizing meaning; this idea is well expressed in the famous distributional hypothesis: “Know the word by the company it keeps”<sup>23</sup>. Thus, in order to further help detect frames shaped by lexical composition, we also ran concordance searches based on LDA top words to automatically collect text extracts for frame analysis. As Van Gorp (2010) notes, “[t]he intention of an inductive framing analysis is to reconstruct the frames that are useful to define a certain topic”<sup>24</sup>. We therefore open-coded together documents sharing similar LDA topic weightings, which tend to display recurring linguistic elements or framing/reasoning devices indicative of frame packages<sup>12,24</sup>.

We further investigated discursive constructs by performing collocation searches using the logDice statistic applied to corpora tokenized by sentence<sup>25-27</sup>.

Our inventory resulting from open-coding comprised manifest framing devices such as catchphrases, lexical choices, visual images, depictions, metaphors, and exemplars; and (often latent) reasoning devices in the form of apparent definitions of the AGW problem, assignments of responsibility for causing it and/or solving it, identifications of solutions, and moral assessments. As Entman, Matthes, and Pellicano (2009) note, a defining feature of a frame is that it “repeatedly invokes the same objects and traits, using identical or synonymous words and symbols...”<sup>28</sup>. The linguistic tools employed in this study are amenable to the detection of such cues, and therefore to the identification and differentiation of frames from other features such as themes, arguments, and assertions.

Digital annotation during open-coding allows us to code the dates of all entries in this inventory. Following Schlichting (2013), this offers insights into how ExxonMobil’s public frames have shifted over time<sup>29</sup>. The primary contribution of our inductive frame analysis, however, is its frame matrix, which may serve as the basis for a coding scheme in future quantitative, deductive, and fully longitudinal content analyses<sup>24,30</sup>.

#### *SI.4.2 Axial coding*

We codify our axial codings with reference to an inventory of discourses that we assembled based on an informal literature review of past studies of AGW communications by fossil fuel interests<sup>24</sup>. A summary of discourses identified by this literature review is provided in section S3.

## **S2. SUPPLEMENTAL DIVERGENT TERM ANALYSIS RESULTS**

### **S2.1. Mobil versus ExxonMobil Corp advertorials**

In section 2.1.1, we note that because both Mobil and ExxonMobil Corp advertorials often promoted doubt about climate science, terms conveying explicit doubt are common to both corpora and so do not appear in table 1. One example of this is the term “debate”, which appears  $n_{EM} = 9$  times in ExxonMobil Corp advertorials and  $n_M = 17$  times in Mobil advertorials. This corresponds to  $FS = 0.37$  and  $G_2 = -1.69$ , indicating statistically insignificant divergence ( $p = 0.24$  and  $0.19$ , respectively). Likewise, “uncertain(/ty/ties)” appears  $n_{EM} = 13$  and  $n_M = 18$  times, equivalent to  $FS = 0.44$  ( $p = 0.59$ ) and  $G_2 = -0.35$  ( $p = 0.55$ ). Other common terms displaying statistically insignificant divergence include “(un)know(/n/ing/ledge)”, “believe”, “compl(ex/exity/icated)”, “answer(s)”, etc.

### **S2.2. Advertorials versus internal and peer-reviewed documents**

In section 2.4, we observe that ExxonMobil’s advertorials statistically overuse terms that reduce AGW to a downstream problem caused by consumer energy demand. We here note that advertorials do, in fact, contain divergent terms of “oil and natural gas” (compared to internal and peer-reviewed publications – see tables 2 and 3) and “fossil fuels” (compared to peer-reviewed publications – see table 3). In the majority of cases, however, these terms are employed in discourses such as Energy Poverty/Prosperity (“Abundant and affordable, fossil fuels have contributed to unprecedented prosperity for much of the human race. In decades to come, the benefits of modern fossil fuel energy will extend even further”<sup>152</sup>); Policy Apocalypse (“World economic health will suffer as nations are forced to switch from fossil fuels...”<sup>181</sup>); and Greenwashing/Symbolic Corporate Environmentalism (“ExxonMobil is also leading the way in increasing safety and reducing marine spills in the oil and natural gas industry.”<sup>137,138</sup>). Such examples do not speak to the cause of – or accountability for – AGW or greenhouse gas emissions. If anything, they generally reinforce the narrative that fossil fuels passively satisfy demand; for example: “[F]ossil fuels...[f]or at least several decades, they will continue to be the major source of the world’s energy needs”<sup>128</sup>. The number of cases concerning responsibility for AGW or greenhouse gases is statistically insignificant even at  $p \leq 0.05$  levels (“oil and natural gas”: LL  $\sim 0.01$ , FS  $\sim 0.53$ ; “fossil fuels”: LL  $\sim 2.79$ , FS  $\sim 0.63$ ). Virtually all such cases appear in advertorials that simultaneously promote doubt about whether AGW is real and human-caused and/or serious and/or solvable.

### **S3. LITERATURE REVIEW OF FOSSIL FUEL INDUSTRY AGW DISCOURSES**

As noted in section S1.3.2, frame package analysis was guided by an informal literature review of existing studies of AGW communications by fossil fuel interests. Table S1 summarizes the results of this meta-analysis of contemporary (~1990–present) discourses. The scope of this review was limited to publications concerning AGW communications by fossil fuel producers. 15 such studies were investigated. For studies regarding discourses of climate denial and delay by a broader range of actors, such as conservative news media, columnists, think tanks, and other industries, see for example refs. <sup>9,13,31–36</sup>. For a review of AGW framing and discourse literature as a whole, see for example ref. <sup>37</sup>. For detailed taxonomies of Doubt Mongering discourse, as labeled in table S1, see ref. <sup>1</sup> and several of the foregoing references.

## S4. FRAME PACKAGE ANALYSIS RESULTS

The following are frame matrices summarizing framing and reasoning devices of each identified frame package.

### S4.1 Scientific Uncertainty Frame

**Table S2.** Frame matrix of Scientific Uncertainty frame package.

Scientific Uncertainty Frame Package		
Reasoning Devices	Description	
Problem	Global warming is unproven	
Cause	Global climate system is complex, science is unsettled	
Moral evaluation	We don't know enough	
Solutions	Wait for better climate science research	
Framing Devices	Discourse	Example/Description
Catchphrases & lexical choices	Climate Risk	"Risk(s) of climate change" "Longterm"
	Doubt Mongering	"Debate" "Gap(s)"
	Scientific/Technological Optimism	"Invest(ing/ment(s))" "Promise"
Visual images	Doubt Mongering	Graphs and charts
	Scientific/Technological Optimism	Graphs and charts
Exemplars	Doubt Mongering	Quotations of contrarian scientists (e.g. Heidelberg Appeal; S Fred Singer)
Depictions	Climate Risk	Amorphous "risk(s)" of AGW
	Scientific/Technological Optimism	Dynamic "breakthrough" university research collaborations
Metaphors	Doubt Mongering	"Weather and climate"; "Climate change: a degree of uncertainty"
Example discourse quotations		
Climate Risk	"[C]limate changes may pose long-term risks. Natural variability and human activity may lead to climate change that could be significant and perhaps both positive and negative." <sup>51</sup>	
Doubt Mongering	"Weather and climate. In the debate over climate change, there is an understandable tendency to use recent weather events to draw conclusions about global warming." <sup>52</sup>	
Scientific/Technological Optimism	"To address the scientific uncertainty, governments, universities and industry should form global research partnerships to fill in the knowledge gap, with the goal of achieving a consensus view within a defined time frame." <sup>53</sup>	

The Scientific Uncertainty frame presents AGW as unproven and, accordingly, advocates additional climate science research before any policy action is taken.

Central to this frame's problem definition and causal attribution is the discourse of 'Doubt Mongering', which promotes false scientific debate about whether AGW is real and human-caused. One example, a 2004 ExxonMobil Corp advertorial entitled "Weather and climate", argued that "In the debate over climate change, there is an understandable tendency to use recent weather events to draw conclusions about global warming"<sup>52</sup>. At work here are the key framing devices of catchphrases (such as "debate") and metaphors (such as "weather and climate"). The advertorial goes on to insist that "in the face of natural variability and complexity, the consequences of change in any single factor, for example greenhouse gases, cannot readily be isolated and prediction becomes difficult... scientific uncertainties continue to limit our ability to make objective, quantitative determinations regarding the human role in recent climate change or the degree and consequences of future change". Visual images (such as graphs and charts) and exemplars (such as quotations of the minority opinions of contrarian scientists) help falsely legitimize such claims.

Discourses of 'Scientific/Technological Optimism' and 'Climate Risk' help further the impression of scientific debate while simultaneously prescribing the moral evaluation that enough is not yet known to take any policy actions, and the solution of further scientific research. "To address the scientific uncertainty", reasons a 2007 advertorial, "governments, universities and industry should form global research partnerships to fill in the knowledge gap, with the goal of

achieving a consensus view within a defined time frame.”<sup>53</sup> Such Scientific/Technological Optimism repeatedly alleges “gap(s)” in scientific knowledge and emphasizes the “promise” of “breakthrough” research collaborations. The “risk” rhetoric that emerges in ExxonMobil Corp advertorials serves similar dual functions of presenting AGW as a risk rather than a reality and of thereby rationalizing research rather than policy action, as discussed in section 3.1. As a 2000 advertorial entitled “Unsettled Science” puts it, “[C]limate changes may pose long-term risks. Natural variability and human activity may lead to climate change that could be significant and perhaps both positive and negative.”<sup>51</sup> ExxonMobil Corp accordingly argue that “future scientific research will help understand how human actions and natural climate change may affect the world and will help determine what actions may be desirable to address the long-term”.

## S4.2 Socioeconomic Threat Frame

**Table S3.** Frame matrix of Socioeconomic Threat frame package.

Socioeconomic Threat Frame Package		
Reasoning Devices	Description	
Problem	Climate policy threatens prosperity	
Cause	Alarmist policy and politics are outrunning science	
Moral evaluation	Binding climate policies are unwarranted and economically dangerous	
Solutions	Voluntary efforts, especially energy efficiency Technology R&D No policy exemptions for developing countries	
Framing Devices	Discourse	Example/Description
Catchphrases & lexical choices	Climate Risk	"Risk(s) of climate change"
		"Longterm"
	Energy Poverty/Prosperity	"Developing/poorer countries/world/nations" "Affordable"
	Free-Market Solutionism	"Voluntary steps" "Free market"
	Policy Apocalypse	"Economic impact" "Jobs/employment"
	Scientific/Technological Optimism	"Develop" "Innovat(e/ion(s))"
	Whataboutism	"Developing/poorer countries/world/nations" "All nations"
Exemplars	Policy Apocalypse	Projected hardships on U.S. economy and livelihoods
	Whataboutism	Projected emissions of developing countries
Depictions	Climate Risk	Amorphous "risk(s)" of AGW
	Energy Poverty/Prosperity	Concrete benefits of energy allegedly in jeopardy Dire forecasts for developing countries
	Free-Market Solutionism	Voluntary, free-market responses
	Policy Apocalypse	Concrete alleged costs of climate policy
	Scientific/Technological Optimism	Company scientists committed to "decades" of technology R&D University research collaborations
Example discourse quotations		
Climate Risk	"Businesses, governments and NGOs are faced with a daunting task: selecting policies that balance economic growth and human development with the risks of climate change." <sup>54,55</sup>	
Energy Poverty/Prosperity	"A global approach [to "addressing the risk of climate change"] is needed that recognizes...the need for developing countries to weigh emissions control against energy-intensive economic development which lowers poverty and improves public health." <sup>56</sup>	
Free-Market Solutionism	"Governments should...harnes[s] free markets and voluntary measures...[and] encourage and promote voluntary actions by industry and citizens that reduce emissions and use energy wisely." <sup>53</sup>	
Policy Apocalypse	"Committing to binding targets and timetables now will alter today's lifestyles and tomorrow's living standards...Carpooling in; sport utility vehicles out. High fuel and electric bills. Factory closures. Job displacement...[T]ax or carbon rationing..." <sup>57</sup>	
Scientific/Technological Optimism	"[W]e believe that technology provides the key avenue to solutions that manage long-term risk and preserve prosperity. [This] will almost certainly require decades..." <sup>58</sup>	
Whataboutism	"At what point will developing nations begin to participate in emission-reduction activities?" <sup>59</sup>	

The Socioeconomic Threat frame argues that binding climate policies (such as the Kyoto Protocol) are alarmist and threaten prosperity, urging voluntary measures instead.

Central to this frame is the discourse of 'Policy Apocalypse', which depicts dramatic socioeconomic decline due to climate policies on what Schlichting (2013) observes to be both national (macro) and individual (micro) economic levels<sup>29</sup>.

On the macro level, catchphrases of Policy Apocalypse articulating the "economic impact" that climate policies would bring, for example on "jobs/employment", were given added credence by exemplar figures from economic studies. One 1997 advertorial, for instance, cited a study by Charles River Associates predicting "an annual drop in gross domestic product ranging from \$105 billion in the year 2010 to \$460 billion in 2030", "depending on the timing and severity of the plan selected" to limit emissions<sup>60</sup>. Another advertorial the following year warned that WEFA, Inc. "estimates the cost of achieving the Kyoto target by 2010 would result in a loss of 2.4 million jobs, a doubling of electricity prices and an annual loss in economic output of \$300 billion..."<sup>61</sup>. ExxonMobil also made broader moral appeals, such as a 2000 advertorial calling on

policymakers to “Do No Harm”<sup>62</sup>. A key thrust of their argument was that policies such as the Kyoto Protocol could “entail enormous transfers of wealth [from the United States] to other countries”.

On the micro level, advertorials depicted damage to individuals’ wealth and wellbeing. “Committing to binding targets and timetables now will alter today’s lifestyles and tomorrow’s living standards...”, said a 1997 advertorial<sup>57</sup>. “Carpooling in; sport utility vehicles out. High fuel and electric bills. Factory closures. Job displacement...[T]ax or carbon rationing...”.

ExxonMobil’s scaremongering is offset by, at best, ‘Climate Risk’ discourse, and at worse, explicit climate denial (which was commonplace through the mid-2000s). As a result of this imbalanced alleged dichotomy, the frame’s moral evaluation is that any ambiguous, uncertain “risk(s)” of AGW are outweighed by severe economic damages threatened by mandatory climate policies. Such policies are therefore unwarranted and economically dangerous.

They are also ineffective, ‘Whataboutism’ discourse argues. For example, quoting a report by The Business Roundtable, Mobil wrote in a 1998 advertorial that “‘Without full participation by developing countries, the Kyoto Protocol will not lead to a net reduction of global...emissions.’ ...The Protocol uses ‘differentiated targets’ for countries to meet, which potentially could put the U.S. at a disadvantage.”<sup>63</sup> Thus, Whataboutism, which also displays elements of discourses that Lamb *et al.* (2020) term “The ‘free rider’ excuse” and “Policy perfectionism”, effectively extends the economic scaremongering arguments of Policy Apocalypse discourse, but does so by directly questioning the efficacy of proposed policies rather than simply highlighting their alleged societal costs<sup>50</sup>. The issue is further confounded by Energy Poverty/Prosperity discourse, which tends to imply that alternative binding policies including developing countries would not be viable either. “Kyoto failed to include developing countries”, said an advertorial in 2000. “Yet poorer countries need more energy if they are to provide economic growth and a better life for their people”, implying that developing countries should not be included after all.

The only solutions, then, according to Discourses of ‘Science/Technology Optimism’ and ‘Free Market Solutionism’, are “voluntary steps”. “[I]t is time to move beyond Kyoto”, the 2000 advertorial above concludes. “[W]e believe that technology provides the key avenue to solutions...”, said a 2002 advertorial<sup>58</sup>. “Governments should...harnes[s] free markets and voluntary measures...”, argued another in 2007<sup>53</sup>.

### S4.3 Fossil Fuel Savior frame

**Table S4.** Frame matrix of Fossil Fuel Savior (FFS) frame package.

Fossil Fuel Savior (FFS) Frame Package		
Reasoning Devices	Description	
Problem	Climate change is a (potential long-term) risk	
Cause	Consumer energy demand	
Moral evaluation	Climate risk is an energy technology/efficiency challenge in pursuit of energy prosperity	
Solutions	Continued fossil fuels for decades to come Technology innovation in a free-market Individualized energy efficiency improvements	
Framing Devices	Discourse	Example/Description
Catchphrases & lexical choices	Climate Risk	"Risk(s) of climate change" "Longterm)"
	Individualized Responsibility	"(Energy) demand" "Energy use" "Needs" "To meet"
	Energy Poverty/Prosperity	"Prosperity" "Poor/poverty/lack"
	Fossil Fuel Solutionism	"Oil and gas/natural gas" "For generations/foreseeable future/several decades/decades to come/next 25 years"
	Policy Apocalypse	"Economic growth/impact" "Wise(r)/prudent/reasonable/responsible/sound(er)"
	Greenwashing/Corporate	"Steps"
	Symbolic Environmentalism	"Tree(s)"
	Scientific/Technological	"New/advanced technolog(y/ies)"
	Optimism	"Solutions"
	Technological Shell-Game	"Natural gas" "Limitations/obstacles/barriers/cannot compete"
	Visual images	Scientific/Technological
	Optimism	Science iconography
Exemplars	Fossil Fuel Solutionism	Conservative clean energy projections
	Greenwashing/Corporate	Donations to environmental initiatives
	Symbolic Environmentalism	Reports of company energy efficiency efforts Corporate social responsibility actions and pledges such as "math and science" "education" initiatives
	Individualized Responsibility	Projected energy demand growth Personal energy conservation tips
Depictions	Climate Risk	Amorphous "risk(s)" of AGW
	Energy Poverty/Prosperity	Concrete benefits of energy allegedly in jeopardy World's poor reliant on fossil fuels for decades to come
	Fossil Fuel Solutionism	Society reliant mostly on fossil fuels for decades to come
	Technological Shell-Game	Renewable energy supply negligible for decades to come
	Scientific/Technological	Photographs of company scientists as face of technology R&D
	Optimism	Dynamic "breakthrough" university research collaborations
Example discourse quotations		
Climate Risk	"[W]e'll need more energy to power our homes, businesses and industries, and to fuel our transportation needs...while addressing the risks posed by rising greenhouse gas emissions..." <sup>64</sup>	
Energy Poverty/Prosperity	"[G]lobal carbon-dioxide emissions are expected to rise through 2030. This is particularly true in developing countries, which will rely on relatively carbon-intensive fuels like coal to meet their needs." <sup>64</sup>	
Fossil Fuel Solutionism	"Oil and gas will be essential to meeting demand." <sup>65</sup>	
Individualized Responsibility	"[G]rowing demand will boost CO <sub>2</sub> emissions." <sup>64</sup>	
Greenwashing/Corporate	"For five years we have partnered with the group American Forests to plant trees...this year the partnership planted its two millionth tree." <sup>66</sup>	
Symbolic Environmentalism	"[W]e believe that technology provides the key avenue to solutions that manage long-term risk and preserve prosperity. [This] will almost certainly require decades..." <sup>58</sup>	
Scientific/Technological	"[T]echnological progress in these conventional fuels ["oil and <u>natural gas</u> "] holds immediate potential to help reduce emissions on a significant scale...[T]his clean and abundant resource [of " <u>natural gas</u> "] is helping meet our energy and environmental goals." <sup>67</sup>	
Optimism		
Technological Shell-Game		

## S5. DISCOURSES OF DELAY

Each of the following tables displays a selection of highly divergent terms in advertorials, by Log-Likelihood ratio ( $G^2$ ) and Frequency Score (FS), identified by frame package analysis as framing devices of each of the discourses displayed in figure 1 of the main text. Definitions of each discourse are provided in the captions of respective tables (see table S1 for supporting literature). P-values: \* <0.005; \*\* <0.05; \*\*\* $\geq$ 0.05; otherwise, <0.001 for all  $G^2$  and FS scores.

**Table S5.** Rhetoric of Climate Risk. Example quotations illustrate how advertorials use divergent terms to present AGW or greenhouse gases as a “(long-term) risk”.

Climate Risk rhetoric						
Advertorials often say:						
	Advertorials	Internal	Peer-reviewed	$G^2$ (Int./P.r.)	FS (Int./P.r.)	Example
risk(s)	49	7	261	72.48 / 56.56	0.93 / 0.8	"Enough is known about climate change to recognize it may pose a legitimate long-term <u>risk</u> , and that more needs to be learned about it." <sup>62</sup>
climate (change) risk(s)/ risk(s) of climate	26	0	10	57.89 / 119.09	1 / 0.98	"It is our view that better scientific understanding of climate change, human influence on it, and the associated risks and possible consequences are needed. We are heavily involved in such scientific research...But we are also taking other actions to minimize the <u>risks of climate change</u> ." <sup>68</sup>
longterm	40	17	282	33.14 / 31.82	0.83 / 0.75	"In releasing this [National Assessment Synthesis] report, the [Clinton] administration seeks to gain support for its own [climate] policies, which could damage the economy and employment while accomplishing little in addressing potential <u>long-term climate risks</u> ." <sup>69</sup>

**Table S6.** Rhetoric of Doubt Mongering. Example quotations illustrate how advertorials use divergent terms to promote doubt about climate science and its implications.

Doubt Mongering rhetoric						
Advertorials often say:						
	Advertorials	Internal	Peer-reviewed	$G^2$ (Int./P.r.)	FS (Int./P.r.)	Example
dont	24	2	0	40.93 / 148.34	0.96 / 1	"We still <u>don't</u> know what role man-made greenhouse gases might play in warming the planet." <sup>57</sup>
improv(e/es/ed/ing/ements)	73	54	500	32.35 / 60.65	0.73 / 0.75	"... <u>improve</u> our understanding of the science of this complex issue." <sup>68</sup>
doom(sday/sdayers)/apocalypse/ hype/scare	11	0	0	24.49 / 67.99	1/1	" <u>Apocalypse</u> no. For the first half of 1992, America was inundated by the media with dire predictions of global warming catastrophes..." <sup>70</sup>
debate	26	12	30	20.05 / 86.15	0.82 / 0.95	"Weather and climate. In the <u>debate</u> over climate change, there is an understandable tendency to use recent weather events to draw conclusions about global warming..." <sup>52</sup>
answer(s)	22	9	22	18.8 / 77.03	0.83 / 0.95	"Within a decade, science is likely to provide more <u>answers</u> on what factors affect global warming..." <sup>71</sup>
believe	21	9	18	17.28 / 77.64	0.83 / 0.96	Quoting Freeman J Dyson: "[C]limate models...are unreliable...[W]e must continue to warn the politicians and the public don't <u>believe</u> the numbers just because they come out of supercomputer" <sup>69</sup>
(un)know(n/ing/ledge)	57	66	330	9.63* / 59.52	0.64* / 0.78	"[F]undamental gaps in <u>knowledge</u> leave scientists unable to make reliable predictions about future [climatic] changes." <sup>51</sup>
gap(s)	11	7	39	6.01** / 18.93	0.76** / 0.86	"...better delineating <u>gaps</u> and uncertainties that limit our current ability to know the extent to which humans are affecting climate and to predict future changes caused by both human and natural forces." <sup>72</sup>
better science/understanding	6	NA	10	NA / 16.85	NA / 0.93	"Concern over global climate change is triggering actions... <u>Better science</u> and flexible timing also need to be part of the mix." <sup>73</sup>
agree(ment)/consensus	35	45	338	4.12** / 15.55	0.61** / 0.68	"[T]here is no <u>consensus</u> on what constitutes "dangerous levels" of emissions nor is there <u>agreement</u> on when, where and how best to reduce their impact." <sup>60</sup>
compl(ex/exity/icated)	18	NA	165	NA / 8.96*	NA / 0.7*	"Climate science remains extraordinarily <u>complex</u> ." <sup>54,55</sup>
natural causes/phenomen(on/a)/ climate/variability/and manmade	16	NA	159	NA / 6.66**	NA / 0.68**	Research "[p]rograms should concentrate on factors that seriously limit current understanding [of AGW]. These include the effects of clouds, aerosols, sea ice, deep-ocean circulation, hydrology and <u>natural climate variability</u> ." <sup>74</sup>

**Table S7. Rhetoric of Energy Poverty/Prosperity.** Example quotations illustrate how advertorials use divergent terms to present energy – and typically, by extension, fossil fuels – as essential to well-being and social justice.

Energy Poverty/Prosperity rhetoric						
Advertorials often say:						
	Advertorials	Internal	Peer-reviewed	G <sup>2</sup> (Int./P.r.)	FS (Int./P.r.)	Example
developing/poorer countries/world/nations challenge(s)	53	3	196	97.01 / 88.01	0.97 / 0.85	"Energy demand is expected to be 35 percent higher in the year 2030...driven largely by people in the <u>developing world</u> seeking higher standards of living." <sup>75</sup>
	56	5	100	94.08 / 151.75	0.96 / 0.92	"A key goal of our citizenship strategy is addressing the <u>challenge</u> of sustainability balancing economic growth, social development and environmental performance while continuing to deliver superior shareholder returns so that future generations are not compromised by actions taken today." <sup>76,77</sup>
prosperity	15	0	1	33.4 / 85.32	1 / 1	"[G]lobal energy needs are rising, with increasing <u>prosperity</u> in the developing world the main driver of greater energy demand (and consequently rising CO <sub>2</sub> emissions) over the coming decades." <sup>56</sup>
social	22	6	201	24.67 / 11.03	0.88 / 0.7	"[E]fforts to control emissions have important economic and <u>social</u> consequences." <sup>74</sup>
affordable	11	0	6	24.49 / 46.47	1 / 0.97	"Balancing the long-term risks of climate change against society's need for unsubsidized but <u>affordable</u> energy..." <sup>58</sup>
living standard(s)/ standard(s) of living/ quality of life	10	0	0	22.27 / 61.81	1 / 1	"[S]cientists work to provide more definitive answers on the impact that these [greenhouse] gases and other factors may have on our climate system. Let's wait for more answers before taking on obligations that could jeopardize better <u>living standards</u> for all." <sup>78</sup>
poor/poverty/lack	11	7	0	6.01** / 67.99	0.76** / 1	"A global approach [to "addressing the risk of climate change"] is needed that recognizes...the need for developing countries to weigh emissions control against energy-intensive economic development which lowers <u>poverty</u> and improves public health." <sup>56</sup>

**Table S8. Rhetoric of Fossil Fuel Solutionism.** Example quotations illustrate how advertorials use divergent terms to present fossil fuels and their industry as an essential and inevitable part of the solution to AGW.

Fossil Fuel Solutionism rhetoric						
Advertorials often say:						
	Advertorials	Internal	Peer-reviewed	G <sup>2</sup> (Int./P.r.)	FS (Int./P.r.)	Example
oil and (natural) gas	28	3	92	45.02 / 51.24	0.95 / 0.86	"As Americans look for ways to access more supplies of reliable, affordable energy while at the same time reducing emissions, answers are emerging from what may seem an unlikely source - the <u>oil and natural gas</u> industry." <sup>67</sup>
clean(er)	14	0	36	31.17 / 30.59	1 / 0.89	"[D]iesel could become a viable player, providing motorists with a <u>clean</u> , efficient option." <sup>79</sup>
through/by/in the year 2030	22	9	113	18.8 / 26.47	0.83 / 0.8	"Wind and solar...meet about 1% of total world demand <u>by 2030</u> . Close to 60% to be met by oil and natural gas." <sup>80-82</sup>
continued/continue to	23	10	123	18.69 / 26.43	0.82 / 0.8	"Oil, natural gas and coal will remain essential...In 2030, these fuels will <u>continue to</u> provide approximately 80 percent of the world's energy..." <sup>64</sup>
for generations/foreseeable future/several decades/decades to come/next 25 years	12	3	28	14.1 / 27.91	0.89 / 0.9	"Battery technology just cannot compete with internal combustion engines today or in the <u>foreseeable future</u> ..." <sup>83</sup>
fossil fuels	24	NA	149	NA / 22.89	NA / 0.77	" <u>Fossil fuels</u> must be relied upon to meet society's immediate and near-term needs." <sup>84</sup>
re(ly/ied)	8	NA	39	NA / 10.19*	NA / 0.81*	"Among the more promising approaches to addressing the risks of climate change are those that <u>rely</u> upon economically attractive actions and advanced technology. One good example is the increasing use of cogeneration units." <sup>85</sup>

**Table S9. Rhetoric of Free-Market Solutionism.** Example quotations illustrate how advertorials use divergent terms to denounce restrictive measures and instead promote voluntary/free-market policies.

Free-Market Solutionism rhetoric						
Advertorials often say:						
	Advertorials	Internal	Peer-reviewed	G <sup>2</sup> (Int./P.r.)	FS (Int./P.r.)	Example
mandat(e/es/ed/ing)	15	1	10	26.72 / 59.99	0.97 / 0.97	"[w]e ask the Kyoto delegates to avoid <u>mandates</u> based on uncertain science..." <sup>53</sup>
voluntarily reduce(d) / voluntary initiative/step/measure/action/ effort/approache/use/usage(s)	12	0	7	26.72 / 49.81	1 / 0.97	"[W]e support <u>voluntary efforts</u> to reduce emissions." <sup>61</sup>
bind(ing)/rigid	11	0	11	24.49 / 38.51	1 / 0.95	"Instead of <u>rigid</u> targets and timetables, governments should consider alternatives, including: adopt consensus objectives; encourage voluntary initiatives and government-industry partnerships..." <sup>86</sup>
market(place/-based)	5	NA	13	NA / 10.84	NA / 0.89*	"[G]overnment policies should support long-term research on alternatives but let the <u>marketplace</u> decide which technical approach will gain commercial and consumer acceptance." <sup>84</sup>
flexible	7	NA	33	NA / 9.24*	NA / 0.82*	"These suggestions...avoid regulatory strait-jackets and invite participation by all nations. Because they are <u>flexible</u> , policies can change as experience and knowledge are gained." <sup>74</sup>

**Table S10. Rhetoric of Greenwashing/Symbolic Corporate Environmentalism.** Example quotations illustrate how advertorials use divergent terms to communicate symbolic corporate environmentalism, including greenwashing. Bowen (2014) defines symbolic corporate environmentalism as “the shared meanings and representations surrounding” “changes made by managers inside organizations that they describe as primarily for environmental reasons”<sup>87</sup>. Greenwashing is a subset of symbolic corporate environmentalism “in which the changes are both ‘merely symbolic’ and deliberately so”.

Greenwashing/Symbolic Corporate Environmentalism rhetoric						
Advertorials often say:						
	Advertorials	Internal	Peer-reviewed	G <sup>2</sup> (Int./P.r.)	FS (Int./P.r.)	Example
percent	104	9	39	175.94 / 478.85	0.96 / 0.98	"Across our operations, we reduced the number of oil spills by 21 <u>percent</u> from 2005 and by an average of over 10 <u>percent</u> annually since 2000." <sup>88</sup>
energy efficien(cy/t)/us(e/age)	56	5	246	94.08 / 79.39	0.96 / 0.83	"We have developed global energy-management system to identify opportunities to further reduce <u>energy use</u> . <u>Energy efficiency</u> has already improved 35 percent in our refineries and chemical plants since the 1970s." <sup>88</sup>
new/advanced technolog(y/ies)	40	2	42	74.58 / 137.51	0.98 / 0.95	"[T]here men and women [at ExxonMobil] are developing amazing <u>new technologies</u> for finding and delivering energy, as well as innovations that will allow us to use energy more efficiently." <sup>89</sup>
steps	36	1	36	71.76 / 126.05	0.99 / 0.95	"[W]e have taken <u>steps</u> to reduce our own emissions and initiate reforestation programs." <sup>90</sup>
cut	19	0	9	42.31 / 83.11	1 / 0.98	"In the last three years, we've <u>cut</u> our carbon emissions by more than one million metric tons..." <sup>91</sup>
invest(ing/ment(s))	27	4	243	39.46 / 13.96	0.93 / 0.7	"[W]e're now making the largest ever <u>investment</u> in independent climate and energy research that is specifically designed to look for new breakthrough technologies." <sup>80-82</sup>
tree(s)	28	5	141	38.26 / 34.44	0.92 / 0.81	"In support of American Forests [charity], Mobil this year will fund the planting of 500,000 <u>trees</u> in watersheds, state and national forests and wildlife refuges..." <sup>92</sup>
gcep	17	0	1	37.85 / 97.44	1 / 1	The "Global Climate and Energy Project ( <u>GCEP</u> ) based at Stanford University...brings together some of the world's best scientific and engineering minds to address this pressing challenge...ExxonMobil is proud to be its lead developer and sponsor..." <sup>93</sup>
hydrogen/fuel cell(s)	26	5	314	34.48 / 6.29**	0.91 / 0.63**	At the "Global Climate and Energy Project (GCEP), initiated at Stanford University in 2002 with the intention of ExxonMobil...[r]esearchers are investigating the use of genetically engineered bacteria to capture solar energy and produce <u>hydrogen</u> ..." <sup>94</sup>
improv(e/es/ed/ing/ements)	73	54	500	32.35 / 60.65	0.73 / 0.75	"Mobil 1 AFE [gasoline] can <u>improve</u> fuel economy by up to 2 percent...if one-third of U.S. motorists reduced their gasoline by 2 percent, almost...8 million tons of CO <sub>2</sub> emissions would be saved every year." <sup>95</sup>
innovat(e/ion(s))	17	1	93	30.93 / 19.02	0.97 / 0.79	"Other <u>innovations</u> are still emerging. One is a new engine technology...The result: up to 30 percent better fuel economy and lower emissions." <sup>96</sup>
fuel economy	13	0	63	28.95 / 16.67	1 / 0.81	"ExxonMobil is taking [steps] to address the risk of climate change. These include[e] working to improve energy efficiency and <u>fuel economy</u> ..." <sup>97</sup>
cogeneration	12	0	26	26.72 / 29.19	1 / 0.91	"We now have interest in 4300 megawatts of energy-efficient <u>cogeneration</u> facilities globally - enough to reduce global carbon-dioxide emissions by over 10.5 million metric tons annually..." <sup>98</sup>
education	12	0	28	26.72 / 27.91	1 / 0.9	"Over the long-term, investments such as these could also yield real progress in developing the new technologies needed to address global challenges such as climate change...By investing more in math and science <u>education</u> , we can...solve tomorrow's tough challenges..." <sup>98</sup>
stanford	14	1	0	24.62 / 86.53	0.97 / 1	"With initial funding of \$225 million [from ExxonMobil and other companies], the Global Climate and Energy Project (GCEP) will unleash the creativity of faculty and students at <u>Stanford</u> and other universities..." Advertorial signed by "Dr. Lynn Orr, GCEP Project Director, <u>Stanford</u> University." <sup>99</sup>
sav(e/ed/ing)	14	1	51	24.62 / 23.55	0.97 / 0.85	" <u>Saving</u> and preserving forests and trees are long-term endeavors. But we are hopeful, and optimistic, that planting trees now will be planting a better future around the world." <sup>100</sup>
protect(/ion/ing)	26	10	109	23.32 / 38.56	0.84 / 0.83	"Many groups work to <u>protect</u> and to expand forests. ExxonMobil is proud to say that we are one of them." <sup>100</sup>
math and science	10	0	0	22.27 / 61.81	1 / 1	"Sustainability means balancing economic, environmental and social goals...[W]e are a leading supporter of <u>math and science</u> education..." <sup>76,77</sup>
plant(ing)	21	7	NA	20.84 / NA	0.86 / NA	"We intend to sponsor several projects to <u>plant</u> and protect trees in the U.S. and internationally." <sup>78</sup>
partner(/ing/ship)	12	1	13	20.47 / 40.76	0.96 / 0.95	"[O]ur scientists and engineers are...[ <u>Partnering</u> with with the U.S. Environmental Protection Agency and Department of Energy in the "Smartway" <u>partnership</u> to improve fuel economy and reduce emissions associated with the transportation of our products." <sup>100</sup>
initiative(s)	18	5	35	19.98 / 46.59	0.88 / 0.92	"Working with leading environmental groups, Mobil will underwrite international projects to plant and protect trees which absorb significant amounts of CO <sub>2</sub> . <u>Initiatives</u> like these, which are good for the environment, can be taken while the debate continues." <sup>101</sup>
operations	11	3	99	12.33 / 5.69**	0.88 / 0.77**	"At ExxonMobil, we are taking action...deploying energy-efficient technologies across our global <u>operations</u> ..." <sup>56</sup>
universit(y/ies)	23	16	9	11.15 / 104.97	0.75 / 0.98	"[W]e are supporting climate-related research at major <u>universities</u> , including Stanford and MIT." <sup>72</sup>
sponsor/fund/invest/underwrite/grant(/ed/ing)	34	41	41	5.04** / 110.65	0.63** / 0.95	"We are <u>funding</u> research into the scientific and economic consequences of climate change." <sup>102</sup>
environment(/al/ally) effort(s) to	84	112	527	8.53* / 79.01	0.61* / 0.77	"We all share the same goal: protecting Earth's <u>environment</u> while raising living standards for all." <sup>101</sup>
	18	11	44	10.34* / 40.65	0.77* / 0.9	"[W]e are a leading supporter of math and science education, including <u>efforts to</u> increase the number of women and minorities studying in these fields." <sup>76,77</sup>

**Table S11. Rhetoric of Individualized Responsibility.** Example quotations illustrate how advertorials use divergent terms to present: (a) consumer demand for energy as the cause of – and culpable for – fossil fuel use, greenhouse gas emissions, and/or AGW; and (b) individual/demand-side actions as accountable for mitigating AGW. By contrast, divergent terms in (bottom) internal and/or peer-reviewed documents often articulate the causality and culpability of fossil fuel combustion.

Advertorials often say:						
	Advertorials	Internal	Peer-reviewed	G <sup>2</sup> (Int./P.r.)	FS (Int./P.r.)	Example
(to) meet	65	2	98	128.34 / 191.64	0.99 / 0.93	"To <u>meet</u> this demand, while addressing the risks posed by rising greenhouse gas emissions, we'll need to call upon broad mix of energy sources." <sup>64</sup>
vehicles	33	0	240	73.48 / 25.02	1 / 0.74	"[T]he cars and trucks we drive aren't just <u>vehicles</u> , they're opportunities to solve the world's energy and environmental challenges." <sup>96</sup>
greenhouse gas emissions	42	7	60	58.9 / 126.97	0.92 / 0.94	"We're supporting research and technology efforts, curtailing our own <u>greenhouse gas emissions</u> and helping customers scale back their emissions of carbon dioxide." <sup>78</sup>
energy efficiency	30	1	152	58.76 / 36.65	0.98 / 0.81	"We have invested \$1.5 billion since 2004 in activities to increase <u>energy efficiency</u> and reduce greenhouse gas emissions. We are on track to improve energy efficiency in our worldwide refining and chemical operations..." <sup>76,77</sup>
cars	24	0	59	53.44 / 54	1 / 0.9	"By enabling <u>cars</u> and trucks to travel farther on a gallon of fuel, drivers not only spend less money per mile, they also emit less carbon dioxide (CO <sub>2</sub> ) per mile." <sup>95</sup>
reduce emissions	23	0	25	51.21 / 78.03	1 / 0.95	"During the fact-finding period, governments should encourage and promote voluntary actions by industry and citizens that <u>reduce emissions</u> and use energy wisely. Governments can do much to raise public awareness of the importance of energy conservation." <sup>53</sup>
consumers	21	0	33	46.76 / 60.7	1 / 0.93	"We also are developing new vehicle technologies that can help <u>consumers</u> use energy more efficiently." <sup>76,77</sup>
world	91	64	338	43.45 / 150.55	0.74 / 0.85	"By 2030, experts predict that the <u>world</u> will require about 60 percent more energy than in 2000...As a result, greenhouse gas emissions are predicted to increase too..." <sup>93</sup>
developing countries	27	3	162	43 / 26.94	0.95 / 0.78	Through 2030, " <u>developing countries</u> ...will rely on relatively carbon-intensive fuels like coal to meet their needs." <sup>64</sup>
transportation	23	2	121	38.87 / 26.93	0.96 / 0.8	"Ongoing advances in vehicle and fuel technology will be critical to meeting global demand for <u>transportation</u> fuels. They will also help address the risk posed by rising greenhouse-gas emissions." <sup>96</sup>
energy use	23	4	83	31.75 / 39	0.92 / 0.85	"Central to any future policy should be the understanding that man-made greenhouse gas emissions arise from essential <u>energy use</u> in the everyday activities of people, governments and businesses." <sup>74</sup>
people	30	11	61	27.87 / 75.73	0.85 / 0.91	"Thus, we're pleased to extend our support of...American Forests...whose "Global Releaf 2000" program is mobilizing <u>people</u> around the world to plant and care for trees." <sup>92</sup>
demand	40	21	422	27.24 / 14.35	0.8 / 0.67	"[I]n the electric power sector, growing <u>demand</u> will boost CO <sub>2</sub> emissions..." <sup>65</sup>
needs	36	22	71	20.69 / 92.45	0.77 / 0.91	"...fossil fuels must be relied upon to meet society's immediate and near-term <u>needs</u> ." <sup>84</sup>
conservation	15	5	66	14.89 / 21.23	0.86 / 0.83	"Prudent measures such as <u>conservation</u> and investment in energy-efficient technology make sense, but embarking on regulatory [climate/energy] policies that may prove wasteful or counterproductive does not." <sup>103</sup>
energy demand	15	14	59	4.38** / 23.59	0.69** / 0.84	"[I]ncreasing prosperity in the developing world [is] the main driver of greater <u>energy demand</u> (and consequently rising CO <sub>2</sub> emissions) over the coming decades." <sup>56</sup>
Internal and/or peer-reviewed documents often say:						
fossil fuel	9	144	359	-66.26 / -4.48**	0.11 / 0.34***	"Release of this amount of CO <sub>2</sub> to the atmosphere raises concern with respect to its effect on the CO <sub>2</sub> greenhouse problem. Global <u>fossil fuel</u> emissions of CO <sub>2</sub> currently amount to about 1.8 x 10 <sup>10</sup> metric tons per year..." <sup>104</sup>
natuna	0	67	NA	-53.36 / NA	0 / NA	"Arrhenius put forth the idea that CO <sub>2</sub> from <u>fossil fuel</u> burning could...warm the Earth...fossil fuel greenhouse warming...fossil fuel greenhouse effect..." <sup>105</sup>
due to	5	89	731	-42.94 / -39.08	0.1 / 0.13	"This would make <u>Natuna</u> the world's largest point source emitter of CO <sub>2</sub> and raises concern for the possible incremental impact of <u>Natuna</u> on the CO <sub>2</sub> greenhouse problem." <sup>104</sup>
fossil fuel combustion	1	48	NA	-30.69 / NA	0.04 / NA	"The CO <sub>2</sub> concentration in the atmosphere has increased...The most widely held theory is that: the increase is <u>due to</u> fossil fuel combustion." <sup>106</sup>
shale	1	41	NA	-25.43 / NA	0.05 / NA	"About three-quarters of the anthropogenic emissions of CO <sub>2</sub> to the atmosphere during the past 20 years is <u>due to</u> fossil fuel burning." <sup>107</sup>
ccs	0	NA	374	NA / -34.82	NA / 0	"[T]here is the potential for our [climate] research to attract the attention of the popular news media because of the connection between Exxon's major business and the role of <u>fossil fuel combustion</u> in contributing to the increase of atmospheric CO <sub>2</sub> ." <sup>108</sup>
source	6	39	322	-9.08* / -7.16**	0.24* / 0.28**	"The quantity of CO <sub>2</sub> emitted by various fuels in shown in Table 1...They show the high CO <sub>2</sub> /energy ratio for coal and <u>shale</u> ...["Shale oil"] is not predicted to be a major future energy source due to...rather large amounts of CO <sub>2</sub> emitted per unit energy generated (see Table 1)." <sup>106</sup>
fossil fuel use	0	13	NA	-10.35* / NA	0** / NA	"CCS includes applying technologies that capture the CO <sub>2</sub> whether generated by combustion of carbon-based fuels or by the separation of CO <sub>2</sub> from natural gas with a high CO <sub>2</sub> concentration." <sup>109</sup>
fossil fuel co2	0	NA	64	NA / -5.96**	NA / 0***	"[Fossil fuel combustion is the only readily identifiable <u>source</u> [of CO <sub>2</sub> ] which is (1) growing at the same rate, (2) large enough to account for the observed increases..." <sup>110</sup>
fossil fuel emissions	0	NA	54	NA / -5.03**	NA / 0***	Table 1 presents "coal combustion" and "natural gas combustion" as the " <u>source[s]</u> " of CO <sub>2</sub> , CH <sub>4</sub> , SO <sub>2</sub> . <sup>111</sup>
						"[F]or scenarios with higher <u>fossil fuel use</u> (hence, higher carbon dioxide emissions..." <sup>107</sup>
						"This long tail on the <u>fossil fuel CO<sub>2</sub></u> forcing of climate may well be more significant to the future glacial/interglacial timescale evolution of Earth's climate..." <sup>112</sup>
						"We use our Integrated Science Model to...estimate the time variation <u>fossil fuel emissions</u> of CO <sub>2</sub> ...required to match the [IPCC] concentration stabilization scenarios." <sup>113</sup>

**Table S12. Rhetoric of Policy Apocalypse.** Example quotations illustrate how advertorials use divergent terms to allege that climate policies will be socioeconomically damaging.

Policy Apocalypse rhetoric						
Advertorials often say:						
	Advertorials	Internal	Peer-reviewed	G <sup>2</sup> (Int./P.r.)	FS (Int./P.r.)	Example
econom(y)ic	148	22	714	216.08 / 190.67	0.93 / 0.81	"We ask the Kyoto delegates to...resist agreements that could inflict great <u>economic</u> pain." <sup>53</sup>
economic growth/impact	29	2	74	51.34 / 63.68	0.97 / 0.89	"The report shows how ill-timed or ill-considered [GHG emissions] abatement measures could stunt world <u>economic growth</u> , unsettle global trading patterns and set the stage for new era of trade protectionism." <sup>60</sup>
cost(s)/ly/liest/lier)	61	32	NA	41.58 / NA	0.8 / NA	"[A]s higher energy <u>costs</u> work their way through the economy, the annual loss in GDP could range from \$150 billion to \$400 billion." <sup>61</sup>
jobs/employment	15	0	40	33.4 / 31.98	1 / 0.89	"WEFA estimates the cost of achieving the Kyoto target by 2010 would result in loss of 24 million <u>jobs</u> ..." <sup>61</sup>
tax(es)	20	2	177	32.72 / 10.7*	0.95 / 0.7	"Most economists tell us that such a step [as the Kyoto Protocol] would damage our economy and almost certainly require large increases in <u>taxes</u> on gas and oil." <sup>62</sup>
livelihood(s)/lifestyle(s)	13	0	42	28.95 / 24.11	1 / 0.87	"How much prosperity are Americans willing to forgo? How many <u>lifestyle</u> changes will they have to make? How much more tax will they pay?" <sup>59</sup>
wise(r)/prudent/reasonable/responsible/sound(er)	39	21	119	25.87 / 75.54	0.79 / 0.87	A " <u>prudent</u> approach to the climate issue must recognize that there is not enough information to justify harming economies and forcing the world's population to endure unwarranted lifestyle changes by dramatically reducing the use of energy now." <sup>62</sup>
disruptive/dislocations/distortions/unsettled	11	0	8	24.49 / 42.87	1 / 0.97	"Concern about the impact of human activity on the global climate...is triggering actions that may create major <u>dislocations</u> unnecessarily." <sup>14</sup>
suffer/saddled/havoc/pain(ful)/grave/fatal/turmoil/jeopardize/harm/hit/inflict/plunge/cripple/wreck(ing)impos(e)ing)	17	3	15	23.33 / 62.23	0.92 / 0.96	"Adopting quick-fix measures [for AGW] at this point could pose <u>grave</u> economic risks for the world." <sup>72</sup>
	16	6	9	14.62 / 67.06	0.85 / 0.97	"As gaps in climate science are being filled, these approaches can lead to real changes in emissions trends without <u>harming</u> economies and lifestyles." <sup>15</sup>
	8	NA	16	NA / 20.38	NA / 0.91	"[T]he impact that some [AGW mitigation] measures could have on jobs and livelihoods will <u>impose</u> extensive burdens on the global community." <sup>16</sup>
consequences	15	NA	81	NA / 17.04	NA / 0.8	"Because of the potentially serious <u>consequences</u> any such [climate action] plan would have on the U.S. economy and peoples livelihoods..." <sup>17</sup>
drastic/rash/premature	6	NA	22	NA / 10.04*	NA / 0.85*	"[T]he jury's still out on whether <u>drastic</u> steps to curb CO <sub>2</sub> emissions are needed." <sup>70</sup>

**Table S13. Rhetoric of Scientific/Technological Optimism.** Example quotations illustrate how advertorials use divergent terms to give primacy to scientific or technological breakthroughs as the solutions to understanding and/or mitigating AGW.

Scientific/Technological Optimism rhetoric						
Advertorials often say:						
	Advertorials	Internal	Peer-reviewed	G <sup>2</sup> (Int./P.r.)	FS (Int./P.r.)	Example
new/advanced technolog(y)ies)	40	2	42	74.58 / 137.51	0.98 / 0.95	"[W]e are excited to be working on breakthrough technology that could advance the use of hydrogen fuel cells. This <u>new technology</u> ...converts traditional hydrocarbon fuels (such as gasoline or diesel) into hydrogen..." <sup>96</sup>
promise	20	0	12	44.53 / 82.39	1 / 0.97	"The <u>promise</u> of technology. One of the brighter hopes in the climate change debate has to be the benefits to be achieved through technology." <sup>118</sup>
invest(ing/ment(s))	27	4	243	39.46 / 13.96	0.93 / 0.7	"[W]e're now making the largest ever <u>investment</u> in independent climate and energy research that is specifically designed to look for new breakthrough technologies." <sup>80-82</sup>
innovat(e/ion(s)) solutions	17	1	93	30.93 / 19.02	0.97 / 0.79	"Support for oil and natural gas <u>innovation</u> can reduce emissions." <sup>67</sup>
	26	7	78	29.36 / 51	0.88 / 0.87	"[W]e believe that technology provides the key avenue to <u>solutions</u> that manage long-term risk and preserve prosperity." <sup>58</sup>
develop	29	32	69	5.64** / 66.62	0.65** / 0.9	"Many respected economists conclude that research to <u>develop</u> new technology offers the most effective near-term means to address the long-term response to climate change." <sup>118</sup>

**Table S14. Rhetoric of Technological Shell-Game.** Example quotations illustrate how advertorials use divergent terms to communicate what Schneider *et al.* (2016) define as “misdirection that relies on strategic ambiguity about the feasibility, costs, and successful implementation of technologies in order to deflect attention from environmental pollution and health concerns”.

Technological Shell-Game rhetoric						
Advertorials often say:						
	Advertorials	Internal	Peer-reviewed	G <sup>2</sup> (Int./P.r.)	FS (Int./P.r.)	Example
natural gas	48	18	334	43.87 / 38.95	0.85 / 0.75	"[T]echnological progress in these conventional fuels [“oil and <u>natural gas</u> ”] holds immediate potential to help reduce emissions on a significant scale...[T]his clean and abundant resource [of “ <u>natural gas</u> ”] is helping meet our energy and environmental goals." <sup>67</sup>
electric vehicles/EVs	16	0	11	35.63 / 63.42	1 / 0.97	"[T]he GAO basically concluded <u>EVs</u> aren't ready. Nor are they likely to become so even in the rosiest of scenarios." <sup>83</sup>
limitations/obstacles/barriers/cannot compete	14	NA	142	NA / 5.54**	NA / 0.67**	"Renewable forms of energy could play role [in the electric power sector], but they have <u>limitations</u> that make them impractical or expensive for most applications." <sup>65</sup>
solar/photovoltaic(s)	31	NA	393	NA / 6.34**	NA / 0.62**	" <u>Solar</u> power is dependent on sunlight availability and is space-intensive. Here again, its potential must be tempered with realism." <sup>119</sup>

**Table S15.** Rhetoric of Whataboutism. Example quotations illustrate how advertorials use divergent terms to point to other actors that produce – or may in the future produce – more greenhouse gas emissions. It is thereby argued that those actors bear significant responsibility for taking action, and that without their participation, climate policies will be unjust (‘free rider’ excuse) or ineffective (policy perfectionism).

Whataboutism rhetoric						
Advertorials often say:						
	Advertorials	Internal	Peer-reviewed	G <sup>2</sup> (Int./P.r.)	F5 (Int./P.r.)	Example
developing/poorer countries/world/nations	53	3	196	97.01 / 88.01	0.97 / 0.85	"Developing countries are not covered by the [Kyoto] Protocol. [Quoting a new report by The Business Roundtable:] "Without full participation by <u>developing countries</u> , the Kyoto Protocol will not lead to a net reduction of global...emissions." <sup>63</sup> ...The Protocol uses "differentiated targets" for countries to meet, which potentially could put the U.S. at a disadvantage." <sup>63</sup>
all nations	11	0	3	24.49 / 53.72	1 / 0.99	"Clearly, curbing greenhouse gases is the responsibility of <u>all nations</u> ." <sup>66</sup>

## **S6. ALGORITHMIC TEXTUAL ANALYSIS OF EXXONMOBIL CORP'S FLAGSHIP REPORTS**

Our key findings concerning ExxonMobil's advertorials are replicated in other ExxonMobil Corp public AGW communications.

We analyzed all of the company's known and available flagship reports concerning AGW spanning 2002-19. Specifically, from ExxonMobil Corp's 2020 listing of 'Publications and reports', we identified reports pertaining, in whole or in part, to AGW, AGW mitigation, and/or greenhouse gas emissions<sup>120</sup>. By way of ExxonMobil Corp webpages (only recent years of reports are made available), digital archives of ExxonMobil Corp webpages (via Wayback Machine), and other online and private collections, we obtained and analyzed the following editions of those reports (see table S1):

- *Corporate Citizenship Reports*, 2002-16 (discontinued after 2016, replaced by *Sustainability Report*)
- *Sustainability Report*, 2017 (this is the only edition at the time of analysis)
- *Outlook For Energy*, 2005-19 (except 2008 and 2011, which could not be located)
- *Energy & Carbon Summary*, 2017-18 (these are the only editions at the time of analysis)
- *Innovating Energy Solutions*, 2019 (this is the only edition at the time of analysis)

In the case of *Corporate Citizenship Reports* and *Outlook For Energy* reports, which are broad in scope, only sections primarily concerned with AGW, AGW mitigation, and/or greenhouse gas emissions were extracted for analysis, as indicated in table S1.

All documents were aggregated into a single corpus, pre-processed (this yielded a flagship report corpus comprising 113,695 words), and algorithmically analyzed according to the same protocols applied to advertorials: corpus comparison to internal and peer-reviewed publications (using frequency score (FS) and Dunning Log-Likelihood (LL) ratio  $G^2$  score); and collocation analysis using the logDice statistic. Notable results of these analyses are summarized in the following subsections.

**Table S16.** Inventory of the five ExxonMobil Corp flagship reports analyzed: *Corporate Citizenship Reports/Sustainability Report*, *Outlook For Energy*, *Energy & Carbon Summary*, and *Innovating Energy Solutions*. Shown for each report are the editions (years) retrieved and the sections (chapter titles and corresponding pages) analyzed. “NA” = report not located. “-” = no report published, to our knowledge, at the time of analysis.

Year	<i>Corporate Citizenship Reports/Sustainability Report</i>	<i>Outlook For Energy</i>	<i>Energy &amp; Carbon Summary</i>	<i>Innovating Energy Solutions</i>
2002	"Addressing climate-change risk"; "Energy research"; "Environmental performance" (p.9-14)	-	-	-
2003	"Greenhouse gas emissions"; "Advanced fuels and vehicle systems research"; "Fuel cell research"; "Global Climate and Energy Project (GCEP)" (p.10-12)	-	-	-
2004	"Climate change" indexed pages (p.3, 22, 24, 25, 29)	-	-	-
2005	"Environmental performance" (p.20-35)	"CO <sub>2</sub> growth"; "Technology critical to efficiency improvements" (p.18-19)	-	-
2006	"Environmental performance" (p.14-23)	"Global CO <sub>2</sub> emissions"; "Technology options for reducing CO <sub>2</sub> "; "CO <sub>2</sub> mitigation options"; "Meeting the world's energy needs" (p.22-25)	-	-
2007	"Environmental performance" (p.14-21)	"World energy and CO <sub>2</sub> emissions"; "Global CO <sub>2</sub> emissions" (p.22-23)	-	-
2008	"Managing climate change risks" (p.30-33)	NA	-	-
2009	"Managing climate change risks" (p.30-35)	"Managing emissions" (p.22-33)	-	-
2010	"Managing climate change risks" (p.32-37)	"Greenhouse gas emissions" (p.32-37)	-	-
2011	"Managing climate change risks" (p.22-25)	NA	-	-
2012	"Managing climate change risks" (p.28-33)	"Emissions" (p.32-35)	-	-
2013	"Managing climate change risks" (p.52-59)	"Emissions" (p.32-35)	-	-
2014	"Managing climate change risks" (p.33-39)	"Emissions" (p.32-33)	-	-
2015	"Managing climate change risks" (p.29-41)	"A shift in the power generation sector"; "Emissions" (p.36-41)	-	-
2016	"Managing climate change risks" (p.16-24)	"Lowering emissions" (p.48-51)	-	-
2017	"Managing climate change risks" (p.16-19)	"Emissions" (p.30-33)	-	-
2018	-	"Emissions"; "Pursuing a 2 °C pathway" (p.29-31, 44-53)	Full report	-
2019	-	"Dual challenge"; "Emissions" (p.3, 37-46)	Full report	Full report

### S6.1. “Risk” rhetoric in ExxonMobil Corp’s flagship reports

FS and LL analyses identify “risk(s)”, “climate change risks”, “risks of climate change”, etc., to be among the most statistically overused terms in ExxonMobil Corp’s flagship reports, compared to both their internal and peer-reviewed publications (table S17). Collocation analysis reveals that across these flagship reports, by far the highest scoring collocate of “climate change” and “global warming” is “risk(s)” (table S18). (Note that, for clarity, we here present the results of FS, LL, and collocation analyses in which all flagship reports were aggregated into a single corpus. Substantively the same results are obtained by treating each type of report as a separate corpus.)

**Table S17.** “Risk” rhetoric: highly divergent terms invoking “risk” in ExxonMobil Corp flagship reports, versus internal and peer-reviewed publications, by Log-Likelihood ratio ( $G^2$ ) and Frequency Score (FS). P-values <0.001 for all  $G^2$  and FS scores.

ExxonMobil Corp’s flagship reports often say:						
	Flagship	Internal	Peer-reviewed	$G^2$ (Int./P.r.)	FS (Int./P.r.)	Example
risk(s)	396	7	261	322.03 / 768.61	0.97 / 0.91	"A global approach to the <u>risk</u> posed by rising greenhouse gas emissions is needed that recognizes energy’s importance to the world’s economies." <sup>121</sup>
climate (change) risk(s)/ risk(s) of climate	213	0	10	203.92 / 768.25	1 / 0.99	"Recognizing the <u>risk of climate</u> change, we are taking actions to improve efficiency and reduce greenhouse gas emissions in our operations." <sup>122</sup>
managing climate change risks	52	0	0	49.78 / 206.76	1 / 1	" <u>Managing climate change risks</u> . Climate change risk management strategy. Society continues to face the dual challenge of meeting the world’s growing energy demand, while simultaneously addressing the risks of climate change." <sup>123</sup>
longterm	100	17	282	31.61 / 41.46	0.78 / 0.69	"ExxonMobil is engaged in the public discussion to create national and international policies to address climate change risks. Recognizing the <u>long-term</u> nature of these risks..." <sup>124</sup>
address the risks of climate	19	0	0	18.19 / 75.55	1 / 1	"Many uncertainties exist concerning the future of energy demand and supply, including potential actions that societies may take to <u>address the risks of climate</u> change." <sup>125</sup>

**Table S18.** Three strongest collocates of “climate change” and “global warming” in Mobil advertorials, ExxonMobil Corp advertorials, and ExxonMobil Corp flagship reports, by logDice score.

Mobil advertorials		ExxonMobil Corp advertorials		Flagship reports	
Collocate	logDice	Collocate	logDice	Collocate	logDice
science	11.46	risk(s)	13.01	risk(s)	13.79
gases	11.31	address	11.86	managing	12.78
debate	11.24	human	11.57	policy	12.72

*S6.2. Discourse of personal responsibility in ExxonMobil Corp’s flagship reports*

Table S19 (top half) collates terms in ExxonMobil Corp’s flagship reports that (a) based on our frame package analysis of advertorials, are characteristic of a Personal Responsibility frame; and (b) are highly divergent between flagship reports and internal and/or peer-reviewed documents according to LL and FS analyses. As with advertorials, we observe that ExxonMobil Corp’s flagship reports disproportionately employ terms that present consumer demand for energy as the cause of fossil fuel production, greenhouse gas emissions, and/or AGW; and disproportionately introduce terms conveying individual and/or demand-side actions as accountable for mitigating AGW. By contrast, Exxon and ExxonMobil Corp’s internal and/or academic communications disproportionately recognize AGW and/or greenhouse gases as also an upstream problem caused by fossil fuel supply and burning.

**Table S19.** Rhetoric of Personal Responsibility: Highly divergent terms in (top) ExxonMobil Corp flagship reports, by Log-Likelihood ratio ( $G^2$ ) and Frequency Score (FS), characteristic of a Personal Responsibility frame. Example quotations illustrate how flagship reports use these terms to disproportionately present: (a) consumer demand for energy as the cause of – and culpable for – fossil fuel use, greenhouse gas emissions, and/or AGW; and (b) individual/demand-side actions as accountable for mitigating AGW. By contrast, divergent terms in (bottom) internal and/or peer-reviewed documents often articulate the causality and culpability of fossil fuel combustion. P-values: \* <0.005; \*\* <0.05; \*\*\* $\geq$ 0.05; otherwise, <0.001 for all  $G^2$  and FS scores.

ExxonMobil Corp's flagship reports often say:						
	Flagship	Internal	Peer-reviewed	$G^2$ (Int./P.r.)	FS (Int./P.r.)	Example
efficient(t/cy/tly)	570	14	809	440.63 / 634.69	0.96 / 0.82	"ExxonMobil is delivering solutions that enable our customers to reduce their emissions and improve their energy efficiency..." <sup>123</sup>
demand (to) meet	455	21	422	304.06 / 718.96	0.93 / 0.87	"Globally, rising energy demand will result in higher energy-related CO <sub>2</sub> emissions through 2030..." <sup>126</sup>
challenge(s)	224	2	98	195.42 / 523.8	0.99 / 0.94	"As we seek to produce oil and natural gas to meet growing global energy demand..." <sup>127</sup>
vehicles	140	5	100	100.2 / 260.12	0.95 / 0.9	"This is society's dual challenge. Billions of people need reliable, affordable energy every day, but their use of energy is contributing to CO <sub>2</sub> emissions." <sup>125</sup>
consumers	83	0	240	79.46 / 32.6	1 / 0.69	"As the number of vehicles in the world continues to rise, energy efficiency in the transportation sector will become increasingly important. According to the International Energy Agency, approximately 90 percent of petroleum-related GHG emissions are generated when customers use our products..." <sup>124</sup>
energy demand	69	0	33	66.06 / 155.66	1 / 0.93	"...the combustion of fuels by consumers generates the majority of GHG emissions..." <sup>121</sup>
reduce emissions the world	135	14	59	63.45 / 315.82	0.86 / 0.94	"Increasingly, the world's CO <sub>2</sub> emissions will be driven by developing nations. Overall, non-OECD emissions are likely to rise about 50 percent, as energy demand rises by about two-thirds." <sup>128</sup>
customers	61	0	25	58.4 / 146.24	1 / 0.94	"[P]rice stability...provides a clear incentive for all consumers to increase efficiency and reduce emissions." <sup>129</sup>
demand growth	149	26	132	45.83 / 242.82	0.78 / 0.88	"...rising greenhouse gas emissions resulting from the world's enormous requirements for fossil fuels..." <sup>130</sup>
global demand	42	0	3	40.21 / 145.84	1 / 0.99	"ExxonMobil develops and produces a range of petroleum-based products that help our customers reduce their greenhouse gas emissions and improve efficiency." <sup>131</sup>
living standards	31	0	5	29.68 / 95.72	1 / 0.98	"Renewables and nuclear energy see strong growth...to meet demand growth through 2040. Natural gas grows the most of any energy type, reaching a quarter of all demand." <sup>132</sup>
natural gas demand	28	0	4	26.81 / 88.4	1 / 0.98	"The benefits of natural gas. Global demand for cleaner-burning natural gas is expected to increase by more than 50 percent by 2030, making it the fastest-growing major energy source for power generation." <sup>124</sup>
footprint	25	0	1	23.93 / 91.22	1 / 0.99	"Close to 85 percent of the increase in CO <sub>2</sub> emissions [through 2030] will come from developing countries where economic growth and improved living standards are creating huge increases in energy demand." <sup>133</sup>
needs	23	0	1	22.02 / 83.43	1 / 0.99	"Natural gas will meet a growing share of our energy needs through 2030...Total natural gas demand in the United States and Europe will follow a similar pattern..." <sup>129</sup>
energy needs	20	0	3	19.15 / 62.6	1 / 0.98	"[T]he core sustainability challenge for the energy industry is how to provide the energy that enables economic development while reducing the environmental footprint associated with energy use." <sup>124</sup>
	89	22	71	17.2 / 155.02	0.71 / 0.89	"Fossil fuels – oil, natural gas and coal – will continue to meet most of the world's needs [through 2030]." <sup>129</sup>
	29	4	6	11.12 / 85.01	0.82* / 0.97	ExxonMobil is "taking action to position ourselves to help meet future global energy needs. For example, we are: Expanding supply of cleaner-burning natural gas..." <sup>127</sup>
Internal and/or peer-reviewed documents often say:						
fossil fuel(s)	15	198	508	-288.59 / -73.18	0.04 / 0.16	"[T]here is general scientific agreement that the most likely manner in which mankind is influencing the global climate is through carbon dioxide release from the burning of fossil fuels." <sup>110</sup>
natuna	2	67	NA	-113.33 / NA	0.02 / NA	"[T]he burning of fossil fuels is linked to both climate change and air pollution..." <sup>134</sup>
fossil fuel combustion	0	48	NA	-92.79 / NA	0 / NA	"This would make Natuna the world's largest point source emitter of CO <sub>2</sub> and raises concern for the possible incremental impact of Natuna on the CO <sub>2</sub> greenhouse problem." <sup>104</sup>
due to	44	89	731	-45.32 / -52.39	0.23 / 0.28	"[T]here is the potential for our [climate] research to attract the attention of the popular news media because of the connection between Exxon's major business and the role of fossil fuel combustion in contributing to the increase of atmospheric CO <sub>2</sub> ." <sup>108</sup>
shale	8	41	NA	-43.3 / NA	0.11 / NA	"The CO <sub>2</sub> concentration in the atmosphere has increased...The most widely held theory is that: the increase is due to fossil fuel combustion." <sup>106</sup>
fossil fuel use	0	13	22	-25.13 / -6.48**	0 / 0***	"About three-quarters of the anthropogenic emissions of CO <sub>2</sub> to the atmosphere during the past 20 years is due to fossil fuel burning." <sup>107</sup>
fossil fuel consumption	0	10	NA	-19.33 / NA	0 / NA	"The quantity of CO <sub>2</sub> emitted by various fuels is shown in Table 1...They show the high CO <sub>2</sub> /energy ratio for coal and shale..." <sup>106</sup>
fossil fuel emissions	0	NA	54	NA / -15.91	NA / 0	"[F]or scenarios with higher fossil fuel use (hence, higher carbon dioxide emissions..." <sup>107</sup>
fossil fuel co2	1	NA	64	NA / -12.5	NA / 0.09*	"The most widely held theory is that...[t]he present trend of fossil fuel consumption will cause dramatic environmental effects before the year 2050." <sup>106</sup>
fossil fuel burning	0	NA	40	NA / -11.78	NA / 0*	"We use our Integrated Science Model to...estimate the time variation fossil fuel emissions of CO <sub>2</sub> ...required to match the [IPCC] concentration stabilization scenarios." <sup>113</sup>
						"This long tail on the fossil fuel CO <sub>2</sub> forcing of climate may well be more significant to the future glacial/interglacial timescale evolution of Earth's climate..." <sup>112</sup>
						"CO <sub>2</sub> emissions from fossil fuel burning are virtually certain to be the dominant factor determining CO <sub>2</sub> concentrations during the 21 <sup>st</sup> century." <sup>135</sup>

## S7. CATALOG OF ANALYZED DOCUMENTS

Raw data (original PDF internal documents, peer-reviewed publications, and advertorials) for this study cannot be reproduced due to copyright restrictions. However, tables S20-22 present catalogs of all 180 analyzed documents, which can be obtained at the following public archives:

- All analyzed advertorials can be downloaded from the ProQuest Historical Newspaper Database<sup>136</sup>. Many can also be downloaded from PolluterWatch<sup>137</sup>.
- All analyzed internal documents can be downloaded from (one or more of) ExxonMobil Corp<sup>138</sup>, *InsideClimate News*<sup>139</sup>, and Climate Investigations Center<sup>140</sup>.
- All analyzed peer-reviewed documents can be obtained from corresponding journals and conference proceedings.

A catalog of analyzed flagship reports is presented in table S16 above.

**Table S20.** Catalog of analyzed advertorials.

Date	Authors	Title
21 December 1972	Mobil Oil	A trio glows in Brooklyn
05 April 1973	Mobil Oil	The profits of doom
16 August 1984	Mobil Oil	Lies they tell our children
03 November 1988	Mobil Oil	musings of a fossil fuel person...
06 July 1989	Mobil Oil	People Who Live in Greenhouses...
09 April 1992	Mobil Oil	Boy, we wish we'd said that!
25 February 1993	Mobil Oil	Apocalypse no
11 May 1995	Mobil Oil	Electric vehicles: a promise too far
28 September 1995	Mobil Oil	The sky is not falling
18 July 1996	Mobil Oil	Less heat, more light on climate change
25 July 1996	Mobil Oil	With Climate Change, What We Don't Know Can't Hurt Us
01 August 1996	Mobil Oil	Climate Change: We're all in this together
12 December 1996	Mobil Oil	A policy agenda for tomorrow
06 March 1997	Mobil Oil	Stop, look and listen before we leap
23 June 1997	Mobil Oil	Climate change: Let's get it right
31 July 1997	Mobil Oil	The Senate speaks
14 August 1997	Mobil Oil	When the facts don't square with the theory, throw out the facts
23 October 1997	Mobil Oil	Global climate change
30 October 1997	Mobil Oil	Reset the alarm
06 November 1997	Mobil Oil	Science: what we know and don't know
13 November 1997	Mobil Oil	Climate change: a prudent approach
20 November 1997	Mobil Oil	Climate change: where we come out
04 December 1997	Mobil Oil	Climate change: a degree of uncertainty
11 December 1997	Mobil Oil	Let's not forget the will of the senate
18 December 1997	Mobil Oil	The Kyoto Conference
29 January 1998	Mobil Oil	Post Kyoto, what's next?
02 April 1998	Mobil Oil	Voluntary 'can do'
10 September 1998	Mobil Oil	The Kyoto Protocol: too many gaps
05 November 1998	Mobil Oil	The Kyoto Protocol: a painful response
15 April 1999	Mobil Oil	Helping Earth breathe easier
10 June 1999	Mobil Oil	King of the road?
29 July 1999	Mobil Oil	Where we are and where we may be heading
05 August 1999	Mobil Oil	Some ways to make a difference
12 August 1999	Mobil Oil	Scenarios for stabilization
19 August 1999	Mobil Oil	Lessons learned
16 March 2000	ExxonMobil Corp	Do no harm
23 March 2000	ExxonMobil Corp	Unsettled Science
30 March 2000	ExxonMobil Corp	The Promise of Technology
06 April 2000	ExxonMobil Corp	The Path Forward on Climate Change
10 August 2000	ExxonMobil Corp	Political cart before a scientific horse
24 August 2000	ExxonMobil Corp	Facts and fundamentals
14 December 2000	ExxonMobil Corp	Fleet changes, but slowly
21 December 2000	ExxonMobil Corp	Planting the future
10 April 2001	ExxonMobil Corp	Moving past Kyoto...
17 April 2001	ExxonMobil Corp	...to a sounder climate policy

03 May 2001	ExxonMobil Corp	Renewable energy: today's basics
10 May 2001	ExxonMobil Corp	Renewable energy: tomorrow's promise
19 July 2001	ExxonMobil Corp	Action, not talk: cogeneration and climate
03 October 2002	ExxonMobil Corp	Managing greenhouse gas emissions
22 November 2002	ExxonMobil Corp	A responsible path forward on climate
06 February 2003	ExxonMobil Corp	The global climate and energy challenge
08 January 2004	ExxonMobil Corp	A century of deep-water research
22 January 2004	ExxonMobil Corp	Weather and climate
05 February 2004	ExxonMobil Corp	Directions for climate research
11 May 2005	ExxonMobil Corp	More Energy and Lower Emissions?
14 June 2005	ExxonMobil Corp	More Energy and Lower Emissions?
07 July 2005	ExxonMobil Corp	More Energy and Lower Emissions?
04 August 2005	ExxonMobil Corp	Research Into Climate Solutions
03 August 2006	ExxonMobil Corp	Changing the Game
19 December 2006	ExxonMobil Corp	Multiplier Effects
25 January 2007	ExxonMobil Corp	Taking action to reduce greenhouse gas emissions
09 February 2007	ExxonMobil Corp	Saving Energy and Reducing Greenhouse Gas Emissions
14 February 2007	ExxonMobil Corp	Let's Talk About Climate Change
15 February 2007	ExxonMobil Corp	Addressing the Risks of Climate Change
16 February 2007	ExxonMobil Corp	Let's Talk About Climate Change
24 May 2007	ExxonMobil Corp	Values at Work
18 October 2007	ExxonMobil Corp	answering energy questions
13 March 2008	ExxonMobil Corp	The Fuels of the Future
03 April 2008	ExxonMobil Corp	Energy Efficiency--Once Quart at a Time
03 June 2008	ExxonMobil Corp	More Energy. Fewer Emissions. With Technology, We Can Do Both
24 June 2008	ExxonMobil Corp	Vehicles of Change
20 January 2009	ExxonMobil Corp	Provide Energy. Protect the Environment. A dual challenge for all of us.
14 April 2009	ExxonMobil Corp	Many Parts Working Together
22 May 2009	ExxonMobil Corp	Citizenship for the Long Term
29 June 2009	ExxonMobil Corp	Citizenship For the Long Term
15 October 2009	ExxonMobil Corp	Tackling Climate Risks With Technology

**Table S21.** Catalog of analyzed internal documents.

Date	Authors	Title
31 October 1977	Shaw, H. to Harrison, J. W.	Environmental Effects of Carbon Dioxide
06 June 1978	Black, J. to Turpin, F. G. (cc: Alpert, N. et al.)	The Greenhouse Effect
07 December 1978	Shaw, H. to David Jr., E. E.	Untitled (request for a credible scientific team)
07 March 1978	Weinberg, H. N. to Gornowski, E. J.	CO2
26 March 1979	Garvey, E. A., Shaw, H., Broecker, W. S., Takahashi, T. presentation to Machta, L.	Proposed Exxon Research Program to Help Assess the Greenhouse Effect
16 October 1979	Mastracchio, R. L. to Hirsch, R. L. (cc: Black, J. F. et al.)	Controlling Atmospheric CO2
19 November 1979	Shaw, H. to Weinberg, H. N. (cc: Werthamer, N. R.)	Research in Atmospheric Science
29 January 1980	Eckelmann, W. R. to O'Loughlin, M. E. J. (cc: David, E. E. et al.)	Exxon's View and Position on "Greenhouse Effect"
09 June 1980	Weinberg, H. N. to Shaw, H. and Werthamer, N. R.	Greenhouse Program
08 July 1980	Werthamer, N. R. to Weinberg, H. N.	CO2 Greenhouse Communications Plan
18 December 1980	Shaw, H. to Kett, R. K. (cc: McCall, P. P. et al.)	Exxon Research and Engineering Company's Technological Forecast CO2 Greenhouse Effect
03 February 1981	Gervasi, G. R. to Northington, G. A. (cc: Preston, R. L. et al.)	CO2 Emissions Natuna Gas Project
05 February 1981	Long, G. H. to Lucceshi, P. J. et al. (cc: Barnum, R. E. et al.)	Atmospheric CO2 Scoping Study
15 May 1981	Shaw, H. to David Jr., E. E. (cc: Barnum, R. E. et al.)	CO2 Position Statement
18 August 1981	Cohen, R. W. to Glass, W. (cc: Weinberg, H. N. et al.)	Untitled (catastrophic effects letter)
18 June 1982	Natkin, A. M. to Weinberg, H. N. (cc: Forshee, M. E. et al.)	CRL/CO2 Greenhouse Program
14 July 1982	Cohen, R. W. to Kimon, P. (cc: Berner, R. et al.)	Untitled (Esso project terminated letter)
21 July 1982	Weinberg, H. N., Cohen, R. W., Callegari, A. J., Flannery, B., et al.	CO2-Greenhouse Effect; Corporate Research Climate Modeling
02 September 1982	Cohen, R. W., Levine, D. G. to Natkin, A. M. (cc: Callegari, A. J. et al.)	Untitled (consensus on CO2 letter)

12 November 1982	Glaser, M. B. to Cohen, R. W. et al.	CO2 "Greenhouse" Effect
17 October 1983	Natkin, A. M. to Preston, R. L. (Esso Eastern) (cc: Gervasi, G. R. et al.)	Untitled (ocean storage environmental concerns letter)
27 October 1983	Gervasi, G. R. to Downing, R. G. et al. (cc: Gates, D. F. et al.)	Background Paper Environmental Issues Natuna Gas Project
1984	Flannery, B., Callegari, A. J., Nair, B., Roberge, W. G.	The Fate of CO2 from the Natuna Gas Project if Disposed of by Subsea Sparging
02 February 1984	Callegari, A. J.	Corporate Research Program in Climate/CO2-Greenhouse
28 March 1984	Shaw, H.	CO2 Greenhouse and Climate Issues (EUSA/ER&E Environmental Conference, Florham Park, New Jersey)
07 May 1985	Shaw, H., Henrikson, F. W. to Lab Directors/Program Managers (cc: Cohen, R. W. et al.)	CR Interactions (handout for June 12th meeting with Lee Raymond)
04 October 1985	Flannery, B. P.	CO2 Greenhouse Update 1985
08 March 1988	Carlson, J. M. to Levine, D. G.	The Greenhouse Effect
02 February 1989	Levine, D. G.	Potential Enhanced Greenhouse Effects, Status and Outlook (Presentation to the Board of Directors of Exxon Corp)
Fall 1989	Flannery, B. P.	Greenhouse Science (CONNECTIONS ExxonMobil publication - "Proprietary information for company use only")
21 December 1994	Bernstein, L. S. to Members of Global Climate Coalition	Primer on Climate Change Science
18 March 2002	Flannery, B. P. to Cooney, P. and Marburger, J. (cc: Randol, A. G.)	Activities

**Table S22.** Catalog of analyzed peer-reviewed publications.

Year	Authors	Title	Publication
1982	Garvey, E. A., Prael, F., Nazimek, K., Shaw, H.	Exxon global CO2 measurement system	IEEE Transactions on Instrumentation and Measurement
1983	Hoffert, M.I., Flannery, B. P., Callegari, A. J., Hseih, C. T., Wiscombe, W.	Evaporation-limited tropical temperatures as a constraint on climate sensitivity	Journals of the Atmospheric Sciences
1984	Flannery, B. P.	Energy balance models incorporating transport of thermal and latent energy	Journals of the Atmospheric Sciences
1984	Flannery, B. P., Callegari, A. J., Hoffert M. I.	Energy balance models incorporating evaporative buffering of equatorial thermal response	Geophysical Monograph Series: Climate Processes and Climate Sensitivity
1985	Flannery, B. P., Callegari, A. J., Hoffert, M. I., Hseih, C. T., Wainger, M. D.	CO2 driven equator-to-pole paleotemperatures: predictions of an energy balance model with and without a tropical evaporation buffer	The Carbon Cycle and Atmospheric CO2: Natural Variations Archean to Present, Geophysical Monograph 32
1985	Hoffert, M. I., Flannery, B. P. (eds. MacCracken, M. C., Luther, F. M.)	Model Projections of the Time-Dependent Response to Increasing Carbon Dioxide	Projecting the Climatic Effects of Increasing Carbon Dioxide, United States Department of Energy
1988	Thomas, E. R., Denton, R. D.	Conceptual studies for CO2/natural gas separation using the controlled freeze zone (CFZ) process	Gas Separation and Purification
1991	Kheshgi, H. S., Hoffert, M. I., Flannery, B. P.	Marine biota effects on the compositional structure of the world oceans	J. Geophys. Res.
1993	Kheshgi, H. S., White, B. S.	Effect of climate variability on estimation of greenhouse parameters: usefulness of a pre-instrumental temperature record	Quaternary Science Reviews
1993	Flannery, B. P., Kheshgi, H. S., Hoffert, M. I., Lapenis, A. G.	Assessing the effectiveness of marine CO2 disposal	Energy Convers. Mgmt
1993	Kheshgi, H. S., White, B. S.	Does recent global warming suggest an enhanced greenhouse effect?	Climatic Change
1994	Jain, A. K., Kheshgi, H. S., Wuebbles, D. J.	Integrated Science Model for Assessment of Climate Change	94-TP59. 08, Air and Waste Management Assoc.; also Lawrence Livermore Nat. Lab., UCRL-JC-116526, Natl. Technical Info Service, US Dept. of

			Commerce. Proceedings of the 87th Annual Meeting of the Air & Waste Management Association
1994	Kheshgi, H. S., Flannery, B. P., Hoffert, M. I., Lapenis, A. G.	The effectiveness of marine CO2 disposal	Energy
1995	Jain, A. K., Kheshgi, H. S., Hoffert, M. I., Wuebbles, D. J.	Distribution of radiocarbon as a test of global carbon cycle models	Global Biogeochem. Cycles
1995	Kheshgi, H. S.	Sequestering atmospheric carbon dioxide by increasing ocean alkalinity	Energy
1996	Santer, B. D., Wigley, T.M.L., Barnett, T.P., Anyamba, E.,..., Kheshgi, H.S. (Contributor), et al.	Detection of Climate Change and Attribution of its Causes	Intergovernmental Panel on Climate Change Second Assessment Report, Chapter 8, Volume I
1996	Kheshgi, H. S., White, B.S.	Modelling ocean carbon cycle with a nonlinear convolution model	Tellus
1996	Kheshgi, H. S., Lapenis, A. G.	Estimating the accuracy of Russian paleotemperature reconstructions	Palaeogeography, Palaeoclimatology, Palaeoecology
1996	Kheshgi, H. S., Jain, A. K., Wuebbles, D. J.	Accounting for the missing carbon sink with the CO2 Fertilization Effect	Climatic Change
1996	Jain, A. K., Kheshgi, H. S., Wuebbles, D. J.	A globally aggregated reconstruction of cycles of carbon and its isotopes	Tellus
1996	Prince, R. C., Kheshgi, H. S.	Longevity in the deep	Trends in Ecology & Evolution
1997	Jain, A. K., Kheshgi, H. S., Wuebbles, D. J.	Is there an imbalance in the global budget of bomb-produced radiocarbon?	Journal of Geophysical Research
1997	Archer, D., Kheshgi, H., Maier-Reimer, E.	Multiple Timescales for the Neutralization of Fossil Fuel CO2	Geophysical Research Letters
1997	Kheshgi, H. S., Schlesinger, M. E., Lapenis, A. G.	Comparison of Paleotemperature Reconstructions as Evidence for the Paleo-Analog Hypothesis	Climatic Change
1997	Kheshgi, H.S., Jain, A. K., Wuebbles, D. J.	Analysis of proposed CO2 emission reductions in the context of stabilization of CO2 concentration	Proceedings of the Air & Waste Management Association's 90th Annual Meeting & Exhibition.
1998	Archer, D., Kheshgi, H., Maier-Reimer, E.	The dynamics of fossil fuel CO2 neutralization by marine CaCO3	Global Biogeochemical Cycles
1998	Hayhoe, K. A. S., Kheshgi, H. S., Jain, A. K., Wuebbles, D. J.	Trade-Offs in Fossil Fuel Use: The Effects of CO2 , CH4 and SO2 Aerosol Emissions on Climate	World Resource Review
1999	Kheshgi, H. S., Jain, A. K., Kotamarthi, V. R. Wuebbles, D. J.	Future Atmospheric Methane Concentrations in the Context of the Stabilization of Greenhouse Gas Concentrations	J. Geophys. Res.
1999	Kheshgi, H. S., Jain, A. K., Wuebbles, D. J.	Model-based estimation of the global carbon budget and its uncertainty from carbon dioxide and carbon isotope records	J. Geophys. Res.,
2000	Kheshgi, H. S., Prince, R. C., Marland, G.	The Potential of Biomass Fuels in the Context of Global Change: Focus on Transportation Fuels	Annual Review of Energy and the Environment
2000	Watson, R.,..., Kheshgi, H. et al. (eds. Watson, R. T. et al.)	Land Use, Land-Use Change, and Forestry	A Special Report of the Intergovernmental Panel on Climate Change
2000	Hayhoe, K. A. S., Jain, A. K., Kheshgi, H. S., Wuebbles, D. J.	Contribution of CH4 to Multi-Gas Reduction Targets: The Impact of Atmospheric Chemistry on GWPs	Non-CO2 Greenhouse Gases: Scientific Understanding, Control and Implementation, 425-432. Proceedings of the Second International Symposium, Noordwijkerhout, The Netherlands, 8-10 September 1999
2001	Bolin, B., Kheshgi, H. S.	On strategies for reducing greenhouse gas emissions	PNAS
2001	Kheshgi, H. S., B. S. White	Testing Distributed Parameter Hypotheses for the Detection of Climate Change	Journal of Climate
2001	Prentice, C., Farquhar, G., Fasham, M., Goulden, M., Heimann, M., Jaramillo, V., Kheshgi, H., Quéré, C. L., Scholes, R., Wallace, D.	The carbon cycle and atmospheric CO2	Intergovernmental Panel on Climate Change Third Assessment Report, Working Group 1, Chapter 3
2001	Mitchell, J. F. B.,..., Kheshgi, H. S.	Detection of Climate Change and	IPCC TAR WGI Ch12

2001	(Contributing Author), et al. Albritton, D. L.,...,Kheshgi, H.S. (Contributing Author), et al.	Attribution of its Causes Technical Summary	Intergovernmental Panel on Climate Change Third Assessment Report, Working Group 1, Summary for Policymakers and Technical Summary
2001	Kauppi, P.,...,Kheshgi, H. S. (Contributing Author), et al.	Technical and Economic Potential of Options to Enhance, Maintain and Manage Biological Carbon Reservoirs and Geo-Engineering	Intergovernmental Panel on Climate Change Third Assessment Report, Working Group 3, Chapter 4
2001	Toth, F. L.,..., Flannery, B. (Lead Author), et al.	Decision Making Frameworks	Intergovernmental Panel on Climate Change Third Assessment Report, Working Group 3, Chapter 10
2002	Hayhoe, K. A. S., Kheshgi, H. S., Jain, A. K., Wuebbles, D. J.	Substitution of natural gas for coal: climatic effects of utility sector emissions	Climatic Change
2002	Hoffert, M. I., Caldeira, K., Benford, G., Criswell, D. R., Green, C., Herzog, H., Jain, A. K., Lackner, K. S., Lewis, J. S., Lightfoot, H. D., Manheimer, W., Mankins, J. C., Mauel, M. E., Perkins, L. J., Schlesinger, M. E., Volk, T., Wigley, T. M. L.	Advanced technology paths to global climate stability: energy for a greenhouse planet	Science
2003	Kheshgi, H. S., Jain, A. K.	Projecting future climate change: implications of carbon cycle model intercomparisons	Global Biogeochemical Cycles
2003	Le Quéré, C., Aumont, O., Bopp, L., Bousquet, P., Ciais, P., Francey, R., Heimann, M., Keeling, C. D., Keeling, R. F., Kheshgi, H., Peylin, P., Piper, S. C., Prentice, I. C., Rayner, P. J.	Two decades of ocean CO2 sink and variability	Tellus
2004	Kheshgi, H. S., Archer, D.	A non-linear convolution model for the evasion of CO2 injected into the deep ocean	Journal of Geophysical Research
2004	Kheshgi, H. S.	Evasion of CO2 injected into the ocean in the context of CO2 stabilization	Energy
2004	Kheshgi, H. S.	Ocean carbon sink duration under stabilization of atmospheric CO2: a 1,000-year time-scale	Geophysical Research Letters
2005	Kheshgi, H. S., Prince, R.	Sequestration of fermentation CO2 from ethanol production	Energy
2005	Kheshgi, H.S., Smith, S.J., Edmonds, J.A.	Emissions and Atmospheric CO2 Stabilization: Long-term Limits and Paths	Mitigation and Adaptation Strategies
2005	Prince, R.C., Kheshgi, H.S.	The photobiological production of hydrogen: potential efficiency and effectiveness as a renewable fuel	Critical Reviews in Microbiology
2005	Caldeira, K., Akai, M., Brewer, P., Chen, B., Haugan, P., Iwama, T., Johnston, P., Kheshgi, H., Li, Q., Ohsumi, T., Poertner, H., Sabine, C., Shirayama, Y., Thomson, J.	Ocean storage (Chapter 6)	IPCC Special Report on Carbon Dioxide Capture and Storage
2007	Barker, T., Bashmakov, I., Alharthi, A., Amann, M., Cifuentes, L., Drexhage, J., Duan, M., Edenhofer, O., Flannery, B., Grubb, M., Hoogwijk, M., Ibitoye, F. I., Jepma, C. J., Pizer, W. A.	Mitigation from a cross-sectoral perspective	Intergovernmental Panel on Climate Change Fourth Assessment Report, Working Group 3, Chapter 11
2007	Kheshgi, H. S. (eds. Schlesinger, M. E., Kheshgi, H., Smith, J. B., de la Chesnaye, F. C., Reilly, J. M., Wilson, T. and Kolstad, C.)	Probabilistic estimates of climate change: methods, assumptions and examples (p. 49-61)	Human-Induced Climate Change: An Interdisciplinary Assessment
2007	Kheshgi, H. S. (Coordinating Editor for Part 1) (eds. Schlesinger, M. E., Kheshgi, H., Smith, J. B., de la Chesnaye, F. C., Reilly, J. M., Wilson, T. and Kolstad, C.)	Part 1, Climate System Science (p. 2-3)	Human-Induced Climate Change: An Interdisciplinary Assessment
2007	Ribeiro, S. K.,..., Kheshgi, H. (Review Editor), et al.	Transport and its infrastructure	Intergovernmental Panel on Climate Change Fourth Assessment Report, Working Group 3, Chapter 5
2009	Lively, R. P., Chance, R. R., Kelley,	Hollow fiber adsorbents for CO2 removal	Ind. Eng. Chem. Res.

	Deckman, H. W., Drese, J. H., Jones, C. W., Koros, W. J.	from flue gas	
2009	Jain, A., Yang, X., Kheshgi, H., McGuire, A. D., Post, W., Kicklighter, D.	Nitrogen attenuation of terrestrial carbon cycle response to global environmental factors	Global Biogeochemical Cycles
2009	Benge, G.	Improving wellbore seal integrity in CO2 injection wells	Energy Procedia
2009	Hershkowitz, F., Deckman, H. W., Frederick, J. W., Fulton, J. W., Socha, R. F.	Pressure swing reforming: a novel process to improve cost and efficiency of CO2 capture in power generation	Energy Procedia
2009	Kheshgi, H. S., Crookshank, S., Cunha, P., Lee, A., Bernstein, L., Siveter, R.	Carbon capture and storage business models	Energy Procedia
2009	Northrop, P. S., Valencia, J. A.	The CFZTM process: a cryogenic method for handling high-CO2 and H2 S gas reserves and facilitating geosequestration of CO2 and acid gases	Energy Procedia
2009	Parker, M. E., Meyer, J. P., Meadows, S.	Carbon dioxide enhanced oil recovery injection operations technologies	Energy Procedia
2009	Ritter, K., Siveter, R., Lev-On, M., Shires, T., Kheshgi, H.	Harmonizing the quantification of greenhouse gas emission reductions through oil and gas industry project guidelines	Energy Procedia
2009	Wilkinson, J., Szafranski, R., Lee, K. -S., Kratzing, C.	Subsurface design considerations for carbon dioxide storage	Energy Procedia
2009	Xiao, Y., Xu, T., Pruess, K.	The effects of gas-fluid-rock interactions on CO2 injection and storage: insights from reactive transport modeling	Energy Procedia
2011	Flannery, B.P.	Comment (on the scale-up of carbon dioxide capture and storage technology systems)	Energy Economics
2011	Burgers, W. F. J., Northrop, P. S., Kheshgi, H. S., Valencia, J. A.	Worldwide development potential for sour gas	Energy Procedia
2011	Parker, M. E., Northrop, S., Vaencia, J. A., Foglesong, R. E., Duncan, W. T.	CO2 management at ExxonMobil's LaBarge field, Wyoming, USA	Energy Procedia
2012	Kheshgi, H., Thomann, H., Bhole, N. B., Hirsh, R. B., Parker, M. E., Teletzke, G. F.	Perspectives on CCS cost and economics	SPE Economics & Management
2014	Allen, R. J., Landuyt, W.	The vertical distribution of black carbon in CMIP5 models: Comparison to observations and the importance of convective transport	J. Geophys. Res. Atmos.
2014	Song, Y., Jain, A. K., Landuyt, W., Kheshgi, H. S., Khanna, M.	Estimates of Biomass Yield for Perennial Bioenergy Grasses in the United States	BioEnergy Research
2014	Fischedick M., Roy, J., Abdel-Aziz, A., Acquaye, A., Allwood, J. M., Ceron, J. - P., Geng, Y., Kheshgi, H., Lanza, A., Perczyk, D., Price, L., Santalla, E., Sheinbaum, C., Tanaka, K. (eds. O. Edenhofer, R. Pichs-Madruga, Y. Sokona, E. Farahani, S. Kadner, K. Seyboth, A. Adler, I. Baum, S. Brunner, P. Eickemeier, B. Kriemann, J. Savolainen, S. Schlömer, C. von Stechow, T. Zwickel and J.C. Minx)	Industry	Intergovernmental Panel on Climate Change Fifth Assessment Report, Working Group 3, Chapter 11
2014	Arent, D. J.,..., Kheshgi, H. (Review Editor), et al.	Key economic sectors and services	Intergovernmental Panel on Climate Change Fifth Assessment Report, Working Group 2, Chapter 10

## SUPPLEMENTAL REFERENCES

1. Supran, G., and Oreskes, N. (2017). Assessing ExxonMobil's climate change communications (1977–2014). *Environ. Res. Lett.* *12*, 084019.
2. Supran, G., and Oreskes, N. (2020). Addendum to “Assessing ExxonMobil's climate change communications (1977–2014).” *Environ. Res. Lett.* *15*, 119401.
3. Exxon (1989). FUEL (Advertisement, industry sponsors possibly include Exxon). *The New York Times Magazine*.
4. I.R.I.S S.A. (2018). Readiris Corporate 17 (v.17.1.3). [www.irislink.com](http://www.irislink.com).
5. R Core Team (2019). R: A language and environment for statistical computing. R Foundation for Statistical Computing. [www.r-project.org](http://www.r-project.org).
6. Grün, B., and Hornik, K. (2011). topicmodels: An R Package for Fitting Topic Models. *J. Stat. Softw.* *40*, 1–30.
7. Grün, B., and Hornik, K. (2018). topicmodels (v0.2-8). (<https://perma.cc/S6PA-CUAC>).
8. Maier, D., Waldherr, A., Miltner, P., Wiedemann, G., Niekler, A., Keinert, A., Pfetsch, B., Heyer, G., Reber, U., Häussler, T., et al. (2018). Applying LDA Topic Modeling in Communication Research: Toward a Valid and Reliable Methodology. *Commun. Methods Meas.* *12*, 93–118.
9. Boussalis, C., and Coan, T.G. (2016). Text-mining the signals of climate change doubt. *Glob. Environ. Chang.* *36*, 89–100.
10. Mimno, D., Wallach, H.M., Talley, E., and Leenders, M. (2011). Optimizing Semantic Coherence in Topic Models. In *Proceedings of the 2011 Conference on Empirical Methods in Natural Language Processing*, pp. 262–272.
11. Sievert, C., and Shirley, K.E. (2014). LDAvis: A method for visualizing and interpreting topics. In *Proceedings of the Workshop on Interactive Language Learning, Visualization, and Interfaces* (Baltimore, MA, USA, 27 June 2014), pp. 63–70.
12. Walter, D., and Ophir, Y. (2019). News Frame Analysis: An Inductive Mixed-method Computational Approach. *Commun. Methods Meas.* *13*, 248–266.
13. Farrell, J. (2015). Corporate funding and ideological polarization about climate change. *Proc. Natl. Acad. Sci.* *113*, 92–97.
14. Krippendorff, K. (2004). Reliability in Content Analysis: Some Common Misconceptions and Recommendations. *Hum. Commun. Res.* *30*, 411–433.
15. Lombard, M., Snyder-Duch, J., and Bracken, C.C. (2002). Content Analysis in Mass Communication - Assessment of Reporting of Intercoder Reliability. *Hum. Commun. Res.* *28*, 587–604.
16. Freelon, D. ReCal2 Intercoder reliability online utility. [dfreelon.org](http://dfreelon.org). <https://perma.cc/T3AN-JETE>.
17. Campbell, J.L., Quincy, C., Osserman, J., and Pedersen, O.K. (2013). Coding in-depth semistructured interviews: Problems of unitization and intercoder reliability and agreement. *Sociol. Methods Res.* *42*, 294–320.
18. Manning, C.D., Raghavan, P., and Schütze, H. (2008). *An introduction to information retrieval*. (Cambridge University Press).
19. QSR International (2019). NVivo 12 (v.12.6.0) Qualitative Data Analysis Software.
20. Balossi, G. (2014). *A Corpus Linguistic Approach to Literary Language and Characterization: Virginia Woolf's The Waves*. (John Benjamins Publishing Company).
21. Rayson, P., and Garside, R. (2000). Comparing Corpora using Frequency Profiling. In *Proceedings of the workshop on comparing corpora*, pp. 1–6. (doi: 10.3115/1117729.1117730).
22. Touri, M., and Koteyko, N. (2015). Using corpus linguistic software in the extraction of news frames: towards a dynamic process of frame analysis in journalistic texts. *Int. J. Soc. Res. Methodol.* *18*, 599–614.
23. Klebanov, B.B., Diermeier, D., Beigman, E., and Diermeier, D. (2008). Automatic

- Annotation of Semantic Fields for Political Science Research. *J. Inf. Technol. Polit.* 5, 95–120.
24. Gorp, B. Van (2009). Strategies to Take Subjectivity Out of Framing Analysis. In *Doing News Framing Analysis: Empirical and Theoretical Perspectives*, J. A. Kuypers and P. D'Angelo, eds. (Routledge), pp. 84–109.
  25. Jaworska, S. (2018). Change But no Climate Change: Discourses of Climate Change in Corporate Social Responsibility Reporting in the Oil Industry. *Int. J. Bus. Commun.* 55, 194–219.
  26. Gablasova, D., Brezina, V., and McEnery, T. (2017). Collocations in Corpus-Based Language Learning Research: Identifying, Comparing, and Interpreting the Evidence. *Lang. Learn.* 67, 155–179.
  27. Rychlý, P. (2008). A Lexicographer-Friendly Association Score. In *Proceedings of Recent Advances in Slavonic Natural Language Processing, RASLAN 2008*, pp. 6–9.
  28. Entman, R.M., Matthes, J., and Pellicano, L. (2008). Nature, sources, and effects of news framing. In *The Handbook of Journalism Studies*, K. Wahl-Jorgensen and T. Hanitzsch, eds. (Routledge), pp. 175–190.
  29. Schlichting, I. (2013). Strategic Framing of Climate Change by Industry Actors: A Meta-analysis. *Environ. Commun.* 7, 493–511.
  30. Van Gorp, B., and van der Goot, M.J. (2012). Sustainable Food and Agriculture: Stakeholder's Frames. *Commun. Cult. Crit.* 5, 127–148.
  31. Elgesem, D., Steskal, L., Diakopoulos, N., Elgesem, D., Steskal, L., and Diakopoulos, N. (2015). Structure and Content of the Discourse on Climate Change in the Blogosphere: The Big Picture. *Environ. Commun.* 9, 169–188.
  32. SkepticalScience.com Climate myths sorted by taxonomy. <https://perma.cc/7LAF-MHEX>.
  33. Elsasser, S.W., and Dunlap, R.E. (2013). Leading Voices in the Denier Choir: Conservative Columnists' Dismissal of Global Warming and Denigration of Climate Science. *Am. Behav. Sci.* 57, 754–776.
  34. McCright, A.M., and Dunlap, R.E. (2000). Challenging global warming as a social problem: An analysis of the conservative movement's counter-claims. *Soc. Probl.* 47, 499–522.
  35. Ferguson, J., de Aguiar, T.R.S., and Fearfull, A. (2016). Corporate response to climate change: language, power and symbolic construction. *Accounting, Audit. Account. J.* 29, 278–304.
  36. Cann, H.W. (2015). Climate Change, Still Challenged: Conservative Think Tanks and Skeptic Frames. In *Annual Meeting of the Western Political Science Association, Las Vegas, April 2–4*, pp. 1–19.
  37. Metag, J. (2016). Content analysis methods for assessing climate change communication and media portrayals. In *Oxford Encyclopedia of Climate Change Communication*, M. Nisbet, S. Ho, E. Markowitz, S. O'Neill, M. S. Schäfer, and J. Thaker, eds. (Oxford University Press, online), pp. 1–34.
  38. Rowlands, I.H. (2000). Beauty and the beast? BP's and Exxon's positions on global climate change. *Environ. Plan. C Gov. Policy* 18, 339–354.
  39. Livesey, S.M. (2002). Global Warming Wars: Rhetorical and Discourse Analytic Approaches to Exxonmobil's Corporate Public Discourse. *J. Bus. Commun.* 39, 117–148.
  40. Smerecnik, K.R., and Renegar, V.R. (2010). Capitalistic Agency: The Rhetoric of BP's Helios Power Campaign. *Environ. Commun.* ISSN 4, 152–171.
  41. Doyle, J. (2011). Where has all the oil gone? BP branding and the discursive elimination of climate change risk. In *Culture, Environment and Eco-Politics*, N. Heffernan and D. A. Wragg, eds. (Cambridge Scholars Publishing), pp. 200–225.
  42. Plec, E., and Pettenger, M. (2012). Greenwashing Consumption: The Didactic Framing of ExxonMobil's Energy Solutions. *Environ. Commun.* 6, 459–476.

43. Robinson, M.L. (2014). *Marketing Big Oil - Brand lessons from the world's largest companies.* (Palgrave Macmillan).
44. Gaither, B.M., Gaither, T.K., Gaither, B.M., and Gaither, T.K. (2016). *Marketplace Advocacy by the U.S. Fossil Fuel Industries: Issues of Representation and Environmental Discourse.* *Mass Commun. Soc.* 19, 585–603.
45. Schneider, J., Schwarze, S., Bsumek, P.K., and Peebles, J. (2016). *Under Pressure - Coal Industry Rhetoric and Neoliberalism.* (Palgrave Macmillan UK).
46. Cahill, S. (2017). *Imagining Alternatives in the Emerald City: The Climate Change Discourse of Transnational Fossil Fuel Corporations* (University of Victoria).
47. Ayling, J. (2017). *A Contest for Legitimacy: The Divestment Movement and the Fossil Fuel.* *Law Policy* 39, 349–371.
48. Scanlan, S.J. (2017). *Framing fracking: scale-shifting and greenwashing risk in the oil and gas industry and gas industry.* *Local Environ.* 22, 1311–1337.
49. Grantham, S., and Vieira Jr, E.T. (2018). *Exxonmobil's social responsibility messaging - 2002-2013 CEO letters.* *Appl. Environ. Educ. Commun.* 17, 266–279.
50. Lamb, W.F., Mattioli, G., Levi, S., Roberts, J.T., Minx, J.C., Müller-hansen, F., Capstick, S., Creutzig, F., Culhane, T., and Steinberger, J.K. (2020). *Discourses of climate delay.* *Glob. Sustain.* 3, 1–5.
51. ExxonMobil (2000). *Unsettled Science* (Advertorial). *The New York Times.*
52. ExxonMobil (2004). *Weather and climate* (Advertorial). *The New York Times.*
53. Mobil (2007). *Climate change: a degree of uncertainty* (Advertorial). *The New York Times.*
54. ExxonMobil (2007). *Let's Talk About Climate Change* (Advertorial, 14 February 2007). *The New York Times.*
55. ExxonMobil (2007). *Let's Talk About Climate Change* (Advertorial, 16 February 2007). *The New York Times.*
56. ExxonMobil (2007). *Addressing the risks of climate change* (Advertorial). *The New York Times.*
57. Mobil (1997). *Reset the alarm* (Advertorial). *New York Times.*
58. ExxonMobil (2002). *A responsible path forward on climate* (Advertorial). *The New York Times.*
59. Mobil (1998). *Post Kyoto, what's next?* (Advertorial). *The New York Times.*
60. Mobil (1997). *Stop, look and listen before we leap* (Advertorial). *The New York Times.*
61. Mobil (1998). *The Kyoto Protocol: a painful response* (Advertorial). *The New York Times.*
62. ExxonMobil (2000). *Do no harm* (Advertorial). *The New York Times.*
63. Mobil (1998). *The Kyoto Protocol: too many gaps* (Advertorial). *The New York Times.*
64. ExxonMobil (2008). *The fuels of the future* (Advertorial). *The New York Times.*
65. Mobil (1999). *Lessons learned* (Advertorial). *The New York Times.*
66. ExxonMobil (2000). *Planting the future.* *The New York Times.*
67. ExxonMobil (2009). *tackling climate risks with technology* (Advertorial). *The New York Times.*
68. ExxonMobil (2002). *Managing greenhouse gas emissions* (Advertorial). *The New York Times.*
69. ExxonMobil (2000). *Political cart before a scientific horse* (Advertorial). *The New York Times.*
70. Mobil (1993). *Apocalypse no* (Advertorial). *The New York Times.*
71. Mobil (1997). *Science: what we know and don't know* (Advertorial). *New York Times.*
72. ExxonMobil (2004). *Directions for climate research* (Advertorial). *The New York Times.*
73. Mobil (1996). *A policy agenda for tomorrow* (Advertorial). *The New York Times.*
74. ExxonMobil (2001). *...to a sounder climate policy* (Advertorial). *The New York Times.*

75. ExxonMobil (2009). Many parts working together - the only way to solve the world's energy challenges (Advertorial). The New York Times.
76. ExxonMobil (2009). citizenship for the long term (Advertorial, 22 May 2009). The New York Times.
77. ExxonMobil (2009). citizenship for the long term (Advertorial, 29 June 2009). The New York Times.
78. ExxonMobil (1997). Climate change: a prudent approach (Advertorial). The New York Times.
79. Mobil (1999). King of the road (Advertorial). The New York Times.
80. ExxonMobil (2005). More Energy and Lower Emissions? (Advertorial, 14 June 2005). The New York Times.
81. ExxonMobil (2005). More Energy and Lower Emissions? (Advertorial, 7 July 2005). The New York Times.
82. ExxonMobil (2005). More Energy and Lower Emissions? (Advertorial, 11 May 2005). The New York Times.
83. Mobil (1995). Electric vehicles: a promise too far (Advertorial). The New York Times.
84. ExxonMobil (2001). Renewable energy: tomorrow's promise (Advertorial). The New York Times.
85. ExxonMobil (2001). Action, not talk: cogeneration and climate (Advertorial). The New York Times.
86. Mobil (1997). Climate change: Let's get it right (Advertorial). The New York Times.
87. Bowen, F. (2014). After Greenwashing: Symbolic Corporate Environmentalism and Society. (Cambridge University Press).
88. ExxonMobil (2007). values at work (Advertorial). The New York Times.
89. ExxonMobil (2008). Next-generation energy (Advertorial). The New York Times.
90. Mobil (1997). The Kyoto conference (Advertorial). The New York Times.
91. Mobil (1998). Voluntary "can do" (Advertorial). The New York Times.
92. Mobil (1999). Helping Earth breathe easier (Advertorial). The New York Times.
93. ExxonMobil (2006). Changing the game (Advertorial). The New York Times.
94. ExxonMobil (2005). Research into climate solutions (Advertorial). The New York Times.
95. ExxonMobil (2008). energy efficiency – one quart at a time (Advertorial). The New York Times.
96. ExxonMobil (2008). Vehicles of change (Advertorial). The New York Times.
97. ExxonMobil (2007). Saving energy and reducing greenhouse gas emissions (Advertorial). The New York Times.
98. ExxonMobil (2006). Multiplier effects (Advertorial). The New York Times.
99. ExxonMobil (2003). The global climate and energy challenge (Advertorial). The New York Times.
100. ExxonMobil (2007). Taking action to reduce greenhouse gas emissions (Advertorial). The New York Times.
101. Mobil (1997). Global climate change (Advertorial). The New York Times.
102. Mobil (1997). Climate change: where we come out (Advertorial). The New York Times.
103. ExxonMobil (2000). Facts and fundamentals (Advertorial). The New York Times.
104. Flannery, B.P., Callegari, A.J., Nair, B., and Roberge, W.G. (1984). The fate of CO<sub>2</sub> from the Natuna gas project if disposed by subsea sparging (Internal Document).
105. Hoffert, M.I., Caldeira, K., Benford, G., Criswell, D.R., Green, C., Herzog, H., Jain, A.K., Kheshgi, H.S., Lackner, K.S., Lewis, J.S., et al. (2002). Advanced Technology Paths to Global Climate Stability: Energy for a Greenhouse Planet. *Science* 298, 981–988.
106. Mastracchio, R.L. (1979). Controlling Atmospheric CO<sub>2</sub> (Internal Document).
107. Albritton, D.L., Allen, M.R., Alfons, P.M., Baede, J.A., Church, U.C., Xiaosu, D., Yihui, D., Ehhalt, D.H., Folland, C.K., Giorgi, F., et al. (2001). Climate change 2001: the

- scientific basis, summary for policymakers. Contribution of Working Group I to the Third Assessment Report of the Intergovernmental Panel on Climate Change.
108. Cohen, R.W., and Levine, D.G. (1982). Untitled (consensus on CO<sub>2</sub> letter) (Internal Document).
  109. Burgers, W.F.J., Northrop, P.S., Kheshgi, H.S., and Valencia, J.A. (2011). Worldwide development potential for sour gas. *Energy Procedia* 4, 2178–2184.
  110. Black, J. (1978). The Greenhouse Effect (Internal Document).
  111. Hayhoe, K., Kheshgi, H.S., Jain, A.K., and Wuebbles, D.J. (2002). Substitution of natural gas for coal: climatic effects of utility sector emissions. *Clim. Change* 54, 107–139.
  112. Archer, D., Kheshgi, H., and Maier-reimer, E. (1998). Dynamics of fossil fuel CO<sub>2</sub> neutralization by marine CaCO<sub>3</sub>. *Global Biogeochem. Cycles* 12, 259–276.
  113. Jain, A.K., Kheshgi, H.S., and Wuebbles, D.J. (1994). Integrated Science Model for Assessment of Climate Change. In 87th Annual Meeting and Exhibition of the Air and Waste Management Association (94-TP59.08).
  114. Mobil (1996). Climate change: We're all in this together (Advertorial). *The New York Times*.
  115. ExxonMobil (2000). The Path Forward on Climate Change (Advertorial). *The New York Times*.
  116. Mobil (1996). Less heat, more light on climate change (Advertorial). *The New York Times*.
  117. Mobil (1997). Let's not forget the will of the Senate (Advertorial). *The New York Times*.
  118. ExxonMobil (2000). The Promise of Technology (Advertorial). *The New York Times*.
  119. ExxonMobil (2001). Renewable energy: today's basics (Advertorial). *The New York Times*.
  120. ExxonMobil (2020). Publications and reports. Exxonmobil Newsroom (accessed 3 August 2020). <https://perma.cc/GRF7-SFGM>.
  121. ExxonMobil (2006). 2006 Corporate Citizenship Report.
  122. ExxonMobil (2005). 2005 Corporate Citizenship Report.
  123. ExxonMobil (2016). 2016 Corporate Citizenship Report.
  124. ExxonMobil (2009). 2009 Corporate Citizenship Report.
  125. ExxonMobil (2019). 2019 Outlook For Energy: A Perspective to 2040.
  126. ExxonMobil (2010). 2010 The Outlook for Energy: A View to 2030.
  127. ExxonMobil (2018). 2018 Energy & Carbon Summary: Positioning for a Lower-Carbon Energy Future.
  128. ExxonMobil (2014). The Outlook for Energy: A View to 2040.
  129. ExxonMobil (2009). Outlook for Energy - A View to 2030.
  130. ExxonMobil (2007). 2007 Corporate Citizenship Report.
  131. ExxonMobil (2019). Innovating Energy Solutions - Research and development highlights.
  132. ExxonMobil (2019). 2019 Energy & Carbon Summary.
  133. ExxonMobil (2005). The Outlook for Energy - A View to 2030.
  134. Barker, T., Bashmakov, I., Alharthi, A., Amann, M., Cifuentes, L., Drexhage, J., Duan, M., and Edenhofer, O. (2007). Mitigation from a cross-sectoral perspective. In *Climate Change 2007: Mitigation. Contribution of Working Group III to the Fourth Assessment Report of the Intergovernmental Panel on Climate Change* (Cambridge University Press).
  135. Prentice, I.C., G.D. Farquhar, M.J.R. Fasham, M.L. Goulden, M. Heimann, V.J. Jaramillo, H.S. Kheshgi, C. Le Quéré, R.J. Scholes, D.W.R.W., Archer, D., Ashmore, M.R., Aumont, O., Baker, D., Battle, M., Bender, M., Bopp, L.P., Bousquet, P., et al. (2001). The Carbon Cycle and Atmospheric Carbon Dioxide. Contribution of Working Group I to the Third Assessment Report of the Intergovernmental Panel on Climate Change, Chapter 3.
  136. ProQuest ProQuest Historical Newspapers Database. <https://search.proquest.com/>.

137. PolluterWatch Exxon and Mobil Ads. <https://perma.cc/8XHW-5GZE>.
138. ExxonMobil Corp. Supporting Materials. <https://perma.cc/D862-KB2N>.
139. ICN Documents (Exxon: The road not taken). InsideClimate News. <https://perma.cc/KCG8-M9ZM>.
140. Climate Investigations Center. Climate Files. [www.climatefiles.com](http://www.climatefiles.com).

## **Bridget Cornell**

---

**From:** David Sive <david.sive@mailatlaw.ch>  
**Sent:** Thursday, April 7, 2022 1:20 PM  
**To:** Joanne Marchetta; John Marshall; Marja Ambler; Katherine Hangeland  
**Subject:** Agenda Item No V.B Verizon/Tahoe Seasons New Telecommunications Facility -- TRPA H.O. Meeting 4/7/2022  
**Attachments:** Council Cancer 2021.pdf; Limiting liability with positioning to minimize negative health effects of cellular phone towers.pdf; Res 2020-103 Deny Appeal Verizon Facility on Saddle Road.pdf

Lest you forget...

## South Lake Tahoe City Council...

**Cancer occurrence by wireless telecommunications systems is comparable to the number of traffic fatalities of 10.92 per 100,000 population per year.<sup>†</sup>**

**When you put cell towers on residential lawns, you will eventually have blood on your hands; it's only a matter of time. You have a moral imperative and legal duty to minimize harm to human life, your lack of doing so will result in...**

**manslaughter.**

**You will have blood on your hands. Ten to twenty years from now, if you are still in town, you will have to look into the eyes of the people and families you hurt and explain to them why you did nothing, having been told the consequences. They will not accept the justification you give, and sorry will not bring back the dead.**

<sup>†</sup> Source:

Frank Barnes and Ben Greenebaum, 'Role of Radical Pairs and Feedback in Weak Radio Frequency Field Effects on Biological Systems', Environmental Research 163 (2018): 165–170. (see p. 169).

[Cody Bass](#) and [Brook Laine](#) now join these ranks with soiled hands after [their vote](#) on the [Tahoe Seasons WTF appeal](#). I hope you've considered the gravity of the situation. You've killed people. [Really](#).



ELSEVIER

Contents lists available at ScienceDirect

Environmental Research

journal homepage: [www.elsevier.com](http://www.elsevier.com)

# Limiting liability with positioning to minimize negative effects of cellular phone towers

J.M. Pearce<sup>a,b,c,\*</sup><sup>a</sup> Department of Electronics and Nanoengineering, School of Electrical Engineering, Aalto University, Espoo, Finland<sup>b</sup> Department of Electrical & Computer Engineering, Michigan Technological University, USA<sup>c</sup> Department of Materials Science & Engineering, Michigan Technological University, USA

## ARTICLE INFO

### Keywords:

**Radiofrequency radiation (RFR)**

Antenna arrays

**Cellular phone base stations****Microwave sickness**

Nonionizing electromagnetic fields

**Environmental pollution****Cancer**

RFR health effects

## ABSTRACT

The use of cellular phones is now ubiquitous (common among even young children in many areas, nearly saturated). The basic operation of cellular phones (radio-frequency radiation (RFR) with cellular phones) and the data needs of the population increase (due to the proliferation of computers to smart phones, this coverage is increasing) and their power output is expected to increase. This paper examines negative human health effects from RFR from cellular phone towers. **enough medical and scientific evidence to warrant the use of cellular phone towers.** In order to protect cell phone users and industries that have caused unintended health effects, the reviewed literature on the effects of RFR from cellular phone towers are closely examined and recommendations for potential future liability.

You have harmed Federally protected [birds](#) and [frogs](#) too!

~David Sive~

**Collin, Wallace, and Middlebrook...**

**Cancer occurrence by wireless telecommunications systems is comparable to the number of traffic fatalities of 10.92 per 100,000 population per year.†**

**When you put cell towers on residential lawns, you will eventually have blood on your hands; it's only a matter of time. You have a moral imperative and legal duty to minimize harm to human life, your lack of doing so will result in...**

**manslaughter.**

**You will have blood on your hands. Ten to twenty years from town, if you are still in town, you will have to look into the eyes of the people and families you hurt and explain to them why you did nothing, having been told the consequences. They will not accept the justification you give, and sorry will not bring back the dead.**

† **Source:**  
Frank Barnes and Ben Greenebaum, 'Role of Radical Pairs and Feedback in Weak Radio Frequency Field Effects on Biological Systems', *Environmental Research* 163 (2018): 165-170. (see p. 169).



## **Resolution 2020-103**

**Adopted by the City of South Lake Tahoe  
City Council**

**November 3, 2020**

### **Deny Appeal of Special Use Permit and Design Review Approval for a Verizon Wireless Telecommunication Facility located at 3901 Saddle Road, File #19-160**

#### **BACKGROUND**

- A. On August 1, 2019, Epic Wireless, representing Verizon Wireless, submitted a General Planning Application for design review and a special use permit, proposing to install a new wireless telecommunication facility on the roof of the Tahoe Seasons Resort building at 3901 Saddle Road.
- B. On July 16, 2020, the City of South Lake City Council Planning Commission held a duly noticed public hearing to consider the proposed wireless telecommunication facility. After receiving public comments, the Planning Commission considered the evidence in the record and voted three in the affirmative with one abstention to take the following action: 1) pass a motion finding the project to be categorically exempt from CEQA pursuant to Section 15303; and 2) approve the design review and grant the special use permit.
- C. On July 31, 2020, the City Clerk received an appeal from Concerned Citizens of South Lake Tahoe appealing the Planning Commission's decision.
- D. On November 3, 2020, the City Council of South Lake Tahoe held a duly noticed public hearing to consider the appeal.

**BASED ON THE FACTS SET FORTH IN THE BACKGROUND, BE IT RESOLVED**, that the City Council of the City of South Lake Tahoe:

- 1. Has considered the public testimony, deliberated, and considered all the evidence in the record; and
- 2. Denies the appeal and upholds the Planning Commission decision to grant a special use permit and design review approval for a Verizon wireless telecommunication facility located at 3901 Saddle Road, File #19-160.

Adopted by the City of South Lake Tahoe City Council on November 3, 2020, by the following vote:

Yes: Bass, Collin, Laine, Middlebrook and Wallace

DocuSigned by:

Jason

Jason Collin, Mayor

Date: 11/10/2020

Attest:

DocuSigned by:

Susan Blankenship, City Clerk

*The presence of electronic signature certifies that the foregoing is a true and correct copy as approved by the South Lake Tahoe City Council*



ELSEVIER

Contents lists available at ScienceDirect

Environmental Research

journal homepage: [www.elsevier.com/locate/envres](http://www.elsevier.com/locate/envres)

# Limiting liability with positioning to minimize negative health effects of cellular phone towers

J.M. Pearce<sup>a,b,c,\*</sup><sup>a</sup> Department of Electronics and Nanoengineering, School of Electrical Engineering, Aalto University, Espoo, Finland<sup>b</sup> Department of Electrical & Computer Engineering, Michigan Technological University, USA<sup>c</sup> Department of Materials Science & Engineering, Michigan Technological University, USA

## ARTICLE INFO

## Keywords:

Radiofrequency radiation (RFR)

Antenna arrays

Cellular phone base stations

Microwave sickness

Nonionizing electromagnetic fields

Environmental pollution

Cancer

RFR health effects

## ABSTRACT

The use of cellular phones is now ubiquitous through most of the adult global population and is increasingly common among even young children in many countries (e.g. Finland, where the market for smart phones is nearly saturated). The basic operation of cellular phone networks demands widespread human exposure to radio-frequency radiation (RFR) with cellular phone base stations providing cellular coverage in most areas. As the data needs of the population increase from the major shift in the source of Internet use from personal computers to smart phones, this coverage is widely predicted to increase. Thus, both the density of base stations and their power output is expected to increase the global human RFR exposure. Although direct causation of negative human health effects from RFR from cellular phone base stations has not been finalized, **there is already enough medical and scientific evidence to warrant long-term liability concerns for companies deploying cellular phone towers.** In order to protect cell phone tower firms from the ramifications of the failed paths of other industries that have caused unintended human harm (e.g. tobacco) this Current Issue summarizes the peer-reviewed literature on the effects of RFR from cellular phone base stations. Specifically the impacts of siting base stations are closely examined and recommendations are made for companies that deploy them to minimize their potential future liability.

## 1. Negative human health effects from proximity to cellular phone base stations

There is a large and growing body of evidence that human exposure to RFR from cellular phone base stations causes negative health effects (Siddoo-Atwal, 2018; Singh et al., 2018; Faisal, et al., 2018) including both i) **neuropsychiatric complaints** such as headache, concentration difficulties, memory changes, dizziness, tremors, depressive symptoms, fatigue and sleep disturbance (Navarro et al., 2003; Hutter et al., 2006; Abdel-Rassoul et al., 2007); and ii) **increased incidence of cancer** and living in proximity to a cell-phone transmitter station (Wolf and Wolf, 2004; Havas, 2017). The mechanism for causing cancer could be from **observed genetic damage using the single cell gel electrophoresis assay** assessed in peripheral blood leukocytes **of individuals residing in the vicinity of a mobile phone base station** and comparing it to that in healthy controls (Gandhi et al., 2014). In epidemiological studies that assessed negative health effects of mobile phone base stations (seven studies explored **the association between base station proximity and neurobehavioral effects** (Navarro et al., 2003; Hutter et al., 2006;

Abdel-Rassoul et al., 2007; Berg-Beckhoff et al., 2009; Blettner et al., 2009; Gadzicka et al., 2006; Santini et al., 2002) **and** three investigated **cancer** (Wolf and Wolf, 2004; Havas, 2017; Levitt and Lai, 2010), 80% reported **increased prevalence of adverse neurobehavioral symptoms or cancer in populations living at distances < 500 m** from base stations (Navarro et al., 2003).

The literature also indicates that **these effects may be cumulative** based on i) mice exposed to **low-intensity RFR** became less reproductive and after five generations of exposure the mice were not able to produce offspring indicating **intergenerational transfer of effects** (Magras and Xenos, 1997); ii) **DNA damage in cells after 24 h exposure to low-intensity RFR, which can lead to gene mutation that accumulates over time** (Phillips et al., 1998) and iii) increased sensitivity to behavior-disruption experiments in rats (D'Andrea et al., 1986) and monkeys (de Lorge, 1984), iv) an increase in permeability of the blood-brain barrier in mice suggesting that **a short-term, high-intensity exposure can produce the same effect as a long-term, low-intensity exposure** (Persson et al., 1997). Studies on short-term exposure generally show no effects. For example, early studies saw no effect from

\* 601 M&M Building, 1400 Townsend Drive, Houghton, MI 49931, USA.

E-mail address: [pearce@mtu.edu](mailto:pearce@mtu.edu).

<https://doi.org/10.1016/j.envres.2019.108845>

Received 26 December 2017; Received in revised form 3 June 2019; Accepted 19 October 2019

0013-9351/ © 2019 Elsevier Inc. All rights reserved.

short-term exposure, however, studies found effects after prolonged, repeated exposure in guinea pigs and rabbits (Takashima et al., 1979).

There are several studies showing the effect intensifies with reduced distance to the cell tower. The first (Santini et al., 2002) found increased symptoms and complaints the closer a person lived to a tower (Santini et al., 2002) and similar results were found in later studies (Navarro et al., 2003; Hutter et al., 2006; Abdel-Rassoul et al., 2007).

## 2. U.S. law unhelpful for preventing future liability

Current U.S. law has created a somewhat peculiar overriding federal preemption that precludes taking the “environmental effects” of RFR into consideration in cell tower siting (see Section 704 of The Telecommunications Act of 1996). The current, U.S. standards are based solely on thermal effects (which do not appear to be a problem) and thus do not mitigate against non-thermal effects (for which there is a growing litany of concern in the medical/scientific community). Due to the findings of many studies briefly summarized above many researchers argue for the revision of standard guidelines for public exposure to RER from mobile phone base station antennas (Abdel-Rassoul et al., 2007; Hardell and Sage, 2008; Khurana et al., 2010). As Roda and Perry summarize (Roda and Perry, 2014), “... because scientific knowledge is incomplete, a precautionary approach is better suited to State obligations under international human rights law.” This is perhaps most forcefully concluded by the *BioInitiative Report* published by the BioInitiative Working Group, which is based on an international research and public policy initiative to give an overview of what is known of biological effects that occur at low-intensity electromagnetic fields exposure. This precautionary approach is gaining favor in Europe, but is less common in the U.S. American companies are therefore ill advised to simply follow “regulatory compliance” on this front, as there appears to be a clear cause for concern in the scientific/medical communities. If causation were to be proven through detailed studies, cellular phone companies would potentially be in position of future legal exposure for causing widespread human health problems and premature death. It is, therefore, in American companies’ best interest to act before government and regulation catches up with the science.

## 3. Current cell tower positioning

Current cell tower locations are chosen based on a “search ring” priority basis of geographic optimum for technical coverage of high concentration of wireless transmissions (e.g. users). This combination of technical parameters (e.g. geography) to enable coverage and dependable service and costs (e.g. positioning on mountaintops on accessibly by helicopter) is then weighed against and local regulations such as local zoning.

To overcome these challenges in urban areas cellphone companies often locate cellphone base stations at schools, because the monthly rental fee (~\$1500) is welcome income for economically-challenged school districts that have influence on local zoning. However, some jurisdictions have already prohibited the placement of cell phone towers near schools or hospitals because of the increased sensitivity of these populations, as in India. Other regions such as Europe (Roda and Perry, 2014) could follow a similar approach. Now even in North America, Canada’s Standing Committee on Health are considering more precautionary approaches to RFR.

## 4. Precautionary cell phone base station positioning

A review article of the health effects near base stations concluded that deployment of base stations should be kept as efficient as possible to minimize exposure of the public to RFR and should not be located less than 500 m from the population, and at a height of 50 m (Levitt and Lai, 2010). This potentially presents a serious challenge to cell phone company RF engineers. However, it is possible to obtain necessary

coverage while at the same time minimizing human exposure at the highest intensities. There are several first steps a cellular phone company can take to minimize human exposure particularly of the most vulnerable populations.

First, voluntarily restrictions can be made on the placement of cellular phone base stations within 500 m of schools and hospitals. This will synchronize base station deployment strategies between regions. This can be done by utilizing the existing hexagon planning map structure of an area with an overlay using an additional semi-automated process with a geographic information system (GIS) (Al-Sahly et al., 2018) such as the Geographic Resources Analysis Support System (GRASS) to identify any regions within 500 m of existing schools and hospitals. All hexagons with schools or hospitals are marked as unusable for RF engineer planning (e.g. colored red). This restriction only makes planning slightly more difficult, but does present a challenge in regions where schools were specifically targeted as base station locations in (e.g. Verizon deployments in the U.S.). Future work is needed to determine if the increased legal exposure warrants the cost of moving existing stations. However, the increased cost to locate future stations away from schools and hospitals should be minimal.

The second technical hurdle is more challenging. Ideally, all cell phone users would have coverage while minimizing the population density near cellular phone base stations (thus minimizing health impacts). This can be planned using GIS tools, freely-accessible U.S. Census data, parcel data and/or satellite images. The population density can be color coded for straightforward decision making for RF engineers. As a cellphone base station costs \$250–350,000 to install in the U.S., using a precautionary approach to potential future regulation can save substantial relocation fees.

The cell phone industry should also consider cell splitting, small cell deployment, beam and null steering antennae as possible technical means for reducing RF exposure. Moreover, more research on cognitive radio should also be conducted, so that the overall RF exposure is reduced. These measures will ultimately benefit the entire telecommunications industry, while potentially significantly reducing global RF pollution.

Finally, exposed companies should consider funding large-scale epidemiological studies with personal dosimeters for strict dose measurement and straight-forward tissue exposure. By quantifying the human medical threat themselves, more appropriate long-term planning can be made to minimize the risk of liability from unintended human harm due to cellular phone base station siting.

## Financial disclosure

The author owns stock in the American Tower Corporation.

## Declaration of competing interest

The author has no conflict of interest.

## References

- Abdel-Rassoul, G., El-Fateh, O.A., Salem, M.A., Michael, A., Farahat, F., El-Batanouny, M., Salem, E., 2007. Neurobehavioral effects among inhabitants around mobile phone base stations. *Neurotoxicology* (Little Rock) 28 (2), 434–440.
- AprilAl-Sahly, A., Hassan, M.M., Al-Rubaian, M., Al-Qurishi, M., 2018. Using GIS for measuring mobile tower radiation on human. In: 2018 1st International Conference on Computer Applications & Information Security (ICCAIS). IEEE, pp. 1–6.
- Berg-Beckhoff, G., Blettner, M., Kowall, B., Breckenkamp, J., Schlehofer, B., Schmiedel, S., et al., 2009. Mobile phone base stations and adverse health effects: phase 2 of a cross-sectional study with measured radio frequency electromagnetic fields. *Occup. Environ. Med.* 66 (2), 124–130.
- Blettner, Maria, Schlehofer, Brigitte, Breckenkamp, Juergen, Kowall, Bernd, Schmiedel, Sven, Reis, Ursula, Potthoff, Peter, Schuez, Joachim, Berg-Beckhoff, Gabriele, 2009. Mobile phone base stations and adverse health effects: phase 1 of a population-based, cross-sectional study in Germany. *Occup. Environ. Med.* 66 (2), 118–123.
- de Lorge, J.O., 1984. Operant behavior and colonic temperature of *Macaca mulatta* exposed to radiofrequency fields at and above resonant frequencies.

- Bioelectromagnetics 5 (2), 233–246. <https://doi.org/10.1002/bem.2250050211>.
- D'Andrea, J.A., DeWitt, J.R., Gandhi, O.P., Stensaas, S., Lords, J.L., Nielson, H.C., 1986. Behavioral and physiological effects of chronic 2450 MHz microwave irradiation of the rat at 0.5 mW/cm<sup>2</sup>. *Bioelectromagnetics* 7 (1), 45–56. <https://doi.org/10.1002/bem.2250070106>.
- OctoberFaisal, M.M.A., Mortuza, M.G., Alam, T., 2018. Cell tower radiation and effect on human body: Bangladesh perspective. In: 2018 International Conference on Innovations in Science, Engineering and Technology (ICISSET). IEEE, pp. 423–426.
- Gadzicka, E., Bortkiewicz, A., Zmyslony, M., Szymczak, W., Szykowska, A., 2006. Assessment of subjective complaints reported by people living near mobile phone base stations. *Biuletyn PTZE Warszawa* 14, 23–26.
- Gandhi, G., Kaur, G., Nisar, U., 2014. A cross-sectional case control study on genetic damage in individuals residing in the vicinity of a mobile phone base station. *Electromagn. Biol. Med.* (0), 1–11.
- Hardell, L., Sage, C., 2008. Biological effects from electromagnetic field exposure and public exposure standards. *Biomed. Pharmacother.* 62 (2), 104–109.
- Havas, M., 2017. Carcinogenic effects of non-ionizing radiation: a paradigm shift. *Clin Oncol* 2, 1278–1279.
- Hutter, H.P., Moshhammer, H., Wallner, P., Kundi, M., 2006. Subjective symptoms, sleeping problems, and cognitive performance in subjects living near mobile phone base stations. *Occup. Environ. Med.* 63 (5), 307–313.
- Khurana, V.G., Hardell, L., Everaert, J., Bortkiewicz, A., Carlberg, M., Ahonen, M., 2010. Epidemiological evidence for a health risk from mobile phone base stations. *Int. J. Occup. Environ. Health* 16 (3), 263–267.
- Levitt, B.B., Lai, H., 2010. Biological effects from exposure to electromagnetic radiation emitted by cell tower base stations and other antenna arrays. *Environ. Rev.* 18, 369–395.
- Magras, I.N., Xenos, T.D., 1997. RF radiation-induced changes in the prenatal development of mice. *Bioelectromagnetics* 18 (6), 455–461. [https://doi.org/10.1002/\(SICI\)1521-186X\(1997\)18:6<455::AID-BEM8>3.0.CO;2-1](https://doi.org/10.1002/(SICI)1521-186X(1997)18:6<455::AID-BEM8>3.0.CO;2-1).
- Navarro, A.E., Sequera, J., Portoleás, M., Gómez-Perretta de Mateo, C., 2003. The microwave syndrome: a preliminary study in Spain. *Electromagn. Biol. Med.* 22 (2–3), 161–169. <https://doi.org/10.1081/JBC-120024625>.
- Persson, B.R.R., Salford, L.G., Brun, A., 1997. Blood–brain barrier permeability in rats exposed to electromagnetic fields used in wireless communication. *Wirel. Netw.* 3 (6), 455–461. <https://doi.org/10.1023/A:1019150510840>.
- Phillips, J.L., Ivaschuk, O., Ishida-Jones, T., Jones, R.A., Campbell-Beachler, M., Haggren, W., 1998. DNA damage in Molt-4 T-lymphoblastoid cells exposed to cellular telephone radiofrequency fields in vitro. *Bioelectrochem. Bioenerg.* 45 (1), 103–110. [https://doi.org/10.1016/S0302-4598\(98\)00074-9](https://doi.org/10.1016/S0302-4598(98)00074-9).
- Roda, C., Perry, S., 2014. Mobile phone infrastructure regulation in Europe: scientific challenges and human rights protection. *Environ. Sci. Policy* 37, 204–214.
- Santini, R., Santini, P., Danze, J.M., Le Ruz, P., Seigne, M., 2002. Enquête sur la sante' de riverains de stations relais de télé-phonie mobile : incidences de la distance et du sexe. *Pathol. Biol.* 50, 369–373. [https://doi.org/10.1016/S0369-8114\(02\)00311-5](https://doi.org/10.1016/S0369-8114(02)00311-5).
- Siddoo-Atwal, C., 2018. Electromagnetic radiation from cellphone towers: a potential health hazard for birds, bees, and humans. *Curr. Underst. Apoptosis: Program. Cell Death* 137.
- Singh, R., Nath, R., Mathur, A.K., Sharma, R.S., 2018. Effect of radiofrequency radiation on reproductive health. *Indian J. Med. Res.* 148 (Suppl 1), S92.
- Takashima, S., Onaral, B., Schwan, H.P., 1979. Effects of modulated RF energy on the EEG of mammalian brain. *Radiat. Environ. Biophys.* 16 (1), 15–27. <https://doi.org/10.1007/BF01326893>.
- Wolf, R., Wolf, D., 2004. Increased incidence of cancer near a cell-phone transmitter station. *Int. J. Cancer Prev.* 1 (2), 123–128.

## **Bridget Cornell**

---

**From:** John Carlos <john.carlos@freedommail.ch>  
**Sent:** Thursday, April 7, 2022 1:38 PM  
**To:** Joanne Marchetta; John Marshall; Marja Ambler; Katherine Hangeland  
**Subject:** H.O. Meeting Agenda Item No V.B Verizon/Tahoe Seasons New Telecommunications Facility  
**Attachments:** Request To Remove Andrew Strain As Hearings Officer.pdf

Dear TRPA Hearings Officer,

We need to establish our independence from the LTVA/Tahoe Chamber/Prosperity Center. † Their idea of prosperity is self-serving, forced, and unwanted in our town. The cell tower fiasco is the culmination of years of abusive overtourism by these organizations.

Hedi Hill-Drum, Patrick Rhamey, and Andrew Strain you are hurting us:



**Steve Teshara, we are a casualty of overtourism:**



Please respect us, our property, our bodily integrity. We don't do this to you, so don't do it to us.

Thank you

John Carlos

† These are surrogates who heavily represent the interests of Edgewood/Tahoe Beach Club, the latter of which [Patrick Rhamey](#) is CEO and [Andrew Strain](#) is a Vice President of Development. Rhamey is a Tahoe Prosperity Center Board-member. Strain is member of Tahoe Chamber's Government Affairs Committee (political action).

APPENDIX II

Appellants' Request To Remove Andrew Strain As Hearings Officer

<p>ATTORNEYS AT LAW</p> <p><b>SWANKIN &amp; TURNER</b></p> <p>5614 CONNECTICUT AVE., N.W. #339 WASHINGTON, D.C. 20015 TEL. 202 462-8800 FAX 202 315-2501</p>	<p>DAVID A. SWANKIN JAMES S. TURNER BETSY E. LEHRFELD CHRISTOPHER B. TURNER JULIAN GRESSER, of Counsel (California only)</p>
--	--

October 7, 2021

**BY E-MAIL**

Joanne S. Marchetta Executive  
Director, TRPA John L.  
Marshall, Esq.  
TRPA General Counsel  
P.O. Box 5310  
Stateline, Nevada 89449

**Re: TRPA File # ERSP2019-0389  
Proposed Verizon monopine cell tower at 1360 Ski Run Boulevard**

Dear Ms. Marchetta and Mr. Marshall:

As you know, we represent Monica Eckenstein, David Benedict, the Environmental Health Trust, Tahoe Stewards, LLC, and Tahoe for Safer Tech in proceedings in opposition to TRPA File # ERSP2019-0389, the proposed Verizon monopine cell tower at 1360 Ski Run Boulevard, South Lake Tahoe City.

We understand that Andrew Strain has been assigned as the TRPA Hearings Officer for the October 14, 2021 Public Hearing on this file. Due to Mr. Strain's current simultaneous employment as both TRPA Hearings Officer and Vice President of Development at the Tahoe Beach Club, and his position as a Member of the Government Affairs Committee of the Tahoe Chamber of Commerce, Mr. Strain has extremely serious conflicts of interest that preclude his serving as a TRPA Hearings Officer on this file. We hereby request that the TRPA immediately appoint a truly independent Hearings Officer whose impartiality cannot be reasonably questioned.

The conflict-of-interest provisions of the Bi-State Compact relevant to TRPA employees such as TRPA Hearings Officer Strain are set forth in Article III(a)(5) of the Bi-State Compact which provide, in relevant part:

(5) Each member and employee of the agency shall disclose his economic interests in the region within 10 days after taking his seat on the governing board or being employed by the agency and shall thereafter disclose any further economic interest which he acquires, as soon as feasible after he acquires it. As used in this paragraph, “economic interests” means:

(A) Any business entity operating in the region in which the member or employee has a direct or indirect investment worth more than one thousand dollars (\$1,000).

(B) Any real property located in the region in which the member or employee has a direct or indirect interest worth more than one thousand dollars (\$1,000).

(C) Any source of income attributable to activities in the region, other than loans by or deposits with a commercial lending institution in the regular course of business, aggregating two hundred fifty dollars (\$250) or more in value received by or promised to the member within the preceding 12 months.

(D) Any business entity operating in the region in which the member or employee is a director, officer, partner, trustee, employee or holds any position of management.

*No member or employee of the agency shall make, or attempt to influence, an agency decision in which he knows or has reason to know he has an economic interest. Members and employees of the agency must disqualify themselves from making or participating in the making of any decision of the agency when it is reasonably foreseeable that the decision will have a material financial effect, distinguishable from its effect on the public generally, on the economic interests of the member or employee. (Emphasis added).*

Mr. Strain, when he serves as a TRPA Hearings Officer, is an employee of the agency. The Compact language above emphatically prohibits TRPA employees, including Mr. Strain, “from making or participating in the making of any decision of the agency when it is reasonably foreseeable that the decision will have a material financial effect, distinguishable from its effect on the public generally, on the economic interests of the member or employee.”

At the same time as he serves as a TRPA Hearings Officer, Mr. Strain remains employed as Vice President of Development at the Tahoe Beach Club, a 143-unit ultra-luxury condominium project with a private members’ club on the shores of Lake Tahoe in Stateline, Nevada. This high-end real estate development enjoyed a record price-setting condominium sale this past summer of \$6 million for a unit located at 17 Beach Club Drive. As the Vice President of Development, Mr. Strain’s duties undoubtedly involve development matters and projects that implicate the TRPA Code of Ordinances and/or require TRPA permitting. As a senior executive of a major real estate developer in the Lake Tahoe Region, it’s rather astonishing, then, that Joanne Marchetta, TRPA’s Executive Director, has appointed Mr. Strain as a TRPA Hearings Officer, given the obvious inherent conflicts of interest between the two simultaneously-held positions.

Mr. Strain’s decisions as a TRPA Hearings Officer, even in matters ostensibly unrelated to those directly affecting his employer, inevitably have a material financial effect on the economic interests of his employer, and therefore, upon himself. To the extent Mr. Strain’s hearing decisions establish TRPA precedent, they affect the course of development of the lands within TRPA’s jurisdiction, including, of course, the properties owned by the Tahoe Beach Club. So it’s difficult to understand how Mr. Strain can serve as a TRPA Hearings Officer in any matter.

But the conflict-of-interest situation is even more egregious in connection with TRPA File # ERSP2019-0389. That’s because Mr. Strain’s boss, Patrick Rhamey, the Chief Executive Officer of the Tahoe Beach Club, has publicly expressed his support for expanding cell tower deployment in the Lake Tahoe Region. Indeed, Mr. Rhamey submitted a written public comment to the City Council of the City of South Lake Tahoe, in an email on May 11, 2020, in advance of the May 12, 2020 City Council meeting. Mr. Rhamey’s written public comment was directed to Agenda Item #12, “Policy Document for Wireless Facility Colocation Modification Submitted for Eligible Facilities Requests.” Mr. Rhamey’s written public comment states as follows:

From: Patrick Rhamey  
To: Public Comment  
Subject: Support for Agenda Item #12  
Date: Monday, May 11, 2020 6:51:50 PM

Please vote yes on Agenda Item #12, cell tower ordinance. It is important for the safety of our residents, visitors, and first responders that they have reliable cell service.

It’s reasonable to infer from Mr. Rhamey’s public comment supporting a wireless telecommunications facility resolution that promotes the expanded deployment of such installations that Mr. Rhamey is predisposed to support the monopine cell tower proposed at 1360 Ski Run Boulevard. Mr. Strain can be expected to act in the interests of his boss and to follow his boss’s lead with respect to Mr. Rhamey’s desire for more reliable cell service in the Lake Tahoe Region. Mr. Strain’s conflict – his need to uphold Mr. Rhamey’s position that the Tahoe Region must support a massive increase in mobile device coverage by approving more cell towers, imposes a fatal bias that poisons his ability to carry out his duty to act as an impartial TRPA Hearings Officer. This bias prevents Mr. Strain from acting in any balanced way as a TRPA Hearings Officer to protect the Public Trust enshrined in the Compact.

Moreover, Mr. Strain may well have an ownership interest in the business that owns/operates the Tahoe Beach Club, and pursuant to Article III(a)(5)(A)-(D), you need to disclose to us Mr. Strain’s “economic interests” in the region immediately.

To make matters even worse, Mr. Strain currently serves as a Member of the Government Affairs Committee of the Tahoe Chamber of Commerce. The Tahoe Chamber partners with the Tahoe Prosperity Center, and the Tahoe Chamber supports and facilitates the Connected Tahoe Initiative, a goal of which is increased cell tower densification in the Lake Tahoe region, no matter the health risks to people and the environment.

Given Mr. Rhamey's public comments supporting an ordinance that furthers expansion of the cellular network footprint in the region, and in particular, cell towers to improve cell service, Mr. Strain's contemporaneous position as Vice President of the Tahoe Beach Club, and his active involvement on the Government Affairs Committee of the Tahoe Chamber, we respectfully demand that Mr. Strain be disqualified from participating in this matter as the TRPA Hearings Officer.

Please let us know immediately whether you intend to replace Mr. Strain as the TRPA Hearings Officer for this matter, and if so, who the new TRPA Hearings Officer will be.

Very truly yours,

*/s/Julian Gresser*

Robert J. Berg

Gregg R. Lien

## Bridget Cornell

---

**From:** Mike Judge <mike.judge@groupoffice.ch>  
**Sent:** Thursday, April 7, 2022 1:09 PM  
**To:** Joanne Marchetta; John Marshall; Marja Ambler; Katherine Hangeland  
**Subject:** Hearings Officer === Meeting Agenda Item No V.B Verizon/Tahoe Seasons New Telecommunications Facility  
**Attachments:** EPA-Norbert-Hankin-to-Newton-RE-FCC-2003.pdf; TPC-CELL TOWER SAFETY\_Disinformation Flyer2\_W.pdf; TPC-CELL TOWER SAFETY\_Disinformation Flyer\_W.pdf; Wireless Hazards\_Washington Spectator.pdf

### SUBMITTED ON BEHALF OF TAHOE RESIDENTS FOR ACTUAL PROSPERITY

Dear TRPA Governing Board, Director, Attorney, Staff, and residents of South Lake Tahoe;

The Tahoe Prosperity Center has apparently hired a graphic designer to manufacture the appended dishonest flyers in a grassroots lobbying effort to secure their cell tower projects. These flyers purport to establish credibility by professing expertise in basic physics, but in doing so, they repeatedly misstate and misspell the basic science concepts—as we will show. They also cite authorities, who do not as a whole, support their claims. They further cite shoddy and unreliable sources such as marketing material. These flyers have the authorship characteristics of someone who is ill-versed in the subject matter, and is only using the material to push a self-serving agenda. It should come as no surprise that Heidi Hill-Drum used to [freelance in public relations \(PR\)](#), a profession almost entirely based upon manipulating people as a means to other ends.

We will now unpack and expose many of the lies on these flyers that are being used to manipulate City officials and the public in order to dismiss facts that are damaging to Heidi's agenda. We quote the attached material and speak nothing but the truth.

#### 1) Economics:

- The distance from a wireless facility has no apparent impact on the sale price of a home. The relationship between the list and sale price of a home matter how close the property was to the wireless facility.

To support Heidi's claim, she cites a single [wireless industry business-to-business \(B2B\) whitepaper](#). As a marketing tool, B2B whitepapers use selected facts and logical arguments to build a case favorable to the company sponsoring the document; they are not academic or peer-reviewed. Essentially ***all*** peer-reviewed studies by academic land economists on the impacts of cell towers on real estate values find a clear adverse relationship between valuation and proximity. Notwithstanding studies, this claim is plainly absurd on its face—[there are obviously a significant number of people who would not want a home with a cell tower on the lawn](#)—and a glossy TPC marketing flyer does not make it credible or true.

Her bold claim that "a wireless facility has no apparent impact on the value of sale price of a home," is clearly poorly sourced, irresponsible, and factually untrue. Fact-check all of the research links below:

[Wireless Towers and Home Values: An Alternative Valuation Approach Using a Spatial Econometric Analysis](#) (Journal of Real Estate Finance & Economics, May 1, 2018)

[The Cost of Convenience: Estimating the Impact of Communication Antennas on Residential Property Values](#) (Land Economics, Feb. 2016)

[Examining invisible urban pollution and its effect on real estate value in New York City](#) (New York Real Estate Journal September 2017)

[Neighborhood Cell Towers & Antennas—Do They Impact a Property's Desirability?](#) (National Institute for Science, Law and Public Policy (NISLAPP) in Washington, D.C.,)

[The effect of distance to cell phone towers on house prices](#) (Appraisal Journal, Fall 2007)

[The Impact of Cell Phone Towers on House Prices in Residential Neighborhoods](#) (The Appraisal Journal, Summer 2005)

["Impact of Communication Towers and Equipment on Nearby Property Values"](#) prepared by Burgoyne Appraisal Company, March 7, 2017

**2) Flyers contain blatant falsehoods about the positions taken by authoritative agencies.** These statements are a particularly heinous public disservice about [an emerging threat to public health](#). Organizations that lie about public health issues have no place in public service. We quote these blatant canards:

The American Cancer Society, the International Agency Research on Cancer and the National Toxicology Program claim that cell towers are unlikely to cause cancer.

Entirely to the contrary, the International Agency for Research on Cancer (IARC) is currently [upgrading its existent assessment that radiofrequency radiation is carcinogenic to humans \(IARC Group 1\)](#).

# Cancer Epidemiology Update, Following the 2011 IARC Evaluation of Radiofrequency Electromagnetic Fields (Monograph 102)

Anthony B Miller <sup>1</sup>, L Lloyd Morgan <sup>2</sup>, Iris Udasin <sup>3</sup>, Devra Lee Davis <sup>4</sup>

Affiliations + expand

PMID: 30196934 DOI: [10.1016/j.envres.2018.06.043](https://doi.org/10.1016/j.envres.2018.06.043)

## Abstract

Epidemiology studies (case-control, cohort, time trend and case studies) published since the International Agency for Research on Cancer (IARC) 2011 categorization of radiofrequency radiation (RFR) from mobile phones and other wireless devices as a possible human carcinogen (Group 2B) are reviewed and summarized. Glioma is an important human cancer found to be associated with RFR in case-control studies conducted in Sweden and France, as well as in some other countries. Increasing glioma incidence trends have been reported in the UK and other countries. Non-malignant effects linked include acoustic neuroma (vestibular Schwannoma) and meningioma. Because they allow detailed consideration of exposure, case-control studies can be superior to cohort studies or other methods in evaluating potential risks for brain cancer. When considered with recent animal experimental evidence, the recent epidemiological studies strengthen and support the conclusion that RFR should be categorized as carcinogenic to humans (IARC Group 1). Opportunistic epidemiologic studies are proposed that can be carried out through cross-sectional analyses of high, medium and low mobile phone users with respect to hearing, vision, memory, reaction time, and other indicators that can easily be assessed through standardized computer-based tests. As exposure data are not uniformly available, billing records should be used whenever available to corroborate reported exposures.

Moreover, the World Health Organization's (WHO) International Agency for the Research on Cancer (IARC) recommends reducing exposure to radiofrequency radiation (RFR) from cell phones. This is stated in their [2011 Press Release](#).

In an identical vein, these flyers egregiously **lie to the public** about the clear stance of **the National Toxicology Program (NTP)** which **expressly found that cell tower radiation causes DNA damage and tumors**. [We quote the federal agency's website:](#)

The NTP studies found that high exposure to RFR (900 MHz) used by cell phones was associated with:

- **Clear evidence of tumors in the hearts of male rats.** The tumors were malignant schwannoma
- **Some evidence of tumors in the brains of male rats.** The tumors were malignant gliomas.
- **Some evidence of tumors in the adrenal glands of male rats.** The tumors were benign, malign pheochromocytoma.

NTP scientists found that RFR exposure was associated with an increase in DNA damage. Specifically, with significant increases in DNA damage in:

- the frontal cortex of the brain in male mice,
- the blood cells of female mice, and
- the hippocampus of male rats.

It was particularly bizarre for the Tahoe "Prosperity" Center to specifically name a National Institute of Health (NIH) program which found **clear evidence** of radiofrequency radiation (RFR) causing tumors, [DNA damage](#), and cancer as exonerating liability for such. **Lying and cheating** are the desperate methods of those who cannot honestly refute a central claim; it is also the signature of persons who have contempt for public consent, honest debate, and places their own selfish interests ahead of the public's.

She makes an identical lie regarding the Environmental Protection Agency. The EPA has not ruled microwave radiation to be safe either. In fact, congress prohibited the EPA from looking into the matter; as a result, it has not issued policy, rules, or regulations on RFR:

## ***Senate Panel: No EMF Work at EPA***

The Senate Committee on Appropriations has cut \$350,000 from the Environmental Protection Agency's (EPA) EMF budget, because, "The committee believes EPA should not engage in EMF activities."

In a September 13 report (No.104-140), the committee also stated: "Section 2118 of the Energy Policy Act of 1992 established a federal program to investigate and report on human health effects from [EMFs]. Congress mandated that this program of research and public communication be managed jointly by the Department of Health and Human Services and the [DOE]. No programmatic role was assigned to EPA, yet EPA has pursued a number of unintegrated activities on EMFs that are of questionable value."

The House committee has already announced plans to cut EPA's low-priority radiation programs, which would include its work on EMFs (see *MWN*, J/A95).

Meanwhile, the EPA is reorganizing; effective October 1, the EMF program is moving to a new division, and Dennis O'Connor, the current EMF team leader, has been reassigned to work on the disposal and cleanup of radioactive waste. No replacement has yet been named.

In a deliberate attempt to stifle EMF regulation, radiation limits were transferred to the FCC which is clearly ill-equipped to deal with human and environmental health issues. Despite this, the EPA did issue the attached letter pertaining to the inadequacy of the FCC radiofrequency radiation exposure guidelines—we quote:

that results from an increase in body temperature. The FCC's exposure is protective of effects arising from a thermal mechanism but not from other mechanisms. Therefore, the generalization by many that the guidelines protect humans from all mechanisms is not justified.

However, there are reports that suggest that potentially adverse health effects may occur. Since EPA's comments were submitted to the FCC in 2001, reporting effects associated with both acute and chronic low-level exposure has increased.

The Tahoe Prosperity Center is wrong in telling City officials and the public that the EPA had determined that radiofrequency radiation is safe. **Heidi Hill-Drum needs to own up to her lies and apologize to the public.**

The National Academies of Sciences, Engineering, and Medicine itself also released [a new report](#) in December 2020 detailing some mechanisms for significant harm from radiofrequency radiation that may have led to traumatic brain injury in [several cases](#).

**3) The flyers contain misspellings, misunderstandings, and misapplications of scientific concepts.** These fall into three general categories: **(A)** confusing electromagnetic radiation with radioactivity; **(B)** not knowing the accepted spelling of fundamental physics terminology; and **(C)** ignorance of the quantum nature of light.

**A)** The author demonstrates she clearly does not possess even an elementary physics understanding of the difference between "radioactive" decay and electromagnetic radiation. There is no "radioactive range" within the electromagnetic spectrum. **Photons are not radioactive.**

For instance, the frequencies that carry x-rays and gamma rays are on the **radioactive range** of the electromagnetic spectrum, and can cause harmful damage to the chemical bonds in our DNA.

**B)** The author misspells "electromagnetic," "frequencies," and is utterly ignorant that a frequency (a measure of cycles per second) doesn't emit anything. Nor is there an "end" to a substantive infinite spectrum that somehow "bounds" a discrete "range," where electromagnetic interactions supposedly take place. All carriers of energy and momentum propagate as waves and exchange energy as particles (and no, she does not understand the concept of Planck frequency).

**Electro Magnetic Spectrum** – The range of frequencies that emit electro magnetic energy. The lower end of the spectrum has low frequencies and longer waves of energy, while higher end has high frequencies and shorter waves.

C) The author demonstrates she clearly does not possess even a basic [high school understanding of conceptual quantum mechanics](#):

**Electromagnetic Energy** – Any energy emitted or absorbed by charged particles traveling through space, anything from visible light to nuclear radiation.

This is an entirely deficient description, because **electromagnetic radiation consists of photons**. These are **uncharged elementary particles**—with zero rest mass which are the quanta of the electromagnetic force—they are responsible for all electromagnetic interactions. **There is extreme irony in all of this**, because quantum energy exchange is the fallacious premise that has led to the misconception that microwaves cannot cause cellular damage. This assumption is entirely wrong because microwave radiation interferes with electron transport mechanisms (eg., [oxidative phosphorylation](#)), and triggers gated ion channels (eg., axons). This causes the buildup of free radicals and oxidative stress, which are an undisputed mechanism of cancer.

It has been greatly understated in this debate that the [penetration depth](#) of electromagnetic radiation is generally a function of wavelength; the longer the wavelength, the greater the penetration—and the lower the photon energy. With respect to biological effects across the entire electromagnetic spectrum, there are compensating harms as one is exposed to either extrema. On the one hand, photon energies that are greater than ionic or chemical bonds will be expected to result in direct molecular breaks. On the other hand, long wavelength radiation—and its low energy photons—penetrates deep into the body; this is where the wave nature of electromagnetic radiation interferes with cellular electron transport mechanisms, which indirectly breaks chemical bonds through free radical generation. It turns out that visible light is in the sweet spot of the spectrum being neither ionizing nor penetrating, making it a choice bandwidth for biological optic systems or "eyes." That said, an entire class of chemicals do have bond energies equivalent to those of visible light photons and are known as "light" or "photo" sensitive.

You can read about this to your hearts content. There are over [3,300 pages of peer-reviewed research in proof of this matter on the City record](#). In addition to this, there is also a list of [1,000 recent peer-reviewed publications](#) on the record. This is a broad body of research by tenured professors across the globe, and cannot be debunked by Cowork Tahoe's capitalistic PhD who laments of having one of the [lowest GRE scores on the scale](#), nearly dropped out of graduate school to become a lawyer, and then misapplied her credentials to sell **crackpot** get-rich-quick startups to gullible investors (cf., Thernos). There is an analogy and lesson in all of this with [Elizabeth Holmes](#), of notorious [Theranos](#) fame. That capitalist used the banner of science to defraud investors, and used similar tactics as Heidi Hill-Drum to discredit anyone who referred to science facts that illuminated her fraud. It appears this affiliated PhD has more in common with Holmes than with a bevy of tenured research professors. That capitalist relies on a strong cellular phone signal to demonstrate her hyped-up tenant monitoring software startup [Jellyswitch](#), and is callous to the fact that the invasive cellular phone network

architecture causes cancer, neuro-psychiatric pathologies, and harms both residential real estate and neighborhood aesthetics.

Distributed Antenna System (DAS) cell network architecture may instead be installed inside landlord businesses where the users and demand are located. [Fiber to the premise](#) implementation of [municipal broadband](#) is the superior solution.

**4)** Last, the Tahoe Prosperity Center relies heavily on a single web page belonging to the American Cancer Society. The **American Cancer Society** is neither an academic authority, nor a professional board, nor a research agency. It is a charity organization, that primarily provides outpatient support to cancer patients, has received widespread criticism for wasting donations on overhead and lobbying, and its stance on cell towers are in conflict with [state](#) and federal health agencies.

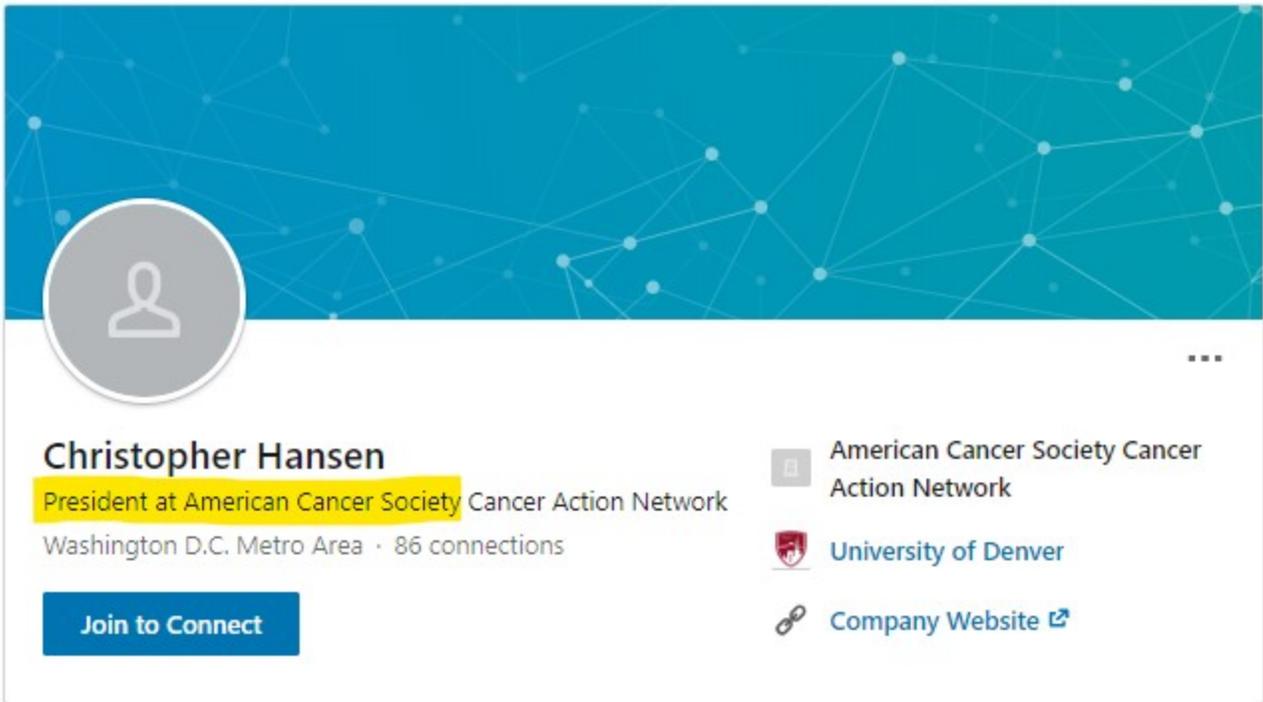
It has been pointed out, that the organization has been apparently hijacked by tech and biochemical companies who control the organization from investigating the carcinogenic nature of their particular industry products. Its policies are chosen by its board of directors, who are capitalists with an agenda to advance their enterprise, not by objective medical scientists. This will be corroborated below:

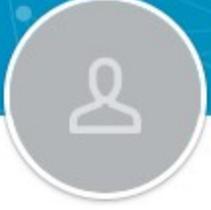
# AMERICAN CANCER SOCIETY CANCER ACTION NETWORK, INC NONPROFIT

**Company Number** [EXTUID\\_2666690](#)  
**Native Company Number** [212917](#)  
**Status** Active  
**Incorporation Date** 4 September 2001 (almost 19 years ago)  
**Company Type** Non-Profit Corporation  
**Jurisdiction** [District of Columbia \(US\)](#)  
**Registered Address** [555 11th Street NW, Suite 300](#)  
Washington  
20004  
District of Columbia  
United States  
**Agent Name** [C T CORPORATION SYSTEM](#)  
**Agent Address** [1015 15th St NW, Suite 1000, Washington, District of Columbia, 20005](#)  
**Directors / Officers** [C T CORPORATION SYSTEM, agent](#)  
[Coleman, P. Kay, governor](#)  
[Coulter, William E. \(Ed\), governor](#)  
[Cullen, Kevin J, governor](#)  
[DuBois, Raymond N, governor](#)  
[Glickman, Dan, governor](#)  
[Hamilton, John W, governor](#)  
[Hansen, Christopher W, governor](#)  
[Jackvony, Bernard A, governor](#)  
[Lopez, Jorge Luis, governor](#)  
[Mann, Maureen G, governor](#)  
[Manna, John J, governor](#)  
[Marquardt, Michael T, governor](#)  
[Meuller, Scarlott K, governor](#)  
[Ngo, Rick Q, governor](#)  
[Philips, Timothy B, executingofficer](#)  
[Philips, Timothy B, governor](#)  
[Reedy, Gary M, governor](#)  
[Underriner, William P, governor](#)  
[Waldholtz, Bruce D, governor](#)  
[Youle, Robert E, governor](#)

Can you see why the board of this [captured](#) organization is not interested in finding carcinogenic threats posed by the **tech sector**?

First-off, the Chairman of the American Cancer Society is a substantive lobbyist for the [Technology Sector](#) which advocates for regulations that favor cell phone and mobile device sales. His strategic interest in the society includes [quelling public concern pertaining to the dangers of EMF](#) currently threatening his tech sector constituents.




**Christopher Hansen**  
 President at American Cancer Society Cancer Action Network  
 Washington D.C. Metro Area · 86 connections

[Join to Connect](#)

- American Cancer Society Cancer Action Network
- University of Denver
- Company Website

## About

Chris Hansen is the President of the TechAmerica Foundation and CEO Emeritus of TechAmerica, the nation's largest association representing all segments of the high-tech industry. In 2008 as the President & CEO of AeA, Mr. Hansen partnered with Phil Bond, then-President & CEO of ITAA, to merge the Associations memberships and services to create TechAmerica.

Before joining AeA in November 2007, Mr. Hansen was AARP's Group Executive Officer for State and National Initiatives. His responsibilities included government relations, advocacy, management of AARP offices in every state, public outreach on key programs, and volunteer management and support. He assumed that position in mid-January 2003, after serving as the organization's Senior Managing Director of Government Relations and Advocacy since March 2002.

Mr. Hansen also worked for 16 years for The Boeing Company. Hansen was known as a political strategist with a broad view and strong interpersonal relations with both Republicans and Democrats. As Boeing's SVP, Government Relations, he directed the company's congressional and executive branch work and its state affairs. Previously, he served as Boeing's VP, Government Relations and VP of Congressional Affairs. Prior to his work with Boeing, Hansen spent 11 years with General Dynamics, where he was Director of Government Relations Operations.

He has held a number of leadership roles in professional organizations, including the U.S. Council for International Business, the National Aeronautics Association, the National Bureau of Asian Research, and currently is a member of the Individual Investors Committee of the New York Stock Exchange. He is also active in philanthropic organizations such as the Wolf Trap Foundation. Mr. Hansen holds a Bachelor of Arts degree in Political Science from the University of Denver and a Master's degree from the American Graduate School of International Management.

...next, the board is directed by a bigwig lobbyist and Clinton cabinet politician:



## Dan Glickman

Former United States Secretary of Agriculture

Daniel Robert Glickman is an American politician, lawyer, lobbyist, and nonprofit leader. He served as the United States Secretary of Agriculture from 1995 until 2001, prior to which he represented Kansas's 4th congressional district as a Democrat in Congress for 18 years.

...as well as politician and former Rhode Island Republican Party chairman:

## Bernard Jackvony

Lawyer

Bernard A. Jackvony is a former lieutenant governor of Rhode Island and a lawyer who specialises in fiduciary litigation. Born in Providence, Rhode Island, he holds a bachelor's degree from Bryant University, a master's degree from Boston University and a J.D. degree from Suffolk University.

...a lobbyist-consultant who is also an expert on fundraising strategies (hint *quid pro quo* gets money!!!!):



30

## Jorge Luis Lopez, Esq.

Book Author "FRONT AND CENTER WITH TRUMP:  
AMERICAS WARTIME PRESIDENT | English & Spanish | Kindle  
& Paperback | New Release Amazon  
Miami, Florida · 500+ connections

 [Jorge Luis Lopez Law Firm](#)

 [University of Miami School of Law](#)

 [Company Website](#)

[Join to Connect](#)

## About

Jorge Luis Lopez, Esq., practicing law since 1987, established his governmental affairs practice, the Jorge Luis Lopez Law Firm in 2007. He is recognized as one of the top lawyers in South Florida and the U.S., an expert in political campaigns and fundraising as well as a dedicated philanthropist for charitable organizations in Miami-Dade and South Florida communities.

As a Government Relations Specialist, he represents the interests of private and public sector clients before federal, state and local governments, advising clients on the development and response to government regulations, procurement cycles, land use, zoning and redistricting. He has represented numerous Florida 100 and Fortune 500 companies, including some of Miami's leading companies, in complex intergovernmental matters.

...another Tech venture capitalist:

Mr. Marquardt has been the chief executive officer of Global Kompass Strategies, Inc. since 2009 after serving as CEO of three other companies over the past 25 years. Having lived and worked in Europe, Asia and the United States, he maintains an international network of corporate and government leaders. Mr. Marquardt is a global advisor to corporations around the world, working closely with senior leadership teams on business development, strategic planning and cybersecurity issues. He also advises boards of directors and audit committees on effective risk management measures, corporate governance, geographic expansion, and emerging digital technology opportunities. Mr. Marquardt has extensive experience with corporate turn-around situations, crisis management, succession planning, foreign due diligence and transformation at the enterprise level. He served as chair of the former South Atlantic Division Board, received the Society's St. George National Award in 2017, and has served on the American Cancer Society Cancer Action Network's Board of Directors since 2014 and the American Cancer Society Board of Directors since 2018.



...then we have a biotech venture Capitalist:

## P Kay Coleman

President & CEO at Del Mar Venture Group, LLC  
Encinitas, California

Experienced Entrepreneur with a demonstrated history of working in the management consulting industry. Skilled in Executive Development, Business Transformation, Strategic Leadership, Change Management, an.

...there is John J. Manna, Jr., Esq., your run-of-the-mill tech venture capitalist:

Mr. Manna received his Bachelor of Arts from Fordham University and his Juris Doctor from Columbia University School of Law and is currently an investor in real estate and technology ventures.

...Big Pharma executive, Gary Reedy:

Prior to taking the helm of the American Cancer Society, Gary had a distinguished 37-year advocacy leader, most recently as worldwide vice president, government affairs and policy spearheaded initiatives to influence global health policy. He previously devoted more than business side of the industry, including senior leadership positions with SmithKline Beecham Johnson. During his tenure at Johnson & Johnson, Gary served as president of Ortho Biotech with annual revenues of more than \$3 billion.

...an apparent Evangelical Superintendent for good measure:

Dr. Coulter earned his Bachelor of Science In Education from Ouachita Baptist University and his Mast Education and Doctorate In Education from the University of Arkansas. He and his wife Lucretia live on a 400-acre farm and are blessed with 7 children and 15 grandchildren.

...A Buick Dealer! We couldn't make this up. However, automotive products are a leading source of carcinogenic exposure.



**WILLIAM P UNDERRINER**

Dealer Principal

William P. Underriner, president and co-owner of Underriner Motors in Billings, Mont., is 2012 chairman of the National Automobile Dealers Association and represents Montana's franchised new-car dealers on the association's board of directors. In the automobile business since 1984, Underriner took over the family business in 2001. He currently owns Honda, Hyundai, Buick and Volvo franchises in Billings. Previously, he served three terms as Treasurer of NADA and on the

Furthermore, the American Cancer Society serves also as a lobbyist organization, [spending 4 million dollars a year](#) of charitable donations in Washington DC:

# American Cancer Society

Top Affiliates: ACS Cancer Action Network

## CONTRIBUTIONS

Ranks 3,537 of 19,115

**\$49,418**

## LOBBYING

Ranks 114 of 5,502 in 2019

**\$4,450,000** in 2019

The aforementioned people control the American Cancer Society and they have conflicting obligations with their respective industries. They partially use this organization as a "Public Relations" platform to deny [the large body of science linking RFR to cancer](#), and control the narrative. The "Tech Sector" makes generous charitable tax-write offs, with *quid pro quo* strings attached to control the narrative on this emerging health threat; this has paid-off beyond their wildest dreams. Technology and capitalism can be a great thing, but less so for unbridled greed [hijacking](#) vulnerable resources.

Notwithstanding, **it is completely inappropriate for a municipality to form its policy entirely off a private charity's webpage.** This would not even pass as an acceptable primary information source for a high school science essay, let alone [a government document](#); basic research and library skills are publicly taught for a reason. The City needs to perform actual **due diligence**, emphasize an inquisitive and professional approach to the public welfare, consult the published scientific literature as well as academic experts, and give heavy weight to the concerns of the public. It is better to be safe than sorry. The Tahoe Prosperity Center has proven itself untrustworthy, and should not be making City policy.

If the Tahoe Prosperity Center were seriously concerned about organic small business prosperity, it's would be advocating for a cellular network architecture of low-power [indoor distributed antenna systems \(DAS\)](#) that supplement seasonal store and restaurant revenue with steady telecom lease income, rather giveaway the town to centralized high-intensity telecom broadcasting which primarily benefits out-of-area corporate titans. The necessary cellular signal emitters should be installed inside places that are the sources of demand, rather than outdoors in neighborhoods, requiring extremely high-intensity broadcasts in futile attempts to penetrate cement infrastructure and facilities. This clutter ought to be installed on the indoor walls of the very businesses that demand towers in other people's residential yards.

If the Tahoe Prosperity center were genuinely concerned about low income internet access as a right, it would be advocating [fiber-to-the-premises](#) form of **municipal broadband**, using the City's bulk buying power to ensure the cheapest possible access to all denizens, the solution commonly implemented in cities directly tackling this issue. However, they are advocating for an **extremely specific telecommunications architecture** that **benefits very specific entities**, has very specific losers, does not even attempt to minimize adverse impacts to residents or the environment, and is using the banner of "charity" sell it.

Submitted on behalf of,

Tahoe Residents for Actual Prosperity



UNITED STATES ENVIRONMENTAL PROTECTION AGENCY  
WASHINGTON, D.C. 20460

JUL 16 2002

OFFICE OF  
AIR AND RADIATION

Ms. Janet Newton  
President  
The EMR Network  
P.O. Box 221  
Marshfield, VT 05658

Dear Ms. Newton:

This is in reply to your letter of January 31, 2002, to the Environmental Protection Agency (EPA) Administrator Whitman, in which you express your concerns about the adequacy of the Federal Communications Commission's (FCC) radiofrequency (RF) radiation exposure guidelines and nonthermal effects of radiofrequency radiation. Another issue that you raise in your letter is the FCC's claim that EPA shares responsibility for recommending RF radiation protection guidelines to the FCC. I hope that my reply will clarify EPA's position with regard to these concerns. I believe that it is correct to say that there is uncertainty about whether or not current guidelines adequately treat nonthermal, prolonged exposures (exposures that may continue on an intermittent basis for many years). The explanation that follows is basically a summary of statements that have been made in other EPA documents and correspondence.

The guidelines currently used by the FCC were adopted by the FCC in 1996. The guidelines were recommended by EPA, with certain reservations, in a letter to Thomas P. Stanley, Chief Engineer, Office of Engineering and Technology, Federal Communications Commission, November 9, 1993, in response to the FCC's request for comments on their Notice of Proposed Rulemaking (NPRM), Guidelines for Evaluating the Environmental Effects of Radiofrequency Radiation (enclosed).

The FCC's current exposure guidelines, as well as those of the Institute of Electrical and Electronics Engineers (IEEE) and the International Commission on Non-ionizing Radiation Protection, are thermally based, and do not apply to chronic, nonthermal exposure situations. They are believed to protect against injury that may be caused by acute exposures that result in tissue heating or electric shock and burn. The hazard level (for frequencies generally at or greater than 3 MHz) is based on a specific absorption dose-rate, SAR, associated with an effect

that results from an increase in body temperature. The FCC's exposure guideline is considered protective of effects arising from a thermal mechanism but not from all possible mechanisms. Therefore, the generalization by many that the guidelines protect human beings from harm by any or all mechanisms is not justified.

These guidelines are based on findings of an adverse effect level of 4 watts per kilogram (W/kg) body weight. This SAR was observed in laboratory research involving acute exposures that elevated the body temperature of animals, including nonhuman primates. The exposure guidelines did not consider information that addresses nonthermal, prolonged exposures, i.e., from research showing effects with implications for possible adversity in situations involving chronic/prolonged, low-level (nonthermal) exposures. Relatively few chronic, low-level exposure studies of laboratory animals and epidemiological studies of human populations have been reported and the majority of these studies do not show obvious adverse health effects. However, there are reports that suggest that potentially adverse health effects, such as cancer, may occur. Since EPA's comments were submitted to the FCC in 1993, the number of studies reporting effects associated with both acute and chronic low-level exposure to RF radiation has increased.

While there is general, although not unanimous, agreement that the database on low-level, long-term exposures is not sufficient to provide a basis for standards development, some contemporary guidelines state explicitly that their adverse-effect level is based on an increase in body temperature and do not claim that the exposure limits protect against both thermal and nonthermal effects. The FCC does not claim that their exposure guidelines provide protection for exposures to which the 4 W/kg SAR basis does not apply, i.e., exposures below the 4 W/kg threshold level that are chronic/prolonged and nonthermal. However, exposures that comply with the FCC's guidelines generally have been represented as "safe" by many of the RF system operators and service providers who must comply with them, even though there is uncertainty about possible risk from nonthermal, intermittent exposures that may continue for years.

The 4 W/kg SAR, a whole-body average, time-average dose-rate, is used to derive dose-rate and exposure limits for situations involving RF radiation exposure of a person's entire body from a relatively remote radiating source. Most people's greatest exposures result from the use of personal communications devices that expose the head. In summary, the current exposure guidelines used by the FCC are based on the effects resulting from whole-body heating, not exposure of and effect on critical organs including the brain and the eyes. In addition, the maximum permitted local SAR limit of 1.6 W/kg for critical organs of the body is related directly to the permitted whole body average SAR (0.08 W/kg), with no explanation given other than to limit heating.

I also have enclosed a letter written in June of 1999 to Mr. Richard Tell, Chair, IEEE SCC28 (SC4) Risk Assessment Work Group, in which the members of the Radiofrequency Interagency Work Group (RFIAWG) identified certain issues that they had determined needed to be addressed in order to provide a strong and credible rationale to support RF exposure guidelines.

**Federal health and safety agencies have not yet developed policies concerning possible risk from long-term, nonthermal exposures.** When developing exposure standards for other physical agents such as toxic substances, health risk uncertainties, with emphasis given to sensitive populations, are often considered. Incorporating information on exposure scenarios involving repeated short duration/nonthermal exposures that may continue over very long periods of time (years), with an exposed population that includes children, the elderly, and people with various debilitating physical and medical conditions, could be beneficial in delineating appropriate protective exposure guidelines.

I appreciate the opportunity to be of service and trust that the information provided is helpful. If you have further questions, my phone number is (202) 564-9235 and e-mail address is [hankin.norbert@epa.gov](mailto:hankin.norbert@epa.gov).

Sincerely,



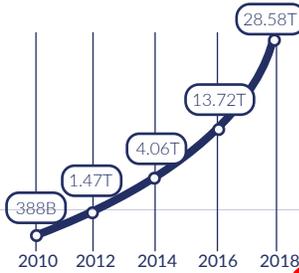
Norbert Hankin  
Center for Science and Risk Assessment  
Radiation Protection Division

Enclosures:

- 1) letter to Thomas P. Stanley, Chief Engineer, Office of Engineering and Technology, Federal Communications Commission, November 9, 1993, in response to the FCC's request for comments on their Notice of Proposed Rulemaking (NPRM), Guidelines for Evaluating the Environmental Effects of Radiofrequency Radiation
- 2) June 1999 letter to Mr. Richard Tell, Chair, IEEE SCC28 (SC4) Risk Assessment Work Group from the Radiofrequency Radiation Interagency Work Group

# TAHOE WIRELESS BROADBAND AND YOU

## REPORTED WIRELESS DATA TRAFFIC (MEGABYTES)



- **Wireless data use almost doubles in just one year.** Wireless data puts the internet in the palm of our hand and allows us to access nearly anything or anyone on the go, and its tremendous value to consumers shows no signs of slowing.
- This year, we saw mobile data grow by **12.89 trillion MBs** to a **total of 28.58 trillion.**
- That's an **82 percent increase** in the last year alone and is more data than was used in the first six and a half years of this decade combined.
- In fact, data use is up over **73 times since 2010.**<sup>1</sup>

## NEED MORE TOWERS



Less Towers = Longer Wait

## STUDIED FOR SAFETY

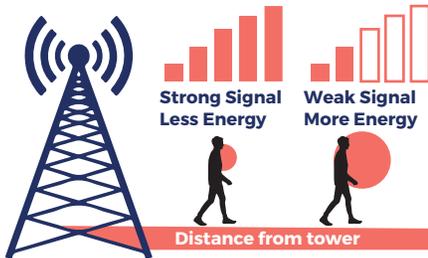


Regulated Levels Are Safe to Humans



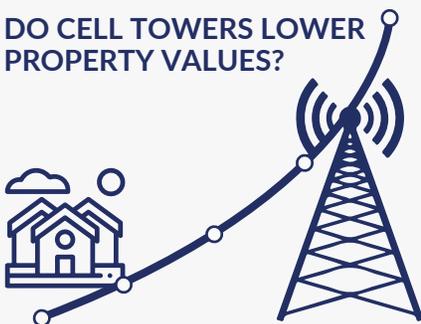
- **A fast wireless network is a critical resource for our community,** and failing to provide fast wireless networks is no different than failing to provide clean drinking water, natural gas, sewage service, or electricity.<sup>3</sup>
- When a disaster occurs people need to know about it. An increasingly large segment of the population use mobile devices instead of landlines. **Receiving a report on mobile devices is vital for emergency preparedness.**
- The World Health Organization (WHO) has classified radio frequency energy as “possibly carcinogenic to humans.” **WHO also states that in the last twenty years no adverse health effects have been established as being caused by mobile phone use.**<sup>2</sup>
- The American Cancer Society, the International Agency for Research on Cancer and the National Toxicology Program claim that **cell towers are unlikely to cause cancer.**

## CELL TOWER SIGNAL



- **Using phones in areas of good reception decreases exposure as it allows the phone to transmit at reduced power.** More towers mean better coverage and hence less electromagnetic field radiation exposure from mobile phones.<sup>2</sup>

## DO CELL TOWERS LOWER PROPERTY VALUES?



- The distance from a wireless facility has no apparent impact on the value or sale price of a home. The relationship between **the list and sale price remained the same no matter how close the property was to the wireless facility.**<sup>5</sup>

## ARE CELLPHONE TOWERS DANGEROUS?

Research by organizations such as the National Institute for Occupational Safety and Health, the environmental Protection Agency (EPA), FCC and others have found **radio frequency energy within the regulated levels are not harmful to humans.**

Radio frequency waves, a form of energy, is released when a mobile device (phone, tablet or laptop) connects with a cell tower.

Different devices create different frequencies on the electromagnetic spectrum. **Some frequencies are harmful to humans while others are not.**

For instance, the frequencies that carry x-rays and gamma rays are on the radioactive end of the electromagnetic spectrum, and can cause harmful damage to the chemical bonds in our DNA.

Radio frequency energy from cell towers and mobile devices is "non-ionizing," similar to radio and television waves.

All cell towers keep radio frequency energy high above the ground. **At ground level, radio frequency energy from towers is thousands of times less than the FCC safe exposure limits.** Other antennas, such as those used for radio and television broadcast transmissions, use power levels that are generally much higher than those used for cellular antennas.<sup>6</sup>

## DEFINITIONS

**Mobile Broadband** – The use of high speed internet via mobile devices (smart phone, tablet or laptop) that utilizes frequencies on the electro magnetic spectrum.

**Electromagnetic Spectrum** – The range of frequencies that emit electromagnetic energy. The lower end of the spectrum has low frequencies and longer waves of energy, while the higher end has high frequencies and shorter waves.

**Electromagnetic Energy** – Any energy emitted or absorbed by charged particles traveling through space, anything from visible light to nuclear reactions.

**Ionizing and Non-ionizing Energy** – Ionizing energy is energy on the high end of the spectrum that is harmful to human DNA. Energies that are on the low end of the spectrum are considered non-ionizing energy and are not harmful to humans.

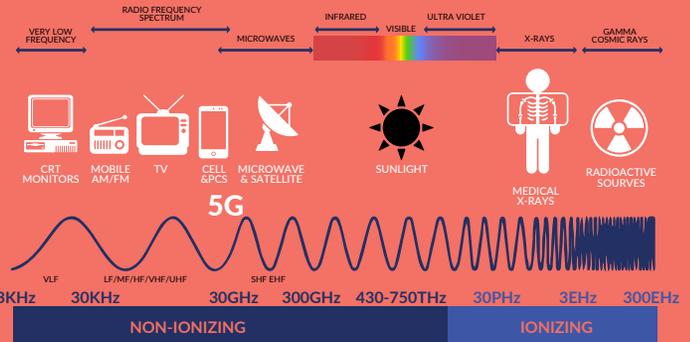
**Radio Frequency Energy** - The range of frequencies on the non-ionizing end of the electromagnetic spectrum used for telecommunications devices such as mobile phones, laptops, radios and television.

## WHAT THE EXPERTS SAY...

A systematic review of existing academic studies on the potential health risks of radio frequency emissions found that **the majority of research on the subject currently indicates no ill-health related to radio frequency energy exposure.**<sup>7</sup>

Research is ongoing. There is consensus that additional research is warranted to address gaps in knowledge, such as the effects of cell phone use over the long-term and on pediatric populations.<sup>8</sup>

### THE ELECTROMAGNETIC SPECTRUM



The electromagnetic spectrum. CNET

## REFERENCES

1. CNET 2019 Annual Survey, [www.cnet.com/news/2019-annual-survey-highlights](http://www.cnet.com/news/2019-annual-survey-highlights)
2. World Health Organization, Electromagnetic fields and public health: mobile Phone Use. WHO Fact Sheet #193. June, 2011. Reviewed October 2014
3. Wireless Emergency Alerts report by the Department of Homeland Security, [www.dhs.gov/sites/default/files/publications/Wireless%20Emergency%20Alerts%20Mobile%20Communication%20Strategy.pdf](http://www.dhs.gov/sites/default/files/publications/Wireless%20Emergency%20Alerts%20Mobile%20Communication%20Strategy.pdf)
4. The American Cancer Society, [www.cancer.org/cancer/cell-phone-towers/othercarcinogens/athome/cellular-phone-towers](http://www.cancer.org/cancer/cell-phone-towers/othercarcinogens/athome/cellular-phone-towers)
5. Joint Ventures Wireless Communications Initiative Study - Wireless Facilities Impact on Property Values, November 2012 [www.jointventure.org/images/stories/pdf/WirelessFacilitiesImpactOnPropertyValues.pdf](http://www.jointventure.org/images/stories/pdf/WirelessFacilitiesImpactOnPropertyValues.pdf)
6. FCC Radio Frequency Safety, [www.transition.fcc.gov/oet/rfsafety/rf-faqs.html](http://www.transition.fcc.gov/oet/rfsafety/rf-faqs.html)
7. Martin Röösli et al., "Systematic Review on the Health Effects of Exposure to Radiofrequency Electromagnetic Fields from Mobile Phone Base Stations," Bulletin of the World Health Organization 88, no. 12 (December 1, 2010): 887–896F.
8. The Food and Drug Administration, [www.fda.gov/Radiation-EmittingProducts/RadiationEmittingProductsandProcedures/HomeBusinessandEntertainment/CellPhones/ucm116335.htm](http://www.fda.gov/Radiation-EmittingProducts/RadiationEmittingProductsandProcedures/HomeBusinessandEntertainment/CellPhones/ucm116335.htm)

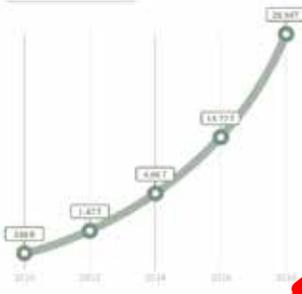


TAHOE PROSPERITY CENTER

[tahoeprosperity.org](http://tahoeprosperity.org)

# SLT WIRELESS BROADBAND AND YOU

## REPORTED WIRELESS DATA TRAFFIC (MEGABYTES)



## October 2019

- **Wireless data use almost doubles in just one year.** Wireless data puts the internet in the palm of our hand and allows us to access nearly anything or anyone on the go, and its tremendous value to consumers shows no signs of slowing.
- This year, we saw mobile data grow by **12.89 trillion MBs** to a **total of 28.58 trillion.**
- That's an over **82 percent increase** in the last year alone and is more data than was used in the first six and a half years of this decade—combined.
- In fact, data use is up over **73x since 2010.**

### Reference point 1. CTIA 2019 Annual Survey

<https://www.ctia.org/news/2019-annual-survey-highlights/>

## NEED MORE TOWERS



Less Towers = Longer Wait

- Using phones in areas of good reception decreases exposure as it allows the phone to transmit at reduced power." More towers mean better coverage and hence less EMF radiation exposure from mobile phones.

### Reference 3. FCC Factsheet No 193. Reviewed October 2014

## STUDIED FOR SAFETY

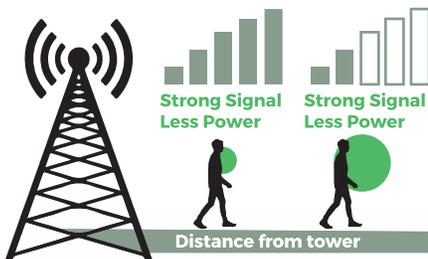


- A fast wireless network is a critical resource for our citizens, and failing to provide them is no different than failing to provide clean drinking water, natural gas, sewage service, or electricity.
- When a disaster occurs, many people need to know about it. An increasingly large segment of the population now uses mobile devices instead of landlines. Receiving an alert on mobile devices is vital for emergency preparedness.

### Reference 2. Wireless Emergency Alert Support by the Department of Homeland Security

[www.dhs.gov/sites/default/files/publications/Wireless\\_Emergency%20Alerts%20Mobile%20Penetration%20Strategy.pdf](http://www.dhs.gov/sites/default/files/publications/Wireless_Emergency%20Alerts%20Mobile%20Penetration%20Strategy.pdf)

## THIS IS NOT A 5G TOWER



- A 5G tower is different than a 4G tower both physically and functionally: more 5G towers are needed to cover the same amount of space, they're much smaller, and they transmit data on an entirely different part of the radio spectrum.

### Reference 4. 5G Cell Towers: Why You See Them and How They Work

<https://www.lifewire.com/5g-cell-towers-4584192>

## Do Cell Towers Lower Property Values?



- The distance from a wireless facility has no apparent impact on the value or sale price of a home. The relationship between the list and sale price remained the same no matter how close the property was to the wireless facility.

### Reference 5. Joint Ventures Wireless Communications Initiative Study Wireless Facilities Impact on Property Values November 2012

<https://jointventure.org/images/stories/pdf/WirelessFacilitiesImpactOnPropertyValues.pdf>

## Are Cellphone Towers Dangerous?

Research by organizations such as the National Institute for Occupational Safety and Health, The Environmental Protection Agency (EPA), FCC and others have found **RF energy within the regulated levels are not harmful to humans.**

Radiofrequency (RF) waves, a form of energy, is released when a mobile device (phone, tablet or laptop) connects with a cell tower.

Different devices create different frequencies on the Electro Magnetic Spectrum. Some frequencies are harmful to humans while others are not.

For instance, the frequencies that cell towers use and gamma rays are on the radioactive range of the electromagnetic spectrum, and can cause harmful damage to the chemical bonds in our DNA.

**RF energy from cell towers and mobile devices is “non-ionizing,” similar to radio and television waves.**

Tall cell towers keep RF energy high above the ground. **At ground level, RF energy from towers is thousands of times less than the FCC safe exposure limits.** Other antennas, such as those used for radio and television broadcast transmissions, use power levels that are generally much higher than those used for cellular antennas.”

## DEFINITIONS & REFERENCES

**Mobile Broadband** – The use of high speed internet via mobile devices (smart phone, tablet or laptop) that utilizes frequencies on the electro magnetic spectrum.

**Electro Magnetic Spectrum** – The range of frequencies that emit electro magnetic energy. The lower end of the spectrum has low frequencies and longer waves of energy, while the higher end has high frequencies and shorter waves.

**Electro Magnetic Energy** – Any energy emitted or absorbed by charged particles traveling through space, anything from visible light to nuclear reactions.

**Ionizing and Non-ionizing Energy** – Ionizing energy is energy on the high end of the spectrum that is harmful to human DNA. Energies that are on the low end of the spectrum are considered non-ionizing energy and are not harmful to humans.

**Radio Frequency (RF) Energy** - The range of frequencies on the non-ionizing end of the electro magnetic spectrum used for telecommunication devices such as mobile phones, laptops, radios and television.

## What the Experts Say...

A systematic review of existing academic studies on the potential health risks of RF emissions found that the majority of research on the subject currently indicates no ill-health related to RF energy exposure.

The World Health Organization (WHO) has classified RF energy as “possibly carcinogenic to humans.” WHO also states that in the last twenty years “no adverse health effects have been established as being caused by mobile phone use.”

The American Cancer Society, the International Agency for Research on Cancer and the National Toxicology Program claim that cell towers are unlikely to cause cancer.

Research is ongoing. There is consensus that additional research is warranted to address gaps in knowledge, such as the effects of cell phone use over the long-term and on pediatric populations.



World Health Organization, Electromagnetic fields and public health: mobile Phones, WHO Fact Sheet #102, June, 2011

FCC Radio Frequency Safety <http://transition.fcc.gov/oet/rfsafety/rf-faqs.html>

The American Cancer Society, <http://www.cancer.org/cancer/cancercauses/othercarcinogens/athome/cellphone Towers>

Martin Rööfli et al., “Systematic Review on the Health Effects of Exposure to Radiofrequency Electromagnetic Fields from Mobile Phone Base Stations,” Bulletin of the World Health Organization, November 1, 2010): 887–896F.

The Food and Drug Administration, <http://www.fda.gov/Radiation-EmittingProducts/RadiationEmittingProductsandProcesses/HomeBusinessandEntertainment/CellPhones/ucm116335.htm>

CTIA 2019 Annual Survey, <https://www.ctia.org/news/2019-annual-survey-highlights/>

Wireless Emergency Alerts Report by the Department of Homeland Security, [www.dhs.gov/sites/default/files/publications/Wireless%20Emergency%20Alerts%20Mobile%20Penetration%20Strategy.pdf](http://www.dhs.gov/sites/default/files/publications/Wireless%20Emergency%20Alerts%20Mobile%20Penetration%20Strategy.pdf)

WHO Factsheet No 193. Reviewed October 2014

5G Cell Towers: Why You See Them and How They Work, <https://www.lifewire.com/5g-cell-towers-4584192>

Joint Ventures Wireless Communications Initiative Study Wireless Facilities Impact on Property Values. November 2012 <https://jointventure.org/images/stories/pdf/WirelessFacilitiesImpactOnPropertyValues.pdf>



TAHOE PROSPERITY CENTER

Read our response to Covid-19. **Get 3 months free** of the Digital Edition.

[Subscribe Now](#)



# The WASHINGTON SPECTATOR

[Politics](#) [World](#) [Culture](#) [Economy](#) [Letters](#) [Latest Issue](#)

[Subscribe](#) [Contact Us](#) [Support](#) [Q](#)

by Barbara Koepfel

Dec 28, 2020 | [Health](#)



PHOTO CREDIT: Verizon Wireless video advertisement

[f Facebook](#)

[Twitter](#)

[✉ Email](#)

If you think your cellphone is safe, have you considered why you believe that? Is it a fact or is it based on carefully crafted messages that you've read or heard?

For the past few decades, the telecom wireless industry and its enthusiasts have heralded cellphones as the greatest achievement of the late 20th and early 21st centuries. But as their use soars, scientists worldwide worry about their hazards and have produced over 2,000 studies that tell a darker tale. They warn that the devices and antennas that power them expose humans and wildlife to nonionizing low-frequency electromagnetic fields—also called cellphone, microwave, or radio-frequency radiation. These studies indicate that when people and animals are exposed, they can develop brain, thyroid gland, prostate gland, acoustic nerve, and breast tumors, and other diseases.

Not surprisingly, the industry argues this type of radiation is safe, because it is unlike the high-frequency ionizing radiation used in X-rays, which can directly damage DNA.

Still, scientists say low frequency doesn't mean harmless. For example, based on data from the U.K. Office of National Statistics, Alasdair Philips, an engineer, scientist, and trustee of Children With Cancer U.K., found that cases of brain tumors (glioblastomas) in Great Britain from 1995 to 2015 mushroomed, from 983 to 2,531.

Why? Philips says, "There's adequate proof that exposure from wireless devices affects cancer cells. Even if they don't start the cancers, they speed up the rate at which the cancer cells multiply. This is true of all the devices—cellphones, tablets, and cordless phones people use in their homes—since they have built-in antennas that communicate with cell towers.

"The exposure is quite significant because people hold their devices near their heads for hours while they stream videos and other materials." He warns that the exposure is particularly potent when the reception is poor: "At such time, the signal's strength can increase by even a millionfold."

Philips says the upsurge in tumors is mainly among those over 50—since this age group typically has more tumors. But, although very few 10-to-15-year-olds get brain tumors, that number is also increasing. He adds that "besides

promoting cancer, microwave radiation makes lower-grade tumors become more aggressive.”

Robert Kane, an electromagnetics engineer who designed and tested wireless devices for Motorola and other firms starting in the 1980s, warned of the dangers in his book *Cellular Telephone: Russian Roulette* (2001). Given his position inside the industry, he was able to confirm that cellphone companies knew their products could harm and even kill, but, like the tobacco, asbestos, and fossil fuel industries, they kept the news quiet. Besides the increased risk of tumors, Kane also described hundreds of studies since the 1950s that found that low-level radiation damaged DNA and tissues and caused loss of memory and motor skills, and cataracts. Kane died of a brain tumor in 2002.

The industry rejects the data. Its main trade group, the Cellular Telecommunications Industry Association (CTIA), states “wireless devices do not pose a public health risk for adults or children.” Although it admits devices and cell towers emit radio-frequency radiation, it says this exposure can only cause acute, short-term overheating of human and animal tissues. But the CTIA also insists this doesn’t happen, because the amount of radiation is minuscule. Instead, it argues that long-term illnesses such as cancer are a fiction of marginal alarmist researchers.

Even the \$30 million, decade-long study by a National Institutes of Health division called the National Toxicology Program, the results of which were released in 2018, didn’t dent industry’s denials. For two years, NTP scientists exposed rats to cellphone radio-frequency radiation and found “clear evidence of cancer in the male rats’ heart cells, some evidence of increased brain gliomas (brain cancer) and adrenal gland tumors, DNA damage in the brains of male and female rats and mice, lower birth weights of female rats’ offspring, and decreased sperm quality.” Ron Melnick, a senior scientist (now retired) at the NTP who led the design of the study, says they also found tumors in the rats’ prostate glands. The numbers were confirmed by a panel of experts.

Still, the story was squashed: the press mostly ignored or dismissed it. And the U.S. watchdog agencies—the Federal Communications Commission and the Food and Drug Administration, which set the safety regulations for wireless devices—disputed the findings. The FDA argued that “the study was not designed to test the safety of cellphone use in humans, so we cannot draw conclusions about the risks [to humans] from it.” Melnick says, “This statement was odd because when we were designing it, the FDA told us an animal study was needed. But when we announced the results, the FDA said, ‘The current safety limits for cellphone exposure, set in 1996, remain acceptable.’” And the FCC concurred.

Melnick sought feedback from scientists outside the NTP and asked one who worked for Motorola to discuss the results. “He refused. He told me we already have lots of studies that don’t show these effects,” Melnick says.

The FDA and FCC claimed the results were skewed because NTP scientists exposed the rats’ entire bodies to higher doses of radiation than cellphones typically emit. But their arguments were countered by scientists at Italy’s Ramazzini Institute (a nonprofit cancer research center in Bologna) who exposed 2,500 rats in the fetus and until their death to lower doses of radiation than those emitted in cellphones. These animals developed the same rare heart cancers.

Why are the deniers so adamant? “It’s all about money, since there are billions, even trillions, at stake,” says Jerry Phillips, a biochemist who directs a science center at the University of Colorado. Indeed, in 2018, global cellphone sales were more than a half-trillion dollars.

The industry is spectacularly successful in ensuring that its message echoes far and wide: its profoundly deep pockets purchase seats at all the right tables in the global and national watchdog agencies, media organizations, and scientific associations—which manage the misinformation. Thus, industry’s billions decide which scientists and studies get funded or defunded, which get quoted or discredited, which agency commissioners bounce back and

forth from telecom companies and corporate law firms, and how dissenters—such as U.S. states and cities—are sued and usually silenced.

At present, the industry and its backers are hyping 5G—the newest generation of devices, following 2G, 3G, and 4G. Online, in newspapers and on television, we are told 5G will change life as we know it—with vastly increased speeds for streaming material and devices that are able to communicate with each other (sometimes called “the internet of things”). The ads also promise that 5G will add \$500 billion to the U.S. economy. Verizon, a key player, even claims it “will help doctors see cancer like never before.”

The scientists worry even more. They say 5G technology uses millimeter waves, along with microwaves (the type in current devices). Because 5G waves can only travel short distances, antennas and towers need to be installed every 300 to 600 feet on every block across the country, to receive and send signals. And this, Philips says, “increases the exposures exponentially.”

Joel Moskowitz, director of the Center for Family and Community Health at the University of California, Berkeley, says “because the technology is so new, we have no way to know about the long-term health effects. But we do know that millimeter waves are absorbed in our skin and on the cornea and can harm the immune, nervous, and cardiovascular systems.”

The U.S. Government Accountability Office agrees—although it buried the warning on page 42 of a report it released this past November. The GAO quotes a National Cancer Society scientist who said “no studies of 5G frequencies have been conducted on the long-term health effects because the technology hasn’t been deployed long or widely enough.” Worse, the scientist warns the effects may not be known “for many years, because some outcomes could take decades to develop.”

Still, the GAO has hyped the 5G debut, as have the other U.S. agencies: It posted a video featuring Tom Wheeler, the former FCC chair and CTIA CEO,

who, not surprisingly, never mentioned the health issues.

However, given the industry's daily drumbeat, there is a dramatic disconnect between the critics' concerns and public awareness. As a result, only 5 percent of U.S. adults worry that cellphones are harmful, and parents buy them for their children: in 2019, 53 percent of children under 12 and 84 percent of teens had them.

Further, few people know that when reception is poor and phones show just one or two bars—say, when users are in subways, elevators, cars, basements, or some rural areas—the devices need more energy to communicate with cell towers and other phones. Philips explained that this leads to a massive increase in exposure. This conclusion was also **noted** in a 2017 California Department of Public Health advisory titled *How to Reduce Exposure to Radiofrequency Energy From Cellphones*, which led the department to warn the public not to use phones in such places.

For their part, the manufacturers and telecom companies don't mention this concern. Instead, they inform users about the proper distance to hold phones from their bodies to avoid excessive exposure (from 5 to 25 millimeters away—about one-fifth of an inch to an inch). But they bury even these modest advisories deep inside the owner manuals.

Moskowitz says, “The problem is that we really don't know what distance is safe for people who use the devices over many years.” Thus, he and other scientists I interviewed said they only use wired landlines at home; and, when out, they carry cellphones in backpacks, brief cases, or tote bags.

However, the industry's message is so widely accepted that contradictory information is routinely discarded. One scientist (who asked for anonymity) told me he recently was asked to advise a state committee about 5G guidelines. “When I tried to tell them about the hazards from the hundreds of thousands or millions of new antennas that will be installed, they weren't interested. Instead, they only looked at materials from a telecom company,

which said the ‘greatest risks from cellphones are traffic deaths due to drivers being distracted.’”

Similarly, when the U.K. National Radiological Protection Board warned, as early as 2000, that people should keep calls short and use hands-free earpieces, the FDA and FCC insisted “the scientific evidence does not show a danger.”

The disconnect was striking at two meetings I attended in Washington D.C. about the coming of 5G. Both had panelists from the D.C. government and industry who championed its benefits. During the Q&A, when someone asked about safety issues, panelists confidently claimed there were “none.”

### **Compromised watchdogs**

How does industry carry it off? First, the watchdog agencies continually reaffirm the industry’s message, and because of their authority, they’re considered objective. Yet their conflicts of interest are pervasive. For example, in 2013, President Obama named Tom Wheeler, the CEO of the main trade group, the CTIA, to chair the FCC. In a 2016 talk, Wheeler said, “We won’t wait for standards to be developed. . . . Instead, we will rely on the private sector to produce them.” On 5G, he told doubters to “stay out of the way. . . . Tens of billions of dollars in economic activity . . . is what’s important.”

President Trump replaced Wheeler with Ajit Pai, a former Verizon legal counsel and attorney at Jenner & Block, which represents the CTIA. As Jenner & Block’s site boasts, “No firm has the experience and credibility we enjoy before the FCC.”

This is not an idle claim. Pai—the regulator in chief—dislikes regulations. In 2018, he repealed the FCC’s net neutrality rules, which, *Los Angeles Times* business columnist Michael Hiltzik noted, “involves billions of dollars in

potential profits for Verizon and other firms.”

Moreover, Pai is determined to quash 5G opponents. In 2018, the FCC issued an order that would force cities to stop blocking companies that were installing 5G antennas. The order also lets the firms sue cities if they don't approve their installation plans in 60 or 90 days. Further, it says that companies needn't wait for health or environmental studies to prove the equipment is safe: instead, they only have to say they comply with FCC rules.

The FDA is just as obliging. Jeffrey Shuren, who heads its Center for Devices and Radiological Health, is an industry loyalist. As Justin Klein, a partner at Vensana, a medical technology venture capital firm, observed, “Shuren has won the trust of the device world through . . . his ‘industry-friendly record.’” A May 2019 CBS news report confirmed this: when France banned certain breast implants that researchers linked to lymphoma in 2019, Shuren said they were safe—and left them on the U.S. market.

Shuren also does not welcome whistleblowers. A 2012 *Orthopedics Journal* story said that when he ran the FDA unit approving new devices, nine of its scientists warned that a CT scanner they were evaluating could cause cancer. Within months, Shuren fired all nine. Two years later, a U.S. congressional committee reported that Shuren had bugged the scientists' computers to record their activities.

In fact, the U.S. federal government thrives on a thriving telecom industry. In *Captured Agency* (a monograph published in 2015 by Harvard's Center for Ethics), journalist Norm Alster wrote that the government had reaped nearly \$100 billion in prior years from selling space on the electromagnetic field spectrum, through which the companies send their signals. Alster says local governments also prosper, collecting an average of 19 percent from users' cellphone bills.

### **Other deniers**

Henry Lai, a University of Washington bioengineer researcher, says the industry's influence is so profound that "even the American Cancer Society accepts its views." So, too, have other respected groups, such as the World Health Organization and the U.S. Centers for Disease Control and Prevention, which repeat the "no radiation problems" refrain.

For example, when the National Toxicology Program released the results of its study—citing cancers in the heart cells, brains, and adrenal glands of laboratory rats exposed to cellphone emissions—an American Cancer Society site said, "Updated Cellphone Study Findings Still Inconclusive," the exact opposite of what the scientists concluded. In fact, the ACS's chief medical officer at the time, Dr. Otis Brawley, said, "The evidence for an association between cellphones and cancer is weak."

Could the ACS have industry ties? I asked Kathi Di Nicola, director of ACS media relations, for its donor list. "We do not release individual or partner giving, unless required by law," she emailed back. But an ACS site called "Our Partners" lists Goldman Sachs, Bank of America, and JP Morgan, whose clients include the telecom giants; other partners are the giants themselves, such as Microsoft, United Technologies, and World Wide Technology.

For its part, the CDC switched its position about wireless dangers without offering any reasons. Theodora Scarato, executive director of the Wyoming-based nonprofit group the Environmental Health Trust, which works with communities and health professionals to promote research and policies, says that, in June 2014, the CDC website recommended "caution in cellphone use" and noted that "more research is needed . . . before we know for sure if cellphones cause cancer."

Just two months later, most of the message had disappeared and was replaced by one line: "There is no scientific evidence that provides a definite answer to that question [can using a cellphone cause cancer?]." Scarato notes that her nonprofit submitted hundreds of Freedom of Information Act requests to the CDC to determine why; in doing so, it learned that the CDC

had hired **Kenneth Foster**, an industry consultant, in 2015, to write that agency's new web pages on the health effects of wireless technology.

The WHO has also straddled both sides. In 2011, just one month after its division the International Agency for Research on Cancer (IARC) defined cellphone radiation as a *possible* human carcinogen, a WHO fact sheet claimed "no adverse health effects have been established." However, Alasdair Philips notes that many IARC scientists now believe the group should revisit the issue and change the assessment from possible to probable.

Further, the WHO consistently adopts the views of the International Commission on Non-Ionizing Radiation Protection, or ICNIRP, which, since its founding in 1992, has argued that electromagnetic frequency, or EMF, radiation can only cause damage by heating body tissues, which, it says, wireless devices don't do. The WHO also defers to the United States (whose position is articulated by the FDA and the FCC), which, until recently, when President Trump cut U.S. funding, was the WHO's largest contributor.

Dariusz Leszczynski, a University of Helsinki biochemist, says ICNIRP's views haven't changed because its current members only choose new members who share their beliefs. His opinion is confirmed by James Lin, a University of Illinois professor of engineering, physiology, and biophysics, who was an ICNIRP member for 12 years. He told me, "If you look at the group's output, it says the same things industry says."

Moreover, many ICNIRP members have serious **conflicts of interest**. While they're supposed to list their income on Declaration of Interests forms, they often don't. For example, Michael Repacholi, an Australian biophysicist and ICNIRP's first chair, also founded a WHO project in 1996 to study cellphone radiation effects. But Louis Slesin, editor of *Microwave News*, reported in 2006 that Repacholi admitted the telecom industry had funded half the WHO project's budget. When he left WHO in 2006, Repacholi soon became an industry consultant.

Andrew Wood, who is on the ICNIRP's Scientific Advisory Group, runs a lab at Swinburne University in Australia supported by the Telstra Corporation, which builds and operates digital networks, provides mobile and internet access, and is that country's largest telecommunications company. Telstra gave Wood's lab some equipment and sent its staff there to test Telstra's products.

Rodney Croft, an ICNIRP member since 2008, told an Australian Broadcasting Corporation news show, "A lot of research . . . has clearly shown there aren't any health effects." However, Croft didn't mention that the research center he directed was created with Telstra funding and lab equipment.

Rene de Seze, in ICNIRP for over a decade, left his Declaration of Interests form completely blank—not listing grants from France Telecom or his work for Motorola.

Even the National Institutes of Health (NIH) has minimized the radiation hazards. For several years, it sponsored *Healthy Building Roundtable* conferences, the last one in 2018. On July 19 and 20, speakers on the Electro Magnetic Frequency (EMF) panel **described** the dangers of wireless devices, circulated material at the conference, and posted it on the NIH-Healthy Buildings Roundtable website. It said, "Current FCC public radiation exposure guidelines were set decades ago, based on the outdated premise that devices need to emit enough heat to raise the temperature of one's skin to cause harm. There are now **over 25,000 articles published**, and the majority of non-industry funded studies show great evidence of biological harm at the non-thermal level."

The message still appeared in September, but by early October, it had disappeared. So, too, had any mention of the EMF panel.

### **The loyal press**

Besides the industry's sway with the agencies, its influence on the press and media means that coverage of wireless devices is almost always upbeat. First, the industry buys full-page ads that promote its services and products and now continually tout 5G. Then there are the owners' personal conflicts. For example, *The New York Times*' largest single stockholder is Carlos Slim—the world's richest man in 2013—who holds 17 percent of the newspaper's stock and whose company, America Movil, is Latin America's biggest telecom provider. And Verizon is partnering with the *Times* on a 5G project.

Most press and media repeat the agencies' positions and debunk or ignore studies that describe the dangers. Since *The New York Times* is America's paper of record, its coverage is instructive.

In a May 2019 *Times* story, "Your 5g phone wont hurt you. But Russia wants you to think so," the journalist William Broad quoted Marvin Ziskin, a Temple University professor of radiology, who claimed, "5G emissions, if anything, should be *safer* [emphasis added] than previous generations' exposure of the body's internal organs." But Ziskin's papers, many co-authored by Kenneth Foster, a professor in the Department of Bioengineering at the University of Pennsylvania, are funded by the Wi-Fi Alliance and the Mobile & Wireless Forum, or MWF, a trade group whose members include Apple, Motorola, Samsung, and Sony. As industry favorites, Foster and Ziskin were invited to chair MWF's 2016 workshop sessions in Belgium, and Foster gave the keynote address.

Broad also quotes David Robert Grimes, whom he identifies as an Oxford University cancer researcher. Besides his statements supporting 5G and wireless devices, Grimes discredits the work of David Carpenter, former dean of SUNY's School of Public Health in Albany who has long warned of cellphone hazards: he claims that "Dr. Carpenter's scariest alarms have been widely dismissed by scientific bodies the world over."

But Grimes isn't a reliable judge. His website has a link to his Oxford work, but the link, when clicked, states, "The page is not found." Grimes's site also

notes his work at Queen's University in Belfast, but, as of December 2019, Queen's no longer listed Grimes in its online directory.

Moreover, Grimes's research is on human consumption of oxygen—not cellphone radiation. And although Broad doesn't mention this, Grimes gets industry funds: in one of his papers, Grimes thanks the NVIDIA Corporation for "generous hardware donations" to his research project on radiotherapy (NVIDIA makes parts for smart phones, tablets, and game systems and had an income of \$4 billion in 2018). Grimes also thanks Cancer Research U.K. for its support—an institute that partners with the Francis Crick Research Institute, whose chair is Baron Edmund John Philip Browne, British Petroleum's former head and now chair of Huawei Technologies U.K.

In July 2019, the *Times* ran another story, titled "5G, Don't Fear the Frequency," under a huge multicolored drawing of panicked people. Broad writes that Bill Curry, a physicist who warns about radiation dangers, produced "flawed reports" about the damage of microwave radiation, which were adopted by "alarmist websites." Again, he quotes Grimes, who states, "If phones are linked to cancer, we'd expect to see a marked uptick. Yet we do not." This assertion contradicts research conducted by Alasdair Philips, who used numbers from the U.K. Cancer Registry to document the increase in aggressive brain tumors.

In fact, Broad's articles reveal consistent biases. In reviewing two books on global warming in 1998, he said, "[W]e live in a great climate experiment, the outcomes of which, good or bad, no one is likely to forecast with any certitude." This assurance came nearly 20 years after a National Academy of Sciences report predicted global warming of 2 to 3.5 degrees Celsius (3.6 to 6.3 degrees Fahrenheit)—with greater increases at high latitudes.

In 2007, Broad called Al Gore's documentary *An Inconvenient Truth* "exaggerated." To prove his point, he quoted Don Easterbrook, a geologist who saw "a lot of inaccuracies." But this is the same Easterbrook who told a Washington State Senate Energy, Environment, and Telecommunications

Committee that “global warming ended in 1998.”

Broad’s science denials resurfaced in October 2019, when he wrote that plastics, a major source of ocean pollution are “less devastating than usually portrayed.” To support this assertion, he quotes a marine chemist who claims that “sunlight can degrade them in centuries or even decades,” not a timeline that accords with sustainable management of the world’s marine and coastal environments.

Although most press and media support the industry’s position, there are some rare exceptions. For example, the *Chicago Tribune* launched its own study to measure the radiation from Apple, Samsung, and Motorola cellphones. In an August 2019 article, the *Tribune* said the testing laboratory found that many models exceeded the FCC exposure standards, “particularly when tested close to the body.”

The *Baltimore Sun*, covering a May 2016 Pediatric Academic Society annual meeting, quoted physicians who warned parents to limit their children’s cellphone use. And in October 2005, a *Florida Sentinel* story noted that researchers worried that “radiation enters users’ heads, and over time might pose serious health risks, including cancer.”

Research and retaliation

Industry’s impact on research is also enormous. Henry Lai, the University of Washington bioengineer researcher, reviewed 326 studies on radio-frequency radiation carried out from 1990 to 2005 and found that half showed harmful biological effects, while half did not. When he checked who funded which ones, the numbers diverged dramatically: of those that were independently funded, 70 percent found harmful effects, while among those funded by industry, only 30 percent reported finding them.

For researchers who refute the message, retaliation is certain. A few examples are useful. John Allis, a physical chemist, and Carl Blackman, a biophysicist, were among a group of scientists at the Environmental

Protection Agency studying low-intensity EMF radiation from the 1970s until the mid-1980s—to determine its effect on brain tissue. Allis says that although ‘low’ sounds benign, it “penetrates more deeply than X-rays.” Since their research predated cellphones, they studied the radiation from electric power lines and the military’s radar installations.

“We exposed newly hatched chickens’ brains to it and found that this changed their brain tissues. It was a crucial discovery that we wanted to study further, but EPA stopped our funds,” Blackman says. He then got Department of Energy support, but it also ended, and his equipment was thrown away.

Why? Allis says that “in the 1980s, the Reagan administration was pushing ‘Star Wars,’ which was thought to need nonionizing radiation to make it work. The scuttlebutt was that Washington didn’t want to know it had negative effects. So it stopped the funds.”

Lai and his research partner, N.P. Singh, a professor of bioengineering at the University of Washington, exposed rats’ brains to radio-frequency radiation at an intensity the FCC said was safe. But after just two hours, the radiation broke or damaged the DNA in their brain cells—which can lead to mutations and cancer. When they published their results in a 1995 issue of *Bioelectromagnetics*, Motorola cut their funds and counterattacked: Slesin posted a leaked memo in a 1997 *MicrowaveNews*, which showed (under [Media Strategy](#), p.13) that Motorola wrote to its public relations firm telling how to discredit them.

Lai and Singh then got a Wireless Technology Research grant (under the trade group CTIA) to continue their studies. But Lai says WTR continually tried to “dictate the design of our experiments.” After many confrontations, George Carlo, WTR’s head, wrote the University of Washington president (Richard McCormick), threatening legal action and telling him to fire Lai and Singh. McCormick refused. The scientists still had NIH funds to continue their research on extremely low-frequency fields, and published a paper in

2005. But it was their last.

Om Gandhi, a University of Utah professor emeritus, studied how humans absorbed cellphone radiation and, by the 1990s, was focusing on children because, as he explains, “their skulls are thinner than adult skulls and they absorb much more.” He also found that for every millimeter closer to their heads people hold their phones, the absorption rate is 15 to 30 percent higher. When he published these results, his funders stopped funding. “Without the grants, I had to close my lab,” he said. Some years later, Devra Davis, an epidemiologist who co-founded the Environmental Health Trust, co-wrote a paper with Gandhi. She says that a five-year-old child’s skull absorbs about 10 times as much radiation as an adult’s skull. But when companies test phones, they use a one-size-fits-all model based on the head size of an adult male.

Jerry Phillips (before he went to the University of Colorado) was at the Veterans Affairs Medical Center in Loma Linda, California, where the team with which he worked got Motorola funds to study EMF radiation. The researchers exposed rats in the fetus and newborns to the radiation and found that under certain conditions, the signals affected brain tissues. “Motorola didn’t want to hear this and told us not to present our results. But we did, anyway,” Phillips says.

After this, the company asked the team to study the DNA breaks that Lai and Singh had found, but he said, “Motorola wanted us to reach different conclusions. What we learned was that different exposures increased *and* decreased DNA damage. Motorola didn’t like this, either, since it wanted to hear that there were no effects. It told us to do more research and not publish our data. A friend at Motorola advised me ‘give Motorola what it wants, or this could harm your career.’

“Although I knew government funds hadn’t been available for such studies for years, I couldn’t work with Motorola’s restrictions. So I took myself off the project. If I hadn’t, Motorola would have. I left California and haven’t done this

type of research since.”

Phillips says Motorola asked several other researchers to disprove what the group at Loma Linda, as well as Lai and Singh, had found about the damage to cells. And some obliged the company. “It’s possible to do this, since the way you design studies determines what you’ll find.

“This is how industry manages to confuse the public. It stops funding research it doesn’t like and promotes the results it likes. It also says the studies cancel each other out.” That is, if some find harmful biological effects and others don’t, then the former don’t count. “This isn’t correct,” Phillips says.

Lai adds that industry enthusiasts always claim there’s a lack of research about the long-term effects, but this isn’t true: over 500 epidemiological and animal studies have shown that cellphone radiation causes biological damage. Lai told Slesin, “The industry says half the studies don’t show effects. But even if this was true, could the other half all be garbage?”

### **Reseachers’ findings**

*Brain tumors and blood leaks* Several scientists have reported on these health problems. Berkeley’s Joel Moskowitz, who writes a blog on electromagnetic radiation, says that in 2017, several journals, such as *Biomedical Research International* and *Neurological Sciences*, published various scientists’ reviews of the many studies carried out on brain tumors. They found that “each reported a ‘statistically significant’ link between heavy cellphone use (of 10 or more years) and brain tumors, especially on the side of the head where people hold their phones (called *ipsilateral* use).”

One review was by Lennart Hardell and Michael Carlberg, whose earlier work on brain tumors is considered the gold standard and was a key reason the International Agency for Research on Cancer classified cellphone radiation as

a possible carcinogen. In their review, Hardell and Carlberg found that the highest risk of glioma—brain cancer—occurred among the heaviest users, and they reported in a 2013 issue of the *International Journal of Oncology* that people using cellphones at least 30 minutes a day for nine years “had nearly three times the glioma incidence. If they started as teenagers or earlier, the risk was four times higher.” They also found meningiomas (slow-growing, mostly nonmalignant brain tumors) and acoustic neuromas (tumors on auditory nerves leading from the inner ear to the brain).

Further, a \$25 million Interphone Study, funded by the European Union and others, was carried out by scientists in Australia, Canada, Denmark, Finland, France, Germany, Israel, Italy, New Zealand, Japan, Norway, Sweden, and the U.K. They compared approximately 5,000 cases of tumors to a similar-size control group. Many of the researchers said the results were consistent with previous studies that showed increased risks for glioma or acoustic neuroma tumors among the heaviest cellphone users.

Two other studies also found serious risks. The French CERNAT study reported in May 2014 that those using phones 30 minutes a day for five years had a higher risk of brain tumors. And a Chinese study by J. Tang (published in *Brain Research* in 2015) found that rats exposed to cellphone radiation had leakage in the blood-brain barrier and cognitive impairment.

*DNA damage* Besides the Lai and Singh studies, the REFLEX study (for which the European Union gave three million Euros to 12 institutions) found that cellphone radiation damaged human cells and DNA. As noted earlier, the NTP study also found DNA damage in rats and mice.

*Thyroid tumors* Berkeley’s Moskowitz says the incidence of thyroid tumors—especially the papillary type, which is the most sensitive to electromagnetic field radiation—is increasing in many countries. He explains that because of the way phones are designed, much of the radiation is directed toward the neck, where the thyroid gland is located. He says the CDC reported a rapid rise of these tumors among children in the United States, and Hardell and his

colleagues wrote about this in 2016. Finally, he says a 2019 Yale University study found increased thyroid cancer among heavy cellphone users.

*Male infertility* The Cleveland Clinic Center for Male Fertility found that when men carried phones in their pants pockets, their sperm were weakened and reduced, which can cause infertility.

*Hypersensitivity* A growing number of physicians and scientists are reporting that some individuals are particularly sensitive to EMF radiation. Their symptoms, which can be quite pronounced, include tinnitus, vertigo, headaches, fatigue, and memory loss.

### **Insurance companies deny coverage**

Interestingly, the risk-averse insurance industry has been reluctant to offer coverage for the companies or those who use the devices. For example, insurance authority Swiss Re classified wireless devices as “high risk,” while Lloyd’s of London underwriters adopted the “Electromagnetic Fields Exclusion Clause”: this means it will not cover “damages or illnesses caused by continuous long-term non-ionizing radiation exposure through mobile phone use.” As journalists Mark Hertsgaard and Mark Dowie noted, in a July 2018 *Guardian* article, they didn’t find a single insurance company that would sell a policy covering cellphone radiation. “Why would we?” one executive told them . . . pointing to over two dozen lawsuits against wireless companies, demanding \$1.9 billion in damages.

### **Countries’ concerns**

Unlike the United States, some countries have tightened their exposure rules. For example, Belgium banned companies from marketing phones specifically designed for children under seven.

Cyprus banned Wi-Fi in nursery schools and kindergartens and launched an

advertising campaign to educate parents. Also, it removed Wi-Fi from Archbishop Makarios hospital.

France, which has the world's strictest limits, banned wireless devices in daycare centers for children under three, required Wi-Fi to be turned off in elementary schools when not in use, and ordered towns to map the locations of antennas, measure their radiation levels, and give this data to the public. Also, it required that ads state the various models' exposure levels (with fines of up to 75,000 Euros if they don't comply); further, the ads may not show children using phones or people holding the devices next to their heads.

India reduced the cell tower radiation limit to one-tenth of the cap recommended by ICNIRP, and some states and cities ordered companies to remove their towers that were located near hospitals and schools.

Israel banned Wi-Fi in kindergartens, limited it in first and second grades to three hours a week, required companies to list the phones' radiation levels, and banned ads that show children using phones. Haifa's school district required computers to be hard-wired.

In Poland, Krakow's mayor distributed free meters to its citizens to measure their devices' exposure levels and tightened zoning rules, which limit the areas where towers can be located.

And in Switzerland, Geneva is one of several cities and towns that placed a moratorium on 5G.

### **States, cities, and scientists fight back**

Alarmed about the hazards from wireless devices, 254 scientists from 44 countries have urged the United Nations to toughen the exposure guidelines and "educate the public about the health risks." The U.N. has not replied.

With the advent of 5G, warnings are even stronger: By October 2020, 407 scientists and physicians appealed to the European Commission “to halt the roll-out of 5G . . . which will substantially increase exposure to radiofrequency electromagnetic fields.” This has also been ignored.

Many U.S. states, cities, and counties also worry. For example, New Hampshire legislators created a commission of experts to study EMF effects. In their report, which was released this November, the experts recommended 15 actions: among the most important, they asked the FCC to study the environmental impact of the 5G antennas and towers and locate them further from schools and homes.

Representative Patrick Abrami, who heads the commission, invited Frank Clegg, Microsoft Canada’s CEO for 14 years, to meet with them. Clegg told them, “The industry only focuses on getting its products to market but doesn’t deal with health and safety issues. It’s self-policing, so we’re seeing a Wild West scenario regarding the guidelines. I’m not aware of a single study which shows 5G technology is safe.”

How did the ex-CEO of Microsoft Canada do such a turnaround? Clegg says, “After I retired in 2005, I talked to scientists and became convinced the devices can harm you. At this point, my wife and I founded Canadians for Safe Technology to raise people’s awareness about the dangers and tell them how to use the devices safely.”

Louisiana legislators are also concerned. They asked their environmental agency to study the 5G safety issues. The problem, Moskowitz says, is that “there are no health studies” specifically on exposure to 5G.

Richard Blumental, senator from Connecticut, shares their concerns. At a February 2019 Commerce Committee hearing on 5G, he blasted the FCC and FDA for “failing to conduct research into the safety of 5G technology . . . instead, deferring to industry. We’re flying blind here.”

Dozens of cities, including Huntington Beach, California; Seattle; and Montgomery County, Maryland, sued the FCC, which they claim has usurped local control in order to promote 5G. They argued that local governments should be able to stop companies from installing thousands of 5G antennas and require that environmental impact studies be made before the companies move forward. But the FCC issued an order to “remove these regulatory barriers.” And it won.

The Environmental Health Trust also took the FCC to court: “The FCC refused to update U.S. radiation guidelines, ignoring the vast number of studies that found harm from low-level radiation emitted by wireless devices and cell towers,” the EHT’s Scarato explains.

The FCC fought back, insisting its 1996 regulations were still adequate. It also repeated its mantra, that 5G will unleash “a wave of entrepreneurship and economic opportunity . . . helping ensure the U.S. wins the global race to 5G.” However, in 2019, the District of Columbia Circuit Court of Appeals **said** the FCC could not eliminate environmental reviews of 5G small-cell infrastructure.

Oral arguments in the EHT case are scheduled for this coming January, but in the meantime, the FCC and telecom companies are forging ahead: the FCC says it can do this—despite local pushback—because the Telecommunications Act of 1996 gives the FCC the sole power to set radiation exposure limits.

Even before the 5G conflict, U.S. cities challenged the industry. In 2010, a San Francisco law required cellphone vendors to warn users about the devices’ radiation and limit their children’s use. CTIA, the trade group, promptly sued, claiming the law violated the sellers’ free speech rights. To flex its economic muscle, CTIA moved its trade show from San Francisco to San Diego. After a three-year fight, the city lost the case in a federal appeals court and backed off—citing the risk of having to pay the industry’s legal fees.

Five years later, Berkeley passed a more limited law that required vendors to

educate users about the safety issues. CTIA sued again, arguing it “violated the sellers’ first amendment rights.” At first, the Circuit Court sided with Berkeley and some vendors complied. But CTIA appealed the decision, arguing that the Berkeley ordinance “over-warned the consumer.” Also, the FCC weighed in that Berkeley didn’t have the right to inform the public about safety concerns because the FCC gave the public all the data it needed. This time, **Berkeley lost**.

Scarato **notes** that Thomas Johnson Jr., the FCC’s general counsel for the Berkeley case, was previously at the law firm of Gibson, Dunn and Crutcher, which represented the CTIA when it sued Berkeley.

### **How users can limit their exposure**

Since wireless devices are here to stay (5.2 billion people use them globally), scientists and health advocates say the best course is to limit people’s exposure. To this end, California’s Department of Public Health says people should use headsets but remove them when not talking, since they release small amounts of radiation even when not in use. Also, they should text instead of talk; carry phones away from their bodies (in backpacks, briefcases, handbags, and tote bags); keep them away from their heads when streaming; and download movies (instead of streaming).

Alasdair Philips, the U.K. scientist, says that modern cellphones use less power and thus emit less radiation than cordless phones (also called satellite phones). But he stresses they are still hazardous and should only be used in areas where reception is strong. Just as important, Philips says, “You should download material, rather than stream it, since streaming emits more radiation. And you should not use ear buds, since these fit deeply inside the ear.”

Warnings from industry executives such as Frank Clegg (Microsoft Canada’s former CEO) are rare. So, too, are those from governments, since the industry lavishes huge sums on the lawmakers. According to the Center for

Responsive Politics, from 1989 to 2017, the industry gave \$101 million to members of Congress and their PACs. Its favorites were Senator John McCain (R-Ariz.), \$2.5 million; Rep. Ed Markey (D-Mass.), \$1.7 million; Rep. Greg Walden (R-Ore.), \$1.6 million; Rep. Fred Upton (R-Mich.), \$1.6million; and Rep. Steny Hoyer (D-Md.), \$1.4 million. The **three most generous donors** were AT&T (\$19.8 million), Comcast (\$14.9 million), and Verizon (\$11.2 million). Moreover, the National Institute on Money in Politics says industry lobbying groups plowed \$93.7 million into local elections in 2018.

As expected, the largesse continues to be rewarded, and a misinformed public continues its love affair with all things wireless.

*Barbara Koepfel is a Washington D.C.-based investigative reporter who covers social, economic, political, and foreign policy issues.*

## Read On:



**Regulators Steamroll  
Health Concerns as the  
Global Economy  
Embraces 5G**



**The Trump Virus**



**Changes in the  
Electorate Signal Close  
Florida Race**



**Letter From New Orleans**

## Share This Story:

 Facebook

 Twitter

 Email

# SUPPORT

Nonprofit and reader-supported, The Washington Spectator reports from the ground on the excesses of the public and private sectors that distort our politics and undermine democracy.



Donate to the Spectator now to support independent media.

## DONATE

## 4 Comments

**William Bruno** on December 28, 2020 at 8:38 PM

Thanks for this! I just subscribed after seeing this!

**Réza Ganjavi** on December 29, 2020 at 12:51 AM

Thank you! Well done! The scam is exposed very well in this article. Here's a list of additional lies and scams exposed:

<https://emfcrisis.yolasite.com/letters.php>

That page includes communications with FCC and FDA — two agencies with deep rooted corruption about the wireless pollution scam.

**Sara** on December 29, 2020 at 8:09 PM

This is so important. Thanks for publishing. We could use a summary of the key points. It's such a long read, hard to conclude what needs to be done, publicly and privately. Would you advocate a moratorium on 5G?

**Morris davidson** on December 30, 2020 at 3:11 AM

Fantastic article. Covered all the bases. A couple of things to add: The original statement by Otis Brawley from the American Cancer Society was this: ACS Responds to New Study Linking Cell Phone Radiation to Cancer [cancer.org/all-.org/NTP2016](http://cancer.org/all-.org/NTP2016)? The U.S. National Toxicology Program (NTP) has released partial results (<http://biorxiv.org/content/biorxiv/early/2016/05/26/055699.full.pdf>) from an animal study of the effect of radiofrequency radiation associated with cell phones. The group found radiofrequency radiation was linked to a higher risk of two cancers. Below is a response from Otis W. Brawley, M.D., American Cancer Society Chief Medical Officer: "For years, the understanding of the potential risk of radiation from cell phones has been hampered by a lack of good science. This report from the National Toxicology Program (NTP) is good science. "The NTP report linking radiofrequency radiation (RFR) to two types of cancer marks a paradigm shift in our understanding of radiation and cancer risk. The findings are unexpected; we wouldn't reasonably expect non-ionizing radiation to cause these tumors. This is a striking example of why serious study is so important in evaluating cancer risk. It's interesting to note that early studies on the link between lung cancer and smoking had similar resistance, since theoretical arguments at the time suggested that there could not be a link." The new report covers only partial findings from the study, but importantly one of the two cancers linked to cell phone radiation was malignant gliomas in the brain. The association with gliomas and acoustic neuromas had been suspected from human epidemiology studies. The second cancer, called schwannoma, is an extremely rare tumor in humans and animals, reducing the possibility that this is a chance finding. And importantly, the study found a 'dose/response' effect: the higher the dose, the larger the effect, a key sign that this association may be real. Second: Kane's book "Cellular Telephone Russian Roulette" was so explosive, industry bought all copies that were released. Third: when George Carlo published the results of the first Motorola studies, Ted Wheeler had his house burned down.

# Submit a Comment

Your email address will not be published. Required fields are marked \*

Comment

NAME \*

EMAIL \*

WEBSITE

SUBMIT COMMENT



Since 1971, the non-profit,

reader-supported *Washington Spectator* has offered independent-minded readers behind-the-scenes insight into significant news stories that are under-reported or ignored by the corporate media.

## WS

### Contributors

Dudley Althaus  
Cyrus Cassells  
Autumn Hayes  
Karen Houppert  
Barbara Koeppel  
Aryeh Neier  
Anne Nelson  
Pamela Newkirk  
Steven Pressman  
Katherine Stewart

## Newsletter

About TWS  
Letters  
Syndication  
Subscribe  
Donate  
Contact Us

We encourage you to **subscribe** either to our digital or print edition, or both, and invite you to consider making a **donation** to *The Spectator* in support of our independent journalism.

© 2021 Washington Spectator. All rights reserved.

## Bridget Cornell

---

**From:** Phillip McGraw <phillip.mcgraw@medmail.ch>  
**Sent:** Thursday, April 7, 2022 12:55 PM  
**To:** Joanne Marchetta; John Marshall; Marja Ambler; Katherine Hangeland  
**Subject:** TRPA Hearings Officer Meeting ( Item No V.B — Verizon/Tahoe Seasons New Telecommunications Facility ) April 7 2022  
**Attachments:** c8618d1b.jpeg; Limiting liability with positioning to minimize negative health effects of cellular phone towers.pdf; In re Flint Water Cases, 2020 WL 6218787 (2020).pdf; In re Flint Water Cases, 969 F.3d 298 (2020).pdf; In re Flint Water Cases, 960 F.3d 820 (2020).pdf; In re Flint Water Cases, 960 F.3d 303 (2020).pdf; In re Flint Water Cases, 384 F.Supp.3d 802 (2019).pdf; Cell Towers—Tahoe Mountain News.pdf; Ex-Governor of Michigan Charged With Neglect in Flint Water Crisis - The New York Times.pdf; Brown v Los Angeles Unified School District, 60 Cal.App.5th 1092 (2021).pdf; Environmental Health Trust v Federal Communications Commission, 2021 WL 3573769 (2021).pdf; City of Portland v United States, 969 F.3d 1020 (2020).pdf; T-Mobile West LLC v City and County of San Francisco, 6 Cal.5th 1107 (2019).pdf

Dear TRPA Hearings Officer,

I have grave concern that the City, County, and Tahoe Regional Planning Agency (TRPA) are not enacting science-based [health](#) and environmental policies. The El Dorado County Public Health officer, Dr. Nancy Williams, has caved-into lowly pressures to base our County health policy upon special economic interests (tourism), rather than [science-based public policy](#). The TRPA has done the same on the environmental front. In your leadership vacuum, Cities in the basin have followed suit.

She did the same during the county's salient December 2020 COVID-19 spike which occurred in concert with a **national crescendo** of cases, and dissembled the economic motives in painting the epidemic as a "data aberration" which was patently false. She unconscionably [petitioned the state's adjudication process to dangerously keep our county in the lower tiers](#), despite the numbers having conspicuously crossed the upper lines. The [Governor shut her down](#).

This is part of a continuous pattern of behavior coming out of her office. She spewed out the same economically motivated health "uncertainty" disinformation about hard data proving the somber health costs of radiation from [cell towers](#). Worse-off, she is not qualified as an economist; it is way cheaper to stay out of trouble than to get out of trouble. Treating [cancer](#) and lost productivity are expensive.

Therefore, it especially falls upon the TRPA and our City to keep us safe. Your rash decisions are [killing us with residential cell tower radiation](#). Literally. As a recent [public comment](#) has pointed out, the interim [DNA damage](#) is clear and measurable.

Applying the peer-reviewed [per capita rate](#) to our City's ~20K population, you get the concrete effects:



During a typical City Council's four-year term, there will be 8-12 new empty seats at dining room tables; that is 8-12 families and their friends heartbroken from the aftermath of cell phone radiation. This is coming from data merely pertaining to 3G & 4G networks, when towers were only on mountain tops and not in neighborhoods. While 3G & 4G are clearly not safe, it is about to get much, much, worse.

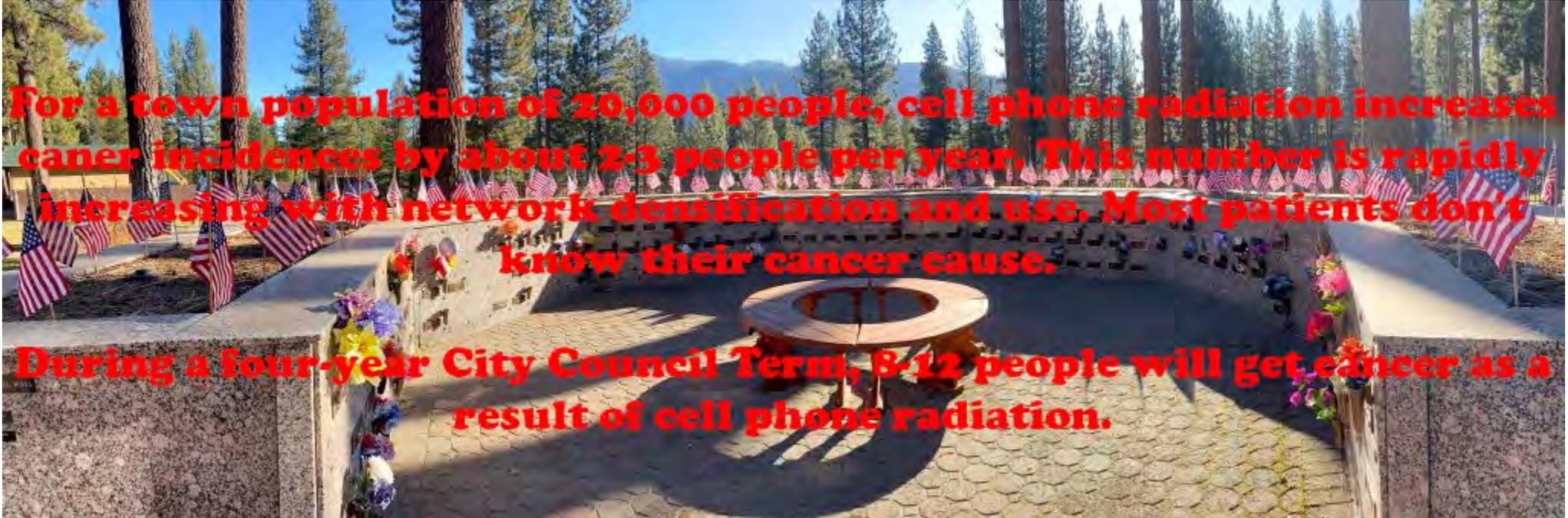
If you multiply the [per capita rate](#) by our life expectancy, you get a rough approximation of the lifetime risk of getting cancer from cell radiation. The results are horrifying. The number is on par with the rate at which women are already getting breast cancer. And *this* cause is 100% preventable.

You are poisoning us by approving Cell towers in close proximity to residences. You really need to stop and reflect on what you are doing. The ends do not justify the means, and the means apparently are an unconstitutional invasion of our [right to bodily integrity](#). Make no mistake, horrible and impermissible deaths will stem from decisions you have already made pertaining to the location and siting of Wireless Telecommunication Facilities (WTFs). This may even result in your being charged criminally and/or [civilly](#) with willful neglect of duty, and violation of our Constitutional right to bodily integrity "under the color of law" ([18 U.S.C. § 242](#); [42 U.S.C. § 1983](#)).

As the instant COVID-19 lesson dictates, **if you procrastinate addressing the problem to the point that we feel real pain, then you are a bad leader.**

Thank you for considering,

Phillip McGraw



**For a town population of 20,000 people, cell phone radiation increases cancer incidences by about 2-3 people per year. This number is rapidly increasing with network densification and use. Most patients don't know their cancer cause.**

**During a four-year City Council Term, 8-12 people will get cancer as a result of cell phone radiation.**

Contents lists available at [ScienceDirect](https://www.sciencedirect.com)

Environmental Research

journal homepage: [www.elsevier.com/locate/envres](http://www.elsevier.com/locate/envres)

## Limiting liability with positioning to minimize negative health effects of cellular phone towers

J.M. Pearce<sup>a,b,c,\*</sup><sup>a</sup> Department of Electronics and Nanoengineering, School of Electrical Engineering, Aalto University, Espoo, Finland<sup>b</sup> Department of Electrical & Computer Engineering, Michigan Technological University, USA<sup>c</sup> Department of Materials Science & Engineering, Michigan Technological University, USA

## ARTICLE INFO

## Keywords:

Radiofrequency radiation (RFR)  
 Antenna arrays  
 Cellular phone base stations  
 Microwave sickness  
 Nonionizing electromagnetic fields  
 Environmental pollution  
 Cancer  
 RFR health effects

## ABSTRACT

The use of cellular phones is now ubiquitous through most of the adult global population and is increasingly common among even young children in many countries (e.g. Finland, where the market for smart phones is nearly saturated). The basic operation of cellular phone networks demands widespread human exposure to radio-frequency radiation (RFR) with cellular phone base stations providing cellular coverage in most areas. As the data needs of the population increase from the major shift in the source of Internet use from personal computers to smart phones, this coverage is widely predicted to increase. Thus, both the density of base stations and their power output is expected to increase the global human RFR exposure. Although direct causation of negative human health effects from RFR from cellular phone base stations has not been finalized, there is already enough medical and scientific evidence to warrant long-term liability concerns for companies deploying cellular phone towers. In order to protect cell phone tower firms from the ramifications of the failed paths of other industries that have caused unintended human harm (e.g. tobacco) this Current Issue summarizes the peer-reviewed literature on the effects of RFR from cellular phone base stations. Specifically the impacts of siting base stations are closely examined and recommendations are made for companies that deploy them to minimize their potential future liability.

### 1. Negative human health effects from proximity to cellular phone base stations

There is a large and growing body of evidence that human exposure to RFR from cellular phone base stations causes negative health effects (Siddoo-Atwal, 2018; Singh et al., 2018; Faisal, et al., 2018) including both i) neuropsychiatric complaints such as headache, concentration difficulties, memory changes, dizziness, tremors, depressive symptoms, fatigue and sleep disturbance (Navarro et al., 2003; Hutter et al., 2006; Abdel-Rassoul et al., 2007); and ii) increased incidence of cancer and living in proximity to a cell-phone transmitter station (Wolf and Wolf, 2004; Havas, 2017). The mechanism for causing cancer could be from observed genetic damage using the single cell gel electrophoresis assay assessed in peripheral blood leukocytes of individuals residing in the vicinity of a mobile phone base station and comparing it to that in healthy controls (Gandhi et al., 2014). In epidemiological studies that assessed negative health effects of mobile phone base stations (seven studies explored the association between base station proximity and neurobehavioral effects (Navarro et al., 2003; Hutter et al., 2006;

Abdel-Rassoul et al., 2007; Berg-Beckhoff et al., 2009; Blettner et al., 2009; Gadzicka et al., 2006; Santini et al., 2002) and three investigated cancer (Wolf and Wolf, 2004; Havas, 2017; Levitt and Lai, 2010), 80% reported increased prevalence of adverse neurobehavioral symptoms or cancer in populations living at distances < 500 m from base stations (Navarro et al., 2003).

The literature also indicates that these effects may be cumulative based on i) mice exposed to low-intensity RFR became less reproductive and after five generations of exposure the mice were not able to produce offspring indicating intergenerational transfer of effects (Magras and Xenos, 1997); ii) DNA damage in cells after 24 h exposure to low-intensity RFR, which can lead to gene mutation that accumulates over time (Phillips et al., 1998) and iii) increased sensitivity to behavior-disruption experiments in rats (D'Andrea et al., 1986) and monkeys (de Lorge, 1984), iv) an increase in permeability of the blood-brain barrier in mice suggesting that a short-term, high-intensity exposure can produce the same effect as a long-term, low-intensity exposure (Persson et al., 1997). Studies on short-term exposure generally show no effects. For example, early studies saw no effect from

\* 601 M&M Building, 1400 Townsend Drive, Houghton, MI 49931, USA.

E-mail address: [pearce@mtu.edu](mailto:pearce@mtu.edu).

<https://doi.org/10.1016/j.envres.2019.108845>

Received 26 December 2017; Received in revised form 3 June 2019; Accepted 19 October 2019

0013-9351/ © 2019 Elsevier Inc. All rights reserved.

short-term exposure, however, studies found effects after prolonged, repeated exposure in guinea pigs and rabbits (Takashima et al., 1979).

There are several studies showing the effect intensifies with reduced distance to the cell tower. The first (Santini et al., 2002) found increased symptoms and complaints the closer a person lived to a tower (Santini et al., 2002) and similar results were found in later studies (Navarro et al., 2003; Hutter et al., 2006; Abdel-Rassoul et al., 2007).

## 2. U.S. law unhelpful for preventing future liability

Current U.S. law has created a somewhat peculiar overriding federal preemption that precludes taking the “environmental effects” of RFR into consideration in cell tower siting (see Section 704 of The Telecommunications Act of 1996). The current, U.S. standards are based solely on thermal effects (which do not appear to be a problem) and thus do not mitigate against non-thermal effects (for which there is a growing litany of concern in the medical/scientific community). Due to the findings of many studies briefly summarized above many researchers argue for the revision of standard guidelines for public exposure to RER from mobile phone base station antennas (Abdel-Rassoul et al., 2007; Hardell and Sage, 2008; Khurana et al., 2010). As Roda and Perry summarize (Roda and Perry, 2014), “... because scientific knowledge is incomplete, a precautionary approach is better suited to State obligations under international human rights law.” This is perhaps most forcefully concluded by the *BioInitiative Report* published by the BioInitiative Working Group, which is based on an international research and public policy initiative to give an overview of what is known of biological effects that occur at low-intensity electromagnetic fields exposure. This precautionary approach is gaining favor in Europe, but is less common in the U.S. American companies are therefore ill advised to simply follow “regulatory compliance” on this front, as there appears to be a clear cause for concern in the scientific/medical communities. If causation were to be proven through detailed studies, cellular phone companies would potentially be in position of future legal exposure for causing widespread human health problems and premature death. It is, therefore, in American companies’ best interest to act before government and regulation catches up with the science.

## 3. Current cell tower positioning

Current cell tower locations are chosen based on a “search ring” priority basis of geographic optimum for technical coverage of high concentration of wireless transmissions (e.g. users). This combination of technical parameters (e.g. geography) to enable coverage and dependable service and costs (e.g. positioning on mountaintops on accessibly by helicopter) is then weighed against and local regulations such as local zoning.

To overcome these challenges in urban areas cellphone companies often locate cellphone base stations at schools, because the monthly rental fee (~\$1500) is welcome income for economically-challenged school districts that have influence on local zoning. However, some jurisdictions have already prohibited the placement of cell phone towers near schools or hospitals because of the increased sensitivity of these populations, as in India. Other regions such as Europe (Roda and Perry, 2014) could follow a similar approach. Now even in North America, Canada’s Standing Committee on Health are considering more precautionary approaches to RFR.

## 4. Precautionary cell phone base station positioning

A review article of the health effects near base stations concluded that deployment of base stations should be kept as efficient as possible to minimize exposure of the public to RFR and should not be located less than 500 m from the population, and at a height of 50 m (Levitt and Lai, 2010). This potentially presents a serious challenge to cell phone company RF engineers. However, it is possible to obtain necessary

coverage while at the same time minimizing human exposure at the highest intensities. There are several first steps a cellular phone company can take to minimize human exposure particularly of the most vulnerable populations.

First, voluntarily restrictions can be made on the placement of cellular phone base stations within 500 m of schools and hospitals. This will synchronize base station deployment strategies between regions. This can be done by utilizing the existing hexagon planning map structure of an area with an overlay using an additional semi-automated process with a geographic information system (GIS) (Al-Sahly et al., 2018) such as the Geographic Resources Analysis Support System (GRASS) to identify any regions within 500 m of existing schools and hospitals. All hexagons with schools or hospitals are marked as unusable for RF engineer planning (e.g. colored red). This restriction only makes planning slightly more difficult, but does present a challenge in regions where schools were specifically targeted as base station locations in (e.g. Verizon deployments in the U.S.). Future work is needed to determine if the increased legal exposure warrants the cost of moving existing stations. However, the increased cost to locate future stations away from schools and hospitals should be minimal.

The second technical hurdle is more challenging. Ideally, all cell phone users would have coverage while minimizing the population density near cellular phone base stations (thus minimizing health impacts). This can be planned using GIS tools, freely-accessible U.S. Census data, parcel data and/or satellite images. The population density can be color coded for straightforward decision making for RF engineers. As a cellphone base station costs \$250–350,000 to install in the U.S., using a precautionary approach to potential future regulation can save substantial relocation fees.

The cell phone industry should also consider cell splitting, small cell deployment, beam and null steering antennae as possible technical means for reducing RF exposure. Moreover, more research on cognitive radio should also be conducted, so that the overall RF exposure is reduced. These measures will ultimately benefit the entire telecommunications industry, while potentially significantly reducing global RF pollution.

Finally, exposed companies should consider funding large-scale epidemiological studies with personal dosimeters for strict dose measurement and straight-forward tissue exposure. By quantifying the human medical threat themselves, more appropriate long-term planning can be made to minimize the risk of liability from unintended human harm due to cellular phone base station siting.

## Financial disclosure

The author owns stock in the American Tower Corporation.

## Declaration of competing interest

The author has no conflict of interest.

## References

- Abdel-Rassoul, G., El-Fateh, O.A., Salem, M.A., Michael, A., Farahat, F., El-Batanouny, M., Salem, E., 2007. Neurobehavioral effects among inhabitants around mobile phone base stations. *Neurotoxicology* (Little Rock) 28 (2), 434–440.
- AprilAl-Sahly, A., Hassan, M.M., Al-Rubaian, M., Al-Qurishi, M., 2018. Using GIS for measuring mobile tower radiation on human. In: 2018 1st International Conference on Computer Applications & Information Security (ICCAIS). IEEE, pp. 1–6.
- Berg-Beckhoff, G., Blettner, M., Kowall, B., Breckenkamp, J., Schlehofer, B., Schmiedel, S., et al., 2009. Mobile phone base stations and adverse health effects: phase 2 of a cross-sectional study with measured radio frequency electromagnetic fields. *Occup. Environ. Med.* 66 (2), 124–130.
- Blettner, Maria, Schlehofer, Brigitte, Breckenkamp, Juergen, Kowall, Bernd, Schmiedel, Sven, Reis, Ursula, Potthoff, Peter, Schuez, Joachim, Berg-Beckhoff, Gabriele, 2009. Mobile phone base stations and adverse health effects: phase 1 of a population-based, cross-sectional study in Germany. *Occup. Environ. Med.* 66 (2), 118–123.
- de Lorge, J.O., 1984. Operant behavior and colonic temperature of *Macaca mulatta* exposed to radiofrequency fields at and above resonant frequencies.

- Bioelectromagnetics 5 (2), 233–246. <https://doi.org/10.1002/bem.2250050211>.
- D'Andrea, J.A., DeWitt, J.R., Gandhi, O.P., Stensaas, S., Lords, J.L., Nielson, H.C., 1986. Behavioral and physiological effects of chronic 2450 MHz microwave irradiation of the rat at 0.5 mW/cm<sup>2</sup>. *Bioelectromagnetics* 7 (1), 45–56. <https://doi.org/10.1002/bem.2250070106>.
- OctoberFaisal, M.M.A., Mortuza, M.G., Alam, T., 2018. Cell tower radiation and effect on human body: Bangladesh perspective. In: 2018 International Conference on Innovations in Science, Engineering and Technology (ICISSET). IEEE, pp. 423–426.
- Gadzicka, E., Bortkiewicz, A., Zmyslony, M., Szymczak, W., Szykowska, A., 2006. Assessment of subjective complaints reported by people living near mobile phone base stations. *Biuletyn PTZE Warszawa* 14, 23–26.
- Gandhi, G., Kaur, G., Nisar, U., 2014. A cross-sectional case control study on genetic damage in individuals residing in the vicinity of a mobile phone base station. *Electromagn. Biol. Med.* (0), 1–11.
- Hardell, L., Sage, C., 2008. Biological effects from electromagnetic field exposure and public exposure standards. *Biomed. Pharmacother.* 62 (2), 104–109.
- Havas, M., 2017. Carcinogenic effects of non-ionizing radiation: a paradigm shift. *Clin Oncol* 2, 1278–1279.
- Hutter, H.P., Moshhammer, H., Wallner, P., Kundi, M., 2006. Subjective symptoms, sleeping problems, and cognitive performance in subjects living near mobile phone base stations. *Occup. Environ. Med.* 63 (5), 307–313.
- Khurana, V.G., Hardell, L., Everaert, J., Bortkiewicz, A., Carlberg, M., Ahonen, M., 2010. Epidemiological evidence for a health risk from mobile phone base stations. *Int. J. Occup. Environ. Health* 16 (3), 263–267.
- Levitt, B.B., Lai, H., 2010. Biological effects from exposure to electromagnetic radiation emitted by cell tower base stations and other antenna arrays. *Environ. Rev.* 18, 369–395.
- Magras, I.N., Xenos, T.D., 1997. RF radiation-induced changes in the prenatal development of mice. *Bioelectromagnetics* 18 (6), 455–461. [https://doi.org/10.1002/\(SICI\)1521-186X\(1997\)18:6<455::AID-BEM8>3.0.CO;2-1](https://doi.org/10.1002/(SICI)1521-186X(1997)18:6<455::AID-BEM8>3.0.CO;2-1).
- Navarro, A.E., Sequera, J., Portoleás, M., Gómez-Perretta de Mateo, C., 2003. The microwave syndrome: a preliminary study in Spain. *Electromagn. Biol. Med.* 22 (2–3), 161–169. <https://doi.org/10.1081/JBC-120024625>.
- Persson, B.R.R., Salford, L.G., Brun, A., 1997. Blood–brain barrier permeability in rats exposed to electromagnetic fields used in wireless communication. *Wirel. Netw.* 3 (6), 455–461. <https://doi.org/10.1023/A:1019150510840>.
- Phillips, J.L., Ivaschuk, O., Ishida-Jones, T., Jones, R.A., Campbell-Beachler, M., Haggren, W., 1998. DNA damage in Molt-4 T-lymphoblastoid cells exposed to cellular telephone radiofrequency fields in vitro. *Bioelectrochem. Bioenerg.* 45 (1), 103–110. [https://doi.org/10.1016/S0302-4598\(98\)00074-9](https://doi.org/10.1016/S0302-4598(98)00074-9).
- Roda, C., Perry, S., 2014. Mobile phone infrastructure regulation in Europe: scientific challenges and human rights protection. *Environ. Sci. Policy* 37, 204–214.
- Santini, R., Santini, P., Danze, J.M., Le Ruz, P., Seigne, M., 2002. Enquête sur la sante' de riverains de stations relais de télé-phonie mobile : incidences de la distance et du sexe. *Pathol. Biol.* 50, 369–373. [https://doi.org/10.1016/S0369-8114\(02\)00311-5](https://doi.org/10.1016/S0369-8114(02)00311-5).
- Siddoo-Atwal, C., 2018. Electromagnetic radiation from cellphone towers: a potential health hazard for birds, bees, and humans. *Curr. Underst. Apoptosis: Program. Cell Death* 137.
- Singh, R., Nath, R., Mathur, A.K., Sharma, R.S., 2018. Effect of radiofrequency radiation on reproductive health. *Indian J. Med. Res.* 148 (Suppl 1), S92.
- Takashima, S., Onaral, B., Schwan, H.P., 1979. Effects of modulated RF energy on the EEG of mammalian brain. *Radiat. Environ. Biophys.* 16 (1), 15–27. <https://doi.org/10.1007/BF01326893>.
- Wolf, R., Wolf, D., 2004. Increased incidence of cancer near a cell-phone transmitter station. *Int. J. Cancer Prev.* 1 (2), 123–128.

2020 WL 6218787

Only the Westlaw citation is currently available.

United States District Court, E.D.  
Michigan, Southern Division.

IN RE FLINT WATER CASES.

This Order Relates to:

Bacon v. Snyder, et al.

Case No. 18-10348

|  
Signed 10/22/2020

**Attorneys and Law Firms**

[James F. Graves](#), Sinas, Dramis, Brake, Boughton and McIntyre, P.C., Lansing, MI, for Deborah Sapolin.

[Philip A. Erickson](#), Plunkett & Cooney, East Lansing, MI, for Lockwood Andrews & Newnam, PC, Lockwood Andrews & Newnam, Inc., Leo A. Daly Company.

[Craig S. Thompson](#), [Jennifer R. Moran](#), Sullivan, Ward, Patton, Gleeson & Felty, P.C., Southfield, MI, for Rowe Professional Services Company.

[James A. Fajen](#), Ann Arbor, MI, [James W. Burdick](#), Burdick Law, P.C., Bloomfield Hills, MI, for Adam Rosenthal.

[Krista A. Jackson](#), [Philip A. Grashoff, Jr.](#), Smith Haughey Rice & Roegge, Grand Rapids, MI, for Stephen Busch.

[Allison M. Collins](#), [Charles E. Barbieri](#), Foster, Swift, Collins & Smith, P.C., Lansing, MI, for Patrick Cook, Michael Prysby.

[Alexander S. Rusek](#), White Law PLLC, Okemos, MI, for Howard Croft.

[Christopher James Marker](#), O'Neill Wallace & Doyle, Saginaw, MI, [Brett T. Meyer](#), Plunkett Cooney, Grand Rapids, MI, for Michael Glasgow.

[Edwar A. Zeineh](#), Law Office of Edwar A. Zeineh, PLLC, Lansing, MI, for Daugherty Johnson.

[Margaret A. Bettenhausen](#), [Nathan A. Gambill](#), [Zachary C. Larsen](#), Michigan Department of Attorney General, [Richard S. Kuhl](#), Assistant Attorney General, Lansing, MI, for Andy Dillon, State of Michigan, [Richard D. Snyder](#).

[Frederick A. Berg](#), Butzel Long, Detroit, MI, [Sheldon H. Klein](#), Butzel Long, Bloomfield Hills, MI, [William Young Kim](#), City of Flint, Flint, MI, for City of Flint.

T. Santino Mateo, Law Offices of T. Santino Mateo, Detroit, MI, for Darnell Earley.

[Barry A. Wolf](#), [Barry A. Wolf](#), Attorney at Law, PLLC, Flint, MI, for Gerald Ambrose.

**OPINION AND ORDER GRANTING IN PART AND DENYING IN PART DEFENDANTS' MOTIONS TO DISMISS PLAINTIFF'S SHORT-FORM COMPLAINT [89, 90, 91, 93]**

[JUDITH E. LEVY](#), United States District Judge

\*1 This is one of the many cases that are collectively referred to as the Flint Water Cases. Plaintiffs allege that Defendants, a combination of private and public individuals and entities, set in motion a chain of events that led to bacteria and lead leaching into the City of Flint's drinking water. Plaintiffs in the various Flint Water Cases claim that Defendants subsequently concealed, ignored, or downplayed the risks that arose from their conduct, causing them serious harm. These plaintiffs contend that the impact of what has since been called the Flint Water Crisis is still with them and continues to cause them problems.

The Plaintiff in this particular case is Deborah Sapolin, personal representative of the Estate of Margaret A. Bacon.<sup>1</sup> In previous Flint Water decisions, the Court has set forth descriptions of each Defendant in these cases, and adopts those descriptions as if fully set forth here. *See In re Flint Water Cases*, 384 F. Supp. 3d 802, 824–825 (E.D. Mich. 2019).

Before the Court are four motions to dismiss. On June 16, 2020, Defendants Lockwood, Andrews & Newnam, Inc. and Lockwood, Andrews & Newnam, P.C. (together, “LAN”) moved to dismiss Plaintiff's complaint. (ECF No. 89.) Defendant Leo A. Daly Company (“LAD”) also moved to dismiss on the same day. (ECF No. 90.) On June 17, 2020, the Michigan Department of Environmental Quality (“MDEQ”) individual Defendants Stephen Busch, Patrick Cook, and Michael Prysby (collectively, “MDEQ Defendants”) moved to dismiss.<sup>2</sup> (ECF No. 91.) And finally, on the same day, Defendants the City of Flint, Darnell Earley, Gerald

Ambrose, Dayne Walling, Howard Croft, Michael Glasgow, and Daugherty Johnson (collectively “City Defendants”) moved to dismiss. (ECF No. 93.) For the reasons set forth below, the Court grants in part and denies in part Defendants’ motions to dismiss the complaint.

### I. Prior Precedent in the Flint Water Cases

This Court has previously adjudicated other motions to dismiss in the Flint Water Cases. First, there was *Guertin v. Michigan*, No. 16-12412, involving individual plaintiffs and many of the same claims and Defendants in the present case. Next, there was *Carthan v. Snyder*, No. 16-10444, a consolidated class action that also involved similar Defendants and claims. Also, there were *Walters v. City of Flint*, No. 17-10164, and *Sirls v. Michigan*, No. 17-10342, which involved individual plaintiffs and the same Master Complaint as the present case.

\*2 Most recently, there were *Brown v. Snyder*, No. 18-10726, and *Marble v. Snyder*, No. 17-12942, which not only involved individual plaintiffs and similar claims, facts, Defendants, and the same Master Complaint as the present case, but also involved *legionella* bacteria, which is the focus of this case.

The Flint Water Cases have already produced several Sixth Circuit opinions. These are binding on this Court and include *Carthan v. Earley*, 960 F.3d 303 (6th Cir. 2020); *Walters v. Flint*, No. 17-10164, 2019 WL 3530874 (6th Cir. August 2, 2019); *Guertin v. Michigan*, 912 F.3d 907 (6th Cir. 2019); *Boler v. Earley*, 865 F.3d 391 (6th Cir. 2017); and *Mays v. City of Flint*, 871 F.3d 437 (6th Cir. 2017).

The Court will also adhere to its own prior decisions where appropriate, including *Guertin v. Michigan*, No. 16-12412, 2017 WL 2418007 (E.D. Mich. June 5, 2017); *Carthan v. Snyder*, 329 F. Supp. 3d 369 (E.D. Mich. 2018); *Carthan v. Snyder*, 384 F. Supp. 3d 802 (E.D. Mich. 2019); and *Walters v. City of Flint*, No. 17-10164, 2019 WL 3530874 (E.D. Mich. Aug. 2, 2019). In particular, it will rely on *Marble v. Snyder*, 453 F. Supp. 3d 970 (E.D. Mich. 2020) and *Brown v. Snyder*, No. 18-10726, 2020 WL 1503256 (E.D. Mich. Mar. 27, 2020) to resolve the current motions where appropriate. This opinion will describe Plaintiff’s legal claims and then explain why a similar or different result is justified based on the factual allegations pleaded here.

### II. Procedural History and Background

#### A. The Master Complaint

As the number of Flint Water Cases increased over the years, the Court entered case management orders to manage the litigation. For example, in early 2018, it appointed and then directed co-liaison lead counsel for the individual plaintiffs to file a Master Complaint that would apply to all pending and future non-class action cases. (*Carthan*, No. 16-10444, ECF No. 347.) The Master Complaint was filed in *Walters*. (*Walters*, No. 17-10164, ECF no.185-2.) The attorneys in each of the individual cases were also ordered to file a Short Form Complaint, adopting only the pertinent allegations from the Master Complaint as they saw fit. The Short Form Complaints also allowed for an Addendum if any Plaintiff wished to allege a new cause of action or include additional Defendants. This would allow the Court to issue opinions that would apply to multiple individuals, rather than to address each case in turn and cause a delay in the administration of justice. This is the procedure that Plaintiff was required to follow in this case.

#### B. Plaintiff’s Operative Short-Form Complaint Filed June 1, 2020

Plaintiff’s operative Short Form Complaint was filed on June 1, 2020 (the “June 2020 Short Form Complaint”). (ECF No. 86.) In it, she fully adopts the relevant facts alleged in the Master Complaint from *Walters*. (*Walters*, No. 17-cv-10164, ECF No. 185-2.) The Master Complaint’s facts, setting forth the background of the Flint Water Crisis, were summarized in this Court’s opinion in *Walters* and will not be reproduced here. *Walters v. City of Flint*, No. 17-cv-10164, 2019 WL 3530874, at \*4–\*11 (E.D. Mich. Aug. 2, 2019). However, as set forth above, unlike *Walters*, Plaintiff does not allege injuries from lead poisoning. Rather, she alleges injuries from Bacon’s exposure to *legionella*.

\*3 Plaintiff’s June 2020 Short Form Complaint involves the following claims against the following Defendants. First, she checked boxes on the short form for the following Defendants.<sup>3</sup>

- Governor Richard D. Snyder<sup>4</sup>
- The City of Flint
- Howard Croft

- Michael Glasgow
- Daugherty Johnson
- Stephen Busch
- Patrick Cook
- Michael Prysby
- Adam Rosenthal
- Andy Dillon
- Lockwood, Andrews & Newnam P.C.
- Lockwood Andres & Newnam, Inc.
- Leo A. Daly Company
- Rowe Professional Services Company and Rowe Engineering (together, “Rowe”)  
(ECF No. 86.)

Next, she checked the boxes on the short form complaint for the following claims:

- Count I: [42 U.S.C. § 1983](#)–14th Amendment, Substantive Due process-State Created Danger
- Count II: [42 U.S.C. § 1983](#)–14th Amendment, Substantive Due Process–Bodily Integrity
- Count IV: [42 U.S.C. § 1983](#) – 5th and 14th Amendments, Equal Protection of the Law–Wealth Based
- Count VIII: Punitive damages
- Count IX: Professional Negligence (LAN PC, LAN Inc. and LAD)
- Count X: Professional Negligence (Rowe)<sup>5</sup>
- Count XIII: Survival and Wrongful Death, [MCL 600.2922](#) (All Defendants)  
(ECF No. 86, PageID.1189–90.)

### C. Plaintiff’s Previous Complaints, Claims, and Defendants

Before analyzing Defendants’ motions to dismiss, it is helpful to set forth some of the background of Plaintiff’s case. Before

she died, Bacon initially filed her lawsuit in the State of Michigan, Genesee County Circuit Court. She amended her complaint on April 26, 2016 (the “April 2016 Complaint”). The Defendants in that case removed it to this Court. (*Bacon v. Rowe et al.*, No. 16-11579, (E.D. Mich. May 3, 2016) (O’Meara, J.)) The following month, in May 2016, Bacon voluntarily dismissed the individual Defendants in that case. (*Id.* at ECF No. 32.) The remaining parties stipulated to remand the case back to the Genesee County Circuit Court, and they stipulated to permit Bacon to file a second amended complaint. (*Id.* at ECF Nos. 34, 35.)

Now back in the Genesee County Circuit Court, Bacon progressed with her second amendment to the complaint, which was titled the First Amended Short Form Complaint, pursuant to the Master Individual Complaint adopted by the Genesee County Circuit Court. On November 9, 2017, she filed her First Amended Short Form (the “November 2017 Complaint”). *Bacon v. Lockwood, Andrews & Newman, P.C. et al.*, No. 17-106692, Consol. Docket No. 17-108646 (Mich. Genesee Cir. Ct. Nov. 9, 2017) (ECF No. 1-1, PageID.47–57; ECF No. 1-3, PageID.59–146.) On January 30, 2018, Defendants jointly removed Bacon’s action to this Court. (ECF No. 1.)

\*4 As set forth above, on March 26, 2018, after this Court’s consolidation and case management orders were entered, Bacon adopted the Master Complaint from *Walters* in full and filed a Short Form Complaint with new allegations and new Defendants (the “March 2018 Short Form Complaint”). (ECF No. 14.)

On April 10, 2018, Bacon unfortunately passed away. (ECF No. 26, 27.) The Court granted a substitution of parties, replacing Bacon with Plaintiff. (ECF No. 31.) Plaintiff and several Defendants then stipulated to dismissal of certain Defendants and certain claims. (ECF Nos. 82, 83.)

On June 1, 2020, Plaintiff filed the operative June 2020 Short Form Complaint.<sup>6</sup> (ECF No. 86.) This complaint differed from her previous complaints in many regards, not only reflecting a new post-death cause of action for wrongful death, but also reflecting several other changes in Defendants and claims.

For example, the June 2020 Short Form Complaint omits some of the claims Bacon previously brought in this case before her death, including: gross negligence, negligent nuisance in fact, public nuisance, intentional nuisance in fact,

intentional infliction of emotional distress, grossly negligent infliction of emotional distress, assault and battery, breach of contract, breach of implied warranty, trespass, unjust enrichment, and a CERCLA violation.

Further, the operative complaint omits Bacon's previous claims against Defendants Daniel Wyant, Laine Shekter Smith, Nick Lyon, State of Michigan, Jeff Wright, Edward Kurtz, Dayne Walling, Veolia LLC, Veolia, Inc., and others. Notably, the June 2020 Short Form Complaint also includes new claims Bacon never brought before: a 41 U.S.C. § 1983 claim based on wealth, a claim for punitive damages, and a state law claim for survival and wrongful death. Finally, Plaintiff did not check the operative complaint's checkbox for "Property Damage," as Bacon had in past iterations of her complaint. (ECF No. 86 PageID.1189.) Nevertheless, Plaintiff filled out paragraph seven of the short form complaint, which instructs that the paragraph should only be filled out "[i]f alleging property damage." (*Id.*, PageID.1188.)

One reason for highlighting this is because Plaintiff requests in her response brief that she be permitted to amend her complaint again if the Court finds her operative complaint fails to state a claim. (ECF No. 105, PageID.1473.)

Although [Federal Rule of Civil Procedure 15\(a\)\(2\)](#) instructs courts to "freely give leave" to amend, this policy does not include arguments made as an aside in a response brief. A "request for leave to amend almost as an aside, to the district court in a memorandum in opposition to the defendant's motion to dismiss is ... not a motion to amend." *Kuyat v. MioMimetic Therapeutics, Inc.*, 747 F.3d 435, 444 (6th Cir. 2014) (citing *La. Sch. Emps.' Ret. Sys. v. Ernst & Young, LLP*, 622 F.3d 471, 486 (6th Cir. 2010)). In *Kuyat*, the Sixth Circuit evaluated language from the plaintiffs in a response brief that stated, "Alternatively, Plaintiffs request leave to amend the Complaint in the event that the Court finds that it falls short of the applicable pleading standards in any respect." (*Id.* at 444.) The plaintiffs in that case did not attach a copy of their proposed amended complaint. Taking these two factors together, the Sixth Circuit found that this type of argument for an amendment, made in a response in opposition to a Rule 12(b)(6) motion is essentially "throwaway language" and that the district court did not abuse its discretion in refusing to allow the plaintiffs to amend. *Id.*

\*5 Here, Plaintiff argues that "if arguendo, Plaintiff has not alleged sufficient facts linking her death to her Legionella

sickness, the appropriate remedy is to grant Plaintiff leave to further amend." (ECF No.105, PageID.1473.) She argues that "any such deficiencies can be readily cured by granting" leave to amend. (*Id.*) Plaintiff did not include her proposed amendment. Instead, she includes a short paragraph stating that Bacon suffered, "numerous severe [infections to her lungs](#) and other parts of her body – which in turn affected her ability to oxygenate and heal from other illnesses that were either pre-existing or contracted after she contracted Legionella sickness." (*Id.* at PageID.1474.) Then, she states that these additional facts "should arguably be sufficient to cure the factual deficiencies." (*Id.*)

Plaintiff's purported factual-support paragraph is not a proposed amendment. Indeed, all it does is raise more questions. What was the nature of Bacon's severe illness, and what other parts of her body besides her lungs were infected? What were her "other illnesses"? Were they contracted before or after her *legionella* exposure and illness? What were her "pre-existing conditions" that are referenced, and how do they tie into her claims? Her response to factual deficiencies raise more questions than answers. In this way, Plaintiff's request to further amend her complaint is not meaningfully different from that which was rejected in *Kuyat*. Accordingly, her request for leave to amend is denied.

### III. Legal Standard

When deciding a motion to dismiss under Federal Rule of Procedure 12(b)(6), the Court must "construe the complaint in the light most favorable to the plaintiff and accept all allegations as true." *Keys v. Humana, Inc.*, 684 F.3d 605, 608 (6th Cir. 2012). "To survive a motion to dismiss, a complaint must contain sufficient factual matter, accepted as true, to 'state a claim to relief that is plausible on its face.'" *Ashcroft v. Iqbal*, 556 U.S. 662, 678 (2009) (quoting *Bell Atlantic Corp. v. Twombly*, 550 U.S. 544, 570 (2007)). A plaintiff's claim is facially plausible "when the plaintiff pleads factual content that allows the court to draw the reasonable inference that the defendant is liable for the misconduct alleged." *Id.* A plausible claim need not contain "detailed factual allegations," but it must contain more than "labels and conclusions" or "a formulaic recitation of the elements of a cause of action." *Twombly*, 550 U.S. at 555.

### IV. Analysis

#### A. Incorporation of Prior Complaints

## 1. The State-Court First Amended Master Long Form Complaint

As an initial matter, Plaintiff did not properly incorporate the items that she references in the June 2020 Short Form Complaint. [Federal Rule of Civil Procedure 10\(c\)](#) governs adoptions by reference, and states, “A statement in a pleading may be adopted by reference elsewhere in the same pleading or in any other pleading or motion. A copy of a written instrument that is an exhibit to a pleading is a part of the pleading for all purposes.” The Sixth Circuit rule that “[m]atters outside the pleadings are not to be considered by a court in ruling on a 12(b)(6) motion to dismiss” applies. [Weiner v. Klais & Co.](#), 108 F.3d 86, 98 (6th Cir. 1997), (overruled on other grounds, [Swierkiwica v. Sorema, N.A.](#), 534 U.S. 506, (2002)).

Plaintiff did not attach the First Amended Long Form Complaint to her pleadings, nor is it anywhere else on her docket. In paragraph twelve of the June 2020 Short Form Complaint, Plaintiff states that she “incorporates herein by reference” the factual allegations and conduct of Defendants “set forth in the *First* Amended Master Long Form Complaint and her Short form Complaint filed in the Genesee County Circuit Court on November 9, 2017, prior to being removed to this Honorable Court on or about January 20, 2018.” (ECF No. 86, PageID.1190 (emphasis added).)

\*6 The state-court *First* Amended Master Long Form Complaint, which she specifically names, is not part of the record in this case. Rather, the state-court *Second* Amended Master Long Form Complaint was the operative state-court long form complaint at the time of removal, and is included on the docket. Plaintiff was clear in her reference to the First Amended Long Form Complaint, and not the Second. Plaintiff does not address this discrepancy or seek to make a correction in her response briefs.<sup>7</sup> Indeed, she does not address this discrepancy at all. Accordingly, she will be taken at her word regarding the item she specifically referenced, and the First Amended Master Long Form Complaint is not incorporated.

## 2. The November 2017 Complaint

Plaintiff does, however, properly incorporate portions of Bacon's state-court November 2017 Complaint. Unlike the state-court First Amended Master Long Form complaint,

the November 2017 Complaint was filed on the docket in this case. (ECF No. 1-2.) However, the November 2017 Complaint is rife with internal inconsistencies, and it involves parties and claims that do not align with the boxes she checked in her June 2020 Short Form Complaint.

For example, in her short form November 2017 Complaint, Bacon sets forth introductory descriptions of individuals identified as governmental defendants, including Dennis Muchmore, Eden Victoria Wells, M.D., Linda Dykema, Nancy Peeler, and Robert Scott. But then, she fails to specifically name them when setting forth her counts. (ECF No. 1-2, PageID.53–56.) Further complicating the puzzle, her November 2017 Complaint incorporates by reference paragraphs of the state-court Second Amended Master Long-Form Complaint. The state-court Second Amended Master Long-Form Complaint does allege counts against these specific individuals. When the two are taken together, it is unclear whether Plaintiff was suing those individuals by incorporation or not since she only partially addressed them in her short form complaint. Yet, it is not the job of this Court to sort this out, particularly since these individuals are not defendants here.

The only relevant paragraphs that the Court can discern in Bacon's November 2017 Complaint are her specific allegations regarding *legionella* exposure. They are set forth below:

2. As a direct and proximate result of using Flint River water in her activities of daily living, Plaintiff Margaret A. Bacon contracted [Legionella pneumonia](#) on or about September 12, 2014 in her home, resulting in lengthy hospitalizations during which she was intubated and placed on mechanical ventilation and treated with intravenous antibiotics.

3. As a direct and proximate result of Plaintiff Margaret Bacon's [Legionella pneumonia](#) and lengthy hospitalizations, mechanical ventilation, and life threatening infections, she suffered severe and permanent injuries and damages.

(ECF No. 1-2, PageID.48.) These are the only paragraphs where Plaintiff provides any detail regarding her *legionella* exposure and illness. The Court will accept as incorporated only those paragraphs set forth above that describe Plaintiff's *legionella* exposure and subsequent illness.

## B. Plaintiff Fails to State a Wrongful Death Claim

\*7 MDEQ Defendants, State Defendants, City Defendants, LAN and LAD's and motions to dismiss Plaintiff's wrongful death claim are granted because Plaintiff did not plead any facts to demonstrate that Bacon's death, or injuries resulting in death, were caused by the wrongful act, neglect, or fault of Defendants. Plaintiff alleges wrongful death under [Michigan Compiled Laws § 600.2922](#). The statute provides for the recovery of damages for a wrongfully caused death, and governs the distribution of wrongful death damages. *Id.* Actions under this statute are derivative and must be brought by the personal representative of the estate. [Mich. Comp. Laws § 600.2922\(2\)](#). The statute states, in relevant part,

Whenever the death of a person, injuries resulting in death, ... *shall be caused by wrongful act, neglect, or fault of another*, and the act, neglect, or fault is such as would, if death had not ensued, have entitled the party injured to maintain an action and recover damages, the person who or the corporation that would have been liable, if death had not ensued, shall be liable to an action for damages....

[Mich. Comp. Laws § 600.2922\(1\)](#) (emphasis added). Plaintiff did not plead any facts to demonstrate that Bacon's death, or injuries resulting in death, were caused by the “wrongful act, neglect, or fault” of Defendants.

In paragraph ten of her June 2020 Short Form Complaint, Plaintiff sets forth her claims. Her claim for survival and wrongful death against all Defendants, is identified as an “additional claim” that is not identified in the Master Complaint from *Walters*. Accordingly, to sustain her additional claim, Plaintiff was required to provide factual support in paragraph twelve.

Paragraph twelve of Plaintiff's June 2020 Short Form Complaint states:

12. If additional claims against the Defendants identified in the Master Long Form Complaint are alleged in paragraph 10, the facts supporting these allegations must be pleaded. Plaintiff asserts the following factual allegations against the Defendants identified in the Master Long Form Complaint:

A. Plaintiff, Deborah Sapolin, as the Personal Representative of the Estate of Margaret A. Bacon, incorporates herein by reference all preceding factual allegations set forth in the First Amended Master Long Form Complaint and her Short Form Complaint filed in

the Genesee County Circuit Court on November 9, 2017, prior to being removed to this Honorable Court on or about January 30, 2018.

B. The conduct of Defendants, as described in the First Amended Master Long Form Complaint and Plaintiff's Short Form Complaint filed in the Genesee County Circuit Court on November 9, 2017, was the proximate cause of Margaret A. Bacon's death.

C. Plaintiff, as Personal Representative of the Estate of Margaret A. Bacon, Deceased, is entitled to the following damages as a result of Defendants' conduct:

- i. All damages recoverable pursuant to Michigan's wrongful death statute, [MCL 600.2922](#);
- ii. Damages for pain and suffering sustained by decedent Margaret A. Bacon before her death on April 10, 2018;
- iii. Loss of past and future earnings;
- iv. Funeral and burial expenses;
- v. Medical and hospital expenses;
- vi. Damages for the loss of society and companionship suffered by Margaret A. Bacon's family members.

(ECF No. 86, PageID.1190–91.)

As set forth above, Plaintiff failed to properly incorporate her state-court complaints except for paragraphs 2–3, which specifically relate to Bacon's *legionella* exposure and illness. However, even the incorporated paragraphs do not provide any facts related to the cause of Bacon's death because they were written before April 2018 when she died.

Therefore, the June 2020 Short Form Complaint was the place to set forth allegations regarding Bacon's cause of death. Yet, the only allegation regarding Bacon's death in the June 2020 Short Form Complaint is Plaintiff's conclusory allegation that Defendants were “the proximate cause of Margaret A. Bacon's death.” (PageID.1190–91.) This bare allegation provides no facts regarding how her 2014 illness, while very serious, contributed to or caused her 2018 death.

\*8 Plaintiff argues in her response briefs that Bacon died “as a result of complications associated with her Legionella sickness.” (ECF No. 101, PageID.1395; ECF No. 102, PageID.1414.) She states that she “advised the court of this” and was given a “green light” to add wrongful death and

survival claims to her short form complaint. (*Id.*) She also defends the brevity of her pleadings, stating that the format of long-and-short-form complaints adopted in this case:

allow each individual Plaintiff to simply check boxes for Defendants and claims, and they are set up in a way that minimizes the amount of additional factual information that each individual Plaintiff must allege in addition to what is already alleged in the Master Complaint. These special pleading requirements clearly supersede any conflicting procedural requirements contained elsewhere. (ECF No. 105, PageID.1471.) She argues that these forms create a “relaxed specificity standard.” (*Id.* at 1472.)

Finally, Plaintiff argues that, even without the “relaxed” standard, her facts were sufficient to support a wrongful death claim:

Her [June 2020] Short Form Complaint clearly identifies that she contracted legionella sickness as a result of being exposed to the contaminated Flint water supply, and that she became violently ill immediately thereafter as a result. Her Complaint further confirms that she subsequently underwent extensive medical treatment *and died with a reasonable short amount of time after contracting Legionella sickness* and undergoing extensive medical treatment for all of the harms that it brought upon her. Given the nature and severity of her legionella sickness and the timing of her death, it is clearly reasonable to infer circumstantially that Plaintiff’s ultimate death was brought on by her legionella sickness.

(ECF No. 105, PageID.1472 (emphasis added).)

This is not an accurate account of the contents of the June 2020 Short Form Complaint. As set forth above, Plaintiff alleges Bacon was exposed to *legionella* in September 2014, and died over three-and-a-half years later in April 2018. Three-and-a-half years is not a “reasonable short amount of time,” particularly when Plaintiff has not provided any additional detail regarding the length of Bacon’s illness, or what harms it brought about that could reasonably lead the Court to conclude that *legionella* exposure, and therefore Defendants, caused her death.

Nor are Plaintiff’s arguments that the streamlined process for long- and-short-form complaints creates a “relaxed” specificity standard persuasive. The short form complaint itself states, “factual support for these allegations must be pleaded.” (ECF No. 86, PageID.1190.) The Master Complaint from *Walters* specifically states that, “[a]ny

separate facts and additional claims of individual Plaintiffs may be set forth as necessary in the actions filed by the respective Plaintiffs.” (*Walters*, No. 17-0164, ECF No.185-2, PageID.5044.) Factual support for a complaint is a basic pleading requirement under [Federal Rule of Civil Procedure 8](#), which is unchanged by the streamlined process in the Flint Water cases.

Accordingly, Defendants’ motions to dismiss Plaintiff’s wrongful death claim are granted. Rowe answered the complaint and did not move to dismiss this claim. However, in light of this decision, Rowe may file a motion under Rule 12(c) within sixty days, which is Monday December 21, 2020.

### C. Plaintiff’s Remaining Long-Form Counts

All of Plaintiff’s remaining claims rely in their entirety on the Master Complaint from *Walters*. (*Walters*, No. 17-10164, ECF No. 86, PageID.1186.) The Court will address each claim in turn as set forth below.

#### 1. State-Created Danger Claim

\*9 Plaintiff alleges that MDEQ Defendants, State Defendants, and City Defendants violated Bacon’s right to be free from a state-created danger. (ECF No. 86, PageID.1189.) The Defendants moved to dismiss. (ECF No. 91, PageID.1307–08); (ECF No. 93, PageID.1327.)

Plaintiff concedes in her response that, “the Court dismissed identical State Created Danger claims” in *Marble* and *Brown*. (ECF No. 104, PageID.1459.) She acknowledges that the Court’s ruling in those cases govern this issue but notes that she disagrees with those rulings. (*Id.*)

For the reasons set forth in *Marble* and *Brown*, Plaintiff’s state-created danger claims are dismissed. [Brown](#), 2020 WL 1503256, at \* 16; [Marble](#), 453 F. Supp. 3d at 988–91.

#### 2. Bodily Integrity Claim

Plaintiff alleges that MDEQ Defendants, State Defendants, and City Defendants violated her right to bodily integrity. (ECF No. 86, PageID.1189.) The Defendants moved to dismiss. (ECF No. 91, PageID. 1296–1306); (ECF No. 93, PageID.1333–1337.)

As in *Marble*, the Court adopts the governing legal standard for a bodily integrity claim set forth previously in *Walters* and *Carthan*:

The right to bodily integrity is a fundamental interest protected by the Due Process Clause of the Fourteenth Amendment. *Guertin*, 912 F.3d at 918–19; *Guertin*, 2017 U.S. Dist. LEXIS 85544, at \*63 (citing *Union Pac. Ry. Co. v. Botsford*, 141 U.S. 250, 251 (1891)). And although violations of the right to bodily integrity usually arise in the context of physical punishment, the scope of the right is not limited to that context. *Kallstrom v. City of Columbus*, 136 F.3d 1055, 1062–63 (6th Cir. 1998). For instance, the “forcible injection of medication into a nonconsenting person’s body represents a substantial interference with that person’s liberty.” *Guertin*, 912 F.3d at 919 (citing *Washington v. Harper*, 494 U.S. 210, 229 (1990)). And “compulsory treatment with anti-psychotic drugs may [also] invade a patient’s interest in bodily integrity.” *Guertin*, 2017 U.S. Dist. LEXIS 85544, at \*66 (citing *Lojuk v. Quandt*, 706 F.2d 1456, 1465–66 (7th Cir. 1983)). The key is whether the intrusion is consensual. See *Guertin*, 912 F.3d at 920. There is no difference between the forced invasion of a person’s body and misleading that person into consuming a substance involuntarily. *Guertin*, 2017 U.S. Dist. LEXIS 85544, at \*71 (citing *Heinrich v. Sweet*, 62 F. Supp. 2d 282, 313–14 (D. Mass. 1999)). As such, officials can violate an individual’s bodily integrity by introducing life-threatening substances into that person’s body without their consent. *Guertin*, 2017 U.S. Dist. LEXIS 85544, at \*65 (citing *Washington*, 494 U.S. at 229).

However, to state a claim, plaintiffs must do more than point to the violation of a protected interest; they must also demonstrate that it was infringed arbitrarily. *Guertin*, 912 F.3d at 922. *But see Range v. Douglas*, 763 F.3d 573, 589 (6th Cir. 2014) (observing that in some contexts government action may violate substantive due process without a liberty interest at stake). And with executive action, as here, only the most egregious conduct can be classified as unconstitutionally arbitrary. *Cty. of Sacramento v. Lewis*, 523 U.S. 833, 846 (1998). In legal terms, the conduct must “shock[ ] the conscience.” *Guertin*, 2017 U.S. Dist. LEXIS 85544, at \*63 (quoting *Lewis*, 523 U.S. at 846).

\*10 Whether government action shocks the conscience depends on the situation. *Ewolski v. City of Brunswick*, 287 F.3d 492, 510 (6th Cir. 2002). Where unforeseen

circumstances demand the immediate judgment of an executive official, liability turns on whether decisions were made “maliciously and sadistically for the very purpose of causing harm.” *Lewis*, 523 U.S. at 852–53 (quoting *Whitley v. Albers*, 475 U.S. 312, 320–21 (1986)). But where an executive official has time for deliberation before acting, conduct taken with “deliberate indifference” to the rights of others “shocks the conscience.” See *Claybrook v. Birchwell*, 199 F.3d 350, 359 (6th Cir. 2000). This case involves the latter of these two situations. And as a result, plaintiffs must demonstrate that (1) officials knew of facts from which they could infer a “substantial risk of serious harm,” (2) that they did infer it, and (3) that they nonetheless acted with indifference, *Range*, 763 F.3d at 591 (citing *Ewolski*, 287 F.3d at 513), demonstrating a callous disregard towards the rights of those affected, *Guertin*, 912 F.3d at 924 (quoting *Schroder v. City of Fort Thomas*, 412 F.3d 724, 730 (6th Cir. 2005)).

*Marble*, 453 F. Supp. 3d at 991–92 (citing *Walters*, 2019 WL 3530874, at \*14–\*15; *Carthan*, 384 F. Supp. 3d at 839–40).

As set forth, the Court’s inquiry is whether each Defendant had knowledge of the facts from which they could infer a substantial risk of serious harm to Bacon, did infer it, and nonetheless acted with indifference demonstrating a callous disregard towards Bacon’s rights. Accordingly, the pertinent time frame for this knowledge-based analysis is the time before Bacon became ill on September 12, 2014. In analyzing the bodily integrity claims in *Walters*, the Court relied upon many facts that occurred after Bacon became ill. Those facts are not applicable to the bodily integrity claim here, which is limited to whether Defendants can be held liable for the conditions that resulted in Bacon contracting *legionella*. For this reason, only conduct undertaken by Defendants before Plaintiff fell ill with *legionella* on September 12, 2014 can be considered in this analysis.

#### a) Legionella Exposure

As an initial matter, in both *Marble* and *Brown*, the Court determined that bodily integrity claims based on *legionella* exposure could proceed on the same bases as claims based on lead exposure. *Marble*, 453 F. Supp. 3d at 992–93; *Brown*, 2020 WL 1503256 at \*5–\*8. It also held that the Defendants’ actions that allegedly hid the Flint water’s lead and *legionella* content implicated the plaintiffs’ right to bodily integrity. *Id.* The Court fully adopts those conclusions in this case.

MDEQ Defendants and State Defendants urge the Court to decide this issue differently here because, they argue, state-level *legionella* exposure regulations do not rest with MDEQ as the state regulator. (ECF No. 91, PageID.1300.) Plaintiff does not address this argument in her response. (ECF No. 104.)

In *Brown*, MDEQ Defendants and State Defendants also argued that *legionella*-related cases should be decided differently from lead injury cases. (Brown, ECF No. 83, PageID.442, 444); (ECF No. 91, PageID.1310–1312.) The Court rejected that argument in *Brown*, explaining:

“[T]his is not a case about the right to a contaminant-free environment or clean water. Rather, this case implicates the consumption of life-threatening substances. Indeed, neither side disagrees that lead and *legionella* are life threatening, nor that plaintiffs ingested these contaminants and others through the water supply.” [*Carthan*,] 384 F. Supp. 3d at 840 (internal citations removed). Similarly, as the Sixth Circuit held in *Guertin*, a related Flint Water Case: “Involuntarily subjecting nonconsenting individuals to foreign substances with no known therapeutic value—often under false pretenses and with deceptive practices hiding the nature of the interference—is a classic example of invading the core of the bodily integrity protection.” *Guertin v. State*, 912 F.3d 907, 920–21 (6th Cir. 2019).

\*11 *Brown*, 2020 WL 1503256, at \* 7. The presence of legionella bacteria was a foreseeable consequence of the April 2014 switch to the Flint River. As such,

Plaintiff plausibly alleges that the presence of *legionella* bacteria in Flint was a foreseeable result of the April 2014 switch to Flint River water. Because Defendants allegedly hid the fact that Flint’s water contained life-threatening substances like lead and *legionella*, and because under state and municipal law, Plaintiff was not permitted to receive water in any other way, Flint Code of Ord. §§ 46-25, 46-26, 46-50(b), Plaintiff’s claim implicates the right to bodily integrity. See *Walters*, 2019 WL 3530874, at \*15.

*Brown*, 2020 WL 1503256, at \* 7. Moreover, the Court’s reasoning regarding exposure in *Brown* applies with the same force here:

The right to bodily integrity is not dependent upon which particular dangerous or even lethal substance came from Flint’s pipes. Defendants made a choice to utilize the long dormant Flint Water Treatment Plant (“FWTP”), knowing that the plant required millions of dollars in upgrades

before it could process the raw water from the Flint River, and that those upgrades would not be implemented.

*Id.* at \*8.

As such, this issue has already been fully litigated in *Marble* and *Brown*. MDEQ Defendants and State Defendants advance no compelling arguments to justify treating *legionella*-related cases differently from lead injury cases. Accordingly, the Court will adhere to its prior decisions in *Marble* and *Brown*.

#### b) Defendants Cook and Dillon

In *Brown*, the Court dismissed the bodily integrity claims against Cook and Dillon because the Master Complaint contained insufficient factual allegations against them that preceded Odie Brown’s death in January 2015. *Brown*, 2020 WL 1503256, at \*9, \*12. If the pre-January 2015 allegations in *Brown* were insufficient, then Plaintiff’s pre-September 2014 allegations here must also fail. For the reasons stated in *Brown*, Plaintiff’s bodily integrity claims against Cook and Dillon are dismissed.<sup>8</sup> See *Brown*, 2020 WL 1503256, at \*9, \*12.

#### c) Defendant Governor Snyder

In contrast to Cook and Dillon, the allegations set forth in *Brown* indicated that Governor Snyder knew of and inferred a substantial risk of serious harm to Flint water users prior to September 2014.

[Governor Snyder] knew that the use of “Flint River water as a primary drinking source had been professionally evaluated and rejected as dangerous and unsafe” in 2011. (*Id.* at PageID.5077.) He also knew that under the plan to create the Karegnondi Water Authority, Flint River water would be used as an interim source of water for the City of Flint. (*Id.*) Plaintiff also alleges that shortly after the switch to Flint River water, the Governor’s office began receiving complaints about the water. (*Id.* at PageID.5085.) There were also numerous press stories about water quality problems in Flint as early as May 2014. (*Id.*) By June of 2014, “[m]any Flint water users reported that the water was making them ill[.]”

\*12 *Id.* (citing *Walters*, No. 17-10164, ECF No. 185-2). As in *Brown*, it is reasonable to infer that because Governor Snyder knew of the significant risks and seriously compromised water quality issues well before then, he knew

of and did infer a substantial risk. *Brown*, 2020 WL 1503256, at \*8. Accordingly, the first two elements of a bodily integrity claim have been adequately plead.

As for the third element of a bodily integrity claim, callous disregard, the Court in *Marble* determined that the plaintiff's claim that Governor Snyder "authorized the switch to the Flint River, knowing that 'there was no agreed upon plan in place to implement the necessary remediation at the FWTP in order to use Flint River water as Flint's sole source of water.'" *Marble*, 453 F. Supp. 3d at 994 (citing Master Complaint from *Walters*, No. 17-10164, ECF No. 185-2, PageID.5077.) The Court in *Marble* also reasoned that "the Governor's continued inaction following the switch reinforces his deliberate indifference." *Id.* Even without the allegations of a cover-up beginning in January 2015, approximately four months after Bacon contracted *legionella*-related illness, Governor Snyder's failure to act for months despite notice of harm shows a callous disregard. The Court came to a similar conclusion in *Brown*, where it also disregarded allegations that took place after January 2015. *Brown*, 2020 WL 1503256, at \*9. Accordingly, Governor Snyder's motion to dismiss is denied and Plaintiff's bodily integrity claim against him may continue.

#### d) Defendants Croft, Glasgow, and Johnson

In *Carthan*, the Court summarized Croft, Glasgow, and Johnson's alleged actions. The following occurred before September 12, 2014, when Bacon contracted illness:

As the transition to the Flint River loomed, [in spring of 2014] all three knew that the FWTP was not ready to process the raw water. And Croft, in particular, was aware of the lead and [Legionnaires' disease](#) issues that followed the transition. Glasgow tested for and found high concentrations of lead in the water. He also recognized that Flint was not using corrosion control treatment and had no legitimate lead and [copper](#) testing in place.... Despite knowing that the FWTP was not ready to process the Flint River water, Croft and Johnson pressured Glasgow to give the green light to the transition [in April 2014].

*Carthan*, 384 F. Supp. 3d at 860. Accordingly, it is reasonable to conclude that these Defendants were aware of the substantial risk of harm facing Bacon, that they did infer it, and that they acted with callous disregard toward her.

In *Walters*, the Court found that the plaintiffs' bodily integrity claims in the Master Complaint contained essentially the same allegations as the *Carthan* complaint with respect to Croft, Johnson, and Glasgow. *Walters*, 2019 WL 3530874, at \*18. Accordingly, for the same reasons set forth in *Carthan* and *Walters*, Croft, Glasgow, and Johnson were aware of the substantial risk of harm facing Bacon.

Analyzing the callous disregard element with respect to these three Defendants again in *Marble*, the Court stated,

[A]ll three Defendants participated in making the switch to the Flint River in April 2014, knowing that the FWTP was not ready to process water. This fact alone is enough to show callous disregard for Bertie Marble's bodily integrity. 453 F. Supp. 3d at 1000. Here, this fact alone is also enough to show Croft, Glasgow, and Johnson's callous disregard for Bacon's bodily integrity. Because these individuals were involved in the switch to the Flint River, knowing full well of the dangers, and the relevant conduct took place prior to September 14, 2014, Plaintiff has stated a bodily integrity claim against Croft, Glasgow, and Johnson.

#### e) Defendants Busch and Prysby

\*13 With respect to Busch and Prysby, the relevant pre-September 2014 facts were also set forth in *Marble*.

Plaintiffs allege that Busch was involved in resolving the regulatory hurdles to using Flint River water. (*Id.* at PageID.5173–5176.) For example, he helped obtain an Administrative Consent Order ("ACO") that was critical to allowing the City of Flint to begin using the FWTP, although the plant was "nowhere near ready to begin distributing water." (*Id.* at PageID.5176.) Plaintiffs allege that Prysby reviewed and approved the permit "that was the last approval necessary for the use of the Flint Water Treatment Plant." (*Id.* at PageID.5081, 5179.)

Moreover, shortly before the switch, the FWTP's water quality supervisor wrote to Prysby and Busch that he had inadequate staff and resources to properly monitor the water. (*Id.* at PageID.5080.) As a result, he informed Prysby and Busch, "I do not anticipate giving the OK to begin sending water out anytime soon. If water is distributed from this plant in the next couple of weeks, it will be against my direction." (*Id.*) But Prysby and Busch did not act on this warning.

*Id.* at 997 (citing Master Complaint from *Walters*, No. 17-10164, ECF No. 185-2.) The Court found that, based on these facts, Busch and Prysby knew of and did infer a substantial risk of serious harm to Flint water users, and showed a callous disregard for Marble's right to bodily integrity. These facts apply with equal force to Bacon. Busch and Prysby argue that “[t]he first allegation that Busch or Prysby had knowledge of a *legionella* issue in Flint is alleged to be March 10, 2015, six months after Plaintiff's alleged contraction.” (ECF No. 91, PageID.1301.) However, the Court has already rejected this contention in *Brown* and *Marble*.

[T]he risks of using Flint River water channeled through the FWTP were substantial. The complaint alleges that many of these MDEQ Defendants knew as early as May 2014 that Flint's water was contaminated in ways that could be life threatening. (*Id.* at PageID.5130–5131, 5140–5141.) Even if the MDEQ Defendants were not aware of *legionella* bacteria in particular by the time of Odie Brown's death, the facts alleged plausibly show that Busch, ... and Prysby were aware of the dangerous condition of the City's water supply before she died.

*Brown*, 2020 WL 1503256, at \* 10 (citing *Walters*, No. 17-10164, ECF No. 185-2); see also *Marble*, 453 F. Supp. 3d at 996–997. Accordingly, Busch and Prysby's motion to dismiss Plaintiff's bodily integrity claim is denied. Plaintiff's bodily integrity claim against Busch and Prysby may continue.

#### f) Defendant Rosenthal

With respect to Rosenthal, the *Walters* Master Complaint—adopted in full by Plaintiff here—contains essentially the same allegations related to the plaintiffs' bodily integrity claims in *Carthan*. *Walters*, 2019 WL 3530874, at \*18. On appeal, the Sixth Circuit summarized Rosenthal's alleged pre-September 2014 conduct that applies equally to Bacon in this case.

On April 16, 2014, the week before the switch to the Flint River, Rosenthal received an email from Michael Glasgow, stating, “[I]t looks as if we will be starting the plant up tomorrow and are being pushed to start distributing water as soon as possible.... I would like to make sure we are monitoring, reporting and meeting requirements before I give the OK to start distributing water.” *Carthan*, 960 F.3d at 314. (citing Amended Complaint in *Carthan*, No. 16-10444, ECF

No. 349, PageID.11804.) And the very next day, Glasgow informed the MDEQ that “the FWTP was not fit to begin operations and that ‘management’ was not listening to him.” *Id.* The Sixth Circuit also noted that, “[b]ack in May 2014, MDEQ officials—including Busch, Prysby, and Rosenthal—knew that [total trihalomethane] levels were above the EPA's maximum contaminant level but did nothing, even as residents raised concerns about the water.” *Id.* at 315 (citing Master Complaint in *Carthan*, No. 16-10444, ECF No. 349, PageID.11813–14.). Moreover, in the summer of 2014, the Michigan Department of Health and Human Services (“MDHHS”) reported an outbreak of *Legionnaires' disease*, which occurs when water droplets contaminated with *legionella* bacteria are inhaled. (*Id.*) These events all took place before Bacon became ill.

\*14 The relevant allegations that the Sixth Circuit in *Carthan* found sufficient to state a claim against Rosenthal for bodily integrity are that Rosenthal was the MDEQ Water Quality Analyst who “did not stop the switch to the Flint River in spite of Glasgow's warning that the FWTP was not ready.” *Id.* at 327. That Rosenthal “knew as early as May 2014 that the water contained high TTHM levels that were above regulation ... and did nothing.” *Id.* These same facts pleaded in the *Walters* Master Complaint are sufficient to show that Rosenthal knew of and did infer a substantial risk of serious harm to Flint water users, including Bacon.<sup>9</sup> Further, these allegations are adequate to show that Rosenthal callously disregarded Bacon's right to bodily integrity. Rosenthal's motion to dismiss is denied, and Plaintiff's claim against him may continue.

#### g) Defendant City of Flint

Plaintiff alleges the City of Flint is liable under 42 U.S.C. § 1983 as a result of the unconstitutional actions taken by Earley and Ambrose. (*Walters*, No. 17-10164, ECF No. 185-2, PageID.5051–52, 5055–56.) Under *Monell v. Dep't of Soc. Servs. of the City of New York*, a plaintiff may bring a § 1983 claim against a city for the unconstitutional conduct of its employees only if the employees' conduct implemented a policy “officially adopted and promulgated by that body's officers.” 436 U.S. 658, 690 (1978). However, a municipality “cannot be held liable solely because it employs a tortfeasor.” *Id.* at 691. Liability will only attach where the policy or custom was the “moving force” behind the constitutional violation. *Powers v. Hamilton Cty. Pub. Def. Comm'n*, 501 F.3d 592, 607 (6th Cir. 2007).

In *Carthan*, the Court held that Earley and Ambrose “were final decisionmakers for Flint with respect to the decision to provide residents with contaminated water.” 384 F. Supp. 3d at 865 (citing *Carthan*, 329 F. Supp. 3d at 421–22). As such, “their actions represented official policy and Flint could be held liable for their conduct insofar as it violated plaintiffs’ rights.” *Carthan*, 329 F. Supp. 3d at 422.

Here, even though Plaintiff has conceded to dismissal of Earley and Ambrose (ECF No. 105, PageID. 1476), she states a claim for *Monell*-based bodily integrity against the City of Flint for the same reasons set forth in *Carthan* and *Brown*. *Carthan*, 329 F. Supp. 3d at 422; *Brown*, 2020 WL 1503256, at \* 14.

### 3. Wealth-Based Equal Protection Claim

Plaintiff alleges that MDEQ Defendants, State Defendants, and City Defendants violated her right to be free from wealth-based discrimination. (ECF No. 86, PageID.1189.) Plaintiff’s wealth-based equal protection allegations are based solely on the allegations set forth in the Master Complaint from *Walters*. In *Walters*, the Court analyzed and dismissed the plaintiffs’ wealth-based discrimination claims because the plaintiffs failed to identify how their treatment differed from a similarly situated class of persons. *Walters*, 2019 WL3530874, at \*20. The Court adopts these conclusions from *Walters*, and Plaintiff’s wealth-based equal protection claim is dismissed.

### 4. Professional Negligence Claim

Plaintiff also alleges a professional negligence claim against Defendants LAN, LAD, and Rowe. (ECF No. 86, PageID.1189.) Only LAN and LAD moved to dismiss. However, neither LAN nor LAD have presented any arguments that differ from the arguments presented and rejected in *Walters*. 2019 WL 3530874, at \*40. For the reasons set forth in *Walters*, LAN’s motion to dismiss is denied. Plaintiff’s claims for professional negligence against LAN and LAD may go forward.

### 5. Punitive Damages Claim

\*15 Plaintiff also incorporates the Punitive Damages claim from the Master Complaint in *Walters* against all Defendants. (ECF No. 86, PageID.1189 (*Walters*, No. 17-10164, ECF No. 185-2, PageID. 5234).) MDEQ Defendants, State Defendants, and LAN move to dismiss. (ECF No. 89, 91.) City Defendants and LAD incorporate their motions to dismiss this claim in other cases. (ECF No. 93, PageID.1327; ECF No. 90, PageID.1206.)

Punitive damages may be awarded in § 1983 actions “when the defendant’s conduct is shown to be motivated by evil motive or intent, or when it involves reckless or callous indifference to the federally protected rights of others.” *King v. Zamiara*, 788 F.3d 207, 216 (6th Cir. 2015) (quoting *Smith v. Wade*, 461 U.S. 30, 56 (1983)). Based on the allegations set forth in the above bodily integrity section of this Opinion and Order, Plaintiff plausibly pleads both recklessness and indifference to the right to bodily integrity against Defendants Snyder, Croft, Glasgow, Johnson, Busch, Prysby, Rosenthal, and the City of Flint (*Monell*-liability). As a result, Plaintiff may continue to seek punitive damages against these Defendants with respect to her remaining § 1983 bodily integrity claim.

Plaintiff also alleges she is entitled to punitive damages because she brought professional negligence claims against LAD and Rowe. Plaintiff acknowledges that these issues were already litigated in *Brown* and *Marble*. There, the Court dismissed the plaintiffs’ claims for punitive damages related to professional negligence, because the plaintiffs in those cases acknowledged that punitive damages are not available for negligence claims. See *Marble*, 453 F. Supp. 3d at 1010; *Brown*, 2020 WL 1503256, at \*16. The result here is no different. Plaintiff’s punitive damages claim against LAN and LAD under a professional negligence theory are dismissed.

Rowe answered the complaint and did not move to dismiss this claim. However, in light of this decision, Rowe may file a motion under Rule 12(c) within sixty days, which is Monday December 21, 2020.

### 6. Joint and Several Liability and Exemplary Damages

Plaintiff acknowledges that her assertions of joint and several liability and exemplary damages are identical to those rejected in *Marble*, *Brown*, and *Walters*. (ECF No. 101, PageID.1394.) While she disagrees with the Court’s rulings in those cases, Plaintiff provides no basis for a different result here. (*Id.*)

The Court agrees that the rulings in *Marble*, *Brown*, and *Walters* apply and dictate the same result here. Accordingly, Plaintiff's assertions of joint and several liability and claim for exemplary damages are dismissed.

#### V. Conclusion

Defendants' motions to dismiss Plaintiffs' Short Form Complaint are granted in part and denied in part. Specifically, Defendants' motions to dismiss:

- Plaintiff's wrongful death claim (Count XIII) is GRANTED as to all Defendants (except Rowe), because Plaintiff did not plead any facts to demonstrate that Bacon's death, or injuries resulting in death, were caused by *legionella* exposure or, in turn, by any wrongful act, neglect or fault Defendants;
- Plaintiff's state-created danger claim (Count I) is GRANTED as to all Defendants;
- Plaintiff's bodily integrity claim (Count II) is GRANTED with respect to Dillon, Cook, and the City of Flint, but DENIED with respect to Snyder, Croft, Glasgow, Johnson, Busch, Prysby, and Rosenthal;
- Plaintiff's wealth-based equal protection claim (Count IV) is granted as to all Defendants;
- \*16 • Plaintiff's punitive damages claim (Count VIII) is GRANTED with respect to Plaintiffs' professional negligence claims against LAN and LAD, but DENIED with respect to Plaintiff's § 1983 claims; and
- Plaintiff's request for exemplary damages and allegations of joint and several liability are GRANTED.

#### Footnotes

- 1 The Court will refer to Ms. Sapolin, as personal representative of Ms. Bacon's estate as Plaintiff, and will refer to Ms. Bacon herself as Bacon.
- 2 Defendants former Governor Richard D. Snyder and Andy Dillon filed a notice of joinder/concurrence in the MDEQ Defendants' motion to dismiss. (ECF No. 92.) (Defendants Snyder and Dillon are, collectively, the "State Defendants.") Defendant Adam Rosenthal also filed a joinder and concurrence in the MDEQ Defendants' motion. (ECF Nos. 97, 99.) Rosenthal will be included in the Court's reference to the "MDEQ Defendants."
- 3 Plaintiff originally included Bradley Wurfel in her operative complaint, but stipulated to his dismissal shortly thereafter. (ECF No. 88.) The operative complaint also named Darnell Earley and Gerald Ambrose. (ECF No. 86.) However, in her response to the City Defendants' motion to dismiss, and as further discussed below, Plaintiff consented to the dismissal of her claims against Earley and Ambrose. (ECF No. 105, PageID.1476.) Accordingly, these three individuals are not included in this list.
- 4 Plaintiff does not specify whether she sues former Governor Snyder in his official or individual capacity. For the sake of consistency with earlier Flint Water decisions, former Governor Snyder will be referred to as Governor Snyder or the

Plaintiff's request that she be given leave to amend is DENIED.

As set forth above, Rowe may file a motion under Rule 12(c) as to Plaintiff's wrongful death and punitive damages claims within sixty days, which is Monday December 21, 2020.

#### VI. Order

IT IS ORDERED THAT,

MDEQ Defendants' motion to dismiss (ECF No. 91) is GRANTED in part and DENIED in part; City Defendants' motion to dismiss (ECF No. 93) is GRANTED in part and DENIED in part; LAN and LAD's motions to dismiss (ECF Nos. 89, 90) are GRANTED in part and DENIED in part.

As a result, Plaintiff's bodily integrity claims against Defendants Snyder, Croft, Glasgow, Johnson, Busch, Prysby, and Rosenthal will proceed; her professional negligence claims against LAN and LAD will proceed; and Plaintiff may continue to request punitive damages with respect to her remaining § 1983 claims. All of Plaintiff's other claims are dismissed except as to Rowe as set forth above.

IT IS SO ORDERED.

#### All Citations

Slip Copy, 2020 WL 6218787

Governor where it appears that the claim against him is in his individual capacity. Where it appears that the claim is against him in his official capacity, the claim is now against Governor Gretchen Whitmer. See *Fed. R. Civ. P. 25(d)*. But, again, for consistency, the Court will still refer to Governor Snyder.

5 Defendant Rowe answered the complaint. (ECF No. 98.)

6 Plaintiff did so pursuant to the Court's order allowing Plaintiffs in any remaining post-*Marble* and post-*Brown legionella* cases to amend their complaints before June 3, 2020. (ECF No. 1150.)

7 Moreover, Plaintiff doubles down on this discrepancy in her sur-reply, claiming that she may have never intended to incorporate the state-court filings at all, where she states:

The MDEQ Defendants have incorrectly represented that Plaintiff is relying on the Master Long Form Complaint filed in the Genesee County Circuit Court. This is simply not true. Plaintiff is relying on her Amended Short Form Complaint ([ECF No.]86), which expressly adopted the Master Long Form Complaint that was filed with this Court in *Walters* ... (ECF No. 111-1, PageID.1527.) Her sur-reply is not a factor in this decision, but it does illustrate more of the inconsistencies she presents in this case.

8 In her response to the City Defendants' motion to dismiss, Plaintiff states that, based on *Brown*, dismissal should be denied against all Defendants "except for Defendants Ambrose and Walling." (ECF No. 105, PageID. 1469–70.) However, Walling is not a defendant in this case (ECF No. 86), so it is unclear why Plaintiff includes this argument. However as to Ambrose, the Court accepts Plaintiff's statement in her response that Defendants' motion to dismiss should be denied except as to Ambrose as a stipulation to his dismissal. Ambrose is dismissed.

9 This conclusion still stands even without the facts cited in *Carthan* regarding the September 2014 MDHHS report regarding [lead poisoning](#) levels in children being higher than usual, the officials' October 2014 realization that bacterial contamination partly stemmed from the over-75-year-old-pipes, or any of the other later-in-time facts. *Carthan*, 960 F.2d at 315.

---

End of Document

© 2020 Thomson Reuters. No claim to original U.S. Government Works.

969 F.3d 298

United States Court of Appeals, Sixth Circuit.

IN RE: FLINT WATER CASES.

Marlana Sirls et al.; Leanne Walters,  
individually and as next friend of  
her children G.w.1, G.w.2, K.M.,  
J.D. et al., Plaintiffs-appellees,

v.

State of Michigan et al., Defendants,  
Stephen Busch, Patrick Cook, Michael  
Prysby, and Bradley Wurfel; Adam  
Rosenthal; Richard Dale Snyder,  
Andy Dillon, and [Gretchen Whitmer](#);  
City of Flint, Michigan, Howard  
Croft, Daugherty Johnson, Michael  
Glasgow, Darnell Earley, and [Gerald  
Ambrose](#), Defendants-Appellants.

19-1961

|

19-1975

|

19-1983

|

19-2000

|

19-2005

|

19-2008

|

19-2011

|

19-2012

|

Argued: June 9, 2020

|

Decided and Filed: August 5, 2020

**Synopsis**

**Background:** Residents brought action against city and government officials, arising out of allegedly causing and covering up the lead poisoning of city residents through their water supply. The United States District Court for the Eastern

District of Michigan, [Judith E. Levy, J.](#), 2019 WL 3530874, granted in part and denied in part city's and officials' motion to dismiss and residents' motion to amend the master complaint, which contained all the various allegations and claims made by plaintiffs across coordinated litigation. City and officials appealed.

**Holdings:** The Court of Appeals, [Moore](#), Circuit Judge, held that:

[1] prior appellate decision addressing same claims at motion-to-dismiss stage controlled, and

[2] former Governor was proper party to action.

Affirmed and remanded.

[Sutton](#), Circuit Judge, filed concurring opinion.

See also [960 F.3d 303](#).

West Headnotes (2)

[1] **Judgment**  [Appellate courts](#)

**Judgment**  [What constitutes identical causes](#)

Prior appellate decision addressing same claims at motion-to-dismiss stage controlled, and thus precluded dismissal of government official in action regarding poisoning of city residents due to switch in water supply, despite contention that allegations against official in subsequent case should have been viewed differently from prior decision; prior decision concluded that same city and state officials plausibly violated city residents' substantive due process right to bodily integrity and were not entitled to qualified immunity, and allegations in subsequent case included that official pressured another to go forward with water switch even though water treatment plant was not ready, and that official stonewalled county health department's attempt to investigate water-quality issues. [U.S. Const. Amend. 5](#).

**[2] States**  **Governor**

Former Governor was proper party to action for damages from decisions that allegedly caused, sustained, and covered up poisoning of city residents due to switch in water supply, despite contention that Governor was too high-ranking to know of risks of switching water supply; Governor was alleged to have coordinated switch of water supplies knowing that water would not be treated for contamination, was alleged to have refused to switch city back to clean water, knowing that residents were being poisoned, and was alleged to have hidden the full extent of the dangers and to have failed to take remedial actions.

\*299 Appeal from the United States District Court for the Eastern District of Michigan at Ann Arbor. No. 5:17-cv-10342—Judith E. Levy, District Judge.

**Attorneys and Law Firms**

ARGUED: [Charles E. Barbieri](#), FOSTER, SWIFT, COLLINS & SMITH, P.C., Lansing, Michigan, for Appellants Busch, Cook, Prysby, and Wurfel. [Margaret A. Bettenhausen](#), OFFICE OF THE MICHIGAN ATTORNEY GENERAL, Lansing, Michigan, for Appellants Snyder, Dillon, and Whitmer. [Christopher J. Marker](#), O'NEIL, WALLACE & DOYLE, P.C., Saginaw, Michigan, for Appellant Glasgow. [Edwar A. Zeineh](#), LAW OFFICE OF EDWAR A. ZEINEH, Lansing, Michigan, for Appellant Johnson. Renner K. Walker, LEVY KONIGSBERG LLP, New York, New York, for Appellees. ON BRIEF: [Charles E. Barbieri](#), [Allison M. Collins](#), FOSTER, SWIFT, COLLINS & SMITH, P.C., Lansing, Michigan, [Jay M. Berger](#), [Michael J. Pattwell](#), CLARK HILL PLC, Lansing, Michigan, [Philip A. Grashoff, Jr.](#), SMITH HAUGHEY RICE & ROEGGE, Grand Rapids, Michigan, for Appellants Busch, Cook, Prysby, and Wurfel. [Margaret A. Bettenhausen](#), [Richard S. Kuhl](#), [Nathan A. Gambill](#), OFFICE OF THE MICHIGAN ATTORNEY GENERAL, Lansing, Michigan, for Appellants Snyder, Dillon, and Whitmer. [Christopher J. Marker](#), O'NEIL, WALLACE & DOYLE, P.C., Saginaw, Michigan, [William Y. Kim](#), CITY OF FLINT, Flint, Michigan, [Edwar A.](#)

[Zeineh](#), LAW OFFICE OF EDWAR A. ZEINEH, Lansing, Michigan, [Frederick A. Berg, Jr.](#), BUTZEL LONG, P.C., Detroit, Michigan, [Sheldon H. Klein](#), BUTZEL LONG, P.C., Bloomfield Hills, Michigan, [Todd R. Perkins](#), THE PERKINS LAW GROUP PLLC, Detroit, Michigan, [Alexander S. Rusek](#), WHITE LAW, PLLC, Okemos, Michigan, [Barry A. Wolf](#), Flint, Michigan, for Appellants City of Flint, Croft, Johnson, Glasgow, Earley, and Ambrose. [James W. Burdick](#), BURDICK LAW, P.C., Bloomfield Hills, Michigan, [James A. Fajen](#), FAJEN AND MILLER, PLLC, Ann Arbor, Michigan, for Appellant Rosenthal. Renner K. Walker, [Corey M. Stern](#), LEVY KONIGSBERG LLP, New York, New York, [Hunter J. Shkolnik](#), NAPOLI SHKOLNIK PLLC, New York, New York, for Appellees.

Before: [MOORE](#), [SUTTON](#), and [WHITE](#), Circuit Judges. [MOORE](#), J., delivered the opinion of the court in which [SUTTON](#) and [WHITE](#), JJ., joined. [SUTTON](#), J. (pp. 304–06), delivered a separate concurring opinion.

**OPINION****KAREN NELSON MOORE.**

\*300 This is an appeal from one of the many strands of the Flint Water Crisis litigation. The City of Flint and City and State officials (collectively, “Defendants-Appellants”) allegedly caused, sustained, and covered up the poisoning of the people of Flint.<sup>1</sup> As Defendants-Appellants for the most part concede that our prior decisions control the outcome of this case, we dispose of their appeal in short order.

LeeAnne Walters and Marlana Sirls (collectively, “Plaintiffs-Appellees”) are part of the coordinated stream of Flint Water Crisis cases brought by individual plaintiffs. *See In re Flint Water Cases (Sirls et al. v. Michigan et al.)*, No. 5:17-cv-10342-JEL-EAS, 2019 WL 3530874, at \*1 (E.D. Mich. Aug. 2, 2019). “Counsel for the plaintiffs in these cases were selected as coliaison lead counsel.” *Id.* On December 15, 2017, counsel filed a “Master Complaint” on the *Walters* docket that contained all the various allegations and claims made by plaintiffs across the coordinated litigation. *See* R. 115 (*Walters* Docket, No. 5:17-cv-10164-JEL-MKM, Master Compl.) (Page ID #1367). Walters and Sirls then filed “short-form” complaints \*301 on their individual dockets on February 1, 2018, charting out the components of the Master Complaint that they were adopting as their own, including named defendants, alleged injuries, and claims. *See* R. 72 (*Sirls* Docket, No. 5:17-cv-10342-JEL-EAS, Short-Form

Compl.) (Page ID #691); R. 124 (*Walters* Docket, No. 5:17-cv-10164-JEL-MKM, Short-Form Compl.) (Page ID #1674). After defendants filed motions to dismiss, plaintiffs moved for leave to amend the Master Complaint, and defendants responded to plaintiffs' motion for leave to amend. See *Sirls*, 2019 WL 3530874, at \*1–2. The district court assessed the motions to dismiss and the motion to amend simultaneously and rendered “a single omnibus decision” on August 2, 2019. *Id.* at \*2.<sup>2</sup> The district court granted in part and denied in part both sets of motions and adopted aspects of the proposed amended Master Complaint as the operative complaint. *Id.* Defendants-Appellants timely appeal from that decision.

Defendants-Appellants in this case are largely the same as those that were parties in *In re Flint Water Cases* (*Waid v. Snyder*), 960 F.3d 303 (6th Cir. 2020).<sup>3</sup> There are no new Defendants-Appellants in this case, the claims at issue are the same, and we again take their appeal at the motion-to-dismiss stage.<sup>4</sup> In *Waid*, we decided that the same City and State officials who are Defendants-Appellants in this case plausibly violated plaintiffs' substantive due process right to bodily integrity and are not entitled to qualified immunity. *Id.* at 311. We additionally rejected the City of Flint's and Governor Whitmer's arguments that the Eleventh Amendment requires their dismissal from the case. *Id.* The full court denied *en banc* rehearing of *Waid* on July 14, 2020. *Waid v. Snyder*, No. 19-1472, slip op. (6th Cir. July 14, 2020) (order). At oral argument in this case, all but one Defendant-Appellant conceded that *Waid* controls our outcome here.<sup>5</sup>

[1] The outlier is Daugherty Johnson, who encourages us to view the allegations against him in this case differently than those levied against him in *Waid*. When pressed at oral argument for any meaningful distinctions between the two sets of allegations, Johnson tried to re-direct our focus to the plausibility of the pleadings. Yet, Johnson acknowledged that, like in *Waid*, the allegations here include that he pressured Michael Glasgow to go forward with the switch to Flint River water even \*302 though the water treatment plant was not ready. See *Waid*, 960 F.3d at 326. And Johnson acknowledged that, like in *Waid*, he is alleged to have stonewalled the county health department's attempt to investigate water-quality issues. See *id.* In light of these key similarities, which formed the basis for our decision with respect to Johnson in *Waid*, we conclude that there is no reason to treat Johnson any differently under the facts alleged in this case.

Separately, Plaintiffs-Appellees, like the plaintiffs in *Waid*, ask that we remand for the district court to decide whether former State Treasurer Andy Dillon should be dismissed in light of the district court's decision in *Brown v. Snyder* (*In re Flint Water Cases*), No. 18-cv-10726, 2020 WL 1503256, at \*9 (E.D. Mich. Mar. 27, 2020). See *Waid*, 960 F.3d at 332. We see no issue with doing so.

[2] Finally, we find it necessary to address the concurrence's criticisms of *Waid*—a stand it takes today after no judge of this court requested a poll for *en banc* rehearing of that case. See *Waid v. Snyder*, No. 19-1472, slip op. (6th Cir. July 14, 2020) (order). *Guertin v. Michigan*, in the concurrence's view, requires that we treat higher-ups differently than officials making decisions on the ground. See 912 F.3d 907 (6th Cir. 2019), *cert. denied*, — U.S. —, 140 S. Ct. 933, 205 L.Ed.2d 522 (2020). According to the concurrence, the majority in *Waid* skirted that command. That is the wrong reading of the law and a faithless reading of the facts.

In theory, the concurrence knows that our job is to “closely examine[ ] the culpability of each defendant to see if they ‘personally’ committed the sort of ‘conscience-shocking’ conduct required to sustain a substantive due process claim.” Conc. Op. at 304 (quoting *Guertin*, 912 F.3d at 929). Yet, the concurrence quickly tumbles out of that test and into one of its own devising: if officials lower in the state-wide hierarchy are entitled to immunity, then higher-up government officials are entitled to immunity, too, according to the concurrence.

The concurrence points out that three high-ranking officials—Daniel Wyant (Director of the Michigan Department of Environmental Quality (“MDEQ”)), Nick Lyon (Director of the Michigan Department of Health and Human Services (“MDHHS”)), and Eden Wells (Chief Medical Executive at the MDHHS)—were dismissed in *Guertin* because they “were too far removed from the relevant conduct to justify allowing the claims against them to proceed.” Conc. Op. at 304–05. The *Guertin* court held that these officials were not among the “chief architects” of the crisis and, accordingly, that they were entitled to immunity. 912 F.3d at 926. *Snyder*, the concurrence contends, is similarly situated.<sup>6</sup>

Wyant is a conceivable comparator for *Snyder*. As director of the MDEQ, Wyant in all likelihood was aware of the crisis and should have done more to stop it. The *Guertin* court dismissed Wyant, however, because the complaint did not ground the theoretical in particular allegations. “Plaintiffs d[id] not plausibly allege Wyant *personally* made decisions

regarding the water-source switch, nor d[id] they allege he *personally* engaged in any other [conscience-shocking] conduct.” *Id.* at 929 (emphasis added). And, of course, Wyant could be held accountable only “for his own conduct, \*303 not the misconduct of his subordinates.” *Id.* For those reasons, the *Guertin* plaintiffs’ claims against Wyant failed. As for Lyon and Wells, the MDHHS was responding to the health crisis created by other State and City officials. Thus, the involvement of the MDHHS, the department that Lyon and Wells oversaw, was attenuated to begin with. In the end, the *Guertin* court not only dismissed Lyon and Wells, but two lower-level MDHHS employees as well. *See id.* at 929–32.

The concurrence concedes that Snyder, unlike the high-ranking officials dismissed in *Guertin*, was personally involved in the decision to switch Flint’s water supply, and that Snyder knew that there was no plan to update Flint’s water treatment plant to make Flint River water safe. “But,” according to the concurrence, “the complaint nowhere alleges that [Snyder] knew or should have known about the risk that move posed.” Conc. Op. at 305. That assertion is hard to fathom.

The plaintiffs in *Waid* alleged that Snyder personally helped coordinate the City of Flint’s switch from clean Detroit Water and Sewerage Department (“DWSD”) water to contaminated Flint River water. *Waid*, 960 F.3d at 330. Plaintiffs-Appellees additionally allege in this case that Snyder not only knew that the Flint River would serve as an interim water source, but that he knew that there were no plans to update the water plant so that it could treat the water properly before making the switch. R. 185-2 (Am. Master Compl. at 35–36, ¶ 109) (Page ID #5076–77). Moreover, Snyder’s staff were told that “the ‘expedited timeframe’ for switching to Flint River water ‘is less than ideal and could lead to some big potential disasters down the road.’ ” *Waid*, 960 F.3d at 314 (internal quotation omitted).

Soon after the switch went into effect, General Motors cut off the Flint River water supply to its engine plant because the water’s chloride levels were so dangerously high that it would corrode the machinery. *Id.* at 315–16. In response, a member of Snyder’s executive staff sent an email to the full team about comments in the media about Flint residents being treated as “lab rats,” and fretting that it might come out that the chemical composition of the water “exceeded health-based water quality standards.” *Id.* at 316 (internal quotation omitted). To prevent the crisis from snowballing, the executive staff member recommended that the Governor’s

office ask the Emergency Manager for the City of Flint to switch Flint back to its prior source of clean water, the DWSD. *Id.* Snyder’s legal counsel agreed that the Flint River water issues were “ ‘downright scary’ ” and “advised that, ‘[t]hey should try to get back on the [DWSD] system as a stopgap ASAP before this thing gets too far out of control.’ ” *Id.* (internal quotation omitted).

Yet, Snyder did not try to switch Flint back to clean water, or to mitigate the crisis with protective equipment, or to acknowledge the dangers of the water contamination—even after his own chief of staff told him that “[t]he water issue continues to be a danger flag.” *See id.* at 318 (internal quotation omitted). Snyder’s office, instead, coordinated with the MDEQ Director of Communications to create political cover for Snyder’s administration. *See id.* Snyder’s office also managed to supply water coolers for State buildings, while refusing to distribute water filters to the people of Flint. *See id.* at 330. From these allegations, it is hard to imagine that Snyder was kept in the dark about the cause and extent of the crisis, and we do not hesitate to conclude that Snyder’s alleged personal actions demonstrated deliberate indifference.

\*304 But the concurrence’s point is not really about the plausibility of the pleadings or whether Snyder’s conduct shocks the conscience. The concurrence’s real issue with *Waid* is that we refused to dismiss a high-ranking government official from the litigation. To escape the *Waid* plaintiffs’ and Plaintiffs-Appellees’ extensive and personal allegations against former Governor Snyder, the concurrence peddles a new appendage to qualified-immunity doctrine that effectively would grant high-ranking officials absolute immunity regardless of the allegations. Snyder, in the concurrence’s view, simply is too high-up to be accountable. Perhaps that would be the case for a different governor in a different set of circumstances. Perhaps that would be the winning argument had the crisis in Flint not been under the public eye and had it not been orchestrated and debated at the highest levels of state government. But on these facts, Snyder is alleged to have coordinated the switch to Flint River water knowing that the water would not be treated for contamination. Snyder is alleged to have refused to switch Flint back to clean water, knowing that the people of Flint were being poisoned. Snyder is alleged to have hidden the full extent of the dangers and to have failed to take remedial actions. On these facts, Snyder is named to defend his own actions, not those of his subordinates, and there is no basis for dismissing him from this case. Thus, although we fail to see

why saying so is relevant to the task at hand, we stand by our decision in *Waid*.

In conclusion, we hold that *Waid* controls our outcome here and accordingly **AFFIRM** the district court's denial of the motions to dismiss as to all Defendants-Appellants except former Treasurer Dillon. We **REMAND** for the district court to decide in the first instance whether Dillon should be dismissed in light of the district court's decision in *Brown*, 2020 WL 1503256, at \*9.

SUTTON, Circuit Judge, concurring.

### CONCURRENCE

Just months ago, we decided *Carthan*, since recaptioned *Waid*, a case involving the same parties pressing the same claims in the same motion-to-dismiss posture. See *In re Flint Water Cases (Waid)*, 960 F.3d 303 (6th Cir. 2020). A majority rejected each defendant's arguments for dismissal. *Id.* at 311. When it comes to resolving this functionally identical dispute, we must follow the same path. That's why I concur.

But in my respectful view, *Waid* erred in allowing claims to proceed against Governor Rick Snyder and Treasurer Andy Dillon. *Waid* came after our first Flint water case, *Guertin v. State*, 912 F.3d 907 (6th Cir. 2019), which allowed substantive due process claims to proceed against several Michigan officials for their role in the crisis. Right or wrong, *Guertin* did not casually assign blame to all named parties. It closely examined the culpability of each defendant to see if they “personally” committed the sort of “conscience-shocking” conduct required to sustain a substantive due process claim. *Id.* at 929. Of the twelve defendants named in *Guertin*, the court allowed claims to proceed against seven. See *id.* at 916. It dismissed the claims against the remaining five. See *id.*

*Guertin* dismissed claims against three high-ranking officials: Daniel Wyant (Director of the Michigan Department of Environmental Quality), Nick Lyon (Director of the Michigan Department of Health and Human Services), and Eden Wells (Chief Medical Executive at the Department of Health and Human Services). See *id.* at 916, 927, 929–31. In each case, it concluded that the officials were too far removed from the relevant conduct to justify allowing \*305 the claims against them to proceed. See *id.* at 929–31. Wyant, it's true, “was aware of some of the issues arising with the water supply.” *Id.* at 929. And he admitted to his department's “colossal failure”

to act. *Id.* But while the conduct of certain “individuals within his department was constitutionally abhorrent,” we found the bulk of the responsibility lay with them. *Id.* And we declined to “hold [him] accountable for ... the[ir] misconduct” despite some knowledge of the danger posed. *Id.*

Lyon and Wells, for their part, were “unjustifiably skeptical” of a study showing the danger posed by the water supply. *Id.* at 930. Lyon “tr[ie]d to discredit [the] study despite his own department's knowledge that it show[ed] a real problem.” *Id.* (quotation omitted). And Wells “discourag[ed] her department [from] look[ing] further” into the study and directed resources toward undermining its conclusions. *Id.* (quotation omitted). Both of those defendants engaged in affirmative conduct that worsened the water crisis. Even so, *Guertin* held, that action “f[ell] well-short of conscience-shocking conduct.” *Id.* at 930–31. Too much separation existed between Lyon and Wells and the “chief architects” of the crisis to hold them to account. *Id.* at 926.

Held to these yardsticks, I cannot see a basis for denying qualified immunity to Governor Snyder. See *Waid*, 960 F.3d at 336, 338 (Murphy, J., concurring in the judgment in part and dissenting in part). No doubt, he “was personally involved in the decisional process which led to the transition” to the contaminated water supply. R. 620-3 at 47 (19-1425). But the complaint nowhere alleges that he knew or should have known about the risk that move posed. The plaintiffs argue that (1) Snyder's “senior executive staff was immediately aware” of issues with the water supply and actively concealed it, *id.* at 65; and (2) Snyder waited too long to declare a state of emergency after hearing troubling reports, *id.* at 151. But *Guertin* forecloses both theories. See *Waid*, 960 F.3d at 338 (Murphy, J., concurring in the judgment in part and dissenting in part). It rejected efforts to impute liability from subordinates to their supervisors. *Id.* And it clarified that high officials resisting early evidence of danger cannot make out a constitutional violation. *Id.* Snyder seems *less* like the scheme's “chief architect[ ]” than Wyant, Lyon, and Wells. *Id.* at 336 (quotation omitted). As Judge Murphy pointed out in his partial dissent in *Waid*: “If Snyder's *subordinates* were too far removed from the crisis to remain defendants, that fact should make us think twice before allowing claims to proceed against an official even further removed.” *Id.* at 337. Snyder played only an attenuated, supervisory role. And while he evidently delayed acting on information, that failure by itself does not give rise to liability. See *id.* at 338.

The plaintiffs no doubt allege that Snyder knew there was not yet an “agreed upon plan in place” for upgrading Flint’s water treatment plant when he “authorized the switch to the Flint River.” R. 185-2 at 34–36 (19-2000). But that hardly suggests that he foresaw that the water “would not be treated for contamination,” Maj. Op. at 304, when the switch actually happened a year later, let alone that he inferred that the switch posed a “substantial risk of serious harm.” *Waid*, 960 F.3d at 336 (Murphy, J., concurring in the judgment in part and dissenting in part) (quotation omitted).

An even weaker case stands against Treasurer Dillon. *See id.* at 338–39. He played a role in the original negotiations that caused Flint to switch to the contaminated water supply. And, yes, he may have \*306 received certain emails discussing complications with the transition to the use of the Flint River water. But none of the emails suggests he knew or should have known of the health risk. Still less do they show that he could

have done anything to stop it. The majority would leave the choice whether to dismiss to the district court. I would stop the litigation here and now.

I do not doubt that Governor Snyder and Treasurer Dillon could have done more to avert the contamination of Flint’s water supply. Not every mistake in governing, however, amounts to a substantive due process violation. To survive a motion to dismiss, the plaintiffs must plead facts indicating that the officials’ conduct “shocks the conscience.” *Id.* at 336 (quotation omitted). On my reading, neither Governor Snyder nor Treasurer Dillon reached that inglorious low. If I had *Waid* before me, I would dissent on that basis. Because I don’t, I concur.

#### All Citations

969 F.3d 298

#### Footnotes

- 1 The Defendants-Appellants party to this appeal are: Darnell Earley, Gerald Ambrose, Howard Croft, Michael Glasgow, Daugherty Johnson, the City of Flint, Richard Dale Snyder (former Governor of Michigan), Andy Dillon (former Treasurer of Michigan), Gretchen Whitmer (present Governor of Michigan), Stephen Busch, Patrick Cook, Michael Prysby, Bradley Wurfel, and Adam Rosenthal.
- 2 The district court followed the same procedure in this case that it had in the underlying decision in *In re Flint Water Cases (Waid v. Snyder)*, 960 F.3d 303 (6th Cir. 2020). We noted in *Waid* that we approved the district court’s omnibus approach in *Waid v. Snyder*, No. 18-1967, slip op., 2019 WL 4121023 (6th Cir. Feb. 19, 2019) (order). *See Waid*, 960 F.3d at 321–22 n.6.
- 3 One other defendant, Liane Shekter-Smith, was a party to the appeal in *Waid*, 960 F.3d 303. Plaintiffs’ claims against Shekter-Smith were dismissed by the district court in this case as time-barred. *See Sirls*, 2019 WL 3530874, at \*11–13.
- 4 One difference, we note, is that Plaintiffs-Appellees in this case allege that they were injured by lead-poisoning only, and not *legionella*. *See* R. 72 (*Sirls* Docket, No. 5:17-cv-10342-JEL-EAS, Short-Form Compl. at 5) (Page ID #695); R. 124 (*Walters* Docket, No. 5:17-cv-10164-JEL-MKM, Short-Form Compl. at 5) (Page ID #1678). The parties, however, do not assign any significance to that difference for purposes of this appeal.
- 5 Our decision in *Waid*, in turn, primarily rested upon *Guertin v. Michigan*, 912 F.3d 907 (6th Cir. 2019), *cert. denied*, — U.S. —, 140 S. Ct. 933, 205 L.Ed.2d 522 (2020), and *Boler v. Earley*, 865 F.3d 391, 412–13 (6th Cir. 2017). *Waid* is our most comprehensive decision to date, in that it addressed allegations against Glasgow, Johnson, Rosenthal, Cook, Snyder, and Dillon (all of whom were not parties in *Guertin*).
- 6 Andy Dillon’s conduct is not worth debating. The parties in *Waid* agreed that it was appropriate to remand the claims against Dillon to the district court to decide whether to dismiss Dillon in light of the district court’s decision to dismiss him in a related case. *See* 960 F.3d at 332. That is what we did in *Waid*, and we do the same here.

960 F.3d 820

United States Court of Appeals, Sixth Circuit.

IN RE: FLINT WATER CASES.

Luke Waid, Parent and Next-Friend  
of SR, a minor; et al., Plaintiffs,  
Elnora Carthan, et al., Plaintiffs-Appellees,  
v.

Darnell Earley, et al., Defendants,  
Richard Dale Snyder, former Governor of  
Michigan; [Andy Dillon](#), former Treasurer  
of Michigan, Defendants-Appellants,  
Veolia North America, Inc., Veolia  
North America, LLC, Veolia North  
America Operating Services,  
LLC, Intervenors-Appellees.

No. 20-1352

Decided and Filed: June 2, 2020

**Synopsis**

**Background:** In consolidated putative class action relating to injuries from city's corroded water, former Governor and former State Treasurer, whose dismissal motion based on qualified immunity had been denied as to § 1983 substantive due process claim for violation of bodily integrity, requested protective order with respect to plaintiffs and private-party defendants taking depositions of them as non-party fact witnesses for separate claims against other defendants. The United States District Court for the Eastern District of Michigan, [Judith E. Levy, J.](#), denied the request. Former Governor and former Treasurer filed interlocutory appeal and filed motion for stay pending appeal

**Holdings:** The Court of Appeals, [Moore](#), Circuit Judge, held that:

[1] former Governor and former Treasurer were not likely to succeed on merits of appeal from denial of protective order;

[2] former Governor and former Treasurer would not suffer irreparable harm absent a stay;

[3] plaintiffs would be harmed by a stay;

[4] public interest weighed against a stay; and

[5] non-final order denying protection from discovery was not immediately appealable under collateral order doctrine.

Motion denied; appeal dismissed.

West Headnotes (15)

[1] **Federal Courts**  **Supersedeas or Stay of Proceedings**

Four interrelated factors are balanced when considering whether to grant a stay pending appeal: (1) the likelihood that the party seeking the stay will prevail on the merits of the appeal; (2) the likelihood that the party seeking the stay will be irreparably harmed absent a stay; (3) the prospect that others will be harmed if the court grants the stay; and (4) the public interest in granting the stay.

[1 Cases that cite this headnote](#)

[2] **Federal Courts**  **Injunction and temporary restraining order cases**

Former Governor and former State Treasurer were not likely to succeed on merits, as factor weighing against stay pending appeal, of their appeal from denial of order for protection, in consolidated putative class action relating to city's corroded water, from depositions of them as non-party fact witnesses with respect to separate claims against other defendants, in which action their dismissal motion based on qualified immunity had been denied as to § 1983 substantive due process claim for violation of bodily integrity; former Governor and former Treasurer incorrectly claimed that they could not be deposed on any matter pending resolution of their qualified-immunity appeal. *U.S. Const. Amend. 14; 42 U.S.C.A. § 1983.*

- [3] **Federal Civil Procedure** — Persons subject  
**Federal Civil Procedure** — Proceedings to obtain

Civil rights actions brought under § 1983 play out in stages in order to shield government officials from the burdens of litigation, and if an official files a motion to dismiss based on qualified immunity, the court must stay discovery until that issue is decided, but if the official is denied qualified immunity on the motion to dismiss, then the plaintiff ordinarily will be entitled to some discovery. [42 U.S.C.A. § 1983](#).

- [4] **Public Employment** — Qualified immunity  
**Public Employment** — Trial, judgment, and relief

Qualified immunity protects government officials only from unnecessary and burdensome discovery or trial proceedings, because qualified immunity is a right to immunity from certain claims, not from litigation in general, and granting qualified immunity on only one of the claims may reduce discovery but it does not eliminate it.

- [5] **Federal Courts** — Depositions and discovery

Discovery rulings, no doubt, are high stakes, but the Court of Appeals usually leaves decisions on how best to manage discovery to the District Court's discretion.

- [6] **Federal Civil Procedure** — Persons subject

It is up to the District Court to take qualified immunity for public officials into account when developing its discovery plan, in order to protect the substance of the qualified immunity defense.

- [7] **Public Employment** — Qualified immunity

Qualified immunity for public officials grants immunity from suit rather than a mere defense to liability.

- [8] **Federal Courts** — Injunction and temporary restraining order cases

Former Governor and former State Treasurer would not suffer irreparable harm absent a stay, as factor weighing against a stay pending their appeal from denial of order for protection, in consolidated putative class action relating to city's corroded water, from depositions of them as non-party fact witnesses with respect to separate claims against other defendants, in which action their dismissal motion based on qualified immunity had been denied as to § 1983 substantive due process claim for violation of bodily integrity; even if former Governor and former Treasurer ultimately were granted qualified immunity, plaintiffs and private-party defendants would still request the depositions because, in their view, the former officials were key factual witnesses regarding other claims. [U.S. Const. Amend. 14](#); [42 U.S.C.A. § 1983](#).

[1 Cases that cite this headnote](#)

- [9] **Federal Courts** — Injunction and temporary restraining order cases

Harm to plaintiffs from issuance of a stay was a factor weighing against a stay pending appeal by former Governor and former State Treasurer from denial of order for protection, in consolidated putative class action relating to city's corroded water, from depositions of them as non-party fact witnesses with respect to separate claims against other defendants, in which action their dismissal motion based on qualified immunity had been denied as to § 1983 substantive due process claim for violation of bodily integrity; plaintiffs alleged ongoing serious health injuries that continued to worsen over time, so a delay in the litigation could have a real effect on their ability to secure a meaningful remedy. [U.S. Const. Amend. 14](#); [42 U.S.C.A. § 1983](#).

- [10] **Federal Courts** — Injunction and temporary restraining order cases

The public interest in developing the facts and resolving the case expeditiously was a factor weighing against a stay pending appeal by former Governor and former State Treasurer from denial of order for protection, in consolidated putative class action relating to city's corroded water, from depositions of them as non-party fact witnesses with respect to separate claims against other defendants, in which action their dismissal motion based on qualified immunity had been denied as to § 1983 substantive due process claim for violation of bodily integrity. *U.S. Const. Amend. 14*; *42 U.S.C.A. § 1983*.

appealable under collateral order doctrine, on interlocutory appeal by former Governor and former State Treasurer from denial of order for protection, in consolidated putative class action relating to city's corroded water, from depositions of them as non-party fact witnesses with respect to separate claims against other defendants, in which action their dismissal motion based on qualified immunity had been denied as to § 1983 substantive due process claim for violation of bodily integrity. *U.S. Const. Amend. 14*; *28 U.S.C.A. § 1291*; *42 U.S.C.A. § 1983*.

**[11] Federal Courts** 🔑 **Interlocutory and Collateral Orders**

When a party appeals something other than the last order possible to be made in a case, a decision of a district court is appealable under the collateral order doctrine if it falls within the small class of decisions that finally determine claims of right separable from, and collateral to, rights asserted in the action, too important to be denied review and too independent of the cause itself to require that appellate consideration be deferred until the whole case is adjudicated. *28 U.S.C.A. § 1291*.

**[12] Federal Courts** 🔑 **Immunity**

The collateral order doctrine entitles government officials to an immediate appeal from a non-final order denying qualified immunity from civil liability. *28 U.S.C.A. § 1291*.

**[13] Federal Courts** 🔑 **Preliminary proceedings; depositions and discovery**

Discovery orders generally are non-final, non-appealable orders. *28 U.S.C.A. § 1291*.

**[14] Federal Courts** 🔑 **Immunity**

Non-final order denying protection from discovery could not be construed as a denial of qualified immunity that would be immediately

**[15] Federal Courts** 🔑 **Immunity**

There are two appeals based upon a public official's claim of qualified immunity from civil liability that can be taken under the collateral order doctrine prior to final judgment: first, after denial of a motion to dismiss on the pleadings, and second, after denial of a motion for summary judgment following discovery. *28 U.S.C.A. § 1291*; *Fed. R. Civ. P. 12, 56*.

\*823 Appeal from the United States District Court for the Eastern District of Michigan at Ann Arbor. Nos. 5:16-cv-10444; 5:16-cv-11247—Judith E. Levy, District Judge.

**Attorneys and Law Firms**

ON MOTIONS: [Richard S. Kuhl](#), [Margaret Bettenhausen](#), [Nathan A. Gambill](#), OFFICE OF THE MICHIGAN ATTORNEY GENERAL, Lansing, Michigan, for Appellants. ON RESPONSE: [Emmy L. Levens](#), COHEN MILSTEIN SELLERS & TOLL PLLC, Washington, D.C., [Paul Novak](#), [Gregory Stamatopoulos](#), WEITZ & LUXENBERG, P.C., Detroit, Michigan, for Plaintiffs-Appellees. [James M. Campbell](#), CAMPBELL CONROY & O'NEIL, P.C., Boston, Massachusetts, for Veolia Appellees.

Before: [MERRITT](#), [MOORE](#), and [MURPHY](#), Circuit Judges.

**OPINION**

KAREN NELSON MOORE, Circuit Judge.

Former State of Michigan Governor Richard Dale Snyder and former State Treasurer Andy Dillon claim that they cannot be deposed as non-party fact witnesses with respect to claims against other defendants in the litigation stemming from the Flint Water Crisis. In their view, they are immune from all discovery until they have exhausted every opportunity for appeal from the district court's denial of their motions to dismiss based on qualified immunity. Meanwhile, other defendants and certain plaintiffs are pursuing discovery on wholly separate claims and have noticed Snyder and Dillon for non-party fact witness depositions. Snyder and Dillon moved for a protective order in the district court to stop the depositions from going forward. When their request was denied, they appealed the district court's discovery order to this court and shortly thereafter requested a stay of the depositions until we resolve their appeal from the denial of a protective order. We **DENY** Snyder's and Dillon's request for a stay of non-party depositions pending resolution of their appeal from the district court's order denying their request for a protective order, and we **DISMISS** for lack of jurisdiction their appeal from the denial of a protective order.

## I. BACKGROUND

This appeal derives from the consolidated putative class action in the *In re Flint Water Cases* litigation.<sup>1</sup> It is one of dozens of ongoing actions brought by individuals, businesses, and putative classes in state and federal court challenging the actions of state and private actors in creating, sustaining, and covering up the Flint Water Crisis. Defendants include government officials from the State of Michigan, the City of Flint, and state agencies. R. 620-3 (Fourth Am. Compl. at 1–2, ¶ 2) (Page ID #17804–05). Defendants also include private \*824 engineering companies like Veolia that are facing claims of professional negligence for failing to explain the need to treat the water properly for corrosion and for lying to the public about the existence and extent of the crisis. *Id.* at 1–3, ¶ 2 (Page ID #17804–06). While government officials like Snyder and Dillon have been litigating the issue of qualified immunity, discovery against private parties like Veolia has proceeded.

On April 1, 2019, the district court ruled on the defendants' motions to dismiss. R. 798 (Op. & Order) (Page ID #21103). The district court granted the government officials' motions

to dismiss plaintiffs' claims alleging 42 U.S.C. § 1983 equal-protection violations, § 1985(3) conspiracy, Michigan's Elliott Larsen Civil Rights Act (“ELCRA”), § 1983 state-created danger, and gross negligence. *Id.* at 128 (Page ID #21230). The district court denied, however, defendants' motions to dismiss plaintiffs' § 1983 bodily-integrity claim on the bases of qualified and absolute immunity. *Id.* at 127 (Page ID #21229). Thus, the only surviving claim against the state defendants, including Snyder and Dillon, is plaintiffs' bodily-integrity claim.

After deciding defendants' motions to dismiss based on qualified immunity, the district court entered a comprehensive case management order (“CMO”) on April 30, 2019, to direct the course of discovery. R. 827 (Case Management Order 4/30/19) (Page ID #22804). The order distinguished between discovery as to parties and non-parties. *See id.* Then, on May 20, 2019, the district court ruled on the state defendants' motion for a stay of discovery pending final resolution of their motions to dismiss based on qualified immunity. R. 861 (Discovery Order 5/20/19) (Page ID #23407).

As context, the district court noted in its May 20, 2019 discovery order that other defendants with no claim to immunity had begun discovery pursuant to the CMO. *Id.* at 1, 7–8 (Page ID #23407, 23413–14). The state defendants, however, sought a stay of “*all* discovery across the Flint Water Cases until their claims of immunity have been decided by this Court, the Sixth Circuit, and the United States Supreme Court, if necessary.” *Id.* at 1–2 (Page ID #23407–08) (emphasis added). The district court granted in part and denied in part the state defendants' request. *Id.* at 2 (Page ID #23408).

The district court recognized that the state defendants must be treated as though they are immune from the claims brought against them until they have exhausted their opportunities to appeal the district court's denial of their motions to dismiss based on immunity. *Id.* at 6–7 (Page ID #23412–13). Accordingly, the district court issued a stay with respect to “discovery on claims for which they continue to litigate the issue of immunity.” *Id.* at 2 (Page ID #23408). Thus, the court ruled, “the state and MDEQ defendants will not be subjected to discovery with respect to the sole allegation against them, which is that they violated plaintiffs' right to bodily integrity, until they have exhausted their opportunities to pursue their qualified immunity claim on appeal.” *Id.* at 6–7 (Page ID #23412–13).

The state defendants' request for a stay of discovery was partly denied in the sense that the state defendants would "be treated as non-parties pending the outcome of their qualified immunity appeals." *Id.* at 5 (Page ID #23411). That meant that they could be subject to discovery requests only as non-party fact witnesses regarding wholly separate claims against other defendants. The district court explained that, "[i]f the state and MDEQ defendants are eventually dismissed as a result of their pending appeals, they will still be required \*825 to respond to discovery as a non-party." *Id.* at 8 (Page ID #23414). Discovery from the state defendants as non-party fact witnesses therefore was "inevitable." *Id.*

Eventually, Snyder, Dillon, and other state defendants received deposition notices from the Veolia defendants and certain plaintiffs. The state defendants promptly moved for a protective order in the district court to stay non-party fact witness depositions until after they exhausted their appeals from the denial of their motions to dismiss on the issue of qualified immunity. *See* R. 1047 (Mot. for Protective Order) (Page ID #26634). The district court denied their request for a protective order and reiterated that they are required to comply with discovery requests as non-parties. R. 1100 (Protective Order Ruling 4/9/20 at 2) (Page ID #27458).

Presently, certain plaintiffs seek to depose former Governor Snyder starting on June 25, 2020, and the Veolia defendants seek to depose former Treasurer Dillon starting on July 7, 2020. When the district court denied their request for a protective order, Snyder and Dillon appealed the denial of a protective order to this court. They informed the district court of their appeal and obtained a statement from the district court that an additional request for a stay of the non-party depositions would be futile. R. 1130 (Conference 5/8/20 at 10–11) (Page ID #27846–47). They then requested a stay from us to stop the depositions from going forward while we decide their appeal from the district court's denial of their request for a protective order against taking the non-party depositions.

We note at the outset that we recently affirmed the district court's denial of qualified immunity as to Snyder, and we remanded for the district court to consider whether to dismiss Dillon in light of the district court's decision in *Brown v. Snyder (In re Flint Water Cases)*, No. 18-cv-10726, 2020 WL 1503256, at \*9 (E.D. Mich. Mar. 27, 2020). *In re Flint Water Cases (Waid v. Snyder)*, 960 F.3d 303, 311–12 (6th Cir. May 22, 2020). We recognize, as the district court did, that our resolution of the qualified immunity issue does not exhaust

Snyder's and Dillon's opportunities for review by the Supreme Court on the qualified immunity issue and, thus, does not render these proceedings moot.

## II. REQUEST FOR A STAY

[1] We have authority over Snyder's and Dillon's request for a stay of the district court's discovery ruling because they have shown that filing an initial motion for a stay in the district court would be futile. *See* FED. R. APP. P. 8(a). We balance four interrelated factors when considering whether to grant a stay: "(1) the likelihood that the party seeking the stay will prevail on the merits of the appeal; (2) the likelihood that the moving party will be irreparably harmed absent a stay; (3) the prospect that others will be harmed if the court grants the stay; and (4) the public interest in granting the stay." *Michigan State A. Philip Randolph Inst. v. Johnson*, 833 F.3d 656, 661 (6th Cir. 2016) (quoting *Mich. Coal. of Radioactive Material Users, Inc. v. Griepentrog*, 945 F.2d 150, 153 (6th Cir. 1991)).

[2] First, Snyder and Dillon are not likely to succeed on appeal from the district court's order denying their request for a protective order. They claim that they cannot be deposed on *any* matter pending resolution of their qualified-immunity appeal. That is incorrect.

[3] Snyder and Dillon stress that qualified immunity protects them from discovery until their claim of entitlement to immunity has been conclusively denied on \*826 their motions to dismiss. The Supreme Court requires that civil-rights actions brought under § 1983 play out in stages to shield government officials from the "burdens of litigation." *Kennedy v. City of Cleveland*, 797 F.2d 297, 299–300 (6th Cir. 1986) (citing *Mitchell v. Forsyth*, 472 U.S. 511, 527, 105 S.Ct. 2806, 86 L.Ed.2d 411 (1985)). If the defendant files a motion to dismiss based on qualified immunity, the court must "stay discovery until that issue is decided." *Id.* at 299. If the defendant is denied qualified immunity on the motion to dismiss, then "the plaintiff ordinarily will be entitled to some discovery." *Crawford-El v. Britton*, 523 U.S. 574, 598, 118 S.Ct. 1584, 140 L.Ed.2d 759 (1998).

[4] Yet, qualified immunity protects government officials from "unnecessary and burdensome discovery or trial proceedings" only. *Id.* at 597–98, 118 S.Ct. 1584; *see also Mitchell*, 472 U.S. at 526, 105 S.Ct. 2806 (explaining that qualified immunity relieves defendants of "the burdens of broad-reaching discovery" (quoting *Harlow v. Fitzgerald*,

457 U.S. 800, 817–18, 102 S.Ct. 2727, 73 L.Ed.2d 396 (1982)) (emphasis added)). The “right to immunity is a right to immunity *from certain claims*, not from litigation in general ....” *Behrens v. Pelletier*, 516 U.S. 299, 312, 116 S.Ct. 834, 133 L.Ed.2d 773 (1996) (emphasis added). “Granting qualified immunity on only one of the claims may reduce discovery but it does not eliminate it.” *McLaurin v. Morton*, 48 F.3d 944, 949 (6th Cir. 1995); see also *Alice L. v. Dusek*, 492 F.3d 563, 565 (5th Cir. 2007) (“To the extent that [the defendant] is subject to discovery requests on claims for which she does not or cannot assert qualified immunity, such discovery requests do not implicate her right to qualified immunity.”).

Here, the district court granted the state defendants effective immunity pending the final resolution of their motions to dismiss based on qualified immunity. In other words, the district court recognized that no discovery may be sought from the state defendants on the claims against them unless and until they are conclusively denied qualified immunity on their motions to dismiss. The district court carefully sculpted a discovery plan that afforded the state defendants their full entitlement to immunity, while permitting other parties to seek discovery from them as fact witnesses on wholly separate claims. The discovery plan would permit state defendants to be deposed as non-party fact witnesses to events regarding separate claims brought against different defendants to prevent the litigation from stalling out for *all* defendants during the pendency of *these state defendants’* appeals of the denial of their motions to dismiss based on qualified immunity. See R. 827 (Case Management Order 4/30/19 at 1) (Page ID #22804); R. 861 (Discovery Order 5/20/19 at 5, 8–10) (Page ID #23411, 23414–16). The state defendants obtained a broad stay from discovery that treats them as though they had already proven their immunity and were dismissed from the case. The district court’s exception to that stay was limited, and it was necessary for discovery to proceed for other parties in the sprawling litigation. Doing so was well within the district court’s discretion.

[5] [6] Discovery rulings, no doubt, are high stakes, but we usually leave decisions on how best to manage discovery to the district court’s discretion. See *Criss v. City of Kent*, 867 F.2d 259, 261 (6th Cir. 1988) (“[I]t is well established that the scope of discovery is within the sound discretion of the trial court.” (quotation omitted)). It is up to the district court to take qualified immunity into account when developing its discovery plan. See *Crawford-El*, 523 U.S. at 597–98, 118 S.Ct. 1584 (“[T]he trial court must exercise its discretion

in a way \*827 that protects the substance of the qualified immunity defense. It must exercise its discretion so that officials are not subjected to unnecessary and burdensome discovery or trial proceedings.”). And, in fact, the district court did so here.

The district court refrained from issuing its discovery plan until after it resolved defendants’ motions to dismiss asserting qualified immunity. See R. 827 (Case Management Order 4/30/19) (Page ID #22804); R. 861 (Discovery Order 5/20/19 at 5) (Page ID #23411). Then, recognizing that its ruling on qualified immunity was subject to appeal, the district court ordered a stay of discovery regarding the sole remaining claim against the state defendants—plaintiffs’ bodily integrity claim. R. 861 (Discovery Order 5/20/19 at 6–7) (Page ID #23412–13). “[T]he state and MDEQ defendants,” the district court ruled, “will not be subjected to discovery with respect to the sole allegation against them, which is that they violated plaintiffs’ right to bodily integrity, until they have exhausted their opportunities to pursue their qualified immunity claim on appeal.” *Id.* In the same order, the district court made a limited exception to the stay to permit discovery from state defendants as non-party fact witnesses to events relevant to entirely separate claims brought against different defendants. *Id.* at 7–8 (Page ID #23413–14). The district court explained that “[p]laintiffs have counts pending against other defendants that have filed answers and are ready to defend their positions.” *Id.* “It follows that the Court can order discovery to proceed with respect to these other defendants.” *Id.* at 8 (Page ID #23414).

[7] The key Supreme Court cases that Snyder and Dillon cite for us—*Mitchell*, *Crawford-El*, and *Harlow*—feature prominently in the district court’s order delineating the state officials’ discovery obligations. The district court recognized that *Mitchell* “established that qualified immunity grants ‘immunity from suit rather than a mere defense to liability.’” *Id.* at 6 (Page ID #23412) (quoting *Mitchell*, 472 U.S. at 526, 105 S.Ct. 2806). “[C]ourts must take care,” the district court wrote, “to ensure that government officials are not subjected to unnecessary and burdensome discovery until issues of immunity have been resolved at the earliest opportunity.” *Id.* (citing *Crawford-El*, 523 U.S. at 597, 118 S.Ct. 1584). Accordingly, the district court struck a balance between the discovery needs of other defendants and the state defendants’ immunity interest: “If the state and MDEQ defendants are eventually dismissed as a result of their pending appeals, they will still be required to respond to discovery as a non-party. So in the interim, this litigation will go forward and the state

and MDEQ defendants are required to respond to discovery requests as if they were already dismissed from the case.” *Id.* at 8 (Page ID #23414).<sup>2</sup>

\*828 We disagree with Snyder and Dillon that the district court’s “non-party” versus “party” distinction is meaningless, or that it permits an end-run around their entitlement to immunity. The district court was clear that no party may seek discovery from the state defendants on the particular claim that they continue to litigate with respect to immunity. *See id.* at 2 (Page ID #23408) (“The state and MDEQ defendants are entitled to a stay of discovery on claims for which they continue to litigate the issue of immunity.”). If these non-party depositions turn out to be a ruse—as Snyder and Dillon assert that they are—Snyder and Dillon are free to object and move for a protective order at the district court level as issues arise. It is inappropriate for us, however, to issue a prophylactic order to stop these depositions from going forward based on hypothetical horrors before a single problematic question has been asked.

For all these reasons, we conclude that Snyder and Dillon are not likely to succeed on their appeal from the district court’s order denying them a protective order.

[8] Second, and for the same reason, Snyder and Dillon will not suffer irreparable harm absent a stay. The district court forbade the noticing parties from using depositions to probe Snyder and Dillon regarding the sole surviving claim against them, which is that they violated plaintiffs’ right to bodily integrity. *See id.* at 5, 8–10 (Page ID #23411, 23414–16). Snyder and Dillon will not be effectively denied their “entitlement not to stand trial or face the other burdens of litigation” if we deny their request for a stay of the district court’s denial of their protective order pending their appeal from that denial. *Mitchell*, 472 U.S. at 526, 105 S.Ct. 2806. Even if Snyder and Dillon ultimately should be granted qualified immunity, the noticing parties would still request these depositions because, in their view, Snyder and Dillon are key factual witnesses regarding other claims. The discovery at issue is not only suitably tailored to the situation, but also inevitable.

We further emphasize that our decision does not leave Snyder and Dillon without a remedy. They may file for a protective order in the district court if they object to the noticing parties’ line of questioning. What they cannot do is ask us to resolve a run-of-the-mill discovery dispute on an interlocutory appeal.

For each of these reasons, Snyder and Dillon will not suffer any irreparable harm absent a stay of the district court’s discovery order.

[9] Third, the noticing parties will be harmed if we grant a stay. The plaintiffs have alleged ongoing serious health injuries that continue to worsen over time. *See In re Flint Water Cases (Waid v. Snyder)*, 960 F.3d 303, 320–21 (6th Cir. 2020). Thus, the progress of the litigation has a real effect on plaintiffs’ ability to secure a meaningful remedy. A delay also could interfere with the scheduled start of the bellwether trials, presently set to begin in January 2021. R. 1150 (Order 5/21/20 at 3) (Page ID #28167). A delay similarly would prejudice the Veolia defendants, who are also invested in the efficient resolution of this case.

[10] Finally, the public interest favors the development of the facts and the expeditious resolution of this case. And as described above, Snyder’s and Dillon’s immunity interest—and the public’s accompanying interest in their immunity—is not at stake in this limited non-party fact witness \*829 discovery. We conclude that all four factors weigh against granting a stay of the district court’s order allowing the non-party depositions to proceed. We accordingly **DENY** Snyder’s and Dillon’s motion for a stay of non-party depositions pending resolution of their appeal from the district court’s order denying their motion for a protective order.

### III. MOTION TO DISMISS

The Veolia Defendants-Appellees have filed a motion to dismiss this appeal No. 20-1352. They assert that we lack jurisdiction under either 28 U.S.C. § 1291 or the collateral order doctrine to review a discovery order. Snyder and Dillon have filed a response, contending that the district court’s discovery order is an implicit denial of their qualified immunity. We conclude that we do not have jurisdiction to entertain Snyder’s and Dillon’s appeal from the district court’s order denying their request for a protective order.

[11] [12] Section 1291 vests us with jurisdiction over appeals from “final decisions of the district courts.” 28 U.S.C. § 1291. When a party appeals something other than “the last order possible to be made in a case,” “a decision of a district court is appealable if it falls within ‘that small class which finally determine claims of right separable from, and collateral to, rights asserted in the action, too important to be denied review and too independent of the cause itself

to require that appellate consideration be deferred until the whole case is adjudicated.” *Mitchell*, 472 U.S. at 524–25, 105 S.Ct. 2806 (quotations omitted). This is known as the collateral order doctrine, and it entitles government officials to an immediate appeal from the denial of qualified immunity. *Id.* at 525–27, 105 S.Ct. 2806.

[13] [14] Because discovery orders generally are non-final, non-appealable orders, even under the collateral order doctrine, *see Coleman v. Am. Red Cross*, 979 F.2d 1135, 1138 (6th Cir. 1992), Snyder and Dillon want us to construe the district court’s order denying their request for a protective order as an implicit order denying them qualified immunity. We do not think that the collateral order doctrine stretches so far.

[15] “[T]here can be two appeals based upon claims of immunity and which can be taken prior to final judgment: first, after denial of a motion to dismiss on the pleadings and, second, after denial of a motion for summary judgment following discovery.” *Sinclair v. Schriber*, 834 F.2d 103, 104 (6th Cir. 1987). Orders regarding discovery do not fit either of these categories and, for that reason, are not independently appealable under the collateral order doctrine. Neither our court nor the Supreme Court has endorsed the extension of the collateral order doctrine that Snyder and Dillon ask for here. We acknowledge that the rationale for permitting government officials to take an immediate appeal from the denial of a motion to dismiss based on qualified immunity rests in part on the concern that forcing officials to wait for a final judgment on the merits would subject them to potentially unwarranted discovery. *See Harlow*, 457 U.S. at 816–18, 102 S.Ct. 2727; *Mitchell*, 472 U.S. at 526, 105 S.Ct. 2806. But these cases simply do not establish an entitlement to an interlocutory appeal from a discovery order itself.

Snyder and Dillon have not pointed to a single case in which we permitted an immediate appeal from a discovery order like the one at issue here. They have, however, pointed to a couple of cases that they claim are close enough. *See Skousen v. Brighton High Sch.*, 305 F.3d 520, 525–26 (6th Cir. 2002); \*830 *Everson v. Leis*, 556 F.3d 484, 490–93 (6th Cir. 2009). In *Skousen* and *Everson*, for example, we held that we had jurisdiction over the district court’s decisions to hold in abeyance summary judgment motions based on qualified immunity pending completion of discovery. *Skousen*, 305 F.3d at 525–26; *Everson*, 556 F.3d at 490–93. There was a question as to whether we had jurisdiction over the orders holding the summary judgment motions in abeyance because

we may entertain an appeal from the denial of summary judgment only if it presents issues solely of law. *Skousen*, 305 F.3d at 525 (citing *Mitchell*, 472 U.S. at 526–28, 105 S.Ct. 2806). We decided that we had jurisdiction to entertain the appeals because, even though the district courts denied summary judgment so that the parties could conduct more discovery, the decisions did not turn on the existence of a genuine issue of material fact. *Id.* at 526, 105 S.Ct. 2806; *Everson*, 556 F.3d at 493. We explained that it did not matter that the order denying summary judgment was styled as an order holding disposition of the motion for summary judgment in abeyance. “If a district court can thwart interlocutory appeal by refusing to address qualified immunity through abeyance rather than dismissal, then the district court can effectively ignore this court’s directive that district courts address qualified immunity promptly.” *Everson*, 556 F.3d at 492. Snyder and Dillon assert that the district court’s discovery order at issue here similarly is tantamount to a denial of qualified immunity. We disagree.

The critical difference between *Skousen/Everson* and this case is that *Skousen* and *Everson* concerned a district court’s delay in ruling on a motion for summary judgment on the issue of qualified immunity. The district courts temporarily denied the defendants’ summary judgment motions to permit additional discovery—but we authorized the appeal because that decision operated, for our purposes, as a denial of summary judgment on the question of qualified immunity. Thus, the orders at issue in *Skousen* and *Everson* fall into *Sinclair*’s second bucket for the types of rulings that are eligible for immediate interlocutory appeal. The orders in those cases were not discovery orders. The collateral order doctrine is already an exception to the general finality rule. Snyder and Dillon are not entitled to appeal any number of discovery matters that they believe have some impact on their immunity interest. We can only imagine the deluge of appeals that would descend upon us if standard discovery orders could so easily be rebranded as final judgments.

Finally, we underscore that the district court’s discovery order fully takes into account the need for a pause in discovery regarding the claim on which Snyder and Dillon assert qualified immunity, and it orders limited discovery as non-party fact witnesses regarding other claims in the litigation. The district court took the state defendants’ immunity seriously. If the noticing parties fail to comply with the district court’s order by pressing an inappropriate line of questioning, Snyder and Dillon may assert their objections in the district court. But ordering Snyder and Dillon to comply

with discovery requests as non-party fact witnesses to events regarding wholly separate claims against different defendants does not, in the abstract, interfere with their immunity.

immunity. We accordingly **DISMISS** for lack of jurisdiction their appeal No. 20-1352.

We reject Snyder's and Dillon's attempt to dress up the district court's discovery order as an implicit denial of qualified

**All Citations**

960 F.3d 820

**Footnotes**

- 1 The facts alleged in the derivative case are set out in our opinion in [In re Flint Water Cases \(Waid v. Snyder\)](#), 960 F.3d 303, 311–23 (6th Cir. 2020).
- 2 The district court's decision to permit discovery from government officials as non-party fact witnesses to events related to claims against other defendants is not out of the ordinary. See, e.g., [Mendia v. Garcia](#), No. 10-cv-03910-MEJ, 2016 WL 3249485, at \*5 (N.D. Cal. June 14, 2016) (“While discovery directed to Defendants as to the *Bivens* claims against them is inappropriate given their pending qualified immunity appeal, ... limited discovery as to these Defendants is appropriate because regardless of whether they are entitled to qualified immunity, they will still need [to] participate in discovery as percipient witnesses related to the FTCA claims against the United States.”); [Harris v. City of Balch Springs](#), 33 F. Supp. 3d 730, 733 (N.D. Tex. 2014) (“The court can think of no legal reason why discovery and pretrial matters may not proceed with respect to [the counts not being appealed on qualified-immunity grounds]. ... [E]ven if the Fifth Circuit were to grant [him] qualified immunity ... he would necessarily be required to testify on behalf of the City regarding [those counts]. ... Whether [he] is subjected to discovery on these counts now or after the resolution of qualified immunity is quite beside the point.”).

---

End of Document

© 2020 Thomson Reuters. No claim to original U.S. Government Works.

960 F.3d 303

United States Court of Appeals, Sixth Circuit.

IN RE: FLINT WATER CASES.

Luke Waid, Parent and Next-Friend  
of SR, a minor, et al., Plaintiffs,

Elnora Carthan et al., Plaintiffs-Appellees,

v.

Darnell Earley, Gerald Ambrose, Howard

Croft, Michael Glasgow, Daugherty

Johnson, and City of Flint, Michigan;

Richard Dale Snyder, former Governor of  
Michigan, [Andy Dillon](#), former Treasurer of  
Michigan, and Gretchen Whitmer, present

Governor of Michigan; Liane Shekter-  
Smith, Stephen Busch, Patrick Cook,

Michael Prysby, and Bradley Wurfel; and  
Adam Rosenthal, Defendants-Appellants.

Nos. 19-1425/1472/1477/1533

Argued: April 27, 2020

Decided and Filed: May 22, 2020

**Synopsis**

**Background:** City residents brought putative class actions under § 1983 against state officials, financially-distressed city that was under emergency management, and city officials, alleging injuries from lead contamination and legionella contamination in city's water supply, and asserting violation of substantive due process right to bodily integrity. The United States District Court for the Eastern District of Michigan, [Judith E. Levy, J., 384 F.Supp.3d 802](#), denied officials' motions for dismissal based on qualified immunity, and denied reconsideration, [2019 WL 8060586](#). Officials filed interlocutory appeal.

**Holdings:** The Court of Appeals, [Moore](#), Circuit Judge, held that:

[1] fact issues existed as to whether emergency managers and city officials had independent knowledge beyond their alleged reliance on opinions from employees of Michigan Department of Environmental Quality (MDEQ) and professional engineering firms;

[2] fact issues existed as to whether MDEQ officials acted based on honest mistakes in law or fact;

[3] fact issues existed as to Governor's reliance on MDEQ and professional engineering firms;

[4] financially-distressed city was not acting as an arm of the State, as would provide basis for city's Eleventh Amendment immunity from suit in federal court; and

[5] *Ex parte Young* exception to a State's Eleventh Amendment immunity from suit in federal court applied to request for prospective injunctive relief against successor Governor.

Affirmed in part and remanded.

[Murphy](#), Circuit Judge, filed an opinion concurring in the judgment and dissenting in part.

West Headnotes (29)

[1] **Federal Courts** ← Immunity

Under the collateral order doctrine, to extent that legal questions were raised by the interlocutory appeals, the Court of Appeals had appellate jurisdiction for state officials' and city officials' interlocutory appeals from denial of their motions to dismiss, based on qualified immunity, in putative class actions brought by city residents under § 1983, alleging deliberate indifference to violations of substantive due process right to bodily integrity, relating to injuries from lead contamination and legionella contamination in city's water supply. *U.S. Const. Amend. 14; 28 U.S.C.A. § 1291; 42 U.S.C.A. § 1983; Fed. R. Civ. P. 12(b)(6)*.

[2] **Federal Courts** ← Immunity

Under the collateral order doctrine, the Court of Appeals had appellate jurisdiction for interlocutory appeals by state's Governor, and financially distressed city that had been under emergency manager control, from denial of their motions to dismiss based on Eleventh Amendment immunity from suit in federal court, in putative class actions brought by city residents under § 1983, alleging violations of the substantive due process right to bodily integrity, relating to injuries from lead contamination and legionella contamination in city's water supply. U.S. Const. Amends. 11, 14; 28 U.S.C.A. § 1291; 42 U.S.C.A. § 1983; Mich. Comp. Laws Ann. § 141.1549; Fed. R. Civ. P. 12(b)(6).

1 Cases that cite this headnote

[3] **Federal Civil Procedure** — Construction of pleadings

**Federal Civil Procedure** — Matters deemed admitted; acceptance as true of allegations in complaint

On a motion to dismiss for failure to state a claim, the court construes the complaint in the light most favorable to plaintiffs, accepts all well-pleaded factual allegations as true, and draws all reasonable inferences in plaintiffs' favor. Fed. R. Civ. P. 12(b)(6).

[4] **Federal Courts** — Immunity

The Court of Appeals reviews de novo a district court's decision to deny qualified immunity to public officials.

[5] **Civil Rights** — Government Agencies and Officers

**Civil Rights** — Good faith and reasonableness; knowledge and clarity of law; motive and intent, in general

**United States** — Qualified immunity in general

Qualified immunity shields federal and state officials from money damages unless a plaintiff pleads facts showing: (1) that the official violated

a statutory or constitutional right, and (2) that the right was clearly established at the time of the challenged conduct.

1 Cases that cite this headnote

[6] **Constitutional Law** — Egregiousness; "shock the conscience" test

The substantive due process aspect of the Fourteenth Amendment protects against conscience-shocking deprivations of liberty. U.S. Const. Amend. 14.

[7] **Constitutional Law** — Personal and bodily rights in general

Violating a person's bodily integrity is a grave deprivation of their liberty, against which deprivation substantive due process provides protection. U.S. Const. Amend. 14.

[8] **Constitutional Law** — Negligence, recklessness, or indifference

The standard for government officials' deliberate indifference to an individual's substantive due process rights is subjective recklessness, and the plaintiff must show that the government officials knew of facts from which they could infer a substantial risk of serious harm, that they did infer it, and that they acted with indifference toward the individual's rights. U.S. Const. Amend. 14.

[9] **Federal Civil Procedure** — Immunity

**Federal Civil Procedure** — Civil rights cases in general

Because the facts at the motion to dismiss stage are undeveloped, it is generally inappropriate for a district court to grant a motion to dismiss on the basis of a public official's qualified immunity from civil liability, and while an official's entitlement to qualified immunity is a threshold question to be resolved at the earliest possible point, that point is usually summary judgment. Fed. R. Civ. P. 12(b)(6), 56.

1 Cases that cite this headnote

**[10] Federal Civil Procedure** 🔑 Fact issues

Issue of whether emergency managers for financially distressed city, city's public works director, and city's utilities administrators had qualified immunity from liability under § 1983 to city residents, for alleged deliberate indifference to residents' substantive due process right to bodily integrity, arising from lead contamination and legionella contamination in city's water supply, could not be resolved at motion to dismiss phase because of factual disputes as to whether those managers and officials had knowledge that city's water supply was causing a public health crisis independent of their alleged reliance on opinions from professional engineering firms and employees of Michigan Department of Environmental Quality (MDEQ). U.S. Const. Amend. 14; 42 U.S.C.A. § 1983; Mich. Comp. Laws Ann. § 141.1549; Fed. R. Civ. P. 12(b)(6).

**[11] Constitutional Law** 🔑 Water, sewer, and irrigation**Water Law** 🔑 Civil claims arising from failure to meet quality standards

City residents plausibly alleged, as required to state a claim, that emergency manager for financially distressed city was deliberately indifferent to their substantive due process right to bodily integrity, in § 1983 actions arising from lead contamination and legionella contamination in city's water supply, by alleging that manager forced an interim switch in city's source of water, during development of new long-term source, when he knew that city's water treatment plant was not ready to treat the water from interim source, that manager directed city officials to lie to the public and tell them that a Legionnaires' disease outbreak was an internal issue at a hospital, and that manager refused to reconnect to old source of water despite his knowledge of water quality issues. U.S. Const. Amend. 14; 42 U.S.C.A. § 1983; Fed. R. Civ. P. 12(b)(6).

1 Cases that cite this headnote

**[12] Constitutional Law** 🔑 Water, sewer, and irrigation**Water Law** 🔑 Civil claims arising from failure to meet quality standards

City residents plausibly alleged, as required to state a claim, that supervisor for city's water treatment plant laboratory was deliberately indifferent to their substantive due process right to bodily integrity, in § 1983 actions arising from lead contamination and legionella contamination in city's water supply, by alleging that supervisor covered up extent of lead contamination, and distorted water quality tests to downplay the extent of lead contamination. U.S. Const. Amend. 14; 42 U.S.C.A. § 1983; Fed. R. Civ. P. 12(b)(6).

**[13] Constitutional Law** 🔑 Water, sewer, and irrigation**Water Law** 🔑 Civil claims arising from failure to meet quality standards

City residents plausibly alleged, as required to state a claim, that city's public works director was deliberately indifferent to their substantive due process right to bodily integrity, in § 1983 actions arising from lead contamination and legionella contamination in city's water supply, by alleging that director permitted switch to interim source of water supply, during development of new long-term source, despite his knowledge that city's water treatment plant was not prepared to deliver safe drinking water, and that director did nothing after learning that Legionnaires' disease outbreak was connected to city's interim source. U.S. Const. Amend. 14; 42 U.S.C.A. § 1983; Fed. R. Civ. P. 12(b)(6).

**[14] Constitutional Law** 🔑 Water, sewer, and irrigation**Water Law** 🔑 Civil claims arising from failure to meet quality standards

City residents plausibly alleged, as required to state a claim, that city's utilities administrator was deliberately indifferent to their substantive due process right to bodily integrity, in § 1983 actions arising from lead contamination and legionella contamination in city's water supply, by alleging that administrator pressured supervisor for city's water treatment plant laboratory, despite supervisor's warnings, to make interim change in source of city's water during development of new long-term source, and that administrator stonewalled county health department's attempts to investigate water quality from interim source and attempts to investigate the outbreak of Legionnaires' disease. [U.S. Const. Amend. 14](#); [42 U.S.C.A. § 1983](#); [Fed. R. Civ. P. 12\(b\)\(6\)](#).

**[15] Federal Civil Procedure** 🔑 Fact issues

Issue of whether officials of Michigan Department of Environmental Quality (MDEQ) had qualified immunity from liability under § 1983 to city residents, for alleged deliberate indifference to their substantive due process right to bodily integrity, arising from lead contamination and legionella contamination in city's water supply, could not be resolved at motion to dismiss phase because of factual disputes as to whether those officials, despite their allegedly honest mistakes in law or fact when interpreting and applying Environmental Protection Agency's (EPA) Lead and Copper Rule, rushed to switch city's interim source of water supply, during development of new long-term source, knowing that interim source would deliver contaminated water, and whether officials cared only about cost to financially-distressed city, not water quality. [U.S. Const. Amend. 14](#); [42 U.S.C.A. § 1983](#); [Fed. R. Civ. P. 12\(b\)\(6\)](#).

**[16] Constitutional Law** 🔑 Water, sewer, and irrigation

**Water Law** 🔑 Civil claims arising from failure to meet quality standards

City residents plausibly alleged, as required to state a claim, that Michigan Department of Environmental Quality's (MDEQ) Chief of Office of Drinking Water and Municipal Assistance was deliberately indifferent to their substantive due process right to bodily integrity, in § 1983 actions arising from lead contamination and legionella contamination in city's water supply, by alleging that Chief, despite knowing of health risks presented by city's switch to interim water source during development of new long-term source, secured the necessary administrative consent order (ACO) so city could borrow funds for development of new source and rushed the interim switch before city's water treatment plant was ready, and by alleging that Chief did nothing to mitigate the problem when city residents reported that city's water from interim source was making them ill, and that Chief instead covered up the crisis. [U.S. Const. Amend. 14](#); [42 U.S.C.A. § 1983](#); [Fed. R. Civ. P. 12\(b\)\(6\)](#).

**[17] Constitutional Law** 🔑 Water, sewer, and irrigation

**Water Law** 🔑 Civil claims arising from failure to meet quality standards

City residents plausibly alleged, as required to state a claim, that Michigan Department of Environmental Quality's (MDEQ) Water Quality Analyst was deliberately indifferent to their substantive due process right to bodily integrity, in § 1983 actions arising from lead contamination and legionella contamination in city's water supply, by alleging that Analyst did not stop city's switch to interim source of water, during development of new long-term source, despite warning from supervisor for city's water treatment plant laboratory that the plant was not ready, that he did nothing after learning that city's water contained levels of total trihalomethanes (TTHM) that were above regulatory limit, and that he distributed a distorted water quality report that was altered to exclude high levels of TTHM. [U.S. Const. Amend. 14](#); [42 U.S.C.A. § 1983](#); [Fed. R. Civ. P. 12\(b\)\(6\)](#).

2 Cases that cite this headnote

[18] **Constitutional Law** 🔑 Water, sewer, and irrigation

**Water Law** 🔑 Civil claims arising from failure to meet quality standards

City residents plausibly alleged, as required to state a claim, that Michigan Department of Environmental Quality's (MDEQ) District Supervisor was deliberately indifferent to their substantive due process right to bodily integrity, in § 1983 actions arising from lead contamination and legionella contamination in city's water supply, by alleging that Supervisor, despite knowing that city's switch to interim source of water during development of new long-term source would require significant water treatment, did not stop the switch even after learning that city's water treatment plant was not ready, and that he lied to Environmental Protection Agency (EPA) employee, when MDEQ came under EPA scrutiny for lead contamination, by telling him that city was using corrosion control. *U.S. Const. Amend. 14*; 42 U.S.C.A. § 1983; Fed. R. Civ. P. 12(b)(6).

2 Cases that cite this headnote

[19] **Constitutional Law** 🔑 Water, sewer, and irrigation

**Water Law** 🔑 Civil claims arising from failure to meet quality standards

City residents plausibly alleged, as required to state a claim, that Michigan Department of Environmental Quality's (MDEQ) engineer was deliberately indifferent to their substantive due process right to bodily integrity, in § 1983 actions arising from lead contamination and legionella contamination in city's water supply, by alleging that engineer, despite warnings from supervisor for city's water treatment plant laboratory, did not stop the switch to city's interim source of water during development of new long-term source, that engineer did nothing in response to Environmental Protection Agency (EPA) employee's report of high lead levels in city's water from interim source, and

that he directed laboratory supervisor to distort water quality tests by excluding high results for lead contamination. *U.S. Const. Amend. 14*; 42 U.S.C.A. § 1983; Fed. R. Civ. P. 12(b)(6).

[20] **Constitutional Law** 🔑 Water, sewer, and irrigation

**Water Law** 🔑 Civil claims arising from failure to meet quality standards

City residents plausibly alleged, as required to state a claim, that Michigan Department of Environmental Quality's (MDEQ) Water Treatment Specialist was deliberately indifferent to their substantive due process right to bodily integrity, in § 1983 actions arising from lead contamination and legionella contamination in city's water supply, by alleging that Specialist initially did nothing in response to Environmental Protection Agency (EPA) employee's report of high lead levels in city's water, and later misled EPA regarding necessity of using corrosion control, by stating that city's water testing results were within regulatory limit for lead. *U.S. Const. Amend. 14*; 42 U.S.C.A. § 1983; Fed. R. Civ. P. 12(b)(6).

[21] **Federal Civil Procedure** 🔑 Construction of pleadings

On a motion to dismiss, when a document attached to the complaint contradicts the complaint's allegations by utterly discrediting the allegations, the document trumps the allegations. Fed. R. Civ. P. 12(b).

[22] **Constitutional Law** 🔑 Water, sewer, and irrigation

**Water Law** 🔑 Civil claims arising from failure to meet quality standards

City residents plausibly alleged, as required to state a claim, that Michigan Department of Environmental Quality's (MDEQ) Director of Communications was deliberately indifferent to their substantive due process right to bodily integrity, in § 1983 actions arising from lead contamination and legionella contamination in

city's water supply, by alleging that when concerns and criticisms reached their peak, Director repeatedly lied to the public and assured them that city's water was safe, while distorting water quality tests and attacking independent whistleblower reports that city was in midst of major public health emergency. *U.S. Const. Amend. 14*; 42 U.S.C.A. § 1983; *Fed. R. Civ. P. 12(b)(6)*.

**[23] Federal Civil Procedure** 🔑 Fact issues

Issue of whether Michigan Governor had qualified immunity from liability under § 1983 to city residents, for alleged deliberate indifference to their substantive due process right to bodily integrity, arising from lead contamination and legionella contamination in city's water supply, could not be resolved at motion to dismiss phase because of factual disputes as to whether Governor's actions or failures to act relied on assessments from Michigan Department of Environmental Quality (MDEQ) and professional engineering firms, and whether Governor's disinformation or inaction arose from legitimate disagreements over nature and extent of problems and the appropriate solution. *U.S. Const. Amend. 14*; 42 U.S.C.A. § 1983; *Fed. R. Civ. P. 12(b)(6)*.

**[24] Constitutional Law** 🔑 Water, sewer, and irrigation

**States** 🔑 Governor

**Water Law** 🔑 Civil claims arising from failure to meet quality standards

City residents plausibly alleged, as required to state a claim, that Governor was deliberately indifferent to their substantive due process right to bodily integrity, in § 1983 actions arising from lead contamination and legionella contamination in city's water supply, by alleging that Governor was personally aware, from public reports and from his own staff, that city's interim source of water during development of new long-term source was contaminated yet he downplayed the problem and delayed in switching back to original source and in declaring a state of

emergency that provided valuable resources to abate the harm in financially-distressed city. *U.S. Const. Amend. 14*; 42 U.S.C.A. § 1983; *Fed. R. Civ. P. 12(b)(6)*.

**[25] Federal Courts** 🔑 Immunity

Whether Eleventh Amendment immunity from suit in federal court exists in any particular case is a question of constitutional law that is reviewed de novo. *U.S. Const. Amend. 11*.

**[26] Federal Courts** 🔑 Municipal corporations; cities

The Eleventh Amendment generally bars suits in federal courts against States, but generally does not bar suits against cities. *U.S. Const. Amend. 11*.

**[27] Federal Courts** 🔑 Municipal corporations; cities

Financially-distressed city was not acting as an arm of the State, as would provide basis for city's Eleventh Amendment immunity from suit in federal court, when city was under State's emergency management, and thus, Eleventh Amendment immunity did not apply to city's alleged deliberate indifference to violation of city residents' substantive due process right to bodily integrity, arising from lead contamination and legionella contamination in city's water supply while city was under State's emergency management. *U.S. Const. Amends. 11, 14*; 42 U.S.C.A. § 1983; *Mich. Comp. Laws Ann. § 141.1549*.

**[28] Federal Courts** 🔑 Suits for injunctive or other prospective or equitable relief; *Ex parte Young* doctrine

**Federal Courts** 🔑 Agencies, officers, and public employees

While a State generally has Eleventh Amendment immunity from suit in federal court, the *Ex parte Young* exception allows plaintiffs

to seek prospective injunctive relief against State actors in their official capacity. *U.S. Const. Amend. 11.*

**[29] Federal Courts** 🔑 Suits for injunctive or other prospective or equitable relief; *Ex parte Young* doctrine

**Federal Courts** 🔑 Other particular entities and individuals

*Ex parte Young* exception to a State's Eleventh Amendment immunity from suit in federal court applied to city residents' request for prospective injunctive relief against successor Governor, in her official capacity, to remediate ongoing harms stemming from lead contamination and legionella contamination in city's water supply that originated during predecessor Governor's term in office, though successor Governor had not personally committed the alleged violations of city residents' substantive due process right to bodily integrity. *U.S. Const. Amends. 11, 14; 42 U.S.C.A. § 1983.*

\*309 Appeal from the United States District Court for the Eastern District of Michigan at Ann Arbor. No. 5:16-cv-10444—Judith E. Levy, District Judge.

#### Attorneys and Law Firms

ARGUED: William Y. Kim, CITY OF FLINT LAW DEPARTMENT, Flint, Michigan, for Appellant City of Flint, and Christopher J. Marker, O'NEIL, WALLACE & DOYLE, P.C., Saginaw, Michigan, for Appellant Glasgow in 19-1425. Margaret A. Bettenhausen, OFFICE OF THE MICHIGAN ATTORNEY GENERAL, Lansing, Michigan for Appellants in 19-1472. Charles E. Barbieri, FOSTER, SWIFT, COLLINS & SMITH, P.C., Lansing, Michigan, for Appellants in 19-1477. James A. Fajen, FAJEN AND MILLER, PLLC, Ann Arbor, Michigan, for Appellant in 19-1533. Samuel R. Bagenstos, Ann Arbor, Michigan, for Appellees. ON BRIEF: William Y. Kim, CITY OF FLINT LAW DEPARTMENT, Flint, Michigan, Frederick A. Berg, Jr., BUTZEL LONG, P.C., Detroit, Michigan, Sheldon H. Klein, Joseph E. Richotte, BUTZEL LONG, P.C., Bloomfield Hills, Michigan, Christopher J. Marker,

O'NEIL, WALLACE & DOYLE, P.C., Saginaw, Michigan, Todd R. Perkins, THE PERKINS LAW GROUP PLLC, Detroit, Michigan, Alexander S. Rusek, WHITE LAW, PLLC, Okemos, Michigan, Barry A. Wolf, Flint, Michigan, Edwar A. Zeineh, LAW OFFICE OF EDWAR A. ZEINEH, Lansing, Michigan, for Appellants in 19-1425. Margaret A. Bettenhausen, Richard S. Kuhl, Nathan A. Gambill, Zachary C. Larsen, OFFICE OF THE MICHIGAN ATTORNEY GENERAL, Lansing, Michigan for Appellants in 19-1472. Charles E. Barbieri, FOSTER, SWIFT, COLLINS & SMITH, P.C., Lansing, Michigan, Michael J. Pattwell, Jay M. Berger, CLARK HILL PLC, Lansing, Michigan, Thaddeus E. Morgan, FRASER, TREBILCOCK, DAVIS & DUNLAP, Lansing, Michigan, Philip A. Grashoff, Jr., SMITH HAUGHEY RICE & ROEGGE, Grand Rapids, Michigan, for Appellants in 19-1477. James A. Fajen, FAJEN AND MILLER, PLLC, Ann Arbor, Michigan, James W. Burdick, BURDICK LAW, P.C., Bloomfield Hills, Michigan, for Appellant in 19-1533. Samuel R. Bagenstos, Ann Arbor, Michigan, for Appellees.

Before: MERRITT, MOORE, and MURPHY, Circuit Judges.

MOORE, J., delivered the opinion of the court in which MERRITT, J., joined. MURPHY, J. (pp. 335-39), delivered a separate opinion concurring in the judgment in part and dissenting in part.

#### CONCURRING IN THE JUDGMENT IN PART AND DISSENTING IN PART

#### OPINION

KAREN NELSON MOORE, Circuit Judge.

\*310 This is a case about the Flint Water Crisis. From 2014 to 2015, City of Flint and Michigan State officials caused, sustained, and covered up the poisoning of an entire community with lead- and *legionella*-contaminated water. The crisis started in April 2014 when the City began delivering Flint River water to its predominantly poor and African-American residents, knowing that it was not treated for corrosion. In a matter of weeks, Flint residents reported that there was something wrong with the way the water looked, tasted, and smelled, and that it was causing rashes. In response, the City treated the water with additional chlorine—exacerbating the corrosion in the old water lines. The corrosion contaminated the water with hazardous levels of

lead and caused an outbreak of [Legionnaires' disease](#). State and City officials failed to stop the delivery of Flint River water and obstinately assured the public that the water was safe, when they knew it was not. Now, Flint residents can expect to see their children permanently developmentally stunted. It has been six years since the start of the crisis and corroded pipes still infect the water and poison the people of Flint. The question before us is whether these Defendants-Appellants allegedly responsible for the crisis are immune from suit.

\*311 This appeal arises out of a consolidated class action in the *In re Flint Water Cases* litigation. It follows from the denial of motions to dismiss certain defendants based on qualified and absolute immunity. The Plaintiffs-Appellees are individuals affected by the Flint Water Crisis.<sup>1</sup> The Defendants-Appellants are City and State officials and the City of Flint.<sup>2</sup> Plaintiffs-Appellees claim that City and State officials' deliberate indifference to their being poisoned violated their substantive due process right to bodily integrity, a constitutional claim we have already recognized in *Guertin v. Michigan*, 912 F.3d 907, 921 (6th Cir. 2019), *cert. denied*, — U.S. —, 140 S. Ct. 933, 205 L.Ed.2d 522 (2020). Acknowledging that *Guertin* controls, Defendants-Appellants contend that their alleged individual conduct does not plausibly amount to a constitutional violation. Or, in the case of the City of Flint and Governor Whitmer,<sup>3</sup> that the Eleventh Amendment requires their dismissal from this action—an argument we rejected in prior appeals. *See Guertin*, 912 F.3d at 936; *Boler v. Earley*, 865 F.3d 391, 412–13 (6th Cir. 2017), *cert. denied*, — U.S. —, 138 S. Ct. 1281, 200 L.Ed.2d 469 (2018).

We **AFFIRM** the district court's denial of the motions to dismiss with respect to every Defendant-Appellant except Treasurer Dillon. We **REMAND** for the district court to decide whether Dillon should be dismissed in light of its decision in *Brown v. Snyder (In re Flint Water Cases)*, No. 18-cv-10726, 2020 WL 1503256, at \*9 (E.D. Mich. Mar. 27, 2020).

## I. BACKGROUND<sup>4</sup>

Plaintiffs allege that, from June 2013 through April 25, 2014, City and State officials created a public health crisis. R. 620-3 (Fourth Am. Compl. at 47–48, ¶ 133) (Page ID #17850–51). Officials “ordered and set in motion the use of

highly corrosive and toxic Flint River water knowing that the [treatment plant] was not ready.” *Id.* “By January 29, 2015, State officials understood that the public health crisis was caused by the corrosion of the entire infrastructure of the Flint water system. Yet no action was taken to warn the public of the health crisis or to correct the harm.” *Id.* at 81, ¶ 238 (Page ID #17884).<sup>5</sup> Accordingly, “the complaint alleges constitutional violations that occurred during two relevant \*312 periods: (1) the period leading up to the April 2014 switch to the Flint River, during which Defendants were callously indifferent to the facts showing that the water would be dangerous; and (2) the 18-month period from April 2014 to October 2015, during which Defendants were callously indifferent to the mounting evidence that the water was actually causing serious harm, including death.” Appellees Br. at 4–5.

### A. The Switch to the Flint River

The City of Flint did not always receive its water from the Flint River. For decades, the City received clean water from Lake Huron through the Detroit Water and Sewerage Department (“DWSD”). R. 620-3 (Fourth Am. Compl. at 34–35, ¶¶ 86–91) (Page ID #17837–38). At some point, however, the City became concerned with the DWSD's cost and decided to look into alternative water sources. *Id.* at 35–37, ¶ 92 (Page ID #17838–40). In 2011, City officials had a study conducted to see if the Flint River could be used as a safe water source if it was processed through the old Flint Water Treatment Plant (“FWTP”). *Id.* at 33–34, ¶¶ 82–86 (Page ID #17836–37); *id.* at 36–37, ¶ 92 (Page ID #17839–40). The reports issued from the study concluded that Flint River water could meet regulatory requirements if properly treated—but that would require over \$69 million in improvements to the FWTP, including improvements that would protect the water from corrosion. *Id.* at 36–37, ¶ 92 (Page ID #17839–40). For the moment, the City decided against switching to the Flint River as its primary drinking source. *Id.* at 37, ¶ 94 (Page ID #17840).

But by August 2012, the City was embroiled in a financial emergency, leading Governor Snyder to appoint Edward Kurtz as Emergency Manager for the City. *Id.* at 38–39, ¶ 101 (Page ID #17841–42). In Michigan, the State can appoint emergency managers to take over financially distressed cities, control their operations, and rein in spending. *See Guertin*, 912 F.3d at 939; R. 620-3 (Fourth Am. Compl. at 39, ¶ 102) (Page ID #17842). To carry out their mission, emergency managers are granted “broad powers” to “act for and in the place and stead of the governing body and the office of

chief administrative officer of the local government.” MICH. COMP. LAWS § 141.1549(2).

As Emergency Manager, Kurtz made a pitch to Governor Snyder and State Treasurer Andy Dillon that the City of Flint should switch to receiving water from an altogether new source, the Karegnondi Water Authority (“KWA”). R. 620-3 (Fourth Am. Compl. at 38–39, ¶ 101–02) (Page ID #17841–42). The DWSD, on the other hand, made its case to Snyder, Dillon, and Kurtz that its water was cheaper and more reliable. *Id.* at 39, ¶ 103 (Page ID #17842). Caught between competing offers, Dillon requested an independent assessment of cost effectiveness for each plan by the engineering firm of Tucker, Young, Jackson and Tull (“TYJT”). *Id.* at 39–40, ¶ 104 (Page ID #17842–43). In February 2013, TYJT informed Dillon that “it would be more cost-effective for Flint on both a short term and long term basis to continue to be supplied with water from DWSD.” *Id.* Accordingly, on March 17, 2013, Treasurer Dillon wrote to Governor Snyder that “the KWA representatives were misrepresenting the benefits of the deal and that the ‘(r)eport that I got is that Flint should stay w DWSD.’ ” *Id.* Then, on March 28, 2013, Dillon reversed course and emailed Snyder recommending that he authorize the City of Flint’s switch to the KWA, noting that Kurtz, the Mayor, and City Council all supported that decision. *Id.* at 41, ¶ 107 (Page ID #17844). Even though the KWA was more costly than the DWSD, the City would be able to borrow \*313 funds to pay its share of the project if it obtained an Administrative Consent Order (“ACO”) from a State Agency attesting to need due to “fire, flood, or other calamity.” *Id.* at 127, ¶¶ 375–76 (Page ID #17930).

The Michigan Department of Environmental Quality (“MDEQ”) ultimately backed the interim plan, even though it knew “that the decision to switch the water source for Flint was not based on a scientific assessment of the suitability of the Flint River water.” *Id.* at 40–41, ¶ 106 (Page ID #17843–44). The MDEQ Deputy Director wrote, “[W]e are in a situation with Emergency Financial Managers so it’s entirely possible that they will be making decisions relative to cost.” *Id.* In March 2013, MDEQ officials, including Stephen Busch and Liane Shekter-Smith, knew that “the use of Flint River water would pose increased health risks to the public ..., the triggering of additional regulatory requirements, and significant upgrades to the Flint Water Treatment Plant.” *Id.* at 40, ¶ 105 (Page ID #17843).

In 2013, both the City of Flint and the City of Detroit were under State emergency management. *Id.* at 42, ¶ 114 (Page ID

#17845). As Governor, “Snyder was briefed on reports from both Flint’s and Detroit’s emergency managers and issued directions to both managers as it related to the transition” away from the DWSD. *Id.* Thus, “Governor Snyder was personally involved in the decisional process which led to the transition from DWSD to the KWA.” *Id.* On April 4, 2013, Snyder’s Chief of Staff emailed him, stating “(a)s you know, the Flint people have requested Dillon’s ok to break away from the DWSD.” *Id.* at 43, ¶ 115 (Page ID #17846). Snyder then instructed Dillon, Kurtz, Detroit’s Emergency Manager, and other key players to have the DWSD submit one last offer to the City of Flint. *Id.* The DWSD did so, Kurtz rejected the offer, and Snyder “authorized Kurtz, through Department of Treasury officials, to enter into a contractual relationship with KWA for the purpose of supplying water to Flint *beginning in mid-year 2016 or 2017.*” *Id.* at 43, ¶¶ 115–18 (Page ID #17846) (emphasis added). The City would need to rely on a water source other than the KWA until then. “At the time the Governor authorized Kurtz to contractually bind Flint to the KWA project, the Governor and State officials knew that the Flint River,” rather than the DWSD, “would be used as an interim source and that the use of the interim source had the backing of Snyder, Andy Dillon, and MDEQ Director Wyant.” *Id.* at 44, ¶ 119 (Page ID #17847).

In June 2013, Dillon, Kurtz, and other key players developed the interim Flint River plan that would supply the City with water from April 25, 2014 until approximately December 2016. *Id.* at 44, ¶ 120 (Page ID #17847). A “critical part” of the interim plan was to upgrade the FWTP so that it could treat the Flint River water and then later treat water delivered through the KWA. *Id.* at 44, ¶ 122 (Page ID #17847). In September 2013, Governor Snyder appointed Darnell Earley as the City of Flint’s new Emergency Manager. *Id.* at 45, ¶ 125 (Page ID #17848). Earley worked to ensure that the interim Flint River plan would not be displaced by a return to the DWSD, even as the FWTP “was deemed unready for service by several people involved with its management.” *Id.* at 51, ¶ 147 (Page ID #17854).

In March 2014, MDEQ officials, led by Chief of the Office of Drinking Water and Municipal Assistance Liane Shekter-Smith, put the interim Flint River plan into motion by ensuring, at the Treasury’s direction, that the City quickly obtain the necessary ACO so that the KWA would not need to stop construction. *See* \*314 *id.* at 45–46, ¶ 128 (Page ID #17848–49); *id.* at 130, ¶¶ 382–83 (Page ID #17933). The ACO “(i) required Flint to make use of the Flint Water Treatment Plant, (ii) attempted to prevent Flint from ever

returning to the DWSD and (iii) mandated Flint to ‘undertake the KWA public improvement project or undertake other public improvement projects to continue to use the Flint River....’ ” *Id.* at 45–46, ¶ 128 (Page ID #17848–49). “After obtaining the ACO, Flint entered a Bond Purchase Agreement allowing it to borrow funds despite being in receivership so that the KWA could move on to the next phase of construction. Unfortunately, the Flint Water Treatment Plant was nowhere near ready to begin distributing water.” *Id.* at 131, ¶ 384 (Page ID #17934).

On March 14, 2014, the associate director of the Governor’s Office of Urban and Metropolitan Initiatives stated in an email to other members of Snyder’s staff that the “expedited timeframe” for switching to Flint River water “is less than ideal and could lead to some big potential disasters down the road.” *Id.* at 45, ¶ 127 (Page ID #17848). His warning went unheeded, as plans stayed in motion.

On April 16, 2014—a week before the date set for the switch to the Flint River—Michael Glasgow, the City’s water treatment plant laboratory and water quality supervisor, emailed MDEQ Water Quality Analyst Adam Rosenthal, “... it looks as if we will be starting the plant up tomorrow and are being pushed to start distributing water as soon as possible.... I would like to make sure we are monitoring, reporting and meeting requirements before I give the OK to start distributing water.” *Id.* at 46, ¶ 129 (Page ID #17849). The next day, Glasgow informed the MDEQ that “the FWTP was not fit to begin operations and that ‘management’ was not listening to him.” *Id.* On April 17, 2014, Glasgow wrote to MDEQ District Supervisor Stephen Busch and MDEQ District 11 (Flint) Engineer Michael Prysby,

I have people above me making plans to distribute water ASAP. I was reluctant before, but after looking at the monitoring schedule and our current staffing, I do not anticipate giving the OK to begin sending water out anytime soon. If water is distributed from this plant in the next couple of weeks, it will be against my direction. I need time to adequately train additional staff and to update our monitoring plans before I will feel we are ready. I will reiterate this to management above me, but they seem to have their own agenda.

*Id.* “Glasgow later told State investigators that he received pressure from superiors—particularly Defendants Johnson and Croft—to begin the switch to the Flint River.” *Id.* at 47, ¶ 130 (Page ID #17850).

MDEQ Water Treatment Specialist Patrick Cook signed the permit that was the last necessary approval for use of the FWTP. *Id.* at 47, ¶ 132 (Page ID #17850).

### **B. Lead Poisoning and Legionnaires’ Disease**

“On April 25, 2014, Flint officially began using the Flint River as its primary water source, despite the fact that the proper preparations had not been made and Glasgow had warned that the FWTP was not ready.” *Id.* at 57, ¶ 164 (Page ID #17860). Flint River water had high chloride levels that, left untreated, would corrode the water pipes and cause lead to “leach into drinking water.” *Id.* at 55, ¶ 161 (Page ID #17858). The MDEQ purportedly believed that it needed to collect data on the water for an entire year, in two consecutive six-month tests, before it could treat the water for corrosion. *Id.* at 95–96, ¶ 290 (Page ID #17898–99). Prior to the switch, City and \*315 MDEQ officials “discussed optimization for lead,” but “decided that having more data was advisable before implementing an optimization method.” *Id.* at 55, ¶ 159 (Page ID #17858). Rather than delay the switch to the Flint River, the City began delivering *untreated* water to its residents.

Within weeks of the switch, residents reported to Shekter-Smith that there was something wrong with the smell, taste, and color of the water, and that it was causing rashes. *Id.* at 57, ¶¶ 165–66 (Page ID #17860). By June 2014, residents were reporting that “the water was making them ill.” *Id.* at 57, ¶ 167 (Page ID #17860). The City and State did nothing. *Id.* “On August 14, 2014, Flint’s water tested above legal limits for total coliform and E. coli bacteria.” *Id.* at 57, ¶ 168 (Page ID #17860). In response, the City issued boil water advisories and treated the water with additional chlorine. *Id.* at 57–58, ¶¶ 168–69 (Page ID #17860–61). Chlorine, however, “as has been well known for decades,” “preferentially reacts with the bare metal [in corroded pipes] instead of attacking solely bacteria.” *Id.* at 57–58, ¶ 169 (Page ID #17860–61). Unsurprisingly, then, the bacterial problem did not abate—so the City added still more chlorine. *Id.* The water then tested high in total trihalomethanes (“TTHM”), a byproduct of chlorine interacting with metal, and a “red flag that the steel in the pipes had been laid bare,” and that lead was leaching into the water. *Id.* at 58, ¶¶ 170–71 (Page ID #17861). Back in May 2014, MDEQ officials—including Busch, Prysby, and Rosenthal—knew that TTHM levels were above the EPA’s maximum contaminant level but did nothing, even as residents raised concerns about the water. *Id.* at 58, ¶ 172 (Page ID #17861). From May 2014 to August 2015, the City sampled the water six times to test for corrosivity, and “[t]he

sampling results all showed that the drinking water was very corrosive.” *Id.* at 62, ¶ 187 (Page ID #17865).

In the summer of 2014, just “[a]s officials were beginning to assess the extent of Flint’s TTHM problems, ... the Michigan Department of Health and Human Services (MDHHS) reported an outbreak of Legionnaires’ disease—another red flag.” *Id.* at 58–59, ¶ 173 (Page ID #17861–62). **Legionnaires’ disease** “is a severe form of **pneumonia**.” *Id.* at 59, ¶ 174 (Page ID #17862). It infects people who inhale or consume water contaminated with *legionella* bacteria. *Id.* “Extensive studies of *legionella* have established that the pathogen enters the water supply when the ‘bio-film’ protecting pipes is stripped away—which is exactly what happened when the River’s corrosive water entered the City’s pipes.” *Id.* When a City officer informed Earley and his then-advisor Gerald Ambrose of the outbreak, Earley responded by “disclaiming any connection between the outbreak and Flint’s water.” *Id.* at 59, ¶ 175 (Page ID #17862). Earley stated that “the City’s ‘message’ should be that the outbreak was ‘an internal issue at McLaren [Hospital] that they are working on with our assistance, not a Flint water problem that we are trying to resolve.’” *Id.*

In September 2014, MDHHS reported that “**lead poisoning** rates ‘were higher than usual for children under age 16 living in the City of Flint during the months of July, August and September, 2014.’” *Id.* at 59–60, ¶ 176 (Page ID #17862–63). And in early October 2014, officials realized that the bacterial contamination partly stemmed from the use of over-75-year-old cast iron pipes that comprised most of the City’s water distribution system. *Id.* at 60, ¶ 177 (Page ID #17863). Still no action.

On October 13, 2014, General Motors stopped using Flint River water at its engine plant out of fear that the high **\*316** levels of chloride would corrode its machinery. *Id.* at 60, ¶ 179 (Page ID #17863). The next day, a member of Governor Snyder’s executive staff wrote to the team:

Now we are getting comments about being lab rats in the media, which are going to be exacerbated when it comes out that after the boil water order, there were chemicals in the water that exceeded health-based water quality standards. I think we should ask the [Emergency Manager] to consider coming back to the Detroit system in full or in part as an interim solution to both the quality, and now the financial, problems that the current solution is causing. *Id.* at 60–61, ¶ 180 (Page ID #17863–64). Snyder’s legal counsel similarly stated that the Flint River water issues are

“downright scary” and “advised that, ‘[t]hey should try to get back on the Detroit system as a stopgap ASAP before this thing gets too far out of control.’” *Id.* at 61, ¶ 182 (Page ID #17864). The executive staff directed MDEQ officials to brief Earley on the water quality issues, *id.* at 60–61, ¶ 180 (Page ID #17863–64), but Earley refused to reconnect to the DWSD, *id.* at 61, ¶ 181 (Page ID #17864).

With their awareness of the dangers of Flint River water only increasing, officials nonetheless failed to disclose the risks to Flint Residents. *Id.* at 80, ¶ 235 (Page ID #17883). “On December 31, 2014, the first round of lead monitoring showed results exceeding the Lead and **Copper** Rule’s action levels for lead, 15 parts per billion.” *Id.* at 61–62, ¶ 183 (Page ID #17864–65). And the samples had not even been drawn from the highest risk homes. *Id.* In January 2015, State officials met to discuss the *legionella* problem. *Id.* at 80, ¶ 233 (Page ID #17883). Around that time, MDEQ Director of Communications Bradley Wurfel wrote in an email, “I don’t want my director to say publicly that the water in Flint is safe until we get back the results of some county health department of epidemiological trace-back work on [the] 41 cases of **Legionnaires’ disease**” diagnosed since the switch to the Flint River. *Id.* at 62, ¶ 184 (Page ID #17865).

On January 9, 2015, the University of Michigan turned off certain water fountains on its Flint campus because tests it conducted revealed high levels of lead. *Id.* at 62, ¶ 185 (Page ID #17865). “That same day, Earley,” again, “refused to return to DWSD water.” *Id.* at 62, ¶ 186 (Page ID #17865). A few days later, Earley resigned as Emergency Manager, and Governor Snyder appointed Gerald Ambrose in his stead. *Id.* at 80, ¶ 234 (Page ID #17883).

On January 21, 2015, State officials had water coolers discreetly installed in State buildings located in Flint, careful not to make their actions known to the public. *Id.* at 80, ¶ 235 (Page ID #17883). On January 27, 2015, the Genesee County Health Department (“GCHD”) reported a likely “association between the spike in **Legionnaires’ disease** reports and the onset of the use of Flint River water.” *Id.* at 81, ¶ 237 (Page ID #17884). The City and State did nothing. *Id.* On January 29, 2015, the DWSD offered Emergency Manager Ambrose “an opportunity to purchase DWSD water at attractive rates ... includ[ing] waiving the re-connection fee.” *Id.* at 81, ¶ 239 (Page ID #17884). Ambrose refused. *Id.* “On February 17, 2015, Flint water users staged public demonstrations demanding that Flint reconnect with DWSD.” *Id.* at 82, ¶ 243 (Page ID #17885). Ambrose again refused. *Id.*

### C. The Coverup

With the crisis growing undeniable, City and State officials attempted to cover it up. They lied to the public and to regulators, and they took no action to protect the people of Flint. *Id.*

\*317 On February 26, 2015, Jennifer Crooks of the EPA followed up on a request from a Flint resident to test her water after she and her family became physically ill, developed rashes, and even experienced hair loss after drinking from the tap. *Id.* at 81, ¶ 240 (Page ID #17884); *id.* at 82–83, ¶ 244 (Page ID #17885–86). Crooks wrote to MDEQ and EPA officials that “the iron contamination was so high that the testing instrumentation could not measure it” and that the water tested for 104 parts per billion (“ppb”) of lead, well over the 15 ppb regulatory maximum. *See id.* at 82–83, ¶ 244 (Page ID #17885–86). Crooks further noted that, with two children under the age of three residing at the house, there were “[b]ig worries here.” *Id.* This prompted another EPA employee, Miguel Del Toral, to wonder whether the City of Flint was implementing optimized corrosion control, and whether the high lead levels were isolated to that one family’s neighborhood or were more widespread. *See id.* at 83, ¶ 245 (Page ID #17886). The EPA shared its concerns with the MDEQ. In response, MDEQ District Supervisor Stephen Busch lied and told Del Toral that the City was using corrosion control. *Id.* at 83, ¶ 246 (Page ID #17886).

“Likewise, [City Utilities Administrator Daugherty] Johnson inhibited efforts by [GCHD] to obtain information about Flint’s water through the Freedom of Information Act (“FOIA”).” *Id.* at 83–84, ¶ 248 (Page ID #17886–87). On January 27, 2015, GCHD requested water-testing information that would help it understand perceived water quality issues and the outbreak of [Legionnaires’ disease](#). *See id.* A week later, Johnson responded that he had not received the FOIA request but would fulfill it as soon as possible. *Id.* Yet, “by March 2015, GCHD still had not received the information they requested by FOIA.” *Id.* The GCHD soon gathered that it was “being stonewalled.” *Id.* at 84, ¶ 250 (Page ID #17887).

By March 2015, Governor Snyder and other State officials knew “that they had a massive public health emergency which probably included widespread [lead poisoning](#) on their hands and began discussing distributing water filters to Flint water users.” *Id.* at 84, ¶ 249 (Page ID #17887). Nevertheless, “these public officials took no action to warn or otherwise protect Plaintiffs and the Class, and continued to conceal from

them and the public the true nature, extent, and severity of the public health crisis.” *Id.* The Governor’s office’s talking points included false statements that the City was practicing corrosion control consistent with federal protocols and that Flint’s water was in compliance with federal lead and [copper](#) rules. *Id.* at 149–50, ¶ 419 (Page ID #17952–53).

On March 10, 2015, the GCHD wrote to Croft, Prysby, Ambrose, the mayor, and other City officials that the threat of *legionella* was serious and tied to Flint River water. *Id.* at 138, ¶ 401 (Page ID #17941). The GCHD official noted that he had requested to meet with the water plant staff and MDEQ to discuss his concerns, but that the water plant staff did not respond and that the MDEQ declined. *Id.* On March 12, 2015, Shekter-Smith emailed MDEQ employees that, “[w]hile the change in source may have created water quality conditions that could provide additional organic nutrient source to support *legionella* growth, there is no evidence or confirmation of *legionella* coming directly from the Water Treatment Plant or in the community water supply distribution system at this time.” *Id.* at 85, ¶ 252 (Page ID #17888). The next day, Shekter-Smith approved a response from Busch to the GCHD that stated the following:

- “conclusions that legionella is coming from the public water system without \*318 the presentations of any substantiating evidence from your epidemiologic investigations appears premature and prejudice toward that end;
- “[i]t is highly unlikely that legionella would be present in treated water coming from the City of Flint water treatment plan[t] given the treatment plant’s use of ozone along with complete treatment and chlorine disinfect contact time to comply with federal surface water treatment rules for potable water;” and
- “there is no direct correlation that can be made to the presence of legionella.”

*Id.* at 85–86, ¶ 253 (Page ID #17888–89). “That same day, Wurfel wrote in an email to Snyder administration officials, ‘Political flank cover out of the City of Flint today regarding the spike in Legionnaire cases.... Also, area ministers put a shot over the bow last night ... with a call for Snyder to declare a state of emergency there and somehow “fix” the water situation....’ ” *Id.* at 86, ¶ 254 (Page ID #17889).

On March 25, 2015, the Flint City Council voted to re-connect to the DWSD. *Id.* at 86, ¶ 255 (Page ID #17889). Ambrose rejected their vote. *Id.*

On April 24, 2015, MDEQ Water Treatment Specialist Patrick Cook admitted in an email to Miguel Del Toral of the EPA that “Flint is currently not practicing corrosion control at the [F]WTP.” *Id.* at 86–87, ¶ 257 (Page ID #17889–90). In the same email, however, Cook “misled the EPA regarding the necessity of using corrosion control in Flint after the switch,” *id.* at 83, ¶ 247 (Page ID #17886), touting distorted water quality test results that showed that the water was within the regulatory limit of 15 ppb for lead, R. 735-3 (Cook Email at 2) (Page ID #20343).

On April 28, 2015, Governor Snyder’s chief of staff told Snyder and other staff members that “[t]he water issue continues to be a danger flag.” R. 620-3 (Fourth Am. Compl. at 87, ¶ 258) (Page ID #17890).

On June 24, 2015, Del Toral released an EPA report (the “Del Toral Report”) warning of high lead levels in Flint water. *Id.* at 87, ¶ 259 (Page ID #17890). On the following day, Del Toral wrote an internal email with respect to the elevated lead in Flint water at EPA stating:

I understand that this is not a comfortable situation, but the State is complicit in this and the public has a right to know what they are doing because it is their children that are being harmed.

*Id.* He “further warned that the failure to inform Flint water users of the elevated lead levels was ‘bordering on criminal neglect.’ ” *Id.* at 87, ¶ 260 (Page ID #17890). The Del Toral Report was shared with MDEQ officials Shekter-Smith, Cook, Busch, and Prysby. *Id.* at 87, ¶ 261 (Page ID #17890). State and City officials did nothing. *Id.* at 88, ¶ 262 (Page ID #17891).

On July 9, 2015, City Utilities Administrator Michael Glasgow emailed MDEQ Water Quality Analyst Adam Rosenthal the following “Key Points” in all caps:

- 1) Flint has lots of lead pipe, no corrosion control treatment, and has had no legitimate LCR testing for at least a year.
- 2) Amongst low income infants, breast feeding rates are lower, and formula use is higher. Many Flint[ ] residents cannot afford to flush due to higher water rates. They cannot afford bottled water. This is an unprecedented situation and EPA needs to take this seriously. Now.

3) We have one child with an elevated blood lead already ...  
In fact, that is the only reason we know about any of the above.

\*319 4) MDEQ is still publicly insisting Flint water has tested safe, is safe, and that [F]lint has no violations of any sort.  
*Id.* at 89, ¶ 267 (Page ID #17892).

“On July 10, 2015, MDEQ [Director of Communications] Brad Wurfel, in an effort to conceal the public health crisis, appeared on public radio and advised listeners that Flint water was safe and that it was not causing ‘any broad problem’ with lead leaching into residential water.” *Id.* at 88, ¶ 265 (Page ID #17891). Wurfel knowingly lied and assured parents in particular that “anyone who is concerned about lead in the drinking water can relax.” *Id.*

On July 22, 2015, Governor Snyder’s Chief of Staff wrote to the Director of MDHHS that residents’ concerns were being “blown off” by the Defendants. *Id.* at 89, ¶ 268 (Page ID #17892). Around the same time, Snyder’s Director of Urban Initiatives spoke to Snyder directly and “advised him of the growing concerns among Flint residents that they were being exposed to toxic levels of lead.” *Id.* at 89, ¶ 269 (Page ID #17892).

On July 24, 2015, Wurfel publicly stated that “residents of Flint do not need to worry about lead in their water supply, and DEQ’s recent sampling does not indicate an imminent health threat from lead or copper.” *Id.* at 89–90, ¶ 270 (Page ID #17892–93). But the sampling Wurfel referenced was “purposefully skewed ... to minimize the crisis.” *Id.* at 90, ¶ 271 (Page ID #17893). Glasgow would later confess that the MDEQ altered water quality reports by removing the highest lead levels—“we threw out bottles everywhere just to collect as many as we can, just to hit our number.” *Id.*; *see also id.* at 91, ¶ 273 (Page ID #17894). Glasgow also “distort[ed] the City’s water test results by instructing residents to run their water—or ‘flush’ it—before testing, and fail[ed] to obtain water from certain houses.” *Id.* at 90–91, ¶ 272 (Page ID #17893–94). He claims that he skewed the samples at Busch’s and Prysby’s direction. *Id.* at 91, ¶ 273 (Page ID #17894).

When a July 2015 water quality report was altered to exclude some high lead levels, Rosenthal forwarded it on. *Id.* Rosenthal was investigated for “willful participation in the manipulation of lead testing results and falsely report[ing]

that the 90th percentile of the results for lead water testing was below the federal action level.” *Id.*

In August 2015, Professor Marc Edwards from Virginia Tech publicly announced that the City of Flint was experiencing a major public health emergency. *Id.* at 91, ¶ 274 (Page ID #17894). Wurfel countered his announcement by stating that Professor Edwards and his team “only just arrived in town and (have) quickly proven the theory they set out to prove, and while the state appreciates academic participation in this discussion, offering broad, dire public health advice based on some quick testing could be seen as fanning political flames irresponsibly.” *Id.* at 92, ¶ 275 (Page ID #17895).

In the summer of 2015, Dr. Mona Hanna-Attisha published her own study to alert Flint residents to the dangers of drinking Flint River water. *Id.* at 93, ¶ 279 (Page ID #17896). Dr. Hanna-Attisha’s study showed a “spike in the percentage of Flint children with elevated blood lead levels from blood drawn in the second and third quarter of 2014.” *Id.* Although MDHHS had data of its own indicating a similar spike, *id.* at 92, ¶ 276 (Page ID #17895), Wurfel lied and stated on September 25, 2015, that “MDHHS officials have re-examined its blood lead level data and the MDHHS statistics do not show the same upward trend documented by Dr. Hanna-Attisha,” *id.* at 94, ¶ 283 (Page ID \*320 #17897). “On September 28, 2015, Wurfel stated publicly that the Flint water crisis was becoming ‘near-hysteria’ because of Dr. Hanna-Attisha’s report. He said that he wouldn’t call her reports ‘irresponsible. I would call them unfortunate.’ Wurfel finished his remarks that day by falsely stating that ‘Flint’s drinking water is safe in that it’s meeting state and federal standards.’” *Id.* at 94, ¶ 284 (Page ID #17897).

Over a year into the crisis, on October 8, 2015, Governor Snyder finally ordered the City of Flint to reconnect with the DWSD. *Id.* at 95, ¶ 287 (Page ID #17898). The City made the switch on October 16, 2015. *Id.* at 95, ¶ 288 (Page ID #17898). On October 18, 2015, the Director of the MDEQ emailed Governor Snyder and admitted that failing to implement optimized corrosion control for an entire year while Flint residents were being poisoned was a mistake. *Id.* at 95–96, ¶ 290 (Page ID #17898–99). The Governor’s own task force on the crisis reported in March 2016 that the Governor’s office failed to act, or even to conduct a comprehensive review of the water situation in Flint, in part because of cost. *Id.* at 150–51, ¶¶ 420–21 (Page ID #17953–54).

#### D. Aftershock

“Flint is currently in a state of crisis: Mayor Karen Weaver declared a State of Emergency on December 14, 2015 and on January 4, 2016, the Genesee County Commissioners declared a State of Emergency.” *Id.* at 96–97, ¶ 294 (Page ID #17899–17900). Governor Snyder did the same on January 5, 2016, but chose not to disclose the threat of *legionella*. *Id.* at 97, ¶ 295 (Page ID #17900). He disclosed that threat for the first time on January 13, 2016, on the same day that he activated the Michigan National Guard to assist the City of Flint. *Id.* at 97, ¶ 296 (Page ID #17900).

The water crisis has created persistent harms. The effects of [lead poisoning](#) are “catastrophic,” particularly for young children. *Id.* at 104–05, ¶ 314 (Page ID #17907–08). “In children, low levels of exposure have been linked to damage to the central and peripheral nervous system, learning disabilities, shorter stature, impaired hearing, and impaired formation and function of blood cells.” *Id.* (quoting EPA). “[L]ead affects children’s brain development resulting in reduced intelligence quotient (IQ), behavioral changes such as shortening of attention span and increased antisocial behavior, and reduced educational attainment... The neurological and behavioral effects of lead are believed to be irreversible.” *Id.* at 105, ¶ 315 (Page ID #17908) (quoting World Health Organization). In some cases, “ingestion of lead can cause seizures, coma and even death.” *Id.* at 105, ¶ 316 (Page ID #17908) (quoting EPA). In pregnant women, the fetus can be exposed to lead in the mother’s body, causing reduced growth and premature birth. *Id.* at 105, ¶ 317 (Page ID #17908). “Flint’s children have suffered specific, measurable damages in the form of lost earning potential. They have also incurred damages in the form of required special educational, medical, sociological, occupational and disability services, and related education assistance programs.” *Id.* at 106–07, ¶ 322 (Page ID #17909–10).

In adults, lead exposure can damage cardiovascular, kidney, and reproductive functions. *Id.* at 107, ¶ 323 (Page ID #17910). A recent study shows a drastic drop in fertility following the water crisis. *Id.* at 107, ¶ 324 (Page ID #17910). “Given the long-lasting risks of lead exposure and the potential for lead sediment to be disturbed and re-mobilized into the water system, Plaintiffs will require regular medical and tap water testing and evaluation, at bare minimum, in accordance with government \*321 standards.” *Id.* at 107, ¶ 325 (Page ID #17910).

“Although the City has begun adding polyphosphate to its system to reduce the leaching of lead from its service lines,

this is unlikely to render Flint’s water safe because many of the pipes have become so corroded that not even phosphate will be able to fully encapsulate the surface of the pipes and prevent lead from leaching into the water supply.” *Id.* at 109, ¶ 331 (Page ID #17912). The same problem applies to home pipes and appliances—meaning that solely replacing municipal pipes will not fix the health crisis. *Id.* at 109–12, ¶¶ 332–40 (Page ID #17912–15); *id.* at 119, ¶ 359 (Page ID #17922). And because “the health effects of lead poisoning often go undetected for some time,” there is a need for “ongoing medical monitoring[,] educational programs[,] and] other remedial programs.” *Id.* at 119–20, ¶ 360 (Page ID #17922–23). In many ways, the crisis has never ended.

### E. Procedural History

This case is a consolidated class action in the *In re Flint Water Cases* litigation. See R. 173 (Order Consolidating Cases) (Page ID #8072). The only claim before us on appeal is Plaintiffs-Appellants’ 42 U.S.C. § 1983 substantive due process claim for deprivation of bodily integrity. The Putative Class includes Flint residents and businesses, but only Flint residents are parties to this appeal. The Defendants include City and State officials, the City of Flint, and private engineering firms, but only the government defendants are parties to this appeal.

Plaintiffs filed their First Amended Consolidated Class Action Complaint on September 29, 2017. R. 214 (1st Am. Compl.) (Page ID #8494). They filed a Second Amended Complaint on October 27, 2017. R. 238 (2d Am. Compl.) (Page ID #8737). Defendants then filed motions to dismiss under Rule 12 of the Federal Rules of Civil Procedure. See R. 273 (Mot. to Dismiss) (Page ID #9797); R. 274 (Mot. to Dismiss) (Page ID #9909); R. 276 (Mot. to Dismiss) (Page ID #9986); R. 277 (Mot. to Dismiss) (Page ID #10111); R. 278 (Mot. to Dismiss) (Page ID #10167); R. 279 (Mot. to Dismiss) (Page ID #10237); R. 281 (Mot. to Dismiss) (Page ID #10644); R. 282 (Mot. to Dismiss) (Page ID #10789); R. 283 (Mot. to Dismiss) (Page ID #10931); R. 294 (Mot. to Dismiss) (Page ID #11358). Before those motions were resolved, Plaintiffs filed a Third Amended Complaint on January 25, 2018. R. 349 (Third Am. Compl.) (Page ID #11759).

On August 1, 2018, the district court issued an opinion and order granting in part and denying in part the motions to dismiss. *Carthan v. Snyder (In re Flint Water Cases)*, 329 F. Supp. 3d 369 (E.D. Mich. 2018). Some Defendants appealed, while others filed motions for reconsideration. R. 560 (Mot.

for Recons.) (Page ID #17043); R. 561 (Mot. for Recons.) (Page ID #17072); R. 570 (Notice of Appeal) (Page ID #17246); R. 573 (Notice of Appeal) (Page ID #17253); R. 575 (Notice of Appeal) (Page ID #17256); R. 579 (Notice of Appeal) (Page ID #17281); R. 589 (Notice of Appeal) (Page ID #17316). We declined to adjudicate the appeals until the district court resolved the motions for reconsideration. Notice of Abeyance, *Waid v. Snyder*, No. 18-1967, slip op., 2019 WL 4121023 (6th Cir. Feb. 19, 2019). But before the district court could resolve those motions, Plaintiffs moved for leave to amend the Complaint, attaching a proposed Fourth Amended Complaint. R. 620 (Mot. for Leave to File Fourth Am. Compl.) (Page ID #17764).

To dispose of the essentially competing motions, the district court “adopted an \*322 unorthodox but necessary plan.” R. 798 (Op. & Order at 5) (Page ID #21107).<sup>6</sup> The district court “interpreted plaintiffs’ motion [for leave to amend the complaint] as a joint motion for relief from judgment and a motion for leave to file an amended complaint. Finding just cause, the Court vacated its August 1 decision on November 9, 2018, so that it could consider plaintiffs’ motion for leave to amend.” *Id.* Because there was significant overlap between the Third Amended Complaint and the proposed Fourth Amended Complaint, and because the standards for leave to amend and Rule 12 dismissal are substantively the same, the district court adjudicated all pending motions in a single opinion and order. *Id.* at 5–6 (Page ID #21107–08). Accordingly, the district court “issue[d] an omnibus opinion and order, adjudicating plaintiffs’ motion for leave to file a fourth amended complaint, and, if successful, defendants’ motions to dismiss it in a single decision.” *Id.* (entered April 1, 2019). The district court granted Defendants-Appellants’ motions to dismiss Plaintiffs’ claims alleging § 1983 equal-protection violations, § 1985(3) conspiracy, Michigan’s Elliott Larsen Civil Rights Act (“ELCRA”), § 1983 state-created danger, and gross negligence. *Id.* at 128 (Page ID #21230). But the district court denied Defendants-Appellants’ motions to dismiss Plaintiffs-Appellees’ § 1983 bodily-integrity claim on the bases of qualified and absolute immunity.<sup>7</sup> *Id.* at 127 (Page ID #21229).

Plaintiffs filed a Motion for Reconsideration of the April 1 order on April 15, 2019, regarding certain claims not at issue on this appeal. R. 809 (Pls. Mot. for Recons.) (Page ID #21864). The district court disposed of that motion on June 11, 2019. R. 880 (Order Den. Pls. Mot. for Recons.) (Page ID #23632).

## II. JURISDICTION

[1] [2] Under the collateral order doctrine, we have jurisdiction over the City and State officials' interlocutory appeals of the district court's denial of qualified immunity to the extent they raise legal questions. *Mitchell v. Forsyth*, 472 U.S. 511, 526–27, 105 S.Ct. 2806, 86 L.Ed.2d 411 (1985); *Bunkley v. City of Detroit*, 902 F.3d 552, 559 (6th Cir. 2018). The collateral order doctrine also provides us with jurisdiction over the City of Flint's and Governor Whitmer's interlocutory appeals from the district court's denial of Eleventh Amendment sovereign immunity. See *Puerto Rico Aqueduct & Sewer Auth. v. Metcalf & Eddy, Inc.*, 506 U.S. 139, 147, 113 S.Ct. 684, 121 L.Ed.2d 605 (1993). We accordingly have jurisdiction over each party's appeal. See 28 U.S.C. § 1291.

## III. DISCUSSION

[3] Defendants-Appellants argue that they are immune from suit and that the district court should have granted their Rule 12(b)(6) motions to dismiss Plaintiffs-Appellees' § 1983 bodily-integrity claim. "Given this procedural posture, we construe the complaint in the light most favorable to plaintiffs, accept all well-pleaded factual allegations as true, and draw all reasonable inferences in plaintiffs' favor." *Guertin*, 912 F.3d at 916. At the same time, Plaintiffs' factual allegations must state a plausible claim. *Id.* (citing \*323 *Bell Atl. Corp. v. Twombly*, 550 U.S. 544, 556–58, 127 S.Ct. 1955, 167 L.Ed.2d 929 (2007)).

Defendants-Appellants are City and State officials, sued in their individual capacities; the City of Flint; and Governor Whitmer, sued in her official capacity. We have already decided key issues of law in this case that came up in separate appeals:

- (1) the creation and cover-up of the Flint Water Crisis violated Flint residents' substantive due process right to bodily integrity, *Guertin*, 912 F.3d at 921;
- (2) that right was clearly established at the time, *id.* at 934;
- (3) the City of Flint is not entitled to Eleventh Amendment immunity, even though it was under State Emergency Manager control during the crisis, *id.* at 936; and

- (4) a request for prospective injunctive relief in the form of compensatory education, medical monitoring, and evaluation services can be pursued against the current Governor in her official capacity under *Ex parte Young*, 209 U.S. 123, 28 S.Ct. 441, 52 L.Ed. 714 (1908), *Boler*, 865 F.3d at 412–13.

Some (but not all) Defendants-Appellants were parties to the *Guertin* appeal and were denied qualified immunity in that case.

### A. Qualified Immunity

[4] [5] The Defendant-Appellant City and State officials argue that qualified immunity shields them from suit. We review de novo a district court's decision to deny qualified immunity. See *Sutton v. Metro. Gov't of Nashville & Davidson Cty.*, 700 F.3d 865, 871 (6th Cir. 2012). "Qualified immunity shields federal and state officials from money damages unless a plaintiff pleads facts showing (1) that the official violated a statutory or constitutional right, and (2) that the right was clearly established at the time of the challenged conduct." *Ashcroft v. al-Kidd*, 563 U.S. 731, 735, 131 S.Ct. 2074, 179 L.Ed.2d 1149 (2011) (internal quotation marks omitted); see also *Guertin*, 912 F.3d at 917. Plaintiffs allege that Defendants-Appellants violated their substantive due process right to bodily integrity. R. 620-3 (Fourth Am. Compl. at 167–69, ¶¶ 463–70) (Page ID #17970–72). Because Plaintiffs do not allege that Defendants-Appellants intended to harm them, Plaintiffs-Appellees must demonstrate that they acted with deliberate indifference. *Guertin*, 912 F.3d at 926. The district court found Plaintiffs' allegations sufficient to state a claim against Defendants-Appellants. R. 798 (Op. & Order at 100) (Page ID #21202).

[6] [7] In *Guertin*, we held that City and State officials' role in creating, sustaining, and covering up the Flint Water Crisis violated Flint residents' right to bodily integrity, *Guertin*, 912 F.3d at 921, and that this right was clearly established at the time, *id.* at 934. The substantive due process clause of the Fourteenth Amendment protects against conscience-shocking deprivations of liberty. *Id.* at 918. Violating a person's bodily integrity is a grave deprivation of their liberty. See *id.* at 918–19. The *Guertin* plaintiffs were deprived of their bodily integrity when government officials forcibly invaded their bodies by misleading them into consuming a life-threatening substance. *Id.* at 920–22. Once that hurdle is met, whether the alleged conduct amounts to deliberate indifference depends on the circumstances, including whether the defendants had time to deliberate, whether there was an

involuntary relationship, and whether there was a legitimate government purpose. See *id.* at 922–26. Each of these factors weighed against the defendants in *Guertin*. *Id.* at 925–26. And what was true there is true here: “the \*324 generally alleged conduct [i]s ... egregious.” *Id.* at 925.

The parties agree that lead and *legionella* are life-threatening substances and that these contaminants spread to residents through the water supply. R. 798 (Op. & Order at 43) (Page ID #21145). Flint residents had no choice but to receive their water through the City’s water plan. See *Guertin*, 912 F.3d at 925 (citing Flint City Charter § 4-203(A); Flint Code of Ord. §§ 46-7, 46-50(b), 46-51, 46-52). On top of that, “various defendants’ assurances of the water’s potability hid the risks, turning residents’ voluntary consumption of a substance vital to subsistence into an involuntary and unknowing act of self-contamination.” *Id.* at 925–26. The Flint Water Crisis was a “predictable harm” set into motion by alleged decisions that “took place over a series of days, weeks, months, and years.” See *id.* at 925. Given officials’ ample time to deliberate, “this known risk cannot be excused on the basis of split-second decision making.” See *id.* Worse, the officials stood their ground. The crisis was undeniable, but they refused to switch the City back to clean water, or even to take the meager step of introducing corrosion control, or even to admit that the water was poisoned. “When such extended opportunities to do better are teamed with protracted failure even to care, indifference is truly shocking.” *County of Sacramento v. Lewis*, 523 U.S. 833, 853, 118 S.Ct. 1708, 140 L.Ed.2d 1043 (1998).

No legitimate government purpose justifies the City and State officials’ actions. See *Guertin*, 912 F.3d at 926. “[J]ealously guarding the public’s purse cannot, under any circumstances, justify the yearlong contamination of an entire community.” *Id.* The question remains whether each Defendant-Appellant’s alleged actions individually amount to deliberate indifference. See *id.*

[8] To state a claim for bodily integrity, Plaintiffs-Appellees must demonstrate that the officials’ actions “shock the conscience”—here, through deliberate indifference. See *Guertin*, 912 F.3d at 922, 926; see also *Claybrook v. Birchwell*, 199 F.3d 350, 359 (6th Cir. 2000). The standard for deliberate indifference is subjective recklessness. *Guertin*, 912 F.3d at 926. “[P]laintiffs must show the government officials ‘knew of facts from which they could infer a substantial risk of serious harm, that they did infer it, and that they acted with indifference toward the individual’s rights.’

” *Id.* (quoting *Range v. Douglas*, 763 F.3d 573, 591 (6th Cir. 2014)).

[9] Critically, this case comes to us at the motion to dismiss stage. The allegations in the Complaint must be taken as true. *Id.* at 916. Some judges of this court have even noted that, because the facts at this stage are yet undeveloped, “it is generally inappropriate for a district court to grant a 12(b) (6) motion to dismiss on the basis of qualified immunity. Although an officer’s entitlement to qualified immunity is a threshold question to be resolved at the earliest possible point, that point is usually summary judgment and not dismissal under Rule 12.” *Wesley v. Campbell*, 779 F.3d 421, 433–34 (6th Cir. 2015) (internal quotation omitted). With these principles in mind, Plaintiffs-Appellees have plausibly alleged that Defendants-Appellants violated their right to bodily integrity.

### 1. City Officials

[10] Defendant-Appellant City Officials include Emergency Managers Earley and Ambrose, Public Works Director Croft, and Utilities Administrators Glasgow and Johnson. The *Guertin* court described Earley, Ambrose, and Croft as “instrumental in creating the crisis.” 912 F.3d at 926. We have not had the opportunity previously \*325 to address the conduct of Glasgow and Johnson.

All of the Defendant-Appellant City Officials argue that they are entitled to qualified immunity because they acted based on professional opinions from MDEQ officials and private engineering firms. See *Butz v. Economou*, 438 U.S. 478, 507, 98 S.Ct. 2894, 57 L.Ed.2d 895 (1978) (“Federal officials will not be liable for mere mistakes in judgment, whether the mistake is one of fact or one of law.”); Appellant Br. (19-1425) at 25, 27–29, 31–34. We have already held, however, that, “[t]o the extent these defendants claim ‘mistakes in judgment’ because they reasonably relied upon the opinions of Michigan Department of Environmental Quality (MDEQ) employees and professional engineering firms, those are facts to be fleshed out during discovery and are not appropriate to resolve at the motion-to-dismiss posture.” *Guertin*, 912 F.3d at 927 (citation omitted). The same reasoning applies here. At this stage, we must credit Plaintiffs’ allegation that the Defendant-Appellant City Officials had independent knowledge that the Flint River water was causing a public health crisis—regardless of what the MDEQ or the engineering firms reported.

### a. Earley

[11] Darnell Earley was Emergency Manager for the City from September 2013 (prior to the crisis) to January 2015 (in the midst of the crisis). Earley forced the switch to Flint River water when he knew that the FWTP was not ready and that it was important that the water be treated. R. 620-3 (Fourth Am. Compl. at 51, ¶ 147) (Page ID #17854); *see also* [Guertin, 912 F.3d at 927](#). Plaintiffs-Appellees also allege that Earley directed City officials to lie to the public and tell them that the [Legionnaires' disease](#) outbreak in the summer of 2014 “was ‘an internal issue at McLaren [Hospital] that they are working on with our assistance, not a Flint water problem that we are trying to resolve.’ ” R. 620-3 (Fourth Am. Compl. at 59, ¶ 175) (Page ID #17862). Even after he was briefed on water quality issues by the MDEQ in the fall of 2014, Earley refused to reconnect to the DWSD. *Id.* at 60–61, ¶¶ 180–81 (Page ID #17863–64). He again refused to reconnect to the DWSD in January 2015, when officials were aware of the lead and *legionella* problems and after the University of Michigan ceased use of Flint River drinking water because of lead contamination. *Id.* at 62, ¶¶ 185–86 (Page ID #17865). These actions plausibly demonstrate deliberate indifference to the crisis that would likely result.

### b. Ambrose

Gerald Ambrose took over as Emergency Manager for the City of Flint in January 2015 (in the midst of the crisis). Prior to that, he had served as Earley’s advisor, and had been notified about the [Legionnaires' disease](#) outbreak in the summer of 2014. *Id.* at 59, ¶ 175 (Page ID #17862). Like Earley, he repeatedly refused to reconnect to the DWSD—showcasing an indifference that was “especially egregious” in light of the undeniable and worsening crisis. *See* [Guertin, 912 F.3d at 927](#). After State officials installed water coolers in Flint offices and the GCHD reported that the outbreak of Legionnaires’ likely was connected to the use of Flint River water, the DWSD offered Ambrose a deal for reconnecting in January 2015. R. 620-3 (Fourth Am. Compl. at 81, ¶ 239) (Page ID #17884). He refused. *Id.* In February 2015, Flint residents publicly demanded reconnecting to the DWSD, and he again refused. *Id.* at 82, ¶ 243 (Page ID #17885). In March 2015, the Flint City Council voted to re-connect to DWSD. *Id.* at 86, ¶ 255 (Page ID #17889). Ambrose rejected their vote. *Id.* City and State officials were well \*326 aware of the crisis by January 2015 and were under the scrutiny of

the GCHD and the EPA by March 2015. Ambrose’s staunch refusal to stop use of Flint River water in spite of what he knew plausibly demonstrates deliberate indifference to the crisis.

### c. Glasgow

[12] Michael Glasgow was a City Utilities Administrator, and the City’s water treatment plant laboratory and water quality supervisor. Prior to making the switch to the Flint River, he knew that the FWTP was not ready and that the City would be distributing contaminated water. *Id.* at 46, ¶ 129 (Page ID #17849). He tried to stop the switch from happening but nevertheless participated in the transition. *Id.* He later told State investigators that Croft and Johnson, who were his superiors, pressured him to make the switch. *Id.* at 47, ¶ 130 (Page ID #17850). Plaintiffs-Appellees concede that Glasgow’s conduct in implementing the switch did not demonstrate deliberate indifference. *See* Oral Argument at 1:12:52–1:13:10.

What Plaintiffs-Appellees take issue with is Glasgow’s later role in covering up the extent of lead contamination. In July 2015, Glasgow wrote to Rosenthal that “Flint has lots of lead pipe, no corrosion control treatment” and that “[t]his is an unprecedented situation and EPA needs to take this seriously. Now.” *Id.* at 89, ¶ 267 (Page ID #17892). Despite what he knew, he distorted water quality tests to downplay the extent of the lead contamination. *Id.* at 89–91, ¶¶ 270–72 (Page ID #17893–94). Glasgow claims that he did so at the direction of MDEQ officials Busch and Prysby. *Id.* at 91, ¶ 273 (Page ID #17894). But as Plaintiffs-Appellees point out, Busch and Prysby were MDEQ (not City) officials who, unlike Croft and Johnson, had no authority over him. The facts, when fully developed, ultimately might show that Glasgow truly was coerced into distorting the water quality tests, so that he cannot be said to have acted with deliberate indifference. But at this stage, the allegations plausibly support a reasonable inference that he did act with deliberate indifference when he helped to cover up the crisis.

### d. Croft

[13] Howard Croft was Public Works Director for the City of Flint. Croft permitted the switch to the Flint River even though he knew that the FWTP was not prepared to deliver safe drinking water. *Id.* at 47, ¶ 130 (Page ID #17850); *see*

also *Guertin*, 912 F.3d at 927. In fact, Glasgow stated that Croft pressured him to make the switch despite Glasgow’s warnings. R. 620-3 (Fourth Am. Compl. at 47, ¶ 130) (Page ID #17850). Croft also knew from the GCHD that the [Legionnaires’ disease](#) outbreak was connected to Flint River water, and he did nothing. *Id.* at 138, ¶ 401 (Page ID #17941). His alleged role in creating and failing to mitigate the crisis plausibly demonstrates deliberate indifference.

### c. Johnson

[14] Daugherty Johnson was another City Utilities Administrator. Along with Croft, he purportedly pressured Glasgow to make the switch to the Flint River despite Glasgow’s warnings. *Id.* at 47, ¶ 130 (Page ID #17850). He also stonewalled the GCHD’s attempt to investigate Flint River water quality issues and the outbreak of [Legionnaires’ disease](#). *Id.* at 83–84, ¶¶ 248–50 (Page ID #17886–87). His alleged role in creating and covering up the crisis plausibly demonstrates deliberate indifference.

## 2. MDEQ Officials

[15] Defendant-Appellant MDEQ Officials include State agency employees who \*327 permitted the switch to the Flint River, distorted water quality tests, and resisted concerns from other agencies like the EPA and the GCHD regarding the quality of Flint River water. In *Guertin*, we stated that the MDEQ Officials—there, Busch, Shekter-Smith, Prysby, and Wurfel—“played a pivotal role in authorizing Flint to use its ill-prepared water treatment plant to distribute drinking water[,] ... falsely assured the public that the water was safe[,] and attempted to refute assertions to the contrary.” 912 F.3d at 927. We have not had the opportunity previously to address the conduct of Rosenthal and Cook.

The MDEQ Officials argue that they decided not to use corrosion control based on a mistaken, but reasonable, interpretation of the EPA Lead and [Copper](#) Rule. Appellant Br. (19-1477) at 3–4, 38, 45. But as we stated in *Guertin*, “[t]o the extent these defendants made ‘honest mistakes in judgment’—in law or fact—in interpreting and applying the Lead and [Copper](#) Rule, that defense is again best reserved for after discovery.” 912 F.3d at 928 (citation omitted). At this stage, we must accept the reasonable inference from Plaintiffs’ allegations that, whatever the MDEQ’s purported justifications for its actions, it rushed the switch to the Flint River knowing it would deliver contaminated water and that

the decision-makers cared only about cost, not water quality. Their purported defense also does not explain why they failed to treat the water after they came under the EPA’s scrutiny, or why they lied to the EPA.

Plaintiffs-Appellees plausibly allege a constitutional violation for each Defendant-Appellant MDEQ Official for the reasons stated below.

### a. Shekter-Smith

[16] Liane Shekter-Smith was the MDEQ Chief of the Office of Drinking Water and Municipal Assistance. Despite knowing that Flint River water presented health risks, *see* R. 620-3 (Fourth Am. Compl. at 40, ¶ 105) (Page ID #17843), she secured the necessary administrative consent order (or ACO) and rushed the switch to the Flint River before the FWTP was ready, *see id.* at 45–46, ¶ 128 (Page ID #17848–49). When reports poured in from residents that something was wrong with the water and that it was making them ill, she did nothing. *See id.* at 57, ¶¶ 165–67 (Page ID #17860). After privately suggesting that the water might be contaminated, *id.* at 85, ¶ 252 (Page ID #17888), she publicly combatted the GCHD’s *legionella* analysis, *id.* at 85–86, ¶¶ 252–53 (Page ID #17888–89). And she did nothing to mitigate the crisis even after the Del Toral Report blew the whistle on high lead levels in Flint’s water. *Id.* at 87–88, ¶¶ 259–62 (Page ID #17890–91). Her alleged role in creating, failing to mitigate, and covering up the crisis plausibly demonstrates deliberate indifference.

### b. Rosenthal

[17] Adam Rosenthal was the MDEQ Water Quality Analyst. He did not stop the switch to the Flint River in spite of Glasgow’s warning that the FWTP was not ready. *Id.* at 46, ¶ 129 (Page ID #17849). He knew as early as May 2014 that the water contained high TTHM levels that were above regulation (and indicated lead contamination), and did nothing. *Id.* at 58, ¶ 172 (Page ID #17861). In July 2015, Glasgow wrote to him that “Flint has lots of lead pipe, no corrosion control treatment” and that “[t]his is an unprecedented situation and EPA needs to take this seriously. Now.” *Id.* at 89, ¶ 267 (Page ID #17892). Yet, Glasgow wrote, “MDEQ is still publicly insisting Flint water has tested safe, is safe, and that [F]lint has no \*328 violations of any sort.” *Id.* Rosenthal, apparently unmoved, soon afterward distributed a distorted water quality report that was altered to exclude high lead levels. *Id.* at

91, ¶ 273 (Page ID #17894). He has also been accused of manipulating and falsely reporting the test results. *Id.* His alleged role in creating, failing to mitigate, and covering up the crisis plausibly demonstrates deliberate indifference.

### c. Busch

[18] Stephen Busch was the MDEQ District Supervisor. Busch knew as early as March 2013 that Flint River water presented health risks and would require significant treatment, *id.* at 40, ¶ 105 (Page ID #17843), but he did not stop the switch to the Flint River even after Glasgow warned him that the FWTP was not ready, *id.* at 46, ¶ 129 (Page ID #17849); *see also Guertin*, 912 F.3d at 927. When the MDEQ came under the EPA’s scrutiny for lead contamination, Busch lied and told Del Toral that the City was using corrosion control. R. 620-3 (Fourth Am. Compl. at 83, ¶ 246) (Page ID #17886); *see also Guertin*, 912 F.3d at 928. Busch claims that he did not lie and that, instead, he simply informed the EPA that the City had a corrosion control program in place, meaning that the City was monitoring the water without treating it. *See* Appellant Br. (19-1477) at 54. That is quibbling with the facts and asks us to do what we cannot at this stage—to view the allegations in the light most favorable to him. *See Guertin*, 912 F.3d at 916. Plaintiffs’ allegation stands.

Busch also lied to the GCHD. He told them that the evidence did not support a connection between the outbreak of Legionnaires’ disease and the switch to the Flint River. R. 620-3 (Fourth Am. Compl. at 85–86, ¶ 253) (Page ID #17888–89). And according to Glasgow, Busch directed him to distort water quality tests to exclude high results for lead contamination. *Id.* at 91, ¶ 273 (Page ID #17894). Busch’s alleged role in creating, failing to mitigate, and covering up the crisis plausibly demonstrates deliberate indifference.

### d. Prysby

[19] Michael Prysby worked under Busch as an MDEQ Engineer for District 11, which serviced the City of Flint. Along with Busch, he did not stop the switch to the Flint River in the face of Glasgow’s warnings, *id.* at 46, ¶ 129 (Page ID #17849); *see also Guertin*, 912 F.3d at 927; he did nothing in response to the Del Toral Report, R. 620-3 (Fourth Am. Compl. at 87–88, ¶¶ 259–62) (Page ID #17890–91); and he purportedly directed Glasgow to distort water quality tests

to exclude high results for lead contamination, *id.* at 91, ¶ 273 (Page ID #17894). His alleged role in creating, failing to mitigate, and covering up the crisis plausibly demonstrates deliberate indifference.

### e. Cook

[20] Patrick Cook was the MDEQ Water Treatment Specialist. He signed the permit that was the last necessary approval for the (rushed) use of Flint River water and the FWTP. *Id.* at 47, ¶ 132 (Page ID #17850). Like other officials, he at first did nothing in response to the Del Toral Report. *Id.* at 87–88, ¶¶ 259–62 (Page ID #17890–91). Then, in April 2015, he admitted in an email to Del Toral that “Flint is currently not practicing corrosion control at the [F]WTP,” *id.* at 86–87, ¶ 257 (Page ID #17889–90), after Busch had lied and told the EPA that the City was using corrosion control, *id.* at 83, ¶ 246 (Page ID #17886). In the same email, however, Cook “misled the EPA regarding the necessity of using corrosion control in Flint after the switch.” *Id.* at 83, ¶ 247 (Page ID #329 #17886). Cook contends that the email itself renders Plaintiffs’ reading of it implausible. Reply Br. (19-1477) at 6–7.

[21] When a document attached to the complaint contradicts the allegations, the document trumps the allegations. *Williams v. CitiMortgage, Inc.*, 498 F. App’x 532, 536 (6th Cir. 2012). For a document to contradict the complaint, it must “utterly discredit” the allegations. *Cagayat v. United Collection Bureau, Inc.*, 952 F.3d 749, 755 (6th Cir. 2020) (quoting *Bailey v. City of Ann Arbor*, 860 F.3d 382, 386–87 (6th Cir. 2017)). The email at issue here does not utterly discredit Plaintiffs’ allegations. Though Cook admits at the start of the email that the City is not using corrosion control, he then states that there was and is no need to do so because the Flint River water’s testing results were within the regulatory limit of 15 ppb for lead. R. 735-3 (Cook Email at 2) (Page ID #20343) (“The first round of samples after switch-over from DWSD ... had 90th percentiles of 6 ppb for Lead... The highest lead result out of the 20 [samples] received [from the second round of testing] thus far is 13 ppb.”). Touting allegedly distorted water quality test results and false compliance plausibly was misleading. Therefore, the district court was right to credit Plaintiffs’ allegations. Cook’s alleged role in creating and covering up the crisis plausibly demonstrates deliberate indifference.<sup>8</sup>

## f. Wurfel

[22] Bradley Wurfel was the MDEQ Director of Communications and was instrumental in the coverup. In the summer of 2015, as concerns and criticism reached their peak, he repeatedly lied to the public and assured them that Flint River water was safe. R. 620-3 (Fourth Am. Compl. at 88–90, ¶¶ 265–70) (Page ID #17891–93); *see also* [Guertin](#), 912 F.3d at 928. He told parents that “anyone who is concerned about lead in the drinking water can relax.” R. 620-3 (Fourth Am. Compl. at 88, ¶ 265) (Page ID #17891). He cited distorted water quality tests as evidence that “residents of Flint do not need to worry about lead in their water supply.” *Id.* at 89–90, ¶ 270 (Page ID #17892–93). He even attacked independent whistleblower reports by Professor Edwards and Dr. Hanna-Attisha that stated that the City of Flint was in the midst of a major public health emergency. He accused Professor Edwards of “quickly prov[ing] the theory [he] set out to prove” and decried the “near-hysteria” resulting from Dr. Hanna-Attisha’s report. *Id.* at 92, ¶ 275 (Page ID #17895); *id.* at 94, ¶¶ 283–84 (Page ID #17897); *see also* [Guertin](#), 912 F.3d at 928.

Wurfel asks us to consider the context and totality of the statements he made, but points to nothing that directly negates Plaintiffs’ allegations. *See* Appellant Br. (19-1477) at 50–52. We will not view the allegations in the light most favorable to the defendant—and that is essentially what Wurfel asks us to do. *See* [Guertin](#), 912 F.3d at 916. We also reject his attempt to reargue his position in [Guertin](#) that “mere” public statements cannot violate a person’s right to bodily integrity. *See* Reply Br. (19-1477) at 11–13. The [Guertin](#) court concluded that public statements like those alleged here did amount to a constitutional \*330 violation. 912 F.3d at 929. That decision controls. Wurfel’s alleged role in covering up the crisis plausibly demonstrates deliberate indifference.

## 3. State Officials

The Defendant-Appellant State Officials sued in their individual capacities are Governor Snyder and Treasurer Dillon. We have not had the opportunity previously to address their conduct. We hold that Plaintiffs-Appellees plausibly allege a constitutional violation as to Snyder, but we refrain from deciding this question for Dillon until the district court has an opportunity to reconsider in light of [Brown v. Snyder \(In re Flint Water Cases\)](#), No. 18-cv-10726, 2020 WL 1503256, at \*9 (E.D. Mich. Mar. 27, 2020).

## a. Governor Snyder

Governor Snyder was in office for the entire relevant time period. He “was personally involved in the decisional process which led to the transition from DWSD to the KWA,” *id.* at 42, ¶ 114 (Page ID #17845), having himself coordinated the switch, *id.* at 43, ¶ 115–18 (Page ID #17846). And he knew that the Flint River would serve as the City’s interim water source until the KWA went online. *Id.* at 44, ¶ 119 (Page ID #17847). Prior to the switch, a member of his staff warned him that it “could lead to some big potential disasters down the road.” *See id.* at 45, ¶ 127 (Page ID #17848). In spite of that warning, Snyder did not stop the switch from going forward.

Soon after the switch, there was evidence of corrosion and accompanying lead and *legionella* contamination. *See id.* at 58–60, 62, ¶¶ 173, 177, 187 (Page ID #17861–63, 17865). On October 13, 2014, General Motors stopped using Flint River water at its engine plant out of fear that the water would corrode its machinery. *Id.* at 60, ¶ 179 (Page ID #17863). The next day, a member of Snyder’s executive staff expressed concern with the reports coming out about the water’s contamination and recommended that they ask the Emergency Manager to switch back to the DWSD “as an interim solution to both the quality, and now the financial, problems that the current solution is causing.” *Id.* at 60–61, ¶ 180 (Page ID #17863–64). Snyder’s legal counsel similarly stated that the dangers posed by Flint River water were “downright scary” and “advised that, ‘[t]hey should try to get back on the Detroit system as a stopgap ASAP before this thing gets too far out of control.’ ” *Id.* at 61, ¶ 182 (Page ID #17864). Snyder evidently was unmoved.

In January 2015, the University of Michigan turned off certain water fountains on its Flint campus after tests revealed high levels of lead contamination. *Id.* at 62, ¶ 185 (Page ID #17865). Around the same time, the GCHD reported a likely “association between the spike in [Legionnaires’ disease](#) reports and the onset of the use of Flint River water.” *Id.* at 81, ¶ 237 (Page ID #17884). Meanwhile, State officials had water coolers discreetly installed in State buildings located in Flint, without announcing their concerns to the public. *Id.* at 80, ¶ 235 (Page ID #17883). At some point in 2015, Snyder met with other government officials to discuss the serious threats posed by lead and *legionella* contamination, and his office even considered distributing water filters to protect

Flint water users. *Id.* at 80, ¶ 233 (Page ID #17883); *id.* at 84, ¶ 249 (Page ID #17887). But ultimately Snyder did nothing.

In addition to public reports from whistleblowers, Snyder's own staff kept him personally apprised of the worsening crisis. In April 2015, Snyder's chief of staff emailed Snyder and other staff members \*331 that "[t]he water issue continues to be a danger flag." *Id.* at 87, ¶ 258 (Page ID #17890). Soon afterward, Snyder's Director of Urban Initiatives spoke to Snyder directly and "advised him of the growing concerns among Flint residents that they were being exposed to toxic levels of lead." *Id.* at 89, ¶ 269 (Page ID #17892). Nothing came of it. All the while, Snyder kept the crisis under wraps and stood by as the public continued to be poisoned. The Governor's own task force eventually would disclose that Snyder failed to act in part because of cost. *Id.* at 150–51, ¶¶ 420–21 (Page ID #17953–54).

Finally, after more than a year into the crisis, Snyder relented and ordered the City of Flint to reconnect with the DWSD on October 8, 2015. *Id.* at 95, ¶ 287 (Page ID #17898). He declared a State of Emergency *three months later* on January 5, 2016, and disclosed the *legionella* problem on January 13, 2016. *Id.* at 97, ¶¶ 295–96 (Page ID #17900). "Without a state of emergency, plaintiffs were denied valuable resources that could have helped abate the harm that they were still suffering." R. 798 (Op. & Order at 46–47) (Page ID #21148–49).

[23] Snyder argues in the first instance that he is entitled to qualified immunity because he acted (or failed to act) in reliance on the MDEQ and engineering firms' assessments. *See* Appellant Br. (19-1472) at 37–40. Again, "those are facts to be fleshed out during discovery and are not appropriate to resolve at the motion-to-dismiss posture." *Guertin*, 912 F.3d at 927 (citations omitted). For the same reason, his defense that any alleged disinformation or inaction arose from legitimate disagreements over "the nature and extent of the problems and the appropriate solution" is misplaced at this stage. *See* Reply Br. (19-1472) at 8–11.

[24] We agree with the district court that the allegations against Governor Snyder are sufficient to state a claim for deliberate indifference. *See* R. 798 (Op. & Order at 39–47) (Page ID #21141–49). Unlike the executive defendants in *Guertin*, Snyder personally contributed to creating this crisis. The executives that we decided should have been dismissed in *Guertin* were Wyant, the Director of the MDEQ; Lyon, the Director of the MDHHS; and Wells, the Chief

Medical Executive of the MDHHS. *Guertin*, 912 F.3d at 929–31. Wyant may have been "aware of some of the issues arising with the water supply post-switch," but there were no plausible allegations that "Wyant personally made decisions regarding the water-source switch" or that "he personally engaged" in other conscience-shocking conduct. *Id.* at 929. As for Lyon and Wells, we noted that "[t]he complaint set[ ] forth no facts connecting Lyon and Wells to the switch to the Flint River or the decision not to treat the water, and there [wa]s no allegation that they took any action causing plaintiffs to consume the lead-contaminated water." *Id.* at 929–30. All that the plaintiffs alleged was a general "fail[ure] to 'protect and notify the public' of the problems with Flint's water," rather than allege a particular action taken by Lyon or Wells that would demonstrate their deliberate indifference. *Id.* at 930.

Plaintiffs' allegations here demonstrate that Governor Snyder personally was aware that Flint River water was contaminated and that he personally made the decision to switch the City from the DWSD to Flint River water. The allegations demonstrate that Snyder personally understood not just from public reports, but from *his own staff*, that Flint residents were being poisoned. Plaintiffs' allegations demonstrate that Snyder downplayed the problem and delayed taking action to protect the people of Flint, first by refusing to \*332 switch back to the DWSD, then by failing to supply Flint residents with protective supplies, and finally by waiting three months after the City connected back to the DWSD to declare a state of emergency. Snyder's alleged role in creating, failing to mitigate, and covering up the crisis plausibly demonstrates deliberate indifference.<sup>9</sup>

#### b. State Treasurer Dillon

Andy Dillon was Treasurer for the State of Michigan when the City was in the process of switching to Flint River water. Dillon was asked to assess the cost effectiveness of staying with the DWSD or switching to the KWA. *See id.* at 39–40, ¶ 104 (Page ID #17842–43). Dillon ultimately recommended to Snyder that the Governor authorize the City to switch to the KWA, after Dillon learned that the City could fund the switch with an ACO that would require use of Flint River water in the interim. *Id.* at 41, ¶ 107 (Page ID #17844). Dillon was part of the core team that developed the interim Flint River plan, *see id.* at 44, ¶ 119 (Page ID #17847), and he knew that the FWTP would need to undergo significant upgrades before it could treat the water properly, *id.* at 44, ¶ 122 (Page ID #17847).

In spite of what he knew, the Treasury pressed the MDEQ to secure the ACO quickly, so that the switch to the Flint River would take place before the FWTP was ready. *Id.* at 130, ¶ 383 (Page ID #17933).

Plaintiffs-Appellees ask that we remand for the district court to decide whether to dismiss Dillon from this case. Defendants-Appellants do not protest that request. After we accepted this appeal, the district court dismissed Dillon as a defendant in a separate Flint Water Crisis case, *Brown v. Snyder (In re Flint Water Cases)*, No. 18-cv-10726, 2020 WL 1503256, at \*9 (E.D. Mich. Mar. 27, 2020). The district court recently discovered that Dillon was not Treasurer at the time of the actual switch to Flint River water in April 2014. *Id.* at \*9 n.13. In light of that, the district court found that Dillon did not have authority over the switch and, therefore, that he cannot be found liable. *Id.* Without passing judgment on that decision, we see no issue with Plaintiffs-Appellees' request that we remand for the district court to decide in the first instance whether to dismiss Dillon in light of that fact. See *Lopez v. Foerster*, 791 F. App'x 582, 586 (6th Cir. 2019) ("Although we have jurisdiction to decide the qualified-immunity question, given the unique circumstances of this case, we remand to the district court to consider the issue in the first instance.").

## B. Eleventh Amendment Immunity

[25] The City of Flint and Governor Whitmer argue that they are entitled to Eleventh Amendment sovereign immunity. "Whether Eleventh Amendment sovereign immunity exists in any particular case is a question of constitutional law that we review *de novo*." *Mingus v. Butler*, 591 F.3d 474, 481 (6th Cir. 2010).

[26] The Eleventh Amendment generally bars suits against the State, but generally does not bar suits against cities. \*333 U.S. CONST. amend. XI ("The judicial power of the United States shall not be construed to extend to any suit in law or equity, commenced or prosecuted against one of the United States by Citizens of another State, or by Citizens or Subjects of any Foreign State."); see also *S&M Brands, Inc. v. Cooper*, 527 F.3d 500, 507 (6th Cir. 2008); *Monell v. Dep't of Soc. Servs.*, 436 U.S. 658, 690 n.54, 98 S.Ct. 2018, 56 L.Ed.2d 611 (1978). Two quirks of immunity doctrine are at play in this appeal. The district court correctly concluded that precedent from prior Flint Water cases precludes the City's and Governor Whitmer's arguments in this case.

## 1. City of Flint

[27] The City argues that it is entitled to Eleventh Amendment sovereign immunity because it was under State emergency management during the events leading up to and during the Flint Water Crisis. "Although municipalities typically do not enjoy sovereign immunity, 'arms of the state' do." Appellant Br. (19-1425) at 36; *Metcalfe*, 506 U.S. at 144, 113 S.Ct. 684 ("[A] State and its 'arms' are, in effect, immune from suit in federal court."). We already foreclosed this argument in *Guertin*. The *Guertin* court held that the City was not acting as an arm of the State when it was under State emergency management and, accordingly, that it was not entitled to sovereign immunity. 912 F.3d at 936. The City acknowledges that *Guertin*'s holding is binding on this panel. Appellant Br. (19-1425) at 36 n.15. It makes its present argument "for the purpose of preserving the issue for further appeal," if any. *Id.* We accordingly note that the City has preserved its argument and that we abide by our decision in *Guertin*.

## 2. Governor Whitmer

Plaintiffs seek prospective injunctive relief against Governor Whitmer in her official capacity to combat the ongoing effects from the violation of their constitutional rights. They accordingly seek "[a]n injunctive order to remediate the harm caused by the Government Defendants' unconstitutional conduct including, but not limited to: repairs of private property and establishment of medical monitoring to provide health care and other appropriate services to Class members for a period of time deemed appropriate by the Court." R. 620-3 (Fourth Am. Compl. at 214) (Page ID #18017). They also seek "[a]ppointment of a monitor who will assist in the development of remedial plans including, but not limited to: early education, education intervention programs, criminal and juvenile justice evaluations." *Id.*

Under Rule 25(d) of the Federal Rules of Civil Procedure, the successor to an officer sued in their official capacity is "automatically substituted as a party." Fed. R. Civ. P. 25(d). When Whitmer succeeded Snyder in January 2019, she automatically became a party to this action in her official capacity as Governor. Whitmer argues that she is entitled to sovereign immunity because Plaintiffs fail to plead a proper *Ex parte Young* claim against her.

[28] The State generally is immune from suit, but *Ex parte Young* provides an exception for plaintiffs seeking prospective injunctive relief against State actors in their

official capacity. 209 U.S. at 156, 28 S.Ct. 441; *S&M Brands*, 527 F.3d at 507. Plaintiffs originally pleaded their *Ex parte Young* claims against Governor Snyder, but since Governor Whitmer has taken office, they have not amended their Complaint to include allegations against her personally. Appellant Br. (19-1472) at 52–53. Whitmer argues that, because the alleged unconstitutional conduct occurred solely in the past, the pleadings are deficient to state a claim for prospective injunctive \*334 relief. Plaintiffs-Appellees point out that we rejected a similar argument by Governor Snyder in *Boler*, a previous Flint Water case. 865 F.3d at 412–14.

[29] Plaintiffs-Appellees seek prospective injunctive relief to remediate the ongoing harms stemming from the Flint Water Crisis. This type of relief is proper under *Ex parte Young*. See *Milliken v. Bradley*, 433 U.S. 267, 290, 97 S.Ct. 2749, 53 L.Ed.2d 745 (1977). In *Milliken*, the Supreme Court held that, under *Ex parte Young*, courts could order newly integrated schools to implement remedial education programs in order to combat the lasting effects of *de jure* school segregation. *Id.* “[T]he victims of Detroit’s *de jure* segregated system will continue to experience the effects of segregation,” the Court reasoned, “until such future time as the remedial programs can help dissipate *the continuing effects of past misconduct.*” *Id.* (emphasis added). Like the remedial education programs at issue in *Milliken*, the remedial measures that Plaintiffs-Appellees request here “are plainly designed to wipe out continuing [harms] produced by” the unconstitutional acts of Defendants-Appellants. See *id.*<sup>10</sup>

What was true in *Boler* remains true today: “Damage to the water pipes has been done, and has ongoing effects.” 865 F.3d at 413. The year-long corrosion of public and private water pipes continues to contaminate the water, and the prolonged and extreme exposure to lead—particularly in children and mothers—will leave lasting developmental effects. See *supra* pp. 320–21. Plaintiffs have alleged ongoing effects from constitutional violations, even if the conduct at issue occurred solely in the past. See *Boler*, 865 F.3d at 413. Moreover, Plaintiffs’ requests for repairs, medical monitoring, educational programs, and criminal and juvenile justice evaluations are identical to those sought and upheld in *Boler*. See *Boler*, 865 F.3d at 413.

Nevertheless, Whitmer argues, this case is different because she personally did not commit the initial constitutional violations and she is not alleged to be deliberately indifferent now. That distinction makes no difference. As

Plaintiffs-Appellees aptly state, “[a]n official-capacity suit for prospective relief is simply the vehicle by which the state can be compelled to fix a constitutional violation” committed in the past that has continuing effects. Appellees Br. at 82 (citing *Lewis v. Clarke*, — U.S. —, 137 S. Ct. 1285, 1290–91, 197 L.Ed.2d 631 (2017)). It does not matter what Whitmer *personally* did or did not do in the past, or even in the present. “Injunctive relief is appropriate here, not because the defendants will be deliberately indifferent again in the future, but because the past deliberate indifference has continuing effects.” *Id.* at 83 (citing *Boler*, 865 F.3d at 413). We conclude that the district court \*335 rightly rejected Whitmer’s Eleventh Amendment argument.

#### IV. CONCLUSION

We **AFFIRM** the district court’s denial of the motions to dismiss with respect to every Defendant-Appellant except Treasurer Dillon. We **REMAND** for the district court to decide whether Dillon should be dismissed in light of its decision in *Brown v. Snyder (In re Flint Water Cases)*, No. 18-cv-10726, 2020 WL 1503256, at \*9 (E.D. Mich. Mar. 27, 2020).

MURPHY, J., concurring in the judgment in part and dissenting in part.

Like other cases that have reached our court, this case arises out of the tragedy known as the Flint water crisis. See *Guertin v. Michigan*, 912 F.3d 907 (6th Cir. 2019); *Boler v. Earley*, 865 F.3d 391 (6th Cir. 2017). The district court held that the plaintiffs’ complaint stated actionable claims against many government actors in Michigan. *Carthan v. Snyder*, 384 F. Supp. 3d 802, 839–43, 857–61 (E.D. Mich. 2019). These government actors have taken this immediate appeal on qualified-immunity grounds. Yet our court recently allowed similar claims to proceed against many of the same actors. *Guertin*, 912 F.3d at 926–32. I joined Judge Kethledge’s dissent from the denial of rehearing en banc in that case. *Guertin v. Michigan*, 924 F.3d 309, 315 (6th Cir. 2019) (Kethledge, J., dissenting from the denial of rehearing en banc). While “the sympathies of every decent person run entirely to the plaintiffs” in all of these cases, I did not believe that the complaint’s allegations met the Supreme Court’s high bar “for prying away an officer’s qualified immunity”—even at the early motion-to-dismiss stage. *Id.* at 315–16; cf. *Ziglar v. Abbasi*, — U.S. —, 137 S. Ct. 1843, 1865–69, 198 L.Ed.2d 290 (2017).

Now, however, *Guertin* is circuit law that we must faithfully follow. And this case's similarities to *Guertin* are striking. This case's plaintiffs? Flint residents who allege serious harm from contaminated water, just as in *Guertin*. Its defendants? State and local actors, many of whom were defendants in *Guertin*. The claim? That these actors violated the same substantive-due-process right to bodily integrity at issue in *Guertin*. The procedural posture? An appeal from the denial of a motion to dismiss the complaint, just as in *Guertin*. The allegations? Largely the same as in *Guertin*—that government actors played various roles in switching Flint's water supply to a contaminated source and then in concealing the water's contaminated nature. The defenses? The same qualified-immunity and sovereign-immunity defenses from *Guertin* (and *Boler*).

What does this background mean for this case? As an initial matter, I would have written the majority opinion “in a different key.” *Guertin*, 924 F.3d at 311 (Sutton, J., concurring in the denial of rehearing en banc). This appeal arises at the pleading stage. We must assume that the complaint's allegations are true even though many remain hotly contested by the defendants. If discovery ends up showing only negligence on their part, the defendants may raise their qualified-immunity defenses at the summary-judgment stage. See *id.* at 315; *Guertin*, 912 F.3d at 935. Still, I agree with most of my colleagues' conclusions. Under *Guertin*, I agree that the substantive-due-process claims must proceed against the defendants from the City of Flint (Emergency Managers Gerald Ambrose and Darnell Earley, Director of Public Works Howard Croft, and Utility Administrators Michael Glasgow and Daugherty Johnson). And I agree that the claims must proceed against the defendants from the Michigan Department \*336 of Environmental Quality (Stephen Busch, Patrick Cook, Michael Prysby, Adam Rosenthal, Liane Shekter-Smith, and Bradley Wurfel). Yet I respectfully disagree with my colleagues over whether *Guertin* permits the claim against former Governor Rick Snyder, and I also would resolve the claim against former Treasurer Andy Dillon now. I read *Guertin* as requiring us to reject the claims against Snyder and Dillon.

Keep in mind that the *Guertin* appeal involved twelve individual defendants, but our court allowed claims to proceed against only seven of them. 912 F.3d at 932. *Guertin* noted that public actors infringe a due-process right to bodily integrity when they injure individuals through conduct that “shocks the conscience.” *Id.* at 918–24. And it

chose a deliberate-indifference test to measure whether the defendants' actions in that case shocked the conscience. *Id.* at 926. That test (which *Guertin* called a “particularly high hurdle”) required the plaintiffs to plausibly allege that the defendants “knew of facts from which they could infer a substantial risk of serious harm, that they did infer it, and that they acted with indifference toward the individual's rights.” *Id.* (internal quotation marks omitted). Importantly, *Guertin* then explained that it must apply this test to “each individual defendant's conduct” because public actors cannot be held vicariously liable for the conduct of others under 42 U.S.C. § 1983. *Id.* at 926, 929.

Why did *Guertin* find this test met for some defendants but not for others? As I read our opinion, it distinguished the actors with the most day-to-day involvement in allegedly causing the crisis (or in allegedly covering it up) from higher-level officials with more supervisory roles or other employees with more tangential roles. See *id.* at 926–32; cf. *Ashcroft v. Iqbal*, 556 U.S. 662, 680–84, 129 S.Ct. 1937, 173 L.Ed.2d 868 (2009).

On the one hand, *Guertin* allowed claims against the City of Flint employees who were allegedly the “chief architects” of the switch to the Flint River and who made that change while knowing that the Flint water-treatment plant was not ready. 912 F.3d at 926. *Guertin* also allowed claims against the “front and center” employees in the Michigan Department of Environmental Quality who allegedly “authorized use of Flint River water with knowledge of its contaminants and then deceived others to hide the fact of contamination.” *Id.* at 927.

On the other hand, *Guertin* rejected a claim against Daniel Wyant, the Director of the Michigan Department of Environmental Quality, who managed these employees and who was “aware of some of the issues” with the water after the transition. *Id.* at 929. The plaintiffs did not allege that he “personally made decisions regarding the water-source switch, nor [did] they allege he personally engaged in any other conduct that [we found] conscience-shocking.” *Id.* Similarly, the court rejected claims against Nick Lyon, the Director of the Michigan Department of Health and Human Services, and another executive in his department. *Id.* at 929–30. While these actors allegedly knew of problems with the water and failed to warn the public, those allegations fell “well-short of conscience-shocking conduct[.]” *Id.* at 930. *Guertin* lastly dismissed claims against two other lower-level employees in that department even though they allegedly sought to hide evidence of the crisis. *Id.* at 931–32. *Guertin*

reasoned that the failure to “blow the whistle” did not suffice to meet its deliberate-indifference test. *Id.* at 932.

Now apply these standards to the thirteen defendants sued in their personal capacities \*337 in this appeal. *Guertin* already denied qualified immunity to seven of them—three defendants with the city (Earley, Ambrose, and Croft) and four with the Michigan Department of Environmental Quality (Busch, Prysby, Shekter-Smith, and Wurfel). See *id.* at 926–29. The complaint in this case makes allegations against these defendants that are analogous to those in *Guertin*. So *Guertin* requires us to allow the claims to proceed against these defendants in this case too.

*Guertin* did not consider two other defendants with the Michigan Department of Environmental Quality (Cook and Rosenthal) and two other defendants with the City of Flint (Glasgow and Johnson). But the complaint’s allegations against these actors fit the profile of those that *Guertin* found to shock the conscience. As I read *Guertin*, the key conscience-shocking allegations against the relevant actors were that they knowingly authorized use of contaminated water and engaged in “lies” by “deceiv[ing] others to hide the fact of contamination.” *Id.* at 929; see *id.* at 927. The complaint in this case asserts similar claims against Cook and Rosenthal. Cook is alleged to have intentionally misled the EPA about the need for corrosion control by knowingly providing the EPA with false information. Compl., R.620-3, PageID#17886. And Rosenthal is alleged to have “willful[ly] participat[ed] in the manipulation of lead testing results[.]” *Id.*, PageID#17894. Similarly, Glasgow allegedly participated in testing that “purposefully skewed the results to minimize the crisis,” wrongly telling residents “to run their water—or ‘flush’ it—before testing[.]” *Id.*, PageID#17893–94. Finally, in the days before the switch to the Flint River water source, Johnson allegedly pressured Glasgow to complete the transition even though Glasgow told him that the Flint plant was not ready to safely operate. *Id.*, PageID#17849–50. Under *Guertin*, these allegations against these defendants are enough.

That leaves the claims against former Governor Snyder and Treasurer Dillon, neither of whom were addressed by *Guertin*. As I see it, both are entitled to qualified immunity under *Guertin*’s own logic. Start with the former governor. From a bird’s-eye view, *Guertin* already dismissed two of Snyder’s cabinet-level officials—Directors Wyant and Lyon—because it viewed them as too far removed from the conscience-shocking conduct. 912 F.3d at 929–32. If

Snyder’s *subordinates* were too far removed from the crisis to remain defendants, that fact should make us think twice before allowing claims to proceed against an official even further removed.

To be sure, we are reviewing a different complaint. But the new allegations against Snyder do not overcome the “particularly high hurdle” that *Guertin* set. *Id.* at 926. Those allegations fall into two general time periods—those before the April 2014 transition to the Flint River water source and those after it. The allegations for both time periods fail to establish an actionable claim.

Before the transition, the complaint at least alleges that Snyder took an action. Sometime in mid-2013, he allegedly approved the transition after subordinates and city officials recommended it to him. Compl., R.620-3, PageID#17842–46. But the complaint fails to plausibly allege facts suggesting that this approval was callously indifferent to a then-known risk of harm. See *Guertin*, 912 F.3d at 926. Indeed, the complaint itself identifies an earlier study suggesting that Flint River water *could* satisfy regulations if the Flint plant received \$69 million in upgrades. Compl., R.620-3, PageID#17839–40. And it also suggests that the switch contemplated upgrades. *Id.*, PageID#17853–59. Nothing in \*338 these allegations takes this claim outside the usual rule that most “governmental policy choices come with risks attached to both of the competing options, and yet ‘it is not a tort for government to govern’ by picking one option over another.” *Guertin*, 912 F.3d at 924–25 (quoting *Schroder v. City of Fort Thomas*, 412 F.3d 724, 729 (6th Cir. 2005)).

In that respect, Snyder’s sign-off is nothing like the conscience-shocking actions allegedly taken by the “chief architects” of the transition. *Id.* at 926. Much later in April 2014, some of those defendants allegedly forced the transition through despite full knowledge that the Flint water-treatment plant was not ready to safely operate. *Id.* Indeed, the complaint alleges that Glasgow initially refused to approve the change but was pressured to proceed anyway. Compl., R.620-3, PageID#17849–50. The complaint makes no equivalent allegations against Snyder. At most, it identifies a March 2014 email from someone in the governor’s office sent “to several others in the governor’s office” suggesting that the expedited time frame was “less than ideal and could lead to some big potential disasters down the road.” *Id.*, PageID#17848. The complaint does not even allege that the governor saw this email. Regardless, *Guertin* held that a claim could not proceed against Director Wyant even though

the complaint alleged that he “was aware of some of the issues arising with the water supply post-switch[.]” *Guertin*, 912 F.3d at 929. Even if Snyder did receive this email, it would establish no more than the general awareness of issues followed by inaction that *Guertin* found insufficient.

After the transition, the complaint alleges that Snyder was “aware of the health crisis” by early 2015, but failed to take any “corrective action” until October 2015 (when he ordered a return to the prior water source) and January 2016 (when he declared a state of emergency). Compl., R.620-3, PageID#17885; *see id.*, PageID#17887, 17891, 17898–17900. The complaint adds that “public assurances provided by members of his Administration that Flint’s water was ‘safe’ were recklessly false, and caused or contributed to the poisoning of Flint’s citizenry.” *Id.*, PageID#17904; *see Carthan*, 384 F. Supp. 3d at 841–43.

In two ways, these allegations are similar to the allegations against Directors Wyant and Lyon that *Guertin* found insufficient. *First*, the complaint asserts no well-pleaded allegations that Snyder himself deceived the public; instead, it raises generic claims of deception against his “Administration.” Compl., R.620-3, PageID#17904. Yet, as with respect to Director Wyant in *Guertin*, even if “the conduct of individuals within his [chain of command] was constitutionally abhorrent, we may only hold [Snyder] accountable for his own conduct, not the misconduct of his subordinates.” *Guertin*, 912 F.3d at 929 (citing *Iqbal*, 556 U.S. at 676–77, 129 S.Ct. 1937). *Second*, the complaint alleges that Snyder knew of the problems and failed to disclose them to the public or to act sooner. Yet, as with respect to Director Lyon in *Guertin*, an alleged “fail[ure] to ‘protect and notify the public’ ” cannot state a claim because substantive due process “is a limitation only on government action.” *Id.*

at 930. I thus would grant Snyder qualified immunity and dismiss him from this suit.

Turn to former Treasurer Dillon. My colleagues remand the claim against him so that the district court may reconsider its earlier decision in light of a later decision granting him qualified immunity in a parallel case. Yet my analysis concerning Governor Snyder requires me to find Dillon entitled to qualified immunity too. The complaint’s only allegations against Dillon \*339 are that he was involved in the mid-2013 negotiations that led to Snyder’s approval to switch Flint’s water source. Compl., R.620-3, PageID#17842–44, 17847, 17851; *Carthan*, 384 F. Supp. 3d at 858. As I explained for Snyder, that decision did not plausibly allege any deliberate indifference to a then-known risk of harm. *See Guertin*, 912 F.3d at 924–25 (citing *Schroder*, 412 F.3d at 729).

\* \* \*

One final loose end: the two sovereign-immunity defenses. For these defenses too, I agree with my colleagues. *Guertin* forecloses the City of Flint’s invocation of sovereign immunity. *See id.* at 936. And *Boler* forecloses Governor Gretchen Whitmer’s contention that the plaintiffs may not seek injunctive relief (identical to the injunctive relief requested in *Boler*) against the governor in her official capacity. *See 865 F.3d at 413.*

All told, then, I respectfully concur in the judgment in part and dissent in part.

#### All Citations

960 F.3d 303

#### Footnotes

- 1 We use the term “Plaintiffs” when referring to all plaintiffs belonging to the putative class, and we use the term “Plaintiffs-Appellees” when referring solely to the plaintiffs that are party to this appeal.
- 2 We use the term “Defendants” when referring to all named defendants, and we use the term “Defendants-Appellants” when referring solely to the defendants that are party to this appeal.
- 3 Governor Whitmer was elected into office in January 2019 and continues to serve as Michigan’s Governor at the time of this writing. For the sake of consistency with its earlier Flint Water decisions, the district court solely referred to Governor Snyder in its opinion, even where claims are made against the present Governor in her official capacity. R. 798 (Op. & Order at 8 n.4) (Page ID #21110).
- 4 The facts are taken from Plaintiffs’ Fourth Amended Complaint, as we take all factual allegations to be true at this stage. *See Guertin*, 912 F.3d at 916.
- 5 Some Defendants-Appellants contend that they were not aware that the water was contaminated. They point out that Plaintiffs themselves allege that private engineering firms provided inaccurate information about water quality

to government officials. See R. 620-3 (Fourth Am. Compl. at 51–80, ¶¶ 148–232) (Page ID #17854–83). But those allegations do not negate the separate allegations that City and State officials nevertheless had knowledge from other sources that the water was contaminated. Therefore, the role of private engineering firms is irrelevant at the motion to dismiss stage.

- 6 We approved this approach in *Waid v. Snyder*, No. 18-1967, slip op., 2019 WL 4121023 (6th Cir. Feb. 19, 2019) (order).
- 7 The district court granted motions to dismiss in favor of some Defendants, who accordingly are not a part of this appeal. R. 798 (Op. & Order at 128) (Page ID #21230). Additionally, Plaintiffs' *Monell* claim was not certified for interlocutory appeal. *Id.* at 109 (Page ID #21211).
- 8 Defendant Cook notified us that the district court dismissed him from a separate Flint Water Crisis case, *Brown v. Snyder (In re Flint Water Cases)*, No. 18-cv-10726, 2020 WL 1503256, at \*12 (E.D. Mich. Mar. 27, 2020). He contends that his dismissal from *Brown* similarly warrants his dismissal here. We disagree. The district court in *Brown* dismissed Cook because his wrongful conduct occurred after the plaintiff's injury in that case. *Id.* at \*10, 12. The plaintiff in *Brown* had died of Legionnaires' disease before Cook allegedly misled the EPA. *Id.* There is no similar timing issue in this case.
- 9 We note, without passing judgment, that the district court dismissed Governor Snyder from the action in *Guertin*. See *Guertin v. Michigan*, No. 16-cv-12412, 2017 WL 2418007, at \*24 (E.D. Mich. June 5, 2017). It did so because there were no plausible allegations in that case that Governor Snyder personally was involved in the decision-making process for using Flint River water. *Id.* The plaintiffs' theory in *Guertin* was that Snyder should be on the hook merely because he appointed the City Managers who helped to create and sustain the crisis. *Id.* The same cannot be said here, as Plaintiffs have alleged Snyder's personal actions and knowledge in great detail.
- 10 Defendants-Appellants argue that we should look to *Green v. Mansour*, 474 U.S. 64, 106 S.Ct. 423, 88 L.Ed.2d 371 (1985)—not *Milliken*—to decide this case. They rely upon the Supreme Court's statement in *Green* that the Eleventh Amendment permits suits against the State only "designed to prevent ongoing violations of federal law." *Id.* at 71, 106 S.Ct. 423. *Green* is not on point. There, the Supreme Court held that plaintiffs could not seek notice relief ancillary to a declaratory judgment under *Ex parte Young* that would, in effect, serve only to provide them with retroactive monetary relief. See *Green*, 474 U.S. at 73, 106 S.Ct. 423 ("The issuance of a declaratory judgment in these circumstances would have much the same effect as a full-fledged award of damages or restitution by the federal court, the latter kinds of relief being of course prohibited by the Eleventh Amendment."). *Green* did not confront the same issue that is involved in this case—whether remedial measures to combat the effects of past constitutional violations are available as a form of prospective injunctive relief under *Ex parte Young*.

384 F.Supp.3d 802  
United States District Court, E.D.  
Michigan, Southern Division.

IN RE FLINT WATER CASES.

This Order Relates to:

Carthan

v.

Snyder

Case No. 16-10444

|

Signed 04/01/2019

**Synopsis**

**Background:** City residents and property owners brought consolidated class actions against various defendants, including state and city government officials, alleging, inter alia, violation of the Equal Protection Clause and the right to bodily integrity under the Fourteenth Amendment pursuant to § 1983, arising from injuries allegedly sustained as a result of the contamination of city's water supply. Residents and property owners moved for leave to file fourth amended complaint, and defendants moved to dismiss.

**Holdings:** The District Court, [Judith E. Levy, J.](#), held that:

[1] proposed amended complaint stated plausible claim against state governor for violation of the right to bodily integrity;

[2] proposed amended complaint failed to state plausible claim for wealth-based discrimination in violation of the Equal Protection Clause;

[3] proposed amended complaint failed to state plausible claim for violation of the Equal Protection Clause based on race discrimination;

[4] proposed amended complaint failed to state plausible claim for conspiracy to interfere with civil rights;

[5] residents stated claim violation of the right to bodily integrity;

[6] residents and owners stated § 1983 *Monell* claim against city; and

[7] residents and owners failed to state a claim for exemplary damages under Michigan law.

Motions granted in part and denied in part.

West Headnotes (103)

[1] **Federal Civil Procedure** 🔑 **Complaint**

When evaluating the interests of justice, for purposes of determining whether leave to amend a complaint is warranted, courts consider various factors, including: (1) undue delay in filing; (2) lack of notice to the opposing party; (3) bad faith by the moving party; (4) repeated failure to cure deficiencies by previous amendments; and (5) undue prejudice to the opposing party. *Fed. R. Civ. P. 15(a)*.

[2] **Federal Civil Procedure** 🔑 **Injustice or prejudice**

**Federal Civil Procedure** 🔑 **Time for amendment**

Mere delay on its own is insufficient to warrant denial of a motion to amend a complaint; instead, courts examine the competing interests of the litigants and the likelihood of prejudice to the non-moving party. *Fed. R. Civ. P. 15(a)*.

[3] **Federal Civil Procedure** 🔑 **Form and sufficiency of amendment; futility**

Regardless of the equities, leave to amend a complaint must be denied if an amendment would be futile. *Fed. R. Civ. P. 15(a)*.

[4] **Federal Civil Procedure** 🔑 **Form and sufficiency of amendment; futility**

A proposed amendment of a complaint is futile if it could not withstand a motion to dismiss for

failure to state a claim. [Fed. R. Civ. P. 12\(b\)\(6\)](#), [15\(a\)](#).

- [5] **Federal Civil Procedure** 🔑 Time for amendment

**Federal Civil Procedure** 🔑 New cause of action in general

Allowing plaintiffs in consolidated class actions to file fourth amended complaint would not prejudice defendants, so as to preclude grant of motion to amend, even though case had been pending for several years; case had complex procedural history and claims and involved an extraordinary number of plaintiffs, defendants resisted start of discovery so that they were unable to claim that they would have been subject to duplicative discovery, plaintiffs did not change their allegations to the extent that defendants would need to completely overhaul their strategy, and amended complaint did not contain new claims so far outside the scope of prior complaint such that granting leave to amend may have later led to confusion. [Fed. R. Civ. P. 15\(a\)](#).

- [6] **Constitutional Law** 🔑 Personal and bodily rights in general

The right to bodily integrity is a fundamental interest protected by the Due Process Clause of the Fourteenth Amendment. [U.S. Const. Amend. 14](#).

- [7] **Constitutional Law** 🔑 Personal and bodily rights in general

Although violations of the right to bodily integrity under the Due Process Clause usually arise in the context of physical punishment, the scope of the right is not limited to that context. [U.S. Const. Amend. 14](#).

1 Cases that cite this headnote

- [8] **Constitutional Law** 🔑 Personal and bodily rights in general

There is no difference between the forced invasion of a person's body with a substance and misleading that person into consuming a substance involuntarily, for purposes of determining whether activity violates the right to bodily integrity under the Due Process Clause. [U.S. Const. Amend. 14](#).

- [9] **Constitutional Law** 🔑 Personal and bodily rights in general

Government officials can violate an individual's bodily integrity in violation of the Due Process Clause by introducing life-threatening substances into that person's body without their consent. [U.S. Const. Amend. 14](#).

- [10] **Constitutional Law** 🔑 Substantive Due Process in General

To state a substantive due process claim, plaintiffs must do more than point to the violation of a protected interest; they must also demonstrate that it was infringed arbitrarily. [U.S. Const. Amend. 14](#).

- [11] **Constitutional Law** 🔑 Egregiousness; "shock the conscience" test

In regards to government executive action, only the most egregious conduct infringing on a protected interest can be classified as unconstitutionally arbitrary, for purposes of stating a substantive due process claim; in legal terms, the conduct must shock the conscience. [U.S. Const. Amend. 14](#).

- [12] **Constitutional Law** 🔑 Purpose or intent

Where unforeseen circumstances demand the immediate judgment of an executive government official, liability for violation of substantive due process turns on whether decisions were made maliciously and sadistically for the very purpose of causing harm. [U.S. Const. Amend. 14](#).

**[13] Constitutional Law** 🔑 Negligence, recklessness, or indifference

Where an executive government official has time for deliberation before acting, conduct taken with deliberate indifference to the rights of others shocks the conscience, for purposes of stating a claim for violation of substantive due process. U.S. Const. Amend. 14.

**[14] Constitutional Law** 🔑 Negligence, recklessness, or indifference

In order to demonstrate that executive government officials had time for deliberation before acting, so that the officials' conduct, taken with deliberate indifference to the rights of others, shocks the conscience for purposes of stating claim for violation of substantive due process, plaintiffs must demonstrate that: (1) officials knew of facts from which they could infer a substantial risk of serious harm; (2) that they did infer it; and (3) that they nonetheless acted with indifference, demonstrating a callous disregard towards the rights of those affected. U.S. Const. Amend. 14.

2 Cases that cite this headnote

**[15] Constitutional Law** 🔑 Water, sewer, and irrigation

**States** 🔑 Governor

**Water Law** 🔑 Civil claims arising from failure to meet quality standards

City residents and property owners' proposed amended complaint plausibly pointed to bodily integrity violation under the Due Process Clause, for purposes of claim in consolidated class actions against state governor, arising from alleged injuries sustained as a result of contamination of city's water supply, where case implicated the consumption of life-threatening substances such as lead and legionella, residents ingested contaminants through water supply, and intrusion was involuntary as the level of lead in the water was hidden and under state and municipal law residents and owners were not

permitted to receive water in any other way. U.S. Const. Amend. 14.

**[16] Constitutional Law** 🔑 Water, sewer, and irrigation

**States** 🔑 Governor

**Water Law** 🔑 Civil claims arising from failure to meet quality standards

City residents and property owners' proposed amended complaint against state governor plausibly alleged that governor's actions were deliberately indifferent and exhibited callous disregard for their right to bodily integrity, so as to shock the conscience for purposes of due process claim for violation of right to bodily integrity pursuant to § 1983, in consolidated class actions arising from alleged injuries resulting from contamination of city's water supply; plaintiffs alleged that governor knew facts from which he could infer a substantial risk of serious harm, such as an outbreak of Legionnaires' disease, that governor inferred risk of harm, such as by meeting with government officials to discuss water's health threat, and that governor acted indifferently to risk of harm, such as by encouraging drinking of the water. U.S. Const. Amend. 14; 42 U.S.C.A. § 1983.

4 Cases that cite this headnote

**[17] Public Employment** 🔑 Qualified immunity

Qualified immunity shields public officials from undue interference with their duties and from potentially disabling threats of liability.

**[18] Civil Rights** 🔑 Good faith and reasonableness; knowledge and clarity of law; motive and intent, in general

Qualified immunity provides protection to government officials who make reasonable yet mistaken decisions that involve open questions of law.

- [19] **Civil Rights** — Good faith and reasonableness; knowledge and clarity of law; motive and intent, in general

A government official cannot avail herself of qualified immunity if the right violated was clearly established at the time of the challenged conduct.

- [20] **Civil Rights** — Good faith and reasonableness; knowledge and clarity of law; motive and intent, in general

For purposes of determining whether government officials are entitled to qualified immunity, if controlling caselaw or a body of persuasive authority has put the constitutional question beyond debate, government officials are on notice that their conduct must conform to an established legal standard.

- [21] **Civil Rights** — States and territories and their officers and agencies

State governor's alleged conduct of knowing that river's water was unsafe for public use, failing to take steps to counter its problems, and assuring public in the meantime that the water was safe, violated city residents' and property owners' clearly established right to bodily integrity, and thus governor was not entitled to qualified immunity from due process claim for violation of the right to bodily integrity pursuant to § 1983, in consolidated class actions arising from alleged injuries resulting from contamination of city's water supply; any reasonable government official should have known that contaminating a community through its public water supply with deliberate indifference was a government invasion of the highest magnitude. *U.S. Const. Amend. 14*; 42 U.S.C.A. § 1983.

- [22] **Constitutional Law** — Similarly situated persons; like circumstances

Broadly speaking, the Equal Protection Clause requires that state officials treat all persons alike,

under like circumstances and like conditions. *U.S. Const. Amend. 14*.

- [23] **Constitutional Law** — Similarly situated persons; like circumstances

When state officials treat similar individuals differently, the Equal Protection Clause demands a justification. *U.S. Const. Amend. 14*.

- [24] **Constitutional Law** — Levels of Scrutiny

Because all state action tends to disfavor some more than others, courts take this practical reality into account by evaluating state action under differing levels of scrutiny, for purposes of a claim under the Equal Protection Clause. *U.S. Const. Amend. 14*.

- [25] **Constitutional Law** — Rational Basis Standard; Reasonableness

If official state conduct neither burdens a fundamental right nor targets a suspect class, courts will uphold it under the Equal Protection Clause so long as it bears a rational relation to some legitimate end. *U.S. Const. Amend. 14*.

- [26] **Constitutional Law** — Poverty or Wealth; The Homeless

A class of less wealthy persons is not a protected class for the purposes of equal protection. *U.S. Const. Amend. 14*.

- [27] **Constitutional Law** — Equal protection

Under rational basis equal protection review, official state decisions are afforded a strong presumption of validity; even at the motion to dismiss stage, this presents a formidable bar for plaintiffs to surmount. *U.S. Const. Amend. 14*.

- [28] **Constitutional Law** — Rational Basis Standard; Reasonableness

To plausibly allege that state action fails under rational basis equal protection review, plaintiffs must negate every conceivable basis which might support the challenged conduct. [U.S. Const. Amend. 14](#).

**[29] Constitutional Law** 🔑 **Rational Basis Standard; Reasonableness**

Under rational basis equal protection review, courts do not consider the wisdom of the challenged state action, and defendants do not need to offer any justification; it is enough that the reviewing court can fairly conceive of one existing. [U.S. Const. Amend. 14](#).

**[30] Constitutional Law** 🔑 **Other particular issues and applications**

**Municipal Corporations** 🔑 **Public works and improvements**

**Water Law** 🔑 **Civil claims arising from failure to meet quality standards**

City residents and property owners' proposed amended complaint against state and city government officials failed to negate every rational basis which supported officials' challenged conduct of creating an interim plan to supply city with river water, while continuing to provide the remainder of county with other water, as required to support claim in consolidated class actions for wealth-based discrimination in violation of the Equal Protection Clause pursuant to § 1983, arising from injuries allegedly sustained as a result of contamination of city's water supply; having city obtain water from river was financially expedient. [U.S. Const. Amend. 14](#); [42 U.S.C.A. § 1983](#).

**[31] Constitutional Law** 🔑 **Other particular issues and applications**

**States** 🔑 **Governor**

City residents and property owners' proposed amended complaint against state governor failed to negate every rational basis which supported governor's challenged conduct of

delaying declaration of state of emergency in city while allegedly promptly doing so in other emergency situations, as required to support claim in consolidated class actions for wealth-based discrimination in violation of the Equal Protection Clause pursuant to § 1983, arising from injuries allegedly sustained as a result of contamination of city's water supply; it was conceivable that governor initially decided not to expend state resources believing that water crisis could have been addressed without them. [U.S. Const. Amend. 14](#); [42 U.S.C.A. § 1983](#).

**[32] Constitutional Law** 🔑 **Discrimination and Classification**

Class-based discrimination is the essence of an equal protection claim. [U.S. Const. Amend. 14](#).

**[33] Constitutional Law** 🔑 **Discrimination and Classification**

**Constitutional Law** 🔑 **Intentional or purposeful action**

**Constitutional Law** 🔑 **Sex or Gender**

In limited situations, a plaintiff does not need to identify a specific group of persons who were treated differently in order to state an equal protection claim; for instance, if government conduct was premised on a protected classification such as race or gender, a showing of discriminatory purpose may suffice. [U.S. Const. Amend. 14](#).

**[34] Constitutional Law** 🔑 **Similarly situated persons; like circumstances**

Outside of the narrow range of cases involved discrimination based on race or gender, plaintiffs must plead sufficient facts from which it can be inferred that defendants treated similarly situated individuals differently in order to state an equal protection claim. [U.S. Const. Amend. 14](#).

**[35] Constitutional Law** 🔑 **Other particular issues and applications**

**Municipal Corporations**  Public works and improvements**Water Law**  Actions

City residents and property owners' proposed amended complaint against state government officials did not explain how treatment by officials, including by allegedly failing to comply with sampling and optimized corrosion control protocols required under state and federal Lead and Copper Rule, differed from that of a similarly situated class of persons, as required to support claim in consolidated class actions for wealth-based discrimination in violation of the Equal Protection Clause pursuant to § 1983, arising from injuries allegedly sustained as a result of contamination of city's water supply; allegations did not explain in anything but conclusory terms how defendants acted differently in other situations, and complaint revealed nothing about possibility that defendants failed to enforce laws on a statewide basis. *U.S. Const. Amend. 14*; 42 *U.S.C.A. § 1983*.

**[36] Constitutional Law**  Race, national origin, or ethnicity

When state action is premised on a racial classification, courts strictly scrutinize the challenged conduct on claims under the Equal Protection Clause. *U.S. Const. Amend. 14*.

**[37] Constitutional Law**  Equal protection

State conduct subject to equal protection strict scrutiny is presumptively invalid; only official action that is narrowly tailored to meet a compelling state interest will survive. *U.S. Const. Amend. 14*.

1 Cases that cite this headnote

**[38] Constitutional Law**  Intentional or purposeful action

Proof of discriminatory intent or purpose is required to show a violation of the Equal Protection Clause on the basis of race discrimination. *U.S. Const. Amend. 14*.

1 Cases that cite this headnote

**[39] Constitutional Law**  Race, national origin, or ethnicity

If discriminatory intent is missing on a claim for violation of the Equal Protection Clause based on race discrimination, such claims are analyzed under rational basis review. *U.S. Const. Amend. 14*.

1 Cases that cite this headnote

**[40] Constitutional Law**  Intentional or purposeful action

In order to demonstrate discriminatory intent, for purposes of stating a claim for violation of the Equal Protection Clause based on race discrimination, the facts must offer more than intent as volition or intent as awareness of consequences; rather, the facts must demonstrate that a decisionmaker selected or reaffirmed a particular course of action at least in part because of, not merely in spite of, its adverse effects upon a particular racial group. *U.S. Const. Amend. 14*.

1 Cases that cite this headnote

**[41] Constitutional Law**  Intentional or purposeful action

At the motion to dismiss stage in a case alleging violation of the Equal Protection Clause based on race discrimination, plaintiffs need only raise an inference of discriminatory purpose; to do so, plaintiffs must demonstrate that the application of a facially neutral law or policy had a discriminatory impact, and sufficient evidence exists to suggest an invidious motive. *U.S. Const. Amend. 14*; *Fed. R. Civ. P. 12(b)*.

**[42] Constitutional Law**  Intentional or purposeful action

To raise an inference of discriminatory purpose, for purposes of stating a claim for violation of the Equal Protection Clause based on race discrimination, the challenged conduct does not need to rest solely on racially discriminatory

purposes, but this must have been a motivating factor. [U.S. Const. Amend. 14.](#)

**[43] Constitutional Law** 🔑 Intentional or purposeful action

Although discriminatory impact is an important starting point to demonstrate discriminatory intent, for purposes of stating a claim for violation of the Equal Protection Clause based on race discrimination, it is rarely enough on its own; instead, courts must conduct a sensitive inquiry into such circumstantial and direct evidence of intent as may be available. [U.S. Const. Amend. 14.](#)

**[44] Constitutional Law** 🔑 Race, National Origin, or Ethnicity

Discriminatory impact alone is sufficient to state a claim for violation of the Equal Protection Clause based on race discrimination in the rarest case where a clear pattern, unexplainable on grounds other than race, emerges from the effect of the state action. [U.S. Const. Amend. 14.](#)

[1 Cases that cite this headnote](#)

**[45] Constitutional Law** 🔑 Intentional or purposeful action

In order to raise an inference of discriminatory purpose, for purposes of stating a claim for violation of the Equal Protection Clause based on race discrimination, plaintiffs can show that a law or policy explicitly classifies on the basis of race. [U.S. Const. Amend. 14.](#)

**[46] Constitutional Law** 🔑 Intentional or purposeful action

Several non-exhaustive factors guide the inquiry to determine whether there is proof of discriminatory intent, for purposes of stating a claim for violation of the Equal Protection Clause based on race discrimination: (1) the historical background of the decision is one evidentiary source, particularly if it reveals a series of official actions taken for invidious

purposes; (2) the specific sequence of events leading up to the challenged decision may shed light on the decisionmaker's purposes; (3) departures from the normal procedural sequence particularly if the factors usually considered important by the decisionmaker strongly favor a decision contrary to the one reached; and (4) the legislative or administrative history especially where there are contemporary statements by members of the decisionmaking body, minutes of its meetings, or reports. [U.S. Const. Amend. 14.](#)

[1 Cases that cite this headnote](#)

**[47] Constitutional Law** 🔑 Other particular issues and applications

**Municipal Corporations** 🔑 Public works and improvements

**Water Law** 🔑 Civil claims arising from failure to meet quality standards

Proposed amended complaint of residents and property owners of city, which had a majority African American population, against state and city government officials, failed to show that race motivated officials' decision to switch city's water supply to river while providing other water to remainder of county, as would have raised inference of discriminatory purpose, so as to support claim in consolidated class actions for violation of the Equal Protection Clause based on race discrimination pursuant to § 1983, arising from injuries allegedly sustained as a result of contamination of city's water supply; alleged facts indicated that cost of water, not racial bias, motivated officials' decision. [U.S. Const. Amend. 14; 42 U.S.C.A. § 1983.](#)

**[48] Constitutional Law** 🔑 Other particular issues and applications

**States** 🔑 Governor

Proposed amended complaint of residents and property owners of city, which had a majority African American population, against state governor, failed to show that race motivated governor's purported decision to treat emergency in city differently than in majority white communities, as would have raised inference

of discriminatory purpose, so as to support claim in consolidated class actions for violation of the Equal Protection Clause based on race discrimination pursuant to § 1983, arising from injuries allegedly sustained from contamination of city's water supply; comparative states of emergency identified in complaint involved drastically different situations, and plaintiffs did not point to clear pattern of discrimination where governor consistently delayed declaring states of emergency in mostly African American areas. [U.S. Const. Amend. 14](#); [42 U.S.C.A. § 1983](#).

[49] **Constitutional Law** 🔑 Other particular issues and applications

**States** 🔑 Particular persons or agencies; scope of employment

**Water Law** 🔑 Primary enforcement responsibility

**Water Law** 🔑 Actions

Proposed amended complaint of residents and property owners of city, which had a majority African American population, against state government officials, failed to show race motivated officials' alleged decision to not enforce certain laws and policies, as would have raised inference of discriminatory purpose, so as to support claim in consolidated class actions for violation of the Equal Protection Clause based on race discrimination pursuant to § 1983, arising from injuries allegedly sustained from contamination of city's water supply, despite contention that officials failed to develop non-discrimination policy required by federal Environmental Protection Agency; allegations did not link officials' decisions to discriminatory intent, and allegations that nonconformities with law and policy never occurred in white communities were conclusory. [U.S. Const. Amend. 14](#); [42 U.S.C.A. § 1983](#).

[50] **Civil Rights** 🔑 Purpose and construction in general

Michigan's Elliott Larsen Civil Rights Act (ELCRA) is aimed at the prejudices and biases borne against persons because of their

membership in a certain class, and seeks to eliminate the effects of offensive or demeaning stereotypes, prejudices, and biases. [Mich. Comp. Laws Ann. § 37.2301 et seq.](#)

[51] **Civil Rights** 🔑 Public Services, Programs, and Benefits

To state a claim under public service provision of Michigan's Elliott Larsen Civil Rights Act (ELCRA), plaintiffs must allege: (1) discrimination based on a protected characteristic (2) by a person, (3) resulting in denial of the full and equal enjoyment of a public service. [Mich. Comp. Laws Ann. § 37.2301](#).

[52] **Civil Rights** 🔑 Public Services, Programs, and Benefits

The public service provision of Michigan's Elliott Larsen Civil Rights Act (ELCRA) uses the same framework to establish discrimination as that used generally under other provisions of the ELCRA. [Mich. Comp. Laws Ann. § 37.2301, et seq.](#)

[53] **Civil Rights** 🔑 Public Services, Programs, and Benefits

To establish discrimination, for purposes of stating a claim under the public service provision of Michigan's Elliott Larsen Civil Rights Act (ELCRA), plaintiffs must show either intentional discrimination directly or raise an inference of discrimination based on a disparate treatment theory. [Mich. Comp. Laws Ann. § 37.2301](#).

[54] **Civil Rights** 🔑 Public Services, Programs, and Benefits

In a case involving allegations of race-based discrimination, for purposes of stating a claim under public service provision of Michigan's Elliott Larsen Civil Rights Act (ELCRA), plaintiffs can plead intentional discrimination by pointing to direct evidence that defendants were pre-disposed to discriminate against people of

a certain race, and that they acted on that predisposition. *Mich. Comp. Laws Ann.* § 37.2301.

**[55] Civil Rights** 🔑 Evidence

“Direct evidence” that defendants were predisposed to discriminate based on race is evidence which, if believed, requires the conclusion that unlawful discrimination was at least a motivating factor in defendants’ actions, for purposes of stating a claim under public service provision of Michigan’s Elliott Larsen Civil Rights Act (ELCRA). *Mich. Comp. Laws Ann.* § 37.2301.

**[56] Civil Rights** 🔑 Evidence

Plaintiffs can raise an inference of racial discrimination, for purposes of a claim under the public service provision of Michigan’s Elliott Larsen Civil Rights Act (ELCRA), by pleading that defendants treated them differently from non-protected individuals under the same or similar circumstances; but they must also point to sufficient indirect evidence from which it can be inferred that race was a motivating factor, even if not the sole factor. *Mich. Comp. Laws Ann.* § 37.2301.

**[57] Civil Rights** 🔑 Public Services, Programs, and Benefits

Proposed amended complaint of residents and property owners of city, which had a majority African American population, against various defendants, including state and city officials, failed to plead sufficient facts to raise an inference of racial discrimination in officials’ conduct, which allegedly included providing city with inferior water compared to rest of county, as required to state plausible claim in consolidated class action under public service provision of Michigan’s Elliott Larsen Civil Rights Act (ELCRA), arising from injuries allegedly sustained as a result of contamination of city’s water supply. *Mich. Comp. Laws Ann.* § 37.2301.

**[58] Conspiracy** 🔑 Certainty, definiteness, and particularity in general

Conspiracy claims must be pled with some degree of specificity; vague and conclusory allegations unsupported by material facts will not be sufficient to state such a claim.

**[59] Conspiracy** 🔑 Equal privileges and immunities; equal protection

**Conspiracy** 🔑 Civil rights conspiracies

To state a claim for conspiracy to interfere with civil rights, plaintiffs must plead facts consistent with (1) a conspiracy between two or more persons, (2) conceived for the purpose of depriving a person or class of people of the equal protection of the laws, (3) an act committed in furtherance of the conspiracy, and (4) that a person was either injured in his or her person or property, or deprived of a right guaranteed by the Constitution. *U.S. Const. Amend. 14*; *42 U.S.C.A.* § 1985(3).

**[60] Conspiracy** 🔑 Intent, motive, or animus

In stating a claim for conspiracy to interfere with civil rights, plaintiffs must demonstrate that the conspiracy was motivated by racial or other constitutionally suspect class-based animus. *42 U.S.C.A.* § 1985(3).

**[61] Conspiracy** 🔑 Civil Rights Conspiracies

Statute governing claims for conspiracy to interfere with civil rights is not a general federal tort law, providing a federal cause of action for every assault and battery. *42 U.S.C.A.* § 1985(3).

**[62] Conspiracy** 🔑 Intent, motive, or animus

The intent requirement, for purposes of stating a claim for conspiracy to interfere with civil rights, ensures that only those conspiracies that aim at a deprivation of the equal enjoyment of rights secured by the law to all are actionable under

the statute governing such claims. 42 U.S.C.A. § 1985(3).

**[63] Conspiracy** — Particular Rights or Privileges; Particular Deprivations

African American city residents and property owners' proposed amended complaint against state and city officials failed to plausibly allege that officials were motivated by racial or any other invidious class-based animus, as required to support claim in consolidated class actions for conspiracy to interfere with civil rights, arising from injuries allegedly sustained as a result of contamination of city's water supply; residents and owners possibly showed impact that historical race discrimination played a role in the city's water crisis, but not that it was a motivating factor. 42 U.S.C.A. § 1985(3).

**[64] Municipal Corporations** — Liability of officers or agents

**Public Employment** — State, local, and other non-federal personnel in general

To identify whether a lower-level public employee was the proximate cause of an injury, so as to preclude immunity from tort liability under Michigan's Government Tort Liability Act (GTLA), courts must: (1) evaluate the conduct and any legal responsibility of the various parties to an accident, where legal responsibility is assessed by determining whether the accident was a foreseeable consequence of an individual's actions, and (2) jointly consider the actions of those legally responsible to determine whose conduct was the one most immediate, efficient, and direct cause of any injury; if the answer is anyone but the employee, the employee can claim immunity. Mich. Comp. Laws Ann. § 691.1401 et seq.

**[65] Public Employment** — Particular torts  
**States** — Personal injuries in general

City residents and property owners' proposed amended complaint did not sufficiently allege that lower-level state government employees

were the proximate cause of their harm, as would have precluded employees' immunity from gross negligence claim under Michigan law under the Michigan's Government Tort Liability Act (GTLA), in consolidated class actions arising from injuries allegedly sustained as a result of contamination of city's water supply, even though complaint stated that employees' conduct was a direct and proximate cause of the injuries; complaint failed to explain why employees were legally responsible for the harm in anything but conclusory terms, and why employees' conduct was the most immediate, efficient, and direct cause preceding the injuries. Mich. Comp. Laws Ann. § 691.1401 et seq.

**[66] Public Employment** — State, local, and other non-federal personnel in general

**States** — Acts or Omissions of Officers, Agents, or Employees

For purposes of a state government employee's immunity from tort claims under Michigan's Government Tort Liability Act (GTLA), the more governmental actors that are involved in causing a massive tort in Michigan, the less likely it is that state tort claims can proceed against the individual government actors. Mich. Comp. Laws Ann. § 691.1401.

**[67] Federal Courts** — Substantiality of federal question

**Federal Courts** — Pleadings and Motions

To survive a motion to dismiss for lack of subject matter jurisdiction, plaintiffs need only show that the complaint alleges a claim under federal law, and that the claim is substantial; this is a relatively light burden. Fed. R. Civ. P. 12(b)(1).

**[68] Federal Courts** — Substantiality of federal question

Dismissal for lack of subject-matter jurisdiction is proper only when the claim is so insubstantial, implausible, foreclosed by prior decisions of United States Supreme Court, or otherwise

completely devoid of merit as not to involve a federal controversy. Fed. R. Civ. P. 12(b)(1).

[69] **Federal Courts** 🔑 Suits for injunctive or other prospective or equitable relief; *Ex parte Young* doctrine

**Federal Courts** 🔑 Agencies, officers, and public employees

*Ex parte Young* doctrine allows plaintiffs to bring claims for prospective injunctive relief against state officials sued in their official capacity to prevent future federal constitutional or statutory violations.

[70] **Federal Courts** 🔑 Waiver by State; Consent

An exception to a state's sovereign immunity is when the state has waived immunity by consenting to the suit. U.S. Const. Amend. 11.

[71] **Federal Courts** 🔑 Suits for injunctive or other prospective or equitable relief; *Ex parte Young* doctrine

**Federal Courts** 🔑 Other particular entities and individuals

City residents and property owners sought injunctive relief against state governor in his official capacity, and thus the *Ex parte Young* exception to sovereign immunity applied to allow claims for injunctive relief against governor for, inter alia, repairs of private property and establishment of medical monitoring, in consolidated class actions arising from injuries allegedly sustained as a result of contamination of city's water supply. U.S. Const. Amend. 11.

[72] **Public Employment** 🔑 Particular torts

**States** 🔑 Particular persons or agencies; scope of employment

Defendants, state Department of Environmental Quality (DEQ) directors, did not explain how their claim of absolute immunity, which was based on immunity granted to federal officials

carrying out discretionary prosecutorial actions, interacted with city residents and property owners' allegations, and thus defendants were not entitled to absolute immunity from suit in consolidated class actions, arising from injuries allegedly sustained as a result of contamination of city's water supply, even though defendants argued they were functionally acting as federal officials despite working for state agency; defendants merely speculated that absolute immunity would apply if claims ultimately proved to be an alleged failure to sufficiently enforce Safe Drinking Water Act or initiate enforcement proceedings against city. Public Health Service Act, § 1401 et seq., 42 U.S.C.A. § 300f et seq.

[73] **Constitutional Law** 🔑 Business organizations; corporations

City property owners were not individuals, as required to support due process claim for violation of the right to bodily integrity pursuant to § 1983 against various defendants, including state and city government officials, in consolidated class actions arising from allegedly sustained as a result of contamination of city's water supply; property owners were businesses. U.S. Const. Amend. 14; 42 U.S.C.A. § 1983.

[74] **Constitutional Law** 🔑 Water, sewer, and irrigation

**States** 🔑 Liabilities of officers for negligence or misconduct

**Water Law** 🔑 Civil claims arising from failure to meet quality standards

City residents sufficiently alleged that state treasurer demonstrated an indifference to the risk of serious harm residents faced, as required to state due process claim against treasurer for violation of the right to bodily integrity pursuant to § 1983, in consolidated class actions arising from injuries allegedly sustained as a result of contamination of city's water supply; allegations included that treasurer knew that river used as city's water source had been rejected as a water source, and that despite this knowledge treasurer

helped to develop an interim plan that saw city transition to river water. [U.S. Const. Amend. 14](#); [42 U.S.C.A. § 1983](#).

[2 Cases that cite this headnote](#)

[75] **Constitutional Law** [🔑](#) [Water, sewer, and irrigation](#)

**States** [🔑](#) [Liabilities of officers for negligence or misconduct](#)

**Water Law** [🔑](#) [Civil claims arising from failure to meet quality standards](#)

City residents failed to allege that former director of state Department of Health and Human Services was deliberately indifferent to the risk of harm that residents faced, as required to state due process claim against director for violation of the right to bodily integrity pursuant to § 1983, in consolidated class actions arising from injuries allegedly sustained as a result of contamination of city's water supply; while director did not make public aware or alert other government departments about information he received regarding lead contamination in city's water, director directed his team to investigate reports and emails which showed his concern, and residents did not allege that director attempted to cover up what was happening. [U.S. Const. Amend. 14](#); [42 U.S.C.A. § 1983](#).

[76] **Constitutional Law** [🔑](#) [Water, sewer, and irrigation](#)

**States** [🔑](#) [Liabilities of officers for negligence or misconduct](#)

**Water Law** [🔑](#) [Civil claims arising from failure to meet quality standards](#)

City residents sufficiently alleged that state Department of Environmental Quality director of communications was deliberately indifferent to the risk of harm that residents faced, as required to state due process claim against director for violation of the right to bodily integrity pursuant to § 1983, in consolidated class actions arising from injuries allegedly sustained as a result of contamination of city's water supply; residents' allegations included that director knew about

outbreak of Legionnaires' disease and that he aware that something was wrong with city's water, that director appeared on radio and television to advise listeners that city's water was safe to consume and bathe in, and that director discredited others who suggested that lead was leaching into city's water. [U.S. Const. Amend. 14](#); [42 U.S.C.A. § 1983](#).

[77] **Constitutional Law** [🔑](#) [Water, sewer, and irrigation](#)

**States** [🔑](#) [Liabilities of officers for negligence or misconduct](#)

**Water Law** [🔑](#) [Civil claims arising from failure to meet quality standards](#)

City residents sufficiently alleged that state Department of Environmental Quality officials were aware of the substantial risk of harm residents faced and were deliberately indifferent to the risk of harm, as required to state due process claim against officials for violation of the right to bodily integrity pursuant to § 1983, in consolidated class actions arising from injuries allegedly sustained as a result of contamination of city's water supply; residents' allegations included that officials knew of risks associated with river water prior to city's transition to river, that one official resolved the regulatory hurdles associated with city's use of river, and that officials took steps to deceive city's residents into continuing to drink and bathe in contaminated water. [U.S. Const. Amend. 14](#); [42 U.S.C.A. § 1983](#).

[2 Cases that cite this headnote](#)

[78] **Constitutional Law** [🔑](#) [Water, sewer, and irrigation](#)

**States** [🔑](#) [Liabilities of officers for negligence or misconduct](#)

**Water Law** [🔑](#) [Civil claims arising from failure to meet quality standards](#)

City residents failed to allege that state Department of Environmental Quality director was deliberately indifferent to the risk of harm that residents faced, as required to state due process claim against director for violation

of the right to bodily integrity pursuant to § 1983, in consolidated class actions arising from injuries allegedly sustained as a result of contamination of city's water supply, even though residents alleged that director was likely aware of the health risks posed by using river as a water source; complaint contained nothing to suggest that director either publicly denied there was a problem with city's water, or that director otherwise encouraged city residents to use contaminated water. *U.S. Const. Amend. 14*; 42 U.S.C.A. § 1983.

[79] **Constitutional Law** 🔑 Water, sewer, and irrigation

**Municipal Corporations** 🔑 Public works and improvements

**Water Law** 🔑 Civil claims arising from failure to meet quality standards

City residents sufficiently alleged that city emergency managers and were aware of the substantial risk of harm residents faced and were deliberately indifferent to the risk of harm, as required to state due process claim against managers for violation of the right to bodily integrity pursuant to § 1983, in consolidated class actions arising from injuries allegedly sustained as a result of contamination of city's water supply; residents' allegations included that managers knew about outbreak of Legionnaires' disease after city transitioned to river water, and that one manager publicly denied any connection between Legionnaires' disease outbreak and city's water despite knowing that other branches of government concluded that there was a link. *U.S. Const. Amend. 14*; 42 U.S.C.A. § 1983.

[80] **Constitutional Law** 🔑 Water, sewer, and irrigation

**Municipal Corporations** 🔑 Public works and improvements

**Water Law** 🔑 Civil claims arising from failure to meet quality standards

City residents sufficiently alleged that city director of public works and utilities administrators were aware of the substantial risk

of harm residents faced and were deliberately indifferent to the risk of harm, as required to state due process claim against director and administrators for violation of the right to bodily integrity pursuant to § 1983, in consolidated class actions arising from injuries allegedly sustained as a result of contamination of city's water supply; residents' allegations included that director was aware of the lead and Legionnaires' disease issues which followed city's transition to river water, that administrator tested for and found high concentrations of lead in the water, and that administrator altered reports to hide high lead concentrations in city's water. *U.S. Const. Amend. 14*; 42 U.S.C.A. § 1983.

2 Cases that cite this headnote

[81] **Constitutional Law** 🔑 Water, sewer, and irrigation

**Counties** 🔑 Acts of officers or agents

**Water Law** 🔑 Civil claims arising from failure to meet quality standards

City residents failed to allege how county drain commissioner either caused or prolonged their exposure to contaminated water, as required to state due process claim against commissioner for violation of the right to bodily integrity pursuant to § 1983, in consolidated class actions arising from injuries allegedly sustained as a result of contamination of city's water supply, even though commissioner may have been aware of the risk of harm residents faced; commissioner was in charge of county's water system but not city's, and residents did not allege that commissioner took steps to deceive residents about safety of city's water or that he otherwise played a role in any coverup. *U.S. Const. Amend. 14*; 42 U.S.C.A. § 1983.

[82] **Civil Rights** 🔑 Liability of Public Employees and Officials

**Constitutional Law** 🔑 Water, sewer, and irrigation

**Municipal Corporations** 🔑 Particular Officers and Official Acts

**Water Law** — Civil claims arising from failure to meet quality standards

City residents failed to allege how city's former emergency manager and city's mayor caused or prolonged residents' exposure to contaminated water, as required to state due process claim against manager and mayor for violation of the right to bodily integrity pursuant to § 1983, in consolidated class actions arising from injuries allegedly sustained as a result of contamination of city's water supply, even though manager may have set in motion chain of events that led to city's transition to river water; manager resigned before transition and lacked control over decision, mayor was stripped of virtually all authority over operations during emergency management, and residents did not allege that defendants deceived residents about the safety of city's water or that they helped coverup the crisis. [U.S. Const. Amend. 14](#).

**[83] Civil Rights** — Color of law; state action

City residents and property owners failed to allege that any act taken by any state actor created or increased the risk of private violence by a third party to residents and owners, as required to state claim against state and city government officials for violation of their right to be free from a state created danger under the Fourteenth Amendment pursuant to § 1983, in consolidated class actions arising from injuries allegedly sustained as a result of contamination of city's water supply; residents and owners only alleged that city residents used the water without knowing the danger it posed. [U.S. Const. Amend. 14](#); [42 U.S.C.A. § 1983](#).

[1 Cases that cite this headnote](#)

**[84] Civil Rights** — Particular Causes of Action

City residents and property owners failed to allege a special danger to a discrete class of individuals, as required to state claim against state and city government officials for violation of their right to be free from a state created danger under the Fourteenth Amendment pursuant to § 1983, in consolidated class actions

arising from injuries allegedly sustained as a result of contamination of city's water supply, even though residents and owners alleged that city's entire population constituted a discrete class; residents and owners' alleged discrete class included those who visited, worked, or passed through city, which was the general public of state residents. [U.S. Const. Amend. 14](#); [42 U.S.C.A. § 1983](#).

[1 Cases that cite this headnote](#)

**[85] Civil Rights** — Governmental Ordinance, Policy, Practice, or Custom

Under *Monell*, a plaintiff can bring a § 1983 claim against a city for the unconstitutional conduct of its employees if the employees' conduct implemented an unofficial custom, or a policy statement, ordinance, regulation, or decision officially adopted and promulgated by that body's officers. § 1983.

**[86] Civil Rights** — Governmental Ordinance, Policy, Practice, or Custom

City emergency managers were state officials whose edicts or acts may fairly have been said to represent official city policy, as required for city residents and property owners to state § 1983 *Monell* claim against city based on managers' alleged due process violation of the right to bodily integrity, in consolidated class actions arising from injuries allegedly sustained as a result of contamination of city's water supply. [U.S. Const. Amend. 14](#); [42 U.S.C.A. § 1983](#).

**[87] Negligence** — Trades, Special Skills and Professions

City resident and minor daughter alleged that they used city water to bathe, wash, and cook during time period that water quality consultant was involved in city's water crisis, so as to support professional negligence claim against consultant under Michigan law, in consolidated class actions arising from injuries allegedly sustained as a result of contamination of city's water supply.

**[88] Federal Civil Procedure** ➡ **Fraud, mistake and condition of mind**

City residents and property owners alleged with sufficient particularity that water quality consultant made fraudulent statements, including that city's water was safe in compliance with drinking water standards, as required to state fraud claim under Michigan law against consultant, in consolidated class actions arising from injuries allegedly sustained as a result of contamination of city's water supply; complaint's allegations included that city's water was not safe or in compliance with drinking water standards, that tainted water specifically caused medical problems apart from standard problems that might have been associated with a population's sensitivity to a clean water supply, and that this information was generally known absent consultant's representations to the contrary. *Fed. R. Civ. P. 9(b)*.

**[89] Federal Civil Procedure** ➡ **Fraud, mistake and condition of mind**

City residents and property owners' allegations that water quality consultant hired by city knew that its representations were made recklessly, which included that city's water was safe in compliance with drinking water standards, were pled with sufficient particularity, as required to state fraud claim under Michigan law against consultant, in consolidated class actions arising from injuries allegedly sustained as a result of contamination of city's water supply, even though residents and owners alleged knowledge based on information and belief; information about consultant's recklessness was solely within knowledge of consultant's decision-makers, and residents and owners alleged a voluminous factual background about the information known to city and to the public at large. *Fed. R. Civ. P. 9(b)*.

**[90] Federal Civil Procedure** ➡ **Fraud, mistake and condition of mind**

City residents and property owners failed to allege reliance on water quality consultant's fraudulent statements with sufficient particularity, which included statements that city's water was safe in compliance with drinking water standards, as required to state fraud claim under Michigan law against consultant, in consolidated class actions arising from injuries allegedly sustained as a result of contamination of city's water supply; phrase "which plaintiffs did" was sole factual allegation in complaint stating that any plaintiff specifically relied on statements in continuing to drink city's water, and that statement lacked any specificity as to when or how any plaintiff heard the allegedly fraudulent statements. *Fed. R. Civ. P. 9(b)*.

**[91] Damages** ➡ **Particular cases**

City residents and property owners failed to allege that water quality and engineering consultants committed an act that was sudden or brief, as required to state claims against consultants for negligent infliction of emotional distress under Michigan law, in consolidated class actions arising from injuries allegedly sustained as a result of contamination of city's water supply; residents and owners alleged that, over a period of months for one consultant and years for the other, the consultants repeatedly failed to properly evaluate or treat city water, which resulted in prolonged injury.

**[92] Negligence** ➡ **Trades, Special Skills and Professions****Negligence** ➡ **Nature and form of remedy**

City residents and property owners claims for negligence under Michigan law against water quality and engineering consultants were only able to be brought as professional negligence claims, in consolidated class actions arising from injuries suffered as a result of contamination of city's water supply; an ordinary layperson would have had little to no knowledge about the appropriate methods and techniques for remediating, containing, and eliminating lead and bacteria in a municipal water supply.

**[93] Negligence** → **Gross negligence**

Gross negligence is not an independent cause of action in Michigan.

**[94] Negligence** → **Gross negligence**  
**Negligence** → **Elements of Negligence**  
**Negligence** → **Defenses and Mitigating Circumstances**

Statutory gross negligence is an affirmative defense to be raised by a defendant under Michigan law; plaintiffs bringing a tort claim must still plead the common law elements of ordinary negligence.

**[95] Damages** → **Nature and Theory of Damages Additional to Compensation**

In Michigan, exemplary damages are a special class of compensatory damages; they are available under limited circumstances to reimburse for a non-economic harm.

**[96] Damages** → **Grounds for Exemplary Damages**

Exemplary damages under Michigan law, which are available under limited circumstances to reimburse for a non-economic harm, only includes losses for the humiliation, sense of outrage, and indignity that results from malicious, willful, and wanton conduct.

**[97] Damages** → **Grounds for Exemplary Damages**

The malicious, willful, and wanton element, for purposes of determining whether exemplary damages are warranted under Michigan law, is equivalent to malice.

**[98] Damages** → **Grounds for Exemplary Damages**

Because damages for mental pain and anxiety are normally included under actual damages, only intentional actions that show a reckless disregard for a plaintiff's rights will suffice to support an award of exemplary damages under Michigan law.

**[99] Damages** → **Grounds for Exemplary Damages**

Mere negligence is insufficient to support an award of exemplary damages under Michigan law; a defendant's conduct must amount to more than a lack of care.

**[100] Damages** → **Grounds for Exemplary Damages**

It is the reprehensibility of a defendant's conduct that intensifies the emotional injury and justifies exemplary damages under Michigan law, not the magnitude of the harm caused.

**[101] Damages** → **Particular cases in general**

City residents and property owners did not state a claim for allegedly malicious, willful, and wanton conduct, or claim exemplary damages for any intentional tort, as required for residents and owners to state claim for exemplary damages under Michigan law against water quality and engineering consultants, in consolidated class actions arising from injuries allegedly sustained as a result of contamination of city's water supply; residents and owners merely alleged that consultants were professionally negligent and that their negligence caused the city's water crisis.

**[102] Federal Civil Procedure** → **Tort Cases in General**

Engineering consultant was not entitled to grant of motion for a more definite statement in action brought by city residents and property owners seeking damages for injuries allegedly sustained as a result of contamination of city's water supply, notwithstanding claim that residents and

owners' failure to distinguish between various related entities made it impossible to tell what each entity did; complaint treated all three companies as a single entity since, based on the corporate structure of the companies, they were indistinguishable for lawsuit's purposes.

**[103] Federal Civil Procedure** ➔ Tort Cases in General

Engineering consultant was not entitled to grant of motion for a more definite statement in action brought by city residents and property owners' seeking damages for injuries allegedly sustained as a result of contamination of city's water supply, notwithstanding claim that contractor was being "lumped in" with defendant water quality consultant with regard to some allegations; complaint clearly specified the actions each defendant took with respect to city's water supply, and complaint sometimes referred to consultants jointly because either both sets of defendants had similar duties, or because similar claims were asserted against both sets of defendants.

**Attorneys and Law Firms**

\*817 **Brian J. McKeen**, McKeen Assoc., Claire D'Lou Vergara, McKeen & Associates, PC, **Gregory Stamatopoulos**, **Paul F. Novak**, Weitz & Luxenberg, P.C., **Julie H. Hurwitz**, **Kathryn Bruner James**, **William H. Goodman**, Goodman and Hurwitz, P.C., Detroit, MI, **Conrad J. Benedetto**, The Law Offices of Conrad J. Benedetto, Philadelphia, PA, **David J. Shea**, Shea Aiello, PLLC, **Keith L. Altman**, Excolo Law PLLC, Southfield, MI, **Deborah A. LaBelle**, Ann Arbor, MI, **Elliot M. Schaktman**, **Marc J. Bern & Partners LLP**, **Hunter Shkolnik**, **Marie Napoli**, Napoli Shkolnik Law PLLC, **Peretz Bronstein**, Bronstein, Gewirtz & Grossman LLC, New York, NY, **Emmy L. Levens**, **Jessica B. Weiner**, Cohen Milstein Sellers and Toll PLLC, Washington, DC, **Esther Berezofsky**, Motley Rice, LLC, **John McNeill Broaddus**, Weitz & Luxenberg PC, Cherry Hill, NJ, **Jayson E. Blake**, McAlpine PC, **Mark L. McAlpine**, McAlpine & McAlpine, Auburn Hills, MI, **Jordan W. Connors**, **Stephen E. Morrissey**, Susman Godfrey L.L.P., Seattle, WA, **Michael L. Pitt**, Pitt,

McGehee, Royal Oak, MI, **Theodore J. Leopold**, Cohen Milstein Sellers & Toll, PLLC, Palm Beach Gardens, FL, for Plaintiffs Luke Waid, Michelle Rodriguez, A Minor.

**Beth M. Rivers**, **Cary S. McGehee**, **Michael L. Pitt**, Pitt McGehee Palmer & Rivers, Royal Oak, MI, **Diana Gjonaj**, **Gregory Stamatopoulos**, **Paul F. Novak**, Weitz Luxenberg, P.C., **Julie H. Hurwitz**, **Kathryn Bruner James**, **William H. Goodman**, Goodman and Hurwitz, P.C., Detroit, MI, **Conrad J. Benedetto**, The Law Offices of Conrad J. Benedetto, Philadelphia, PA, **David J. Shea**, Shea Aiello, PLLC, **Keith L. Altman**, Excolo Law PLLC, Southfield, MI, **Deborah A. LaBelle**, Ann Arbor, MI, **Elliot M. Schaktman**, **Marc J. Bern & Partners LLP**, **Hunter Shkolnik**, Napoli Shkolnik Law PLLC, **Peretz Bronstein**, Bronstein, Gewirtz & Grossman LLC, New York, NY, **Esther Berezofsky**, Motley Rice, LLC, **John McNeill Broaddus**, Weitz & Luxenberg PC, Cherry Hill, NJ, **Jayson E. Blake**, McAlpine PC, **Mark L. McAlpine**, McAlpine & McAlpine, Auburn Hills, MI, **Jordan W. Connors**, Susman Godfrey L.L.P., Seattle, WA, **Emmy L. Levens**, **Jessica B. Weiner**, Cohen Milstein Sellers and Toll PLLC, Washington, DC, for Plaintiff Rhonda Kelso.

**Conrad J. Benedetto**, The Law Offices of Conrad J. Benedetto, Philadelphia, PA, **David J. Shea**, Shea Aiello, PLLC, **Keith L. Altman**, Excolo Law PLLC, Southfield, MI, **Deborah A. LaBelle**, Ann Arbor, MI, **Elliot M. Schaktman**, **Marc J. Bern & Partners LLP**, **Hunter Shkolnik**, Napoli Shkolnik Law PLLC, **Peretz Bronstein**, Bronstein, Gewirtz & Grossman LLC, New York, NY, **Esther Berezofsky**, Motley Rice, LLC, **John McNeill Broaddus**, Weitz & Luxenberg PC, Cherry Hill, NJ, **Gregory Stamatopoulos**, **Paul F. Novak**, Weitz & Luxenberg, P.C., Detroit, MI, **Jayson E. Blake**, McAlpine PC, **Mark L. McAlpine**, McAlpine & McAlpine, Auburn Hills, MI, **Jordan W. Connors**, Susman Godfrey L.L.P., Seattle, WA, **Michael L. Pitt**, Pitt, McGehee, Royal Oak, MI, **Emmy L. Levens**, **Jessica B. Weiner**, Cohen Milstein Sellers and Toll PLLC, Washington, DC, for Plaintiffs Tiantha Williams, Barbara Davis, Darrell Davis, Marilyn Bryson.

**Susan L. Burke**, **William H. Murphy, III**, **William H. Murphy, Jr.**, **Jessica Hamman Meeder**, Murphy Falcon Murphy Koch Xinis, Baltimore, MD, **Conrad J. Benedetto**, The Law Offices of Conrad J. Benedetto, Philadelphia, PA, **David J. Shea**, Shea Aiello, PLLC, **Keith L. Altman**, Excolo Law PLLC, Southfield, MI, **Deborah A. LaBelle**, Ann Arbor, MI, **Elliot M. Schaktman**, **Marc J. Bern & Partners LLP**, **Hunter Shkolnik**, Napoli Shkolnik Law PLLC, **Peretz Bronstein**, Bronstein, Gewirtz & Grossman LLC, New York, NY, **Esther Berezofsky**, Motley Rice, LLC, **John McNeill**

[Broaddus](#), Weitz & Luxenberg PC, Cherry Hill, NJ, [Gregory Stamatopoulos](#), [Paul F. Novak](#), Weitz & Luxenberg, P.C., Detroit, MI, [Jayson E. Blake](#), McAlpine PC, [Mark L. McAlpine](#), McAlpine & McAlpine, Auburn Hills, MI, [Jordan W. Connors](#), Susman Godfrey L.L.P., Seattle, WA, [Michael L. Pitt](#), Pitt, McGehee, Royal Oak, MI, [Nicholas Adam Szokoly](#), Murphy, Falcon & Murphy, P.A., Baltimore, MD, [Valdemar L. Washington](#), Flint, MI, [Emmy L. Levens](#), [Jessica B. Weiner](#), Cohen Milstein Sellers and Toll PLLC, Washington, DC, for Plaintiff EPCO Sales, LLC.

[David J. Shea](#), Shea Aiello, PLLC, [Keith L. Altman](#), Excolo Law PLLC, Southfield, MI, [Susan L. Burke](#), [William H. Murphy, III](#), [William H. Murphy, Jr.](#), [Jessica Hamman Meeder](#), Murphy Falcon Murphy Koch Xinis Baltimore, MD, [Conrad J. Benedetto](#), The Law Offices of Conrad J. Benedetto, Philadelphia, PA, [Elliot M. Schaktman](#), [Marc J. Bern & Partners LLP](#), New York, NY, [Jessica B. Weiner](#), Cohen Milstein Sellers & Toll PLLC, Washington, DC, [Nicholas Adam Szokoly](#), Murphy, Falcon & Murphy, P.A., Baltimore, MD, [Valdemar L. Washington](#), Flint, MI, for Plaintiffs Beatrice Boler, Allina Anderson.

[Todd J. Weglarz](#), Fieger, Fieger, Kenney & Harington, PC, Southfield, MI, for Plaintiffs Cholyanda Brown, Gradine Rogers.

[David J. Shea](#), Shea Aiello, PLLC, Southfield, MI, [Hunter Shkolnik](#), Napoli Shkolnik Law PLLC, New York, NY, for Plaintiffs Myia McMillian, A Minor Child, Amanda Fetterman, Melissa Lightfoot, Patricia Funches, Tiesha Tipton.

[David J. Shea](#), Shea Aiello, PLLC, Southfield, MI, [Hunter Shkolnik](#), Napoli Shkolnik Law PLLC, New York, NY, [Emmy L. Levens](#), [Jessica B. Weiner](#), Cohen Milstein Sellers and Toll PLLC, Washington, DC, [Gregory Stamatopoulos](#), [Paul F. Novak](#), Weitz & Luxenberg, P.C., Detroit, MI, [Jordan W. Connors](#), Susman Godfrey L.L.P., Seattle, WA, [Michael L. Pitt](#), Pitt, McGehee, Royal Oak, MI, for Plaintiff Amber Brown.

[David J. Shea](#), Shea Aiello, PLLC, Southfield, MI, for Plaintiff Mary Mathes.

[David J. Shea](#), Shea Aiello, PLLC, Southfield, MI, [Emmy L. Levens](#), [Jessica B. Weiner](#), [Kit A. Pierson](#), Cohen Milstein Sellers and Toll PLLC, Washington, DC, [Jordan W. Connors](#), [Stephen E. Morrissey](#), Susman Godfrey L.L.P., Seattle, WA, [Theodore J. Leopold](#), Cohen Milstein Sellers & Toll, PLLC, Palm Beach Gardens, FL, [Gregory Stamatopoulos](#), [Paul F.](#)

[Novak](#), Weitz & Luxenberg, P.C., Detroit, MI, [Michael L. Pitt](#), Pitt, McGehee, Royal Oak, MI, for Plaintiff Frances Gilcreast.

[David J. Shea](#), Shea Aiello, PLLC, Southfield, MI, [Emmy L. Levens](#), [Kit A. Pierson](#), Cohen Milstein Sellers and Toll PLLC, Washington, DC, [Jordan W. Connors](#), [Stephen E. Morrissey](#), Susman Godfrey L.L.P., Seattle, WA, [Theodore J. Leopold](#), Cohen Milstein Sellers & Toll, PLLC, Palm Beach Gardens, FL, for Plaintiffs Vivian Kelley, Estella A. Simpkins.

[Solomon M. Radner](#), Madeline M. Sinkovich, Excolo Law, PLLC, [Ari Kresch](#), Southfield, MI, [Chet W. Kern](#), Katz & Kern, LLP, [Marc J. Bern](#), Bern Ripka LLP, [Debra J. Humphrey](#), [Elliot M. Schaktman](#), [Ryan Steven Sharp](#), [Marc J. Bern & Partners LLP](#), New York, NY, for Plaintiff Lawrence Washington, Jr.

[Solomon M. Radner](#), Madeline M. Sinkovich, Excolo Law, PLLC, [Ari Kresch](#), Southfield, MI, [Chet W. Kern](#), Katz & Kern, LLP, [Marc J. Bern](#), Bern Ripka LLP, [Ryan Steven Sharp](#), [Marc J. Bern](#) and Partners LLP, New York, NY, for Plaintiffs Taylor Washington, Morgan Washington, Chloe Washington, Madison Washington.

[Solomon M. Radner](#), Madeline M. Sinkovich, Excolo Law, PLLC, [Ari Kresch](#), Southfield, MI, [Chet W. Kern](#), Katz & Kern, LLP, [Marc J. Bern](#), Bern Ripka LLP, [Debra J. Humphrey](#), [Ryan Steven Sharp](#), [Marc J. Bern & Partners LLP](#), New York, NY, for Plaintiff Lawrence Washington.

[Solomon M. Radner](#), Excolo Law, PLLC, [Ari Kresch](#), Southfield, MI, [Chet W. Kern](#), Katz & Kern, LLP, [Marc J. Bern](#), Bern Ripka LLP, New York, NY, for Plaintiffs Christa Godin, Angel Arrand, Auston Arrand, William Zietz, III, Roosevelt Cameron, Deborah Smith, Aby Ndoeye, Shanika Mixon, Robin Davis.

[David J. Shea](#), Shea Aiello, PLLC, Southfield, MI, [Emmy L. Levens](#), Cohen Milstein Sellers and Toll PLLC, Washington, DC, [Hunter Shkolnik](#), Napoli Shkolnik Law PLLC, New York, NY, [Jordan W. Connors](#), [Stephen E. Morrissey](#), Susman Godfrey L.L.P., Seattle, WA, [Theodore J. Leopold](#), Cohen Milstein Sellers & Toll, PLLC, Palm Beach Gardens, FL, for Plaintiffs Tiffany Davenport, Merin Johnson, Gregory Miller, Twylla Walker.

[Emmy L. Levens](#), Cohen Milstein Sellers and Toll PLLC, Washington, DC, [Hunter Shkolnik](#), Napoli Shkolnik Law PLLC, New York, NY, for Plaintiffs B.W., E.D., M. N., D.W.

[Andrew P. Abood](#), Abood Law Firm, East Lansing, MI, [Beth M. Rivers](#), [Cary S. McGehee](#), [Michael L. Pitt](#), Pitt McGehee Palmer & Rivers, Royal Oak, MI, [Cynthia M. Lindsey](#), Cynthia Lindsey & Associates, [Gregory Stamatopoulos](#), [Paul F. Novak](#), Weitz & Luxenberg, P.C., [Julie H. Hurwitz](#), [Kathryn Bruner James](#), [William H. Goodman](#), Goodman and Hurwitz, P.C., Detroit, MI, [Deborah A. LaBelle](#), Ann Arbor, MI, [Emmy L. Levens](#), [Jessica B. Weiner](#), Cohen Milstein Sellers and Toll PLLC, Washington, DC, [Esther Berezofsky](#), Motley Rice, LLC, Cherry Hill, NJ, [Hunter Shkolnik](#), Napoli Shkolnik Law PLLC, New York, NY, [Jordan W. Connors](#), [Stephen E. Morrissey](#), Susman Godfrey L.L.P., Seattle, WA, Teresa Ann Caine Bingman, Law Offices of Teresa A. Bingman, Okemos, MI, [Theodore J. Leopold](#), Cohen Milstein Sellers & Toll, PLLC, Palm Beach Gardens, FL, for Plaintiffs Village Shores LLC, Angelos Coney Island Palace, Inc., Jan Burgess, David Munoz, Joshua Boggess, Michael Snyder, Sherry Mulherin, Laquisha Jacobs, Zhanna Gardin, Eddie Hammond.

[Esther Berezofsky](#), Motley Rice, LLC, Cherry Hill, NJ, [Valdemar L. Washington](#), Flint, MI, [Mark R. Cuker](#), Cuker Law Firm, LLC, Philadelphia, PA, for Plaintiffs Tommie Lowery Sr., Tommie Lowery, Jr., Isiah Lowery, Marcus Lowery, Sandra York.

[Esther Berezofsky](#), Motley Rice, LLC, Cherry Hill, NJ, [Valdemar L. Washington](#), Flint, MI, for Plaintiffs Dorsey Ross, Jr., Eugene Nelson, Bernie Nelson.

[Hunter Shkolnik](#), Napoli Shkolnik Law PLLC, New York, NY, for Plaintiffs Tonya Rogers, A. H., Z.R., Carl Johnson, William Thomas, Chantal Travis, J.T., Y.T., D.T., Renee Anderson, Shalandra Green, Z.T., Aloysius Garrow.

[Beth M. Rivers](#), [Cary S. McGehee](#), [Michael L. Pitt](#), Pitt McGehee Palmer & Rivers, Royal Oak, MI, [David J. Shea](#), Shea Aiello, PLLC, Southfield, MI, [Deborah A. LaBelle](#), Ann Arbor, MI, [Diana Gjonaj](#), [Gregory Stamatopoulos](#), [Paul F. Novak](#), Weitz Luxenberg, P.C., [Julie H. Hurwitz](#), [Kathryn Bruner James](#), [William H. Goodman](#), Goodman and Hurwitz, P.C., Detroit, MI, for Plaintiffs Melissa Mays, Michael A. Mays, Keith John Pemberton, Jacqueline Pemberton.

[Beth M. Rivers](#), [Cary S. McGehee](#), [Michael L. Pitt](#), Pitt McGehee Palmer & Rivers, Royal Oak, MI, [David J. Shea](#), Shea Aiello, PLLC, Southfield, MI, [Deborah A. LaBelle](#), Ann Arbor, MI, [Diana Gjonaj](#), [Gregory Stamatopoulos](#), [Paul F. Novak](#), Weitz Luxenberg, P.C., [Julie H. Hurwitz](#), [Kathryn Bruner James](#), [William H. Goodman](#), Goodman and Hurwitz,

P.C., Detroit, MI, [Emmy L. Levens](#), [Jessica B. Weiner](#), Cohen Milstein Sellers and Toll PLLC, Washington, DC, [Jordan W. Connors](#), Susman Godfrey L.L.P., Seattle, WA, for Plaintiff Elnora Carthan.

[Beth M. Rivers](#), [Cary S. McGehee](#), [Michael L. Pitt](#), Pitt McGehee Palmer & Rivers, Royal Oak, MI, [Deborah A. LaBelle](#), Ann Arbor, MI, [Gregory Stamatopoulos](#), [Paul F. Novak](#), Weitz & Luxenberg, P.C., Detroit, MI, for Plaintiffs Brandyn Carpenter, Jess Carpenter, Kimberly Carpenter, Adam Murphy, Christina Murphy.

[Paul F. Novak](#), Weitz & Luxenberg, P.C., Detroit, MI, for Plaintiff a minor child.

[Paul F. Novak](#), Weitz & Luxenberg, P.C., Detroit, MI, [Deborah A. LaBelle](#), Ann Arbor, MI, for Plaintiffs a minor child CM1, a minor child CM2, a minor child CM3.

[David J. Shea](#), Shea Aiello, PLLC, Southfield, MI, [Susan L. Burke](#), [William H. Murphy, III](#), [William H. Murphy, Jr.](#), [Jessica Hamman Meeder](#), Murphy Falcon Murphy Koch Xinis Baltimore, MD, [Nicholas Adam Szokoly](#), Murphy, Falcon & Murphy, P.A., Baltimore, MD, [Valdemar L. Washington](#), Flint, MI, for Plaintiff Rev. Edwin Anderson.

[Eugene Driker](#), [Todd R. Mendel](#), [Morley Witus](#), Barris, Sott, Denn & Driker, PLLC, Detroit, MI, [Margaret A. Bettenhausen](#), [Nathan A. Gambill](#), [Zachary C. Larsen](#), Michigan Department of Attorney General, [Richard S. Kuhl](#), Assistant Attorney General, Lansing, MI, for Defendant Richard D. Snyder.

[Margaret A. Bettenhausen](#), [Nathan A. Gambill](#), [Zachary C. Larsen](#), Michigan Department of Attorney General, [Richard S. Kuhl](#), Assistant Attorney General, Lansing, MI, for Defendants State of Michigan, Michigan Department of Environmental Quality, Dennis Muchmore, Michigan Department of Health and Human Services, Eden Wells, Nick Lyon, Linda Dykema, Andrew Dillon.

[Charles E. Barbieri](#), Foster, Swift, [Michael J. Pattwell](#), Clark Hill, PLC, Lansing, MI, [Christopher B. Clare](#), Clark Hill PIC, Washington, DC, [Jay M. Berger](#), Clark Hill, Detroit, MI, [Jordan S. Bolton](#), Clark Hill PLC, Birmingham, MI, for Defendants Daniel Wyant, Bradley Wurfel.

[Charles E. Barbieri](#), Foster, Swift, [Michael H. Perry](#), [Thaddeus E. Morgan](#), Fraser, Trebilcock, Lansing, MI, for Defendant Liane Shekter-Smith.

[James A. Fajen](#), Ann Arbor, MI, [James W. Burdick](#), Burdick Law, P.C., Bloomfield Hills, MI, [Lisa L. Dwyer](#), Detroit, MI, for Defendant Adam Rosenthal.

[Charles E. Barbieri](#), Foster, Swift, Lansing, MI, [Courtney B. Ciullo](#), [Dennis K. Egan](#), Kotz, Sangster, Wysocki & Berg, P.C., Detroit, MI, [Krista A. Jackson](#), [Philip A. Grashoff, Jr.](#), Smith Haughey Rice & Roegge, Grand Rapids, MI, for Defendant Stephen Busch.

[Allison M. Collins](#), [Charles E. Barbieri](#), Foster, Swift, Collins & Smith, P.C., Lansing, MI, for Defendants Patrick Cook, Michael Prysby.

[Angela N. Wheeler](#), [William Young Kim](#), City of Flint, Flint, MI, [Todd Russell Perkins](#), Detroit, MI, for Defendant Darnell Earley.

[Barry A. Wolf](#), [Barry A. Wolf](#), Attorney at Law, PLLC, [William Young Kim](#), [Angela N. Wheeler](#), City of Flint, Flint, MI, for Defendant Gerald Ambrose.

[Angela N. Wheeler](#), [William Young Kim](#), City of Flint, Flint, MI, for Defendant Dayne Walling.

[Alexander S. Rusek](#), White Law PLLC, Okemos, MI, [Angela N. Wheeler](#), [William Young Kim](#), City of Flint, Flint, MI, for Defendant Howard Croft.

[Angela N. Wheeler](#), [William Young Kim](#), City of Flint, Flint, MI, [Brett T. Meyer](#), Plunkett Cooney, Grand Rapids, MI, [Gregory W. Mair](#), [Christopher James Marker](#), O'Neill Wallace & Doyle, Saginaw, MI, for Defendant Michael Glasgow.

[Edwar A. Zeineh](#), Law Office of Edwar A. Zeineh, PLLC, Lansing, MI, [David W. Meyers](#), Lexington, MI, for Defendant Daugherty Johnson.

[Angela N. Wheeler](#), [William Young Kim](#), City of Flint, Flint, MI, [Frederick A. Berg](#), Butzel Long, Detroit, MI, [Sheldon H. Klein](#), Butzel Long, Bloomfield Hills, MI, Stacy L.E. Oakes, Oakes Law Group, PLLC, Saginaw, MI, for Defendant City of Flint.

[Michael J. Gildner](#), Simen, Figura, Flint, MI, for Defendant Edward Kurtz.

[Cheryl A. Bush](#), [Michael R. Williams](#), Bush, Seyferth & Paige, PLLC, Troy, MI, [James M. Campbell](#), Campbell, Campbell, [John A.K. Grunert](#), Campbell, Conroy & O'Neil, P.C., Boston, MA, for Defendants Veolia North America, Inc.,

Veolia North America, LLC, Veolia Water North America Operating Services, LLC.

[James M. Campbell](#), Campbell, Campbell, [John A.K. Grunert](#), Campbell, Conroy & O'Neil, P.C., Boston, MA, [Cheryl A. Bush](#), [Michael R. Williams](#), Bush, Seyferth & Paige, PLLC, Troy, MI, for Defendant Veolia North America Operating Services, LLC.

[David C. Kent](#), Drinker Biddle Reath LLP, Dallas, TX, [Robert G. Kamenec](#), [Karen E. Beach](#), Plunkett & Cooney, Bloomfield Hills, MI, [Philip A. Erickson](#), Plunkett & Cooney, East Lansing, MI, for Defendants Lockwood, Andrews & Newnam, P.C., Leo A. Daly Company.

[Philip A. Erickson](#), Plunkett & Cooney, East Lansing, MI, for Defendants Lan Inc., Leo Daily.

[David C. Kent](#), Drinker Biddle Reath LLP, Dallas, TX, [Philip A. Erickson](#), Plunkett & Cooney, East Lansing, MI, [Robert G. Kamenec](#), Plunkett & Cooney, Bloomfield Hills, MI, for Defendants Lockwood, Andrews & Newman, Inc., Leo A. Daly Corp., Lockwood, Andrews & Newman, P.C.

[Michael S. Cafferty](#), Michael S. Cafferty & Assoc., Detroit, MI, [Richard S. Kuhl](#), Assistant Attorney General, Lansing, MI, for Defendant Nancy Peeler.

Kurt E. Krause, Michigan State University Office of the General Counsel, [Mary Chartier-Mittendorf](#), East Lansing, MI, for Defendant Robert Scott.

[Craig S. Thompson](#), [Jennifer R. Moran](#), Sullivan, Ward, Southfield, MI, for Defendant Rowe Professional Service Company.

[Margaret A. Bettenhausen](#), [Zachary C. Larsen](#), Michigan Department of Attorney General, Lansing, MI, for Defendant R. Kevin Clinton.

[William Young Kim](#), City of Flint, Flint, MI, for Defendant Mike Brown.

[Gregory M. Meihn](#), [John Stephen Gilliam](#), [Matthew Wise](#), Foley & Mansfield, PLLP, Ferndale, MI, [Kevin K. Kilby](#), [Stacy J. Belisle](#), [Thomas J. McGraw](#), McGraw Morris P.C., Troy, MI, [Joseph F. Galvin](#), Genesee County Drain Commissioners Office, Flint, MI, for Defendant Jeff Wright.

[Angela N. Wheeler](#), [William Young Kim](#), City of Flint, Flint, MI, [Edwar A. Zeineh](#), Law Office of Edwar A. Zeineh, PLLC,

Lansing, MI, [David W. Meyers](#), Lexington, MI, for Defendant Daugherty Johnson.

[David C. Kent](#), Drinker Biddle Reath LLP, Dallas, TX, [Philip A. Erickson](#), Plunkett & Cooney, East Lansing, MI, for Defendant Lockwood.

[David C. Kent](#), Drinker Biddle Reath LLP, Dallas, TX, [Philip A. Erickson](#), Plunkett & Cooney, East Lansing, MI, [Robert G. Kamenech](#), [Karen E. Beach](#), Plunkett & Cooney, Bloomfield Hills, MI, for Defendant Lockwood, Andrews & Newnam, Inc.

**OPINION AND ORDER GRANTING IN PART AND DENYING IN PART PUTATIVE CLASS PLAINTIFFS' MOTION FOR LEAVE TO FILE AN AMENDED COMPLAINT [620] AND GRANTING IN PART AND DENYING IN PART DEFENDANTS' MOTIONS TO DISMISS**

[JUDITH E. LEVY](#), United States District Judge

\*822 This is a class action lawsuit that is part of the litigation collectively referred to as the Flint Water Cases. To those following these cases, the facts are by now well known. Plaintiffs, residents and property owners in Flint, Michigan, were exposed to lead, legionella, and other contaminants within the municipal water supply. They allege that defendants, a collection of government officials and private parties, caused or prolonged this exposure, injuring them and damaging their property. In this opinion and order, the Court will address the following: plaintiffs' motion for leave to file a fourth amended complaint and defendants' motions to dismiss the entire case.

**Table of Contents**

**I. Procedural History...823**

**II. Motion for Leave to Amend the Complaint...824**

**a. Background...824**

**b. Standard of Review...837**

**c. Threshold Issues...837**

**d. Main Analysis...838**

**1. Bodily Integrity...839**

**2. Equal Protection...842**

**3. Elliott Larsen Civil Rights Act...849**

**4. Conspiracy...851**

**5. Gross Negligence...852**

**6. Res Judicata and Statute of Limitations...854**

**e. Conclusion...854**

**III. Motions to Dismiss...854**

**a. Background...854**

**b. Standard of Review...855**

**c. Threshold Issues...855**

**d. Main Analysis...857**

**i. Federal Claims...857**

**1. Bodily Integrity...857**

\*823 **2. Equal Protection...861**

**3. Conspiracy...862**

**4. State-Created Danger...862**

**5. *Monell* Liability...865**

**ii. State Claims...866**

**1. ELCRA...866**

**2. Professional Negligence...866**

**3. Fraud...866**

**4. Negligent Infliction of Emotional Distress...869**

**5. Negligence...871**

**6. Gross Negligence...871**

**7. Exemplary Damages...872**

**e. Conclusion and Order...873**

**I. Procedural History**

Although this lawsuit is still in its early phases, its procedural history is complicated. This case was consolidated with eight other Flint water class action complaints on July 27, 2017.

(Dkt. 173.)<sup>1</sup> In September 2017, plaintiffs filed their first amended consolidated class action complaint. (Dkt. 214.) A second amended complaint followed less than a month later. (Dkt. 238.) Then, plaintiffs filed a third amended complaint on January 25, 2018. (Dkt. 349.)

Defendants filed motions to dismiss the complaint under [Federal Rules of Civil Procedure 12\(b\)\(1\) and \(6\)](#). (Dkts. 273, 274, 276–79, 281–83, 294.) On August 1, 2018, after hearing oral argument on the motions to dismiss, the Court issued an opinion and order granting defendants' motions in part and denying them in part. *Carthan v. Snyder (In re Flint Water Cases)*, 329 F.Supp.3d 369 (E.D. Mich. 2018), *vacated*, No. 16-cv-10444, 2018 U.S. Dist. LEXIS 192371 (E.D. Mich. Nov. 9, 2018). Several defendants appealed part of the ruling to the Sixth Circuit Court of Appeals (Dkts. 570, 573, 575, 579, 589); others filed motions for reconsideration. (Dkts. 560–61.) And under the prevailing rules, the Sixth Circuit awaited the resolution of the motions for reconsideration prior to taking jurisdiction. *Carthan v. Snyder*, No. 18-1967 (6th Cir. Aug. 28, 2018).

Plaintiffs filed a motion for leave to amend the complaint for a fourth time before the Court had resolved the pending motions for reconsideration. (Dkt. 620.) The Court granted leave for plaintiffs in other Flint water lawsuits to file similar motions,<sup>2</sup> and, as a result, there was a significant risk that the Flint Water Cases would proceed in a piecemeal fashion. (Dkt. 670.) The Court was managing filings from more than 150 lawyers and coordinating with related federal and state cases. Having different lawsuits proceed on divergent allegations and differing claims would further complicate the case. Such a scenario would also burden defendants, who would be unable to wage consistent defenses. In the interest of handling the cases in a consistent manner, the Court interpreted plaintiffs' motion as a joint motion for relief from judgment and a motion for leave to file an amended complaint. Finding just cause, the Court vacated its August 1 decision on November 9, 2018, so that it could consider plaintiffs' motion for leave to amend. (*Id.*)<sup>3</sup>

**\*824** To fulfill its duty to ensure that the litigation proceeds efficiently, the Court adopted an unorthodox but necessary plan. Noting that there was significant overlap between the proposed fourth amended complaint and the third amended complaint, and that a motion for leave to amend and a motion to dismiss turn on substantively the same standard, the Court determined that it would treat defendants' responses to the motion for leave to amend as addenda to their previously

filed motions to dismiss. (Dkt. 714.) The Court would then issue an omnibus opinion and order, adjudicating plaintiffs' motion for leave to file a fourth amended complaint, and, if successful, defendants' motions to dismiss it in a single decision. Mindful of the parties' rights, the Court also gave plaintiffs and defendants the opportunity to file supplemental briefing. (*Id.*)

The Court now addresses plaintiffs' motion for leave to amend the complaint and defendants' motions to dismiss. Accordingly, this opinion and order will proceed as follows: Part II will address plaintiffs' motion for leave to file a fourth amended complaint, and, for the reasons set forth below, the Court will grant it in part and deny it in part. In Part III, rather than wait for plaintiffs to file their amended complaint, the Court will adopt the fourth amended complaint as the operative complaint and rule on defendants' motions to dismiss it. The Court's ruling in Part II will be preclusive, so any claim found lacking there will not be addressed separately in Part III. The result will be that the fourth amended complaint, to the extent permitted below, will become the operative complaint for the purposes of this litigation.

## II. Motion for Leave to Amend the Complaint

### a. Background

#### i. The Parties

There are three types of named plaintiffs. First, the following Flint residents claim personal injury and property damage: Elnora Carthan, a seventy-two year old African American woman; Rhonda Kelso, a fifty-four year old African American woman who also represents the interests of her minor child; Darrell and Barbara Davis, both African Americans; Marilyn Bryson, a fifty-eight year old African American woman; and David Munoz, a Hispanic man.

Next, the following Flint residents only claim personal injury: Michael Snyder, the personal representative of the estate of John Snyder, who received medical treatment in Flint before his death; Tiantha Williams, a forty-year old African American woman and her minor child; and Amber Brown and her minor child.

Finally, the following individuals only claim property damage: Frances Gilcreast, on behalf of her Flint real estate partnership FG&S Investments; EPCO Sales, a domestic

limited liability company located in Flint; and Angelo's Coney Island Palace, a Michigan corporation located in Flint.

The named defendants can be separated into two groups: government and private defendants. First, the government defendants include the state defendants Rick Snyder, the former Governor of Michigan;<sup>4</sup> \*825 the State of Michigan; Andy Dillon, then-Treasurer for the State of Michigan; Nick Lyon, the previous Director of the Michigan Department of Health and Human Services (MDHHS); and Nancy Peeler, former MDHHS Director for the Program for Maternal, Infant, and Early Childhood Home Visiting.

The government defendants also include the Michigan Department of Environmental Quality (MDEQ) defendants Daniel Wyant, Director of the MDEQ; Liane Shekter-Smith, MDEQ Chief of the Office of Drinking Water and Municipal Assistance; Adam Rosenthal, an MDEQ Water Quality Analyst; Stephen Busch, an MDEQ District Supervisor; Patrick Cook, an MDEQ Water Treatment Specialist; Michael Prysby, an MDEQ Engineer assigned to MDEQ District 11 (where Flint is located); and Bradley Wurfel, the MDEQ Director of Communications.

Additionally, the government defendants include the city defendants Edward Kurtz, Flint's Emergency Manager from August 2012 to July 2013; Darnell Early, Emergency Manager from September 2013 to January 2015; Gerald Ambrose, Emergency Manager from January 2015 to April 2015; Dayne Walling, Mayor of Flint from August 2009 to November 2015; Howard Croft, Flint's former Director of Public Works; Michael Glasgow, Flint's former Utilities Administrator; Daugherty Johnson, another former Utilities Administrator; and the City of Flint.

Finally, Jeffrey Wright is also a government defendant. Wright is the Genesee County Drain Commissioner and current Chief Executive Officer of the Karegnondi Water Authority (KWA).

The private defendants include Lockwood, Andrews & Newman, PC, Lockwood Andrews & Newman, Inc., and the Leo. A. Daly Company (collectively "LAN"); and Veolia, LLC, Veolia, Inc., and Veolia Water (collectively "Veolia"). LAN performed consultancy work in Flint related to the water supply transition to the Flint River, whereas Veolia performed consultancy work in Flint after the transition, in February and March 2015.

## ii. Facts As Pleaded in the Third Amended Complaint

What follows is a summary of the facts set forth in the third amendment complaint, as summarized in the Court's vacated August 1, 2018, opinion and order:

An 1897 city ordinance required that all water pipes in Flint be made of lead. (Dkt. 349 at 38.) In 1917, the Flint Water Treatment Plant (FWTP) was constructed, and drew water from the Flint River as Flint's primary water source until 1964, when it went dormant. (*Id.*) From 1964 through 2014, users of municipal water in Flint, Michigan received their water through the Detroit Water and Sewerage Department (DWSD). (*Id.* at 38.) In 2014, Flint's water supply switched back to the Flint River, and the water was treated at the FWTP. (*Id.* at 54.) This case concerns the decision to return to the Flint River as Flint's primary water source in 2014, and the alleged injuries that arose from that switch.

Beginning in the 1990s, Flint, along with other local governments relying on the DWSD water supply, had concerns about the cost of that supply, and began studying the viability of alternative water supplies. (*Id.* at 38.) In 2001, Michigan's Department of Natural Resources noted that businesses along the Flint River had permits to discharge industrial and mining runoff, as well as petroleum and gasoline cleanups. (*Id.* at 39.) In 2004, a study by the United States Geological Survey, the [MDEQ], and the Flint Water Utilities Department determined \*826 that the Flint River was a highly sensitive drinking water source susceptible to contamination. (*Id.*) In 2006 and 2009, Flint and other local governments commissioned a study from LAN regarding the viability of continuing to purchase water from the DWSD or constructing a new pipeline, which would be administered by what would later be known as the [KWA]. (*Id.*)

In 2011, Flint commissioned a study by Rowe Engineering and LAN to determine if the Flint River could be safely used as a water supply. (*Id.*) The study determined that water from the Flint River would require more treatment than water from Lake Huron, and that proper treatment for Flint River water would require upgrades to the FWTP. (*Id.*) The report included an addendum that set forth over sixty-nine million dollars in improvements that would be necessary to use Flint River water through the FWTP, including the use of corrosion control chemicals. (*Id.* at 40.)

In August 2012, Michigan Governor Rick Snyder appointed Edward Kurtz as Flint's Emergency Manager, following the declaration of a financial emergency in Flint. (*Id.* at 40.) Emergency managers may be appointed by the governor of Michigan “to address a financial emergency within” a local government, subject to the limitations in Michigan Public Act 436 of 2012. [M.C.L. § 141.1549\(1\)](#).

Upon appointment, an emergency manager shall act for and in the place and stead of the governing body and the office of chief administrative officer of the local government. The emergency manager shall have broad powers in receivership to rectify the financial emergency and to assure the fiscal accountability of the local government and the local government's capacity to provide or cause to be provided necessary governmental services essential to the public health, safety, and welfare. Following appointment of an emergency manager and during the pendency of receivership, the governing body and the chief administrative officer of the local government shall not exercise any of the powers of those offices except as may be specifically authorized in writing by the emergency manager or as otherwise provided by this act and are subject to any conditions required by the emergency manager.

[M.C.L. § 141.1549\(2\)](#).

In November 2012, Kurtz suggested to State of Michigan Treasurer Andy Dillon that Flint join the proposed KWA under the belief that doing so would save money over continuing to purchase water from the DWSD. (Dkt. 349 at 40.) The KWA was to be an administrative body overseeing a pipeline that would use Lake Huron water for the areas it serviced. (*Id.* at 39.) Genesee County Drain Commissioner Jeff Wright had encouraged the formation of the KWA in 2009. (*Id.* at 40.)

DWSD argued throughout 2012 that Flint should not join the KWA based on cost and reliability projections. (*Id.*) It made these arguments to Governor [Snyder], Wright, Kurtz, Dillon, and then-Mayor of Flint Dayne Walling. (*Id.*) During that period, Wright consistently argued to Kurtz, Dillon, and Governor Snyder that the DWSD studies were wrong. (*Id.* at 41.) In late 2012, Dillon requested that an independent engineering firm assess the cost effectiveness of joining the KWA. (*Id.*) The firm concluded that remaining with DWSD was more cost-effective both in the short and long term. (*Id.*) On March 17, 2013, Dillon e-mailed Governor Snyder and stated that the KWA

advocates were misrepresenting \*827 the benefits of a switch, and that the “[r]eport I got is that Flint should stay w [sic] DWSD.” (*Id.*)

On March 26, 2013, MDEQ District Supervisor Stephen Busch sent an e-mail to MDEQ Director Daniel Wyant and MDEQ Chief of the Office of Drinking Water and Municipal Assistance Liane Shekter-Smith setting forth risks associated with using the Flint River as Flint's drinking water source. (*Id.*) The e-mail stated that the water posed increased health risks, including a microbial risk, a risk of trihalomethane (known as “Total Trihalomethanes” or “TTHM”) exposure, and would come with additional regulatory requirements, including significant upgrades to the FWTP. (*Id.* at 41–42.)

On March 27, 2013, MDEQ officials acknowledged that the decision to stay with the DWSD or switch to the Flint River was not based on the scientifically determined suitability of the water, but instead that it was “entirely possible that they will be making decisions relative to cost,” in the words of MDEQ Deputy Director Jim Sygo. (*Id.* at 42.)

On March 28, 2013, Dillon e-mailed Governor Snyder and other officials, and recommended that the state “support the City of Flint's decision to join the KWA,” and that all relevant officials supported the move. (*Id.* at 42–43.) During this period, Governor Snyder was personally involved in the decision-making process. (*Id.* at 43.) On April 4, 2013, Governor Snyder's Chief of Staff Dennis Muchmore informed Governor Snyder that “[a]s you know, the Flint people have requested Dillon's ok to break away from the DWSD.” (*Id.*) Governor Snyder then instructed his Chief of Staff, Dillon, the Emergency Manager of Detroit Kevin Orr, DWSD, and Kurtz to solicit an additional offer from the DWSD before permitting the transition away from the DWSD. (*Id.*)

DWSD submitted its final proposal later in April. (*Id.*) Kurtz and Orr, according to an e-mail from a Senior Policy Advisor in the Michigan Department of Treasury, determined that Flint would not accept the DWSD offer. (*Id.* at 44.) Governor Snyder's Executive Director forwarded the e-mail to Governor Snyder on April 29, 2013, and stated that it “[l]ooks like they adhered to the plan.” (*Id.*)

Following this communication, Governor Snyder authorized Kurtz to enter into a contractual relationship with the KWA beginning in mid-2016. (*Id.*) At the time

Governor Snyder authorized the switch, he did so knowing the Flint River would be used as an interim source. (*Id.*) In June 2013, Dillon, Kurtz, Wright, and Walling developed an interim plan to govern the provision of water to Flint between April 25, 2014, and October 2016. (*Id.*)

On June 10, 2013, LAN submitted a proposal to Flint for upgrading the FWTP. (*Id.* at 48.) The proposal included a “Scope of Services” section that proposed upgrades to the FWTP that would permit “use of the Flint River as a water supply,” and a “Standards of Performance” section that promised LAN would “exercise independent judgment” and “perform its duties under this contract in accordance with sound professional practices.” (*Id.* at 49.) Flint retained LAN to advise it on the water source transition through 2015. (*Id.* at 49–50.)

On June 29, 2013, LAN met with representatives from Flint, the Genesee County Drain Commissioner's Office, and MDEQ to discuss logistics related to the transition to the Flint River as Flint's primary water source. (*Id.* at 50.) \*828 At that meeting, the participants determined that the Flint River was a viable water source, if more difficult to treat, and that upgrades could be made to the FWTP to properly treat the water. (*Id.*) The parties also determined that it was possible to conduct proper quality control with LAN's assistance, the FWTP did not have the capacity to meet the needs of both Flint and Genesee County, and the transition could occur by April or May of 2014. (*Id.* at 51.) LAN agreed to present a comprehensive project proposal with cost estimates. (*Id.*) LAN ultimately provided engineering services for the transition from July 2013 until the transition occurred on April 25, 2014, including creating the plans and specification for the transition. (*Id.* at 53–54.)

Kurtz resigned from his Emergency Manager position effective July 2013. (*Id.* at 45.) Following Michael Brown serving as Emergency Manager for two months, Darnell Earley was appointed as Emergency Manager for Flint in September 2013. (*Id.*) Part of Earley's job included making sure Flint was in compliance with state and federal laws governing safe drinking water. (*Id.*)

The transition to the Flint River continued. On March 14, 2014, Brian Larkin, then associate director of the Governor's Office of Urban and Metropolitan Initiatives, sent an e-mail to others in the Governor's office stating that the timeframe for switching water supplies was “less than

ideal and could lead to some big potential disasters down the road.” (*Id.* at 45–46.)

On March 20, 2014, MDEQ Chief of the Office of Drinking Water and Municipal Assistance Liane Shekter-Smith ensured that the City of Flint received an Administrative Consent Order requiring use of the FWTP, mandating Flint take steps to continue use of Flint River water or take steps to join the KWA, and attempting to prevent Flint's return to use of the DWSD. (*Id.* at 46.) Shekter-Smith had been warned nearly a year earlier about the potential dangers of switching Flint's water supply to the Flint River. (*Id.*)

In April 2014, LAN, Flint, and MDEQ officials discussed optimization for lead in the water supply, and decided to seek more data before implementing an optimization method. (*Id.* at 52.)

On April 16, 2014, former Flint Utility Administrator Michael Glasgow had informed MDEQ Water Analyst Adam Rosenthal that he would like additional time to ensure the FWTP was meeting requirements before giving the okay to distribute water from it. (*Id.* at 46.) On April 17, 2014, Glasgow informed MDEQ that the FWTP was not fit to begin operation, and that “management” refused to listen to his warnings. (*Id.*) On April 18, 2014, Glasgow wrote to Busch and MDEQ Engineer Michael Prysby and informed them that although he was receiving pressure to begin distributing water, he would not give the okay to do so, because he did not feel that staff was trained or proper monitoring was in place. (*Id.* at 46–47.) Glasgow felt that “management” had its “own agenda.” (*Id.* at 47.) Glasgow later told investigators that former Flint Director of Public Works Howard Croft and former Flint Utilities Administrator Daugherty Johnson pressured Glasgow to approve and begin the switch to Flint River water. (*Id.*)

At some point in 2014, MDEQ Water Treatment Specialist Patrick Cook signed the final permit necessary to restart use of the FWTP with the Flint River as the city's primary water source. (*Id.* at 48.) The FWTP officially went into service and began delivering Flint \*829 River water to Flint water users on April 25, 2014. (*Id.*)

When the transition occurred, Flint's water treatment system was not prepared to safely deliver Flint River water to users. The river was contaminated with rock-salt chlorides from treatment of roads in and around Flint during past winters. (*Id.* at 52.) Chlorides are corrosive, and water must be treated to neutralize their corrosive properties. (*Id.*) This is particularly true in a city like Flint,

where most of Flint's water mains are over seventy-five years old and made of cast iron, leaving them subject to internal corrosion called "tuberculation." (*Id.* at 57.) Tuberculation leads to the development of "biofilms," which are layers of bacteria attached to the interior pipe wall. (*Id.*) Although LAN provided professional engineering services related to the transition, and those services included ensuring the safety of the water from the Flint River, it did not recommend treatment of the water to prevent corrosion of the pipes. (*Id.* at 53.)

Within weeks of the transition to Flint River water, residents of Flint began complaining about the smell, taste, and color of the drinking water. (*Id.* at 54.) Shekter-Smith received many of those complaints, including one forwarded from an Environmental Protection Agency (EPA) employee regarding rashes linked to the Flint River water. (*Id.*) Complaints and symptoms related to consumption of the water continued, and, on August 14, 2014, Flint water tested above legal limits for coliform and E. coli bacteria. (*Id.* at 55.) Flint issued boil water advisories on August 16, 2014, and September 5, 2014. (*Id.*)

In response to these issues, Flint treated the water with additional chlorine. (*Id.*) However, because Flint's old water lines were corroded, chlorine attacked the bare metal, rather than the bacteria, leading to further corrosion and the release of TTHM into the water supply. (*Id.*) A PowerPoint presentation circulated among MDEQ officials in March and April 2015, including Busch, Prysby, and Rosenthal, showed that MDEQ officials knew as early as May 2014 that Flint water contained elevated levels of TTHM. (*Id.*)

In the summer of 2014, MDHHS reported an outbreak of [Legionnaires' disease](#) in Flint. (*Id.* at 56.) [Legionnaires' disease](#) infects humans when water droplets containing legionella bacteria are inhaled or legionella-contaminated water is consumed. (*Id.*) Legionella can enter a water supply when the biofilm attached to a water pipe is stripped away, as happened when the Flint River water entered the city's pipes, and more chlorine was added to treat the water. (*Id.*)

On October 3, 2014, Flint's Public Information Officer informed Earley and Ambrose about the spike in Legionnaires' cases via e-mail. (*Id.*) Earley responded by denying any connection between Flint water and the outbreak, and stated that the city's message should be that the outbreak was an internal issue at McLaren Hospital.

(*Id.*) MDHHS personnel did not agree with Earley's message. (*Id.* at 57.)

In September 2014, elevated blood lead levels were beginning to be noted in children under the age of sixteen who were living in Flint. (*Id.*) By October 1, 2014, it was known that the iron pipes making up most of Flint's water distribution system [were] one of the causes of the contamination of the water. (*Id.*)

On October 13, 2014, General Motors stopped the use of Flint River water at its engine plant due to the corrosive **\*830** nature of the water. (*Id.*) Governor Snyder's executive staff was immediately aware of the problem, and on October 14, 2014, Governor Snyder's Deputy Legal Counsel and Senior Policy Advisor Valerie Brader wrote an e-mail in which she suggested asking Earley to "consider coming back to the [DWSD] in full or in part as an interim solution to both the quality, and now the financial, problems that the current solution is causing." (*Id.*) Brader intentionally did not distribute this message to MDEQ officials so that it would be exempted from the Freedom of Information Act [ (FOIA) ], but she did coordinate discussions with Earley and officials at MDEQ. (*Id.* at 58.) In response to this e-mail, Earley rejected the idea of returning to the DWSD on October 14, 2014. (*Id.*) On October 15, 2014, Governor Snyder's Legal Counsel, Michael Gadola, stated that use of the Flint River as a water source was "downright scary," and that Flint "should try to get back on the Detroit system as a stopgap ASAP before this thing gets too far out of control." (*Id.* at 59.)

By November 2014, LAN knew of the need to analyze the cause of the high TTHM levels in Flint water. (*Id.* at 60.) On November 26, 2014, LAN issued a twenty-page Operational Evaluation Report regarding the transition, which addressed compliance with EPA and MDEQ regulations, but did not address the potential for lead contamination resulting from the corrosive water flowing through the lead pipes in Flint's water system. (*Id.*)

By December 31, 2014, lead monitoring showed water testing results exceeding the federal Lead and [Copper Rule's](#) action level for lead, which is 15 parts per billion (ppb). (*Id.* at 59.) On January 9, 2015, University of Michigan – Flint water tests revealed elevated lead levels in two locations on campus, which led the University to turn off certain water fountains. (*Id.*) On January 9, 2015, Earley again refused to return Flint to the DWSD. (*Id.*)

On January 13, 2015 Earley resigned as Emergency Manager for Flint, and was replaced by Gerald Ambrose. (*Id.* at 78.) On January 29, 2015, DWSD offered Ambrose an opportunity to reconnect to the DWSD water supply, with the re-connection fee waived. Ambrose rejected the offer. (*Id.* at 79.)

In January 2015, LeeAnn Walters, a Flint homeowner, contacted the EPA regarding complaints that Flint River water was making her and her family physically ill. (*Id.*) On January 21, 2015, the State of Michigan ordered water coolers to be installed in state buildings operating in Flint, but did not share this information with the public. (*Id.* at 78.) On January 27, 2015, Flint received notice from the Genesee County Health Department that it believed the spike in [Legionnaires' disease](#) cases was linked to the switch to Flint River water. (*Id.*) On January 28, 2015, MDHHS Director Nick Lyon received materials from an MDHHS epidemiologist showing the 2014 outbreak of [Legionnaires' disease](#) in Genesee County. (*Id.*)

On February 26, 2015, Jennifer Crooks, an EPA employee, e-mailed MDEQ and EPA employees regarding Walters' complaints of black sediment in her water. (*Id.* at 80.) The e-mail noted very high testing results for iron contamination, and noted that Glasgow suggested testing for lead and [copper](#), which resulted in test findings of 104 ppb, well over the federal action levels of 15 ppb. (*Id.*) The e-mail also noted that the high presence of lead was a sign that there were other contaminants in the water, as well. (*Id.*) That day, Crooks **\*831** also sent an e-mail to MDEQ and EPA representatives, opining that the black sediment from Walters' water was actually lead, and questioning whether the issue was more widespread. (*Id.* at 80–81.) Crooks also wondered if Flint was using optimal corrosion control. (*Id.* at 81.) On February 27, 2015, Busch told [Miguel] Del Toral [at the EPA] that Flint was using corrosion control, which was false. (*Id.*)

At some point, Flint issued a request for proposals for engineering companies to serve as a water quality consultant to the city. (*Id.* at 60–61.) Flint sought a consultant who could review and evaluate the City's water treatment process and its procedures to maintain and improve water quality, to recommend ways to maintain compliance with state and federal agencies, and to assist Flint in implementing those recommendations. (*Id.* at 61.) In February 2015, Veolia was hired to be Flint's water quality consultant. (*Id.*) The contract retaining Veolia stated

that Flint would rely on the “professional reputation, experience, certification, and ability” of Veolia. (*Id.* at 62.)

On February 10, 2015, Veolia and Flint issued a joint press release that touted Veolia's expertise in “handling challenging river water sources,” and notifying the public of Veolia's role in evaluating Flint's water treatment processes. (*Id.*) On February 10 and 12, 2015, executives at Veolia made statements professing the expertise of the companies and promising to address the issues with Flint's water. (*Id.* at 62–63.)

On February 18, 2015, Veolia made an interim report to Flint's City Council. (*Id.* at 63.) The report indicated that Flint's water was “in compliance with drinking water standards,” but that the discoloration of the water “raises questions.” (*Id.*) The report also stated that medical issues arising from consumption of the water were explained by the fact that “[s]ome people may be sensitive to any water.” (*Id.* at 64.) LAN also released a report addressing TTHM concerns, but that report did not analyze the causes of the high TTHM levels. (*Id.* at 66.)

On March 12, 2015, Veolia issued a final Water Quality Report. (*Id.* at 64.) That report was based on a 160-hour assessment of the FWTP, Flint's distribution system, and related administrative and financial aspects of Flint's water system. (*Id.*) The report found that Flint water was in compliance with state and federal water quality regulations, despite public concerns about the color and quality of the water. (*Id.*) The report also recommended that Flint add polyphosphates to the water supply to minimize the discoloration from iron in the pipes, but that discoloration might happen because of regular breaks and maintenance on the pipes. (*Id.* at 64–65.) But polyphosphates only addressed issues with the iron pipes, and were not a solution to the issues with the lead pipes. (*Id.* at 65.)

Meanwhile, Cook told the EPA that Flint was using corrosion control with Flint River water, and forwarded information he knew to be false to the EPA to back up the contention. (*Id.* at 81.) On January 27, 2015, James Henry, Environmental Health Supervisor at the Genesee County Health Department, filed a [FOIA] request with Flint to obtain information about Flint's water supply. (*Id.*) Johnson stated on February 5, 2015, that he had not received the request and would fulfill it as soon as possible. However, he had not done so by March 2015. (*Id.* at 82.) On March 10, 2015, Henry expressed public concern that Flint and the

State of Michigan \*832 were stonewalling his requests for information. (*Id.*)

On March 12, 2015, Shekter-Smith e-mailed Wurfel and MDEQ employees Jim Sygo and Sarah Howes to discuss a FOIA request related to legionella and stated that although the switch to the Flint River may have created conditions that supported legionella growth, there was no evidence that legionella was coming directly from the FWTP or Flint's water distribution system at the time. (*Id.* at 83.) On March 13, 2015, Busch made statements that denied any provable connection between the switch to Flint River water and the presence of legionella bacteria in that water supply, and Shekter-Smith approved them. (*Id.*) During March, members of Governor Snyder's office were aware of mobilization by Flint area pastors focused on the odor and appearance of Flint water, and of a request by those pastors for water filters. (*Id.* at 84.)

On March 25, 2015, the Flint City Council voted to reconnect to the DWSD, but Ambrose rejected that vote. (*Id.*) On April 24, 2015, almost exactly one year after the switch to Flint River water, Cook e-mailed ... Del Toral ... and informed him, in contradiction of Cook's earlier representations, that Flint was not practicing corrosion control at the FWTP. (*Id.*) On June 24, 2015, Del Toral issued a report noting high lead levels in Flint and the State of Michigan's complicity in both the high lead levels and the failure to inform users of Flint's water supply. (*Id.* at 84–85.) The report was shared with Shekter-Smith, Cook, Busch, and Prysby, but neither they nor any other public official named as a defendant in this lawsuit took measures to effectively address any danger identified in the report. (*Id.* at 85.)

Between June 30, 2015, and July 2, 2015, Walling and EPA Region 5 Director Dr. Susan Hedman discussed the report, and Hedman stated that it was a preliminary draft from which it would be premature to draw any conclusions. (*Id.*)

On July 9, 2015, Glasgow sent an e-mail to Rosenthal describing the clear and undeniable issues that Flint's lead-and bacteria-tainted water was causing. (*Id.* at 86.) On July 10, 2015, Wurfel appeared on public radio and made knowingly false statements asserting that Flint River water was safe and causing no “broad problem[s]” with elevated lead levels in the water. (*Id.* at 85–86.) On July 22, 2015, Governor Snyder's Chief of Staff wrote to Lyon and stated that the concerns of Flint water users were being “blown off” by the defendants. (*Id.* at 87.) On July 24, 2015, Wurfel

again falsely stated that there were no worries about lead or copper contamination in Flint's water supply. (*Id.*)

In that July 24, 2015 statement, Wurfel referenced sampling of the water supply by MDEQ, but that sampling was skewed, and did not resample most lower-lead homes between 2014 and 2015, or any high-lead homes between 2014 and 2015. (*Id.*) The sampling actually covered up high-lead samples. (*Id.* at 88.) Glasgow ultimately pleaded no contest to willful neglect of duty after being accused of distorting the water test results by asking residents of Flint to run or flush their water before testing, and of failing to obtain water samples from certain houses. (*Id.*)

During this time period, Glasgow also stated that Busch and Prysby directed him to alter water quality reports to remove the highest lead levels. (*Id.*) Rosenthal also allegedly manipulated test results, including a July 28, 2015 report \*833 from which Rosenthal excluded high lead-level tests. (*Id.* at 88–89.)

In August 2015, Professor Marc Edwards of Virginia Tech, who had been testing Flint River water, announced that he believed there was serious lead contamination of the Flint water system, which constituted a major public health emergency. (*Id.* at 89.) In response, Wurfel attempted to discredit Edwards' statements by calling the testing “quick” and implying that it was irresponsible. (*Id.*)

By late 2014 or early 2015, Lyon also knew about the increase in children with elevated blood lead levels and Legionnaires' disease cases, but did not report these findings to the public or other government officials, or take any steps to otherwise intervene. (*Id.* at 89–90.) In the summer of 2015, Dr. Mona Hanna-Attisha used data from Hurley Hospital in Flint to note a rise in the number of Flint children with elevated blood lead levels in the second and third quarters of 2014 to publish a study, the purpose of which was to alert Flint water users about the health risks associated with the water. (*Id.* at 90.) The governmental defendants immediately accused Dr. Hanna-Attisha of providing false information to the public. (*Id.*) On September 28, 2015, Lyon directed his staff to provide an analysis rebutting Dr. Hanna-Attisha's findings and portraying the rise in elevated blood lead levels as normal results corresponding to seasonal fluctuations. (*Id.* at 90–91.) Throughout September 2015, Wurfel and the MDEQ continued to issue false statements claiming the water in Flint was safe, and that the people sounding alarms about

Flint's water quality were mistaken or "rogue." (*Id.* at 91–92.)

On October 2, 2015, the State of Michigan announced that it would create a Flint Water Advisory Task Force and provide water filters to Flint water users. (*Id.* at 92.) On October 8, 2015, Governor Snyder ordered Flint to reconnect to the DWSD, and that reconnection took place on October 16, 2015. (*Id.*) On October 18, 2015, Wyant e-mailed Governor Snyder and admitted that MDEQ made a mistake in not implementing optimized corrosion control from the beginning. (*Id.* at 93.) On October 19, 2015, the City of Flint Technical Advisory committee listed LAN as the "owner" of the "corrosion control" issue. (*Id.*)

Current Flint Mayor Karen Weaver declared a state of emergency in Flint on December 14, 2015. (*Id.* at 94.) On January 4, 2016, the Genesee County Commissioners likewise declared a state of emergency; Governor Snyder did so on January 5, 2016, and activated the Michigan National Guard to assist Flint on January 13, 2016. (*Id.*)

[Carthan](#), 329 F.Supp.3d at 382–89.

### iii. Additional Allegations in the Proposed Fourth Amended Complaint

The proposed fourth amended complaint adds new factual allegations as follows. During the middle of the twentieth century, Flint's water supply was taken from the Flint River and was of poor quality due to the presence of fecal coliform bacteria and contaminants. (Dkt. 620-3 at 38–39.) Because of these environmental concerns, Flint began evaluating different water sources in the 1960s. (*Id.*) Flint eventually mothballed the FWTP around 1965, and entered into an agreement to receive its water from DWSD. (*Id.*) This agreement gave Flint the exclusive right to sell DWSD water to the remainder of Genesee County. (*Id.*) And the Genesee County Drain Commission (GCDC) contracted \*834 with Flint to buy DWSD water in order to resell it to local customers. (*Id.*)

In 1973, the GCDC updated its contract with Flint. (*Id.* at 39.) The GCDC had to accept water from Flint as delivered, so long as it met "all requirements of the various State Regulatory Agencies." (*Id.* at 39–40.) In return, Flint was required to sell water "generally sufficient to supply the County's system use." (*Id.* at 40.) In 2003, the contract was again updated. (*Id.*) It similarly required the "City ... to sell water to the [GCDC] in such quantities as will meet

the demands of the County Agency's customers" and "the [GCDC] agree[d] to purchase water exclusively from the City[.]" (*Id.*) This was the situation until 2014, when Flint transitioned back to the Flint River as its source of water. (*Id.* at 52–53.)

Flint was not the only municipality looking to switch its water supply in the early 2000s. Various communities in the same region had formed the KWA in 2009 to explore this possibility. (*Id.* at 42.) Specifically, the KWA was aiming to construct a new water pipeline connected to Lake Huron. (*Id.*) Walling was elected as the KWA's chair, and Wright was elected its Chief Executive Officer. (*Id.*) Wright later stated that he was motivated to establish the KWA at least in part because DWSD was "the poster child for Detroit corruption." (*Id.* at 43.)

To be viable, the KWA would have to construct a water intake at Lake Huron and sixty-three miles of pipeline. (*Id.*) The system would have to supply sixty million gallons of water each day. (*Id.*) It was estimated that Genesee County would require forty-two million gallons a day and Flint eighteen. (*Id.*) The projected capital cost ran to approximately \$ 300 million, of which Flint would shoulder eighty-five million and serve 34.2% of the debt. (*Id.*) Unlike the treated water supplied by DWSD, the KWA water would be raw and would require considerable treatment before use. (*Id.*)

By 2013, Wright had secured long-term commitments from most of its members to purchase their future water from the KWA. (*Id.* at 46.) The commitments were necessary to fund the bonds required to finance the project. (*Id.*) However, at that time, Flint had not committed to the KWA. (*Id.*) And Wright knew that it was doubtful the financing would be successful without Flint's backing. (*Id.* at 46–47.) So beginning in March and April of that year, Wright aggressively argued the case for Flint joining the KWA. (*Id.*) All officials involved in the decision knew that the FWTP would have to be upgraded to process the future supply of raw water. (*Id.* at 47.)

Yet there was a problem with Flint's participation in the KWA: how would it pay for its share of the costs? Under state law, Flint could not issue new bonds because it was in financial receivership. (*Id.* at 55.) An Administrative Consent Order (ACO) would permit Flint to circumvent this problem. (*Id.* at 129–32.) But the only way Flint could obtain an ACO was from a state agency as a result of an emergency. (*Id.* at 132.) Accordingly, Flint began pursuing an ACO from the MDEQ.

(*Id.* at 133.) And although initially hesitant, the MDEQ began to help with the ACO even though there was no prerequisite emergency. (*Id.* at 133–34.) The MDEQ executed the ACO on March 20, 2014. (*Id.* at 136.) Pursuant to its terms, Flint was bound to adopt the interim plan to use the Flint River as its water source. (*Id.*) The bond issue that followed allowed the KWA project to move forward. (*Id.*)

When developing the interim plan, a group of people including defendants Wright, Dillon, and Walling recognized that the FWTP could not process enough \*835 water for all of Genesee County. (*Id.* at 53.) So together with other officials, these defendants eventually planned for Flint to receive Flint River water while Genesee County would continue to receive DWSD water. (*Id.*) This was despite evidence that the use of the Flint River would “[p]ose an increased microbial risk to public health,” “an increased risk of disinfection by-product (carcinogen) exposure,” and “[r]equire significant enhancements to treatment [sic] at the [FWTP.]” (*Id.* at 131.)

As a necessary part of the interim plan, MDEQ officials issued an operating permit for the FWTP in April 2014. (*Id.* at 52.) However, they did so without following the required procedures. (*Id.* at 126.) Federal law requires states to “review and approve the addition of a new source or long-term change in water treatment[.]” (*Id.* at 136.) In turn, Michigan law regulates the MDEQ’s authority to issue permits that impact the State’s water systems. Before issuing a permit for certain water sources, the MDEQ is required to provide a public comment period of “not less than 45 days.” (*Id.* at 136–37.) This regulation applied to the FWTP permitting process. (*Id.* at 137.) Publicly, the MDEQ stated that “the city would just continue to buy water from [DWSD]” if no permit was issued in time. (*Id.* at 138.) And Flint officials assured people that there would be a series of open forums to permit public questions. (*Id.*) But when Flint eventually submitted its permit application on March 31, 2013, it was approved in a matter of days, without an opportunity for public comment. (*Id.* at 138–39.)

Flint transitioned to the Flint River on April 25, 2014. (*Id.* at 62.) And in the months that followed, members of Governor Snyder’s senior staff began to discuss the possibility of lead contamination. (*Id.* at 104–05.) In March 2015, a Flint resident wrote an open letter to Walling stating that “the water is dangerous to our health!” (*Id.* at 158.) Ambrose eventually received the letter, forwarding it to Flint’s public relations firm simply stating, “[w]elcome to Monday.” (*Id.*)

Civic groups tried to get the government defendants to take the issue seriously. (*Id.* at 158–59.) But officials dismissed their concerns as unwarranted. (*Id.*)

Even as the EPA continued to uncover evidence of lead contamination and some senior members of Governor Snyder’s administration voiced the view that the problem was not being taken seriously (*id.* at 159–60), many officials continued to deny that anything was wrong. (*Id.* at 160–62.) Instead, they put it down to “old time negative racial experiences.” (*Id.* at 161.) Several officials recommended that more money be spent on public relations to combat the issue, rather than looking to resolve the underlying problem. (*Id.* at 159.)

On April 28, 2015, Governor Snyder received an e-mail from his chief of staff advising him that the water issue in Flint continued to be a “danger flag” for the administration. (*Id.* at 92.) Later that summer, the Governor’s Director of Urban Initiatives discussed with him the growing concerns among Flint residents that they were being exposed to contaminated water. (*Id.* at 94.) And on September 25, the Governor’s chief of staff again e-mailed the Governor to discuss the issue of lead exposure and the potential political implications. (*Id.* at 103.) In the same communication, the chief of staff opined that the residents of Flint were having their concerns about water quality inappropriately dismissed. (*Id.* at 104.) The Governor received this e-mail almost a week before he publicly acknowledged in October 2015 that Flint’s water was contaminated with lead. (*Id.* 103.) In the meantime, MDEQ officials continued to tell the public that “the drinking water distributed to city customers \*836 currently meets all drinking water standards[.]” (*Id.* at 162.)

Following his public acknowledgment of the crisis in October 2015, the Governor was told in December that in addition to elevated lead levels, Flint residents were also at risk of legionella exposure. (*Id.* at 101.) Despite all this knowledge, Governor Snyder did not disclose this risk when he declared a state of emergency on January 5, 2016. (*Id.* at 102.) It was not until January 13 that he publicly admitted that Flint’s water contained legionella bacteria. (*Id.*) He did this while activating the Michigan National Guard to assist the people of Flint. (*Id.*) On January 14, Governor Snyder asked the federal government to issue an emergency declaration. (*Id.*) The federal government did so two days later. (*Id.*)

In the aftermath of the crisis, facts about how the MDEQ dealt with the disaster came to light. For example, the MDEQ failed

to comply with various rules and regulations. (*Id.* at 126.) State law requires the MDEQ to notify the public if a source of water is found to be out of compliance. (*Id.* at 142.) In August 2014, MDEQ officials discussed whether a Flint boil water advisory was caused by a sampling error in a test or a high fecal coliform result. (*Id.*) It was far from certain that sampling error was the culprit. (*Id.*) And although the MDEQ suspected that the water was contaminated, no one made an effort to investigate the issue or notify the public. (*Id.* at 143–44.)

Additionally, the MDEQ lacked a nondiscrimination policy required by federal law. (*Id.* at 126.) In 1992, the EPA found that the MDEQ had discriminated against Flint's majority African American population in the public participation processes for a power station permit. (*Id.* at 145–46.) In conducting its investigation, the EPA concluded that the MDEQ had insufficient formalized safeguards to protect against operational discrimination as required by federal regulations. (*Id.* at 146.) In 2014, the EPA informed the MDEQ that it was still not in compliance and needed to have in place a non-discrimination policy. (*Id.* at 147.) And even when the MDEQ provided the EPA with a written policy, the EPA determined that it was legally insufficient. (*Id.*) The same defective policy remains in place. (*Id.*)

Throughout the crisis and its aftermath, the concerns and fears of the people of Flint were not taken seriously. In the opinion of some government officials, residents “making noise about civil unrest, violence, [Michigan State Police] shootings and an [emergency manager], [were] the naysayers[.]” (*Id.* at 156.) These officials believed that too many of them had “their handout [sic] and their voices raised,” and caving to their demands was part of the reason that Flint had been “placed in receivership twice in the past decade.” (*Id.* at 156–57.) In the view of these officials, the problem an “entitlement mentality.” (*Id.* at 157.) Ultimately, the Task Force charged with investigating the causes of the crisis summed it up: “Flint residents, who are majority Black or African American and among the most impoverished of any metropolitan area in the United States, did not enjoy the same degree of protection from environmental and health hazards as that provided to other communities.”<sup>5</sup> (*Id.* at 165.)

#### iv. Prior Flint Water Cases

Litigation from the Flint Water Cases has already resulted in several opinions from the Sixth Circuit. The Court must

\*837 follow these as they are binding precedent, including *Guertin v. Michigan*, 912 F.3d 907 (6th Cir. 2019); *Boler v. Earley*, 865 F.3d 391 (6th Cir. 2017); and *Mays v. City of Flint*, 871 F.3d 437 (6th Cir. 2017).<sup>6</sup> Other decisions have been issued by this Court, which will be adhered to where appropriate. This includes *Guertin v. Michigan*, No. 16-cv-12412, 2017 WL 2418007, 2017 U.S. Dist. LEXIS 85544 (E.D. Mich. June 5, 2017), and the Court's vacated August 1, 2018 opinion in the present case. 329 F.Supp.3d 369.

#### b. Standard of Review

[1] [2] Plaintiffs seek leave to amend the complaint under Federal Rule of Civil Procedure 15(a)(2). Rule 15(a)(2) states that “a party may amend its pleading only with ... the court's leave.”<sup>7</sup> However, “court[s] should freely give leave when justice so requires.” Fed. R. Civ. P. 15(a)(2); see also *Leisure Caviar, LLC v. U.S. Fish & Wildlife Serv.*, 616 F.3d 612, 615 (6th Cir. 2010) (noting that Rule 15(a) requests are normally liberally granted). And when evaluating the interests of justice, courts consider various factors. These include “‘[u]ndue delay in filing, lack of notice to the opposing party, bad faith by the moving party, repeated failure to cure deficiencies by previous amendments, [and] undue prejudice to the opposing party[.]’” *Wade v. Knoxville Utils. Bd.*, 259 F.3d 452, 458–59 (6th Cir. 2001) (quoting *Head v. Jellico Hous. Auth.*, 870 F.2d 1117, 1123 (6th Cir. 1989)). Mere delay on its own is insufficient to warrant denial. *Oleson v. United States*, 27 F. App'x 566, 569 (6th Cir. 2001). Instead, courts examine the competing interests of the litigants and the likelihood of prejudice to the non-moving party. See *Morse*, 290 F.3d at 799.

[3] [4] Yet regardless of the equities, leave must be denied if an amendment would be futile. *Parchman v. SLM Corp.*, 896 F.3d 728, 736, 738 (6th Cir. 2018) (citing *Foman v. Davis*, 371 U.S. 178, 182, 83 S.Ct. 227, 9 L.Ed.2d 222 (1962)). And a “proposed amendment is futile if [it] could not withstand a Rule 12(b)(6) motion[.]” *Beydoun v. Sessions*, 871 F.3d 459, 469 (6th Cir. 2017). Under Rule 12(b)(6), the Court must “construe the complaint in the light most favorable to the plaintiff and accept all allegations as true.” *Keys v. Humana, Inc.*, 684 F.3d 605, 608 (6th Cir. 2012). “To survive a motion to dismiss, a complaint must contain sufficient factual matter, accepted as true, to state a claim to relief that is plausible.” *Ashcroft v. Iqbal*, 556 U.S. 662, 678, 129 S.Ct. 1937, 173 L.Ed.2d 868 (2009). A plausible claim need not contain “detailed factual allegations,” but it must

contain more than “labels and conclusions” or “a formulaic recitation of the elements of a cause of action.” *Bell Atl. Corp. v. Twombly*, 550 U.S. 544, 555, 127 S.Ct. 1955, 167 L.Ed.2d 929 (2007). Ultimately, a claim is only facially plausible “when the plaintiff pleads factual content that allows the court to draw the reasonable inference that the defendant is liable for the misconduct alleged.” *Iqbal*, 556 U.S. at 678, 129 S.Ct. 1937.

### c. Threshold Issues

#### i. Jurisdiction

Several parties question the Court's jurisdiction to rule on plaintiffs' motion. (Dkt. 635 at 4–5; Dkt. 650 at 1–2; Dkt. 653 \*838 at 12–13; Dkt. 655 at 12–14; Dkt. 657 at 11–12.) They argue that the Court was divested of jurisdiction by appeals filed in response to its August 1, 2018 opinion and order. (*Id.*) However, as explained in its November 9, 2018 order, the Court retained jurisdiction. (Dkt. 670.) And the Sixth Circuit recently affirmed this position, dismissing defendants' appeals in the process. *Carthan v. Snyder*, No. 18-1967 (6th Cir. Feb. 19, 2019). As such, the Court retains the authority to rule on the present motion.

#### ii. Class Definitions

Plaintiffs seek leave to amend the class definitions. The third amended complaint broadly defined the class as including all individuals and entities who were exposed to Flint's contaminated water and who experienced injuries or damages to their persons or property. (Dkt. 349 at 112–13.) The fourth amended complaint keeps this general definition but adds a subclass of African Americans. (Dkt. 620-3 at 167–68.) Plaintiffs also further divide the proposed classes into a series of even smaller subclasses based on property damage, personal injury, injunctive relief, and a set of common issues relating to liability and causation. (*Id.* at 167–72.)

Class certification should occur “at an early practicable time.” Fed. R. Civ. P. 23(c)(1)(A). The Court is currently preparing a comprehensive case management order that will set forth the timeframe for consideration of class definitions and certification. Therefore, plaintiffs may amend the complaint to include the amended class definitions with the understanding that this issue will be revisited.

### d. Main Analysis

Plaintiffs seek leave to amend the following six counts:

Count	Claim	Defendants
I	42 U.S.C. § 1983 - Bodily Integrity	All government defendants
II-III	42 U.S.C. § 1983 - Equal Protection	Snyder, Dillon, Wright, Ambrose, Kurtz, Earley, Wyant, Shekter-Smith, Prysby, and Busch
IV	42 U.S.C. § 1985(3) - Conspiracy	Snyder, Dillon, Wright, Ambrose, Kurtz, and Earley
V	Elliott-Larsen Civil Rights Act	Snyder, Dillon, Wright, Ambrose, Kurtz, Earley, The City of Flint, Wyant, Shekter-Smith, Prysby, and Busch
XIV	Gross Negligence	Snyder, Dillon, Lyon, Shekter-Smith, Rosenthal, Busch, Cook, Prysby, Wurfel, Wright, Kurtz, Earley, Ambrose, Croft, Johnson, and Glasgow.

#### i. Competing Interests and Likelihood of Prejudice

[5] Some of the defendants argue that leave to amend the complaint should be denied because plaintiffs' request is unduly delayed. (Dkt. 651 at 19–20; Dkt. 653 at 33–34). The Court disagrees.

It is true that the present case has been pending for several years. This is now plaintiffs' fourth amended complaint, and if \*839 this were a routine case, their attempt to amend the pleadings again might be unusual. But this litigation is far from routine. The harm alleged and the number of parties involved are extraordinary. What started out as a series of individual suits has become a large consolidated action. And the complex nature of the claims coupled with less than straightforward procedure must be considered. This weighs in plaintiffs' favor.

Conversely, defendants do not explain how they will be prejudiced. Having resisted the start of discovery, they cannot claim that they will be subject to duplicative discovery. See *Morse*, 290 F.3d at 800–01. Plaintiffs have not changed their allegations so much that defendants will need to completely overhaul their strategy. See *Prather v. Dayton Power & Light Co.*, 918 F.2d 1255, 1259 (6th Cir. 1990). And the fourth amended complaint does not contain new claims so far outside the scope of the third amended complaint such that granting leave to amend may later lead to confusion. See *Lover v. D.C.*, 248 F.R.D. 319, 323 (D.D.C. 2008). As a result, leave to amend will not be denied on the basis of prejudice. Instead, the Court will examine each count for futility.

## ii. Futility of Amendments

### 1. Bodily Integrity

Plaintiffs first seek leave to amend their bodily integrity claim brought under § 1983 against defendant Governor Snyder. (Dkt. 620-1 at 13.) In their view, the newly pleaded allegations establish that the Governor was aware of the significant risks posed by the Flint River water as early as April 2015, but he did nothing to inform Flint's residents until the crisis could no longer be denied many months later. (*Id.* at 14–15.) Additionally, Governor Snyder not only denied the crisis in the intervening period, but later played down the risks for months after having publicly acknowledged the disaster. (*Id.*) Because plaintiffs' state a plausible bodily integrity claim against Governor Snyder, granting leave to include it would not be futile.

#### a. Constitutional Violation

[6] [7] [8] [9] The right to bodily integrity is a fundamental interest protected by the Due Process Clause of the Fourteenth Amendment. *Guertin*, 912 F.3d at 918–19; *Guertin*, 2017 WL 2418007, at \*21, 2017 U.S. Dist. LEXIS 85544, at \*63 (citing *Union Pac. Ry. Co. v. Botsford*, 141 U.S. 250, 251, 11 S.Ct. 1000, 35 L.Ed. 734 (1891)). And although violations of the right to bodily integrity usually arise in the context of physical punishment, the scope of the right is not limited to that context. *Kallstrom v. City of Columbus*, 136 F.3d 1055, 1062–63 (6th Cir. 1998). For instance, the “forcible injection of medication into a nonconsenting person's body represents a substantial interference with that person's liberty[.]” *Guertin*, 912 F.3d at 919 (citing *Washington v. Harper*, 494 U.S. 210, 229, 110 S.Ct. 1028, 108 L.Ed.2d 178 (1990)). And “compulsory treatment with anti-psychotic drugs may [also] invade a patient's interest in bodily integrity[.]” *Guertin*, 2017 WL 2418007, at \*22, 2017 U.S. Dist. LEXIS 85544, at \*66 (citing *Lojuk v. Quandt*, 706 F.2d 1456, 1465–66 (7th Cir. 1983)). The key is whether the intrusion is consensual. See *Guertin*, 912 F.3d at 920. There is no difference between the forced invasion of a person's body and misleading that person into consuming a substance involuntarily. *Guertin*, 2017 WL 2418007, at \*24, 2017 U.S. Dist. LEXIS 85544, at \*71 (citing *Heinrich v. Sweet*, 62 F.Supp.2d 282, 313–14 (D.

Mass. 1999)). As such, officials can violate an individual's bodily integrity by introducing life-threatening substances into that person's body without their consent. *Guertin*, 2017 WL 2418007, at \*22, 2017 U.S. Dist. LEXIS 85544, at \*65 (citing \*840 *Washington*, 494 U.S. at 229, 110 S.Ct. 1028).

[10] [11] However, to state a claim, plaintiffs must do more than point to the violation of a protected interest; they must also demonstrate that it was infringed arbitrarily. *Guertin*, 912 F.3d at 922. *But see Range v. Douglas*, 763 F.3d 573, 589 (6th Cir. 2014) (observing that in some contexts government action may violate substantive due process without a liberty interest at stake). And with executive action, as here, only the most egregious conduct can be classified as unconstitutionally arbitrary. *Cty. of Sacramento v. Lewis*, 523 U.S. 833, 846, 118 S.Ct. 1708, 140 L.Ed.2d 1043 (1998). In legal terms, the conduct must “shock[ ] the conscience.” *Guertin*, 2017 WL 2418007, at \*21, 2017 U.S. Dist. LEXIS 85544, at \*63 (quoting *Lewis*, 523 U.S. at 846, 118 S.Ct. 1708).

[12] [13] [14] Whether government action shocks the conscience depends on the situation. *Ewolski v. City of Brunswick*, 287 F.3d 492, 510 (6th Cir. 2002). Where unforeseen circumstances demand the immediate judgment of an executive official, liability turns on whether decisions were made “maliciously and sadistically for the very purpose of causing harm.” *Lewis*, 523 U.S. at 852–53, 118 S.Ct. 1708 (quoting *Whitley v. Albers*, 475 U.S. 312, 320–21, 106 S.Ct. 1078, 89 L.Ed.2d 251 (1986)). But where an executive official has time for deliberation before acting, conduct taken with “deliberate indifference” to the rights of others “shocks the conscience.” See *Claybrook v. Birchwell*, 199 F.3d 350, 359 (6th Cir. 2000). This case involves the latter of these two situations. And as a result, plaintiffs must demonstrate that (1) officials knew of facts from which they could infer a “substantial risk of serious harm,” (2) that they did infer it, and (3) that they nonetheless acted with indifference, *Range*, 763 F.3d at 591 (citing *Ewolski*, 287 F.3d at 513), demonstrating a callous disregard towards the rights of those affected, *Guertin*, 912 F.3d at 924 (quoting *Schroder v. City of Fort Thomas*, 412 F.3d 724, 730 (6th Cir. 2005)).

[15] As a preliminary matter, plaintiffs point to a bodily integrity violation. This is not a case about the right to a contaminant-free environment or clean water. *But see Guertin*, 912 F.3d at 955–57 (McKeague, J., dissenting). Rather, this case implicates the consumption of life-threatening substances. Indeed, neither side disagrees that

lead and legionella are life threatening, nor that plaintiffs ingested these contaminants and others through the water supply. This intrusion was also involuntary. “[I]t was involuntary because defendants hid from plaintiffs that Flint’s water contained dangerous levels of lead,” *Guertin*, 2017 WL 2418007, at \*24, 2017 U.S. Dist. LEXIS 85544, at \*71, and, “because under state and municipal law, plaintiffs were not permitted to receive water in any other way[.]” *id.* (citing Flint Code of Ord. §§ 46-25, 46-26, 46-50(b)). Plaintiffs’ claim therefore implicates the right to bodily integrity.

[16] Plaintiffs also plead facts which, when taken as true, show that Governor Snyder was deliberately indifferent. First, plaintiffs plausibly allege that Governor Snyder knew of facts from which he could infer that plaintiffs faced a substantial risk of serious harm. As early as March 2014, members of the Governor’s administration were warning that transitioning to the Flint River could lead to a potential disaster. Initial warning signs included an outbreak of *Legionnaires’ disease* in the Flint area. And by October 2014, senior staff, including the Governor’s Chief of Staff, were discussing the need to return to DWSD water because of a growing awareness that the treated Flint River water did \*841 not meet established quality standards. In July 2015, this clamor continued to build when the Governor’s Chief of Staff wrote that concerns over lead contamination were being inappropriately dismissed. There was also a public outcry. Concerned religious leaders informed the administration of problems with the Flint River. News articles discussed lead in Flint’s drinking water. And General Motors stopped using Flint water because it was corroding machinery. Considering the seriousness of the potential problem, the widespread reports, and the seniority of the government staff involved, it is reasonable to infer from plaintiffs’ allegations that Governor Snyder was aware of this information. As a result, the Governor possessed sufficient facts from which he could have deduced that plaintiffs faced a substantial risk of serious harm from the Flint River.

Second, plaintiffs successfully claim that Governor Snyder did in fact infer that plaintiffs faced such a risk of harm. In January 2015, the Governor met with other government officials to discuss the ongoing threat to public health posed by legionella bacteria in the Flint River water. A couple of months later, the Governor and his staff discussed whether to distribute water filters to Flint residents as a form of mitigation against possible contamination. At the same time, the Governor’s Chief of Staff informed the Governor that the water issue in Flint continued to be “a danger flag” and was something that needed addressing sooner rather than later.

(*Id.* at 92.) And in the summer, a senior member of the administration spoke with Governor Snyder about the fear that Flint’s residents were being exposed to toxic levels of lead through the Flint River water. So when plaintiffs state that by February 2015, the Governor was fully aware of a public health threat posed by the water supply in Flint, and that by July 2015, at the very latest, the Governor knew that the water supply was contaminated, these conclusions are supported by well-pleaded factual allegations. It is reasonable to infer that Governor Snyder knew that the residents of Flint faced a substantial risk of serious harm emanating from the water.

Third, plaintiffs plausibly state that the Governor acted indifferently to the risk of harm they faced, demonstrating a callous disregard for their right to bodily integrity. This indifference manifested itself in two ways. Initially, the Governor was indifferent because instead of mitigating the risk of harm caused by the contaminated water, he covered it up. In private, he worried about the need to return Flint to DWSD water and the political implications of the crisis. But in public, he denied all knowledge, despite being aware of the developing crisis. As a result, plaintiffs were lured into a false sense of security. They could have taken protective measures, if only they had known what the Governor knew. Instead, the Governor misled them into assuming that nothing was wrong. Governor Snyder’s administration even encouraged them to continue to drink and bathe in the water.

Subsequently, the Governor continued to show indifference to the risk of harm plaintiffs faced. Even once he acknowledged the crisis, he downplayed the risks that plaintiffs faced. By October 2015, the Governor had publicly admitted that the water was contaminated and Flint had returned to DWSD water. Yet the Governor still waited many months to declare a state of emergency. This was despite local area leaders requesting such a declaration as far back as March 2015. Without a state of emergency, plaintiffs were denied valuable resources that could have helped abate the harm that they were still suffering. It is reasonable to infer that the rationale for \*842 the delay was in part because the Governor wanted to act as if the issue was resolved. But by downplaying the continuing risk of harm, the Governor undermined efforts to enact protective measures. And as with his initial form of indifference, this led to plaintiffs involuntarily ingesting lead and other contaminants, violating their bodily integrity.

These two ways of showing indifference represent a continuum of actions, more powerful combined than when viewed in isolation. They depict indifference in the form of

deception, from the Governor's unwillingness to admit the crisis, to his downplaying of its severity once it became public knowledge. Viewed as a whole, the allegations plausibly describe “conscience shocking” conduct. Governor Snyder's actions were deliberately indifference and exhibited a callous disregard for plaintiffs' right to bodily integrity.<sup>8</sup>

## b. Qualified Immunity

[17] [18] [19] [20] Although plaintiffs plausibly plead that Governor Snyder violated their right to bodily integrity, qualified immunity shields public officials “from undue interference with their duties and from potentially disabling threats of liability.” *Harlow v. Fitzgerald*, 457 U.S. 800, 806, 102 S.Ct. 2727, 73 L.Ed.2d 396 (1982). It provides protection to government officials who make reasonable yet mistaken decisions that involve open questions of law. *Ashcroft v. al-Kidd*, 563 U.S. 731, 743, 131 S.Ct. 2074, 179 L.Ed.2d 1149 (2011). But an official cannot avail herself of qualified immunity if the right violated was “clearly established at the time of the challenged conduct.” *Guertin*, 912 F.3d at 917 (quoting *al-Kidd*, 563 U.S. at 741–42, 131 S.Ct. 2074). If controlling caselaw or a body of persuasive authority has put the constitutional question beyond debate, government officials are on notice that their conduct must conform to an established legal standard. *Id.* at 932.

[21] As the Sixth Circuit recently held, the right to bodily integrity was clearly established at the time of the challenged conduct. *Id.* at 932–35. “Knowing the Flint River water was unsafe for public use,” failing to take “steps to counter its problems, and assuring the public in the meantime that it was safe” was “ ‘conduct that would alert a reasonable person to the likelihood of personal liability.’ ” *Id.* at 933 (quoting *Scicluna v. Wells*, 345 F.3d 441, 446 (6th Cir. 2003)). In other words, any reasonable official should have known that “contaminat[ing] a community through its public water supply with deliberate indifference is a government invasion of the highest magnitude.” *Id.* As a result, the Governor is not entitled to qualified immunity.

## 2. Equal Protection

Plaintiffs also seek leave to revise their equal protection claims under § 1983. \*843 Plaintiffs' third amended complaint included two equal protection counts, one alleging discrimination on the basis of race and the other on wealth.

(Dkt. 349 at 119, 123.) Under both counts, plaintiffs alleged that defendants Snyder, Dillon, Wright, Walling, Ambrose, Kurtz, and Earley developed and executed an interim plan to deliver contaminated water to the predominantly poor African American residents of Flint, while providing the mostly white higher income residents of Genesee County with safe water. (*Id.* at 120–21, 124–25.)

The fourth amended complaint makes two important changes. First, only those plaintiffs who are African American allege race discrimination. (Dkt. 620-3 at 174, 180.) Second, both counts are broken into three theories of liability: (1) like in the third amended complaint, defendants Snyder, Dillon, Wright, Ambrose, Kurtz, and Earley violated their right to equal protection by providing Flint with contaminated water while supplying the remainder of Genesee County with clean water (*id.* at 174–76, 181–83);<sup>9</sup> (2) Governor Snyder violated their right to equal protection by delaying his decision to declare a state of emergency in Flint while promptly doing so in other emergency situations (*id.* at 177–78, 183–84); and (3) MDEQ defendants Wyant, Shekter-Smith, Prysby, and Busch violated their right to equal protection by not enforcing certain laws and regulations in Flint. (*Id.*) For the reasons that follow, the fourth amended complaint fails to state an equal protection claim under any of these theories, and so granting leave to amend the complaint to include these claims would be futile.

\*

[22] [23] [24] [25] “The Equal Protection Clause of the Fourteenth Amendment commands that no state shall ‘deny to any person within its jurisdiction the equal protection of the laws[.]’ ” *City of Cleburne v. Cleburne Living Ctr.*, 473 U.S. 432, 439, 105 S.Ct. 3249, 87 L.Ed.2d 313 (1985) (citing *Plyler v. Doe*, 457 U.S. 202, 216, 102 S.Ct. 2382, 72 L.Ed.2d 786 (1982)). Broadly speaking, it requires that state officials treat all persons alike, under like circumstances and like conditions. *Cleburne*, 473 U.S. at 439, 105 S.Ct. 3249; see also *Rondigo, L.L.C. v. Twp. of Richmond*, 641 F.3d 673, 682 (6th Cir. 2011). When officials treat similar individuals differently, the Equal Protection Clause demands a justification. *Engquist v. Or. Dep't of Agric.*, 553 U.S. 591, 602, 128 S.Ct. 2146, 170 L.Ed.2d 975 (2008). But because all state action tends to disfavor some more than others, courts take this practical reality into account by evaluating state action under differing levels of scrutiny. See *Breck v. Michigan*, 203 F.3d 392, 395 (6th Cir. 2000). If official conduct “neither burdens a fundamental right nor targets a suspect class,” courts will uphold it “so long as it bears a

rational relation to some legitimate end.” *Romer v. Evans*, 517 U.S. 620, 631, 116 S.Ct. 1620, 134 L.Ed.2d 855 (1996); see also *Radvansky v. City of Olmsted Falls*, 395 F.3d 291, 312 (6th Cir. 2005) (citing *Vacco v. Quill*, 521 U.S. 793, 799, 117 S.Ct. 2293, 138 L.Ed.2d 834 (1997)).

#### a. Wealth-Based Discrimination

[26] Plaintiffs fail to state a claim that defendants violated their right to equal protection on the basis of wealth discrimination. A class of less wealthy persons is \*844 not a protected class for the purposes of equal protection. *Molina-Crespo v. U.S. Merit Sys. Prot. Bd.*, 547 F.3d 651, 660 (6th Cir. 2008). The challenged conduct will therefore be upheld if it satisfies a rational basis. *Romer*, 517 U.S. at 631, 116 S.Ct. 1620.

[27] [28] [29] Under rational basis review, official decisions are afforded a strong presumption of validity. See *Walker v. Bain*, 257 F.3d 660, 668 (6th Cir. 2001). And even at the motion to dismiss stage, this presents a formidable bar for plaintiffs to surmount. *Theile v. Michigan*, 891 F.3d 240, 243 (6th Cir. 2018). To plausibly allege that state action fails under rational basis review, plaintiffs must negate “every conceivable basis” which might support the challenged conduct. *Davis v. Prison Health Servs.*, 679 F.3d 433, 438 (6th Cir. 2012). Courts do not consider the wisdom of the challenged action. *Theile*, 891 F.3d at 244 (citing *Breck*, 203 F.3d at 395). And defendants do not need to offer any justification. *Walker*, 257 F.3d at 668. It is enough that the reviewing court can fairly conceive of one existing. *Id.*

[30] As outlined above, plaintiffs’ first theory is that defendants Snyder, Dillon, Wright, Ambrose, Kurtz, and Earley created an interim plan to supply Flint with Flint River water, while continuing to provide the remainder of Genesee County with DWSD water. (Dkt. 620-3 at 181–82.) In plaintiffs’ view, there was no rational basis for this decision. (*Id.* at 182–83.)

Even assuming that Flint and the remainder of Genesee County were similarly situated for equal protection purposes, there are many rational reasons that could justify providing only Flint with Flint River water. The KWA could not proceed without Flint’s participation. Flint’s participation was contingent on the FWTP’s ability to process the raw water that the KWA pipeline would provide, and upgrading the FWTP would cost millions. One key way defendants

could accomplish this was to stop paying for the relatively expensive DWSD water and to start taking water from the Flint River. Indeed, even plaintiffs allege that this was a critical part of the interim plan.

In hindsight, this was a terrible decision. It placed financial interests above the health and safety of Flint’s residents. Assuming the allegations are true, defendants harmed plaintiffs in the pursuit of fiscal expedience. But the Court cannot consider the wisdom of the decision. And it does not matter that defendants may have had other options available to them. It only matters that there is a rational basis for the decision. As such, plaintiffs’ first theory fails to state a claim.

[31] Plaintiffs’ second theory fails for a similar reason. They draw a comparison between Flint and other communities with respect to emergencies across the state. Governor Snyder allegedly waited several months to declare a state of emergency in Flint from the date he publicly acknowledged the seriousness of the problem. (Dkt. 620-3 at 102). With other disasters, he typically acted within days. (*Id.* at 152–53.) Plaintiffs again argue that there was no rational basis for this difference in treatment. (*Id.* at 183.)

Again, even assuming that Flint and these other disaster-struck communities were similarly situated for equal protection purposes, there is a conceivable rational basis for treating them differently. In part, plaintiffs were harmed by the Governor’s delay in declaring a state of emergency because it limited their access to state resources to remedy the problem. It is thus conceivable that the Governor initially decided not to expend these resources, believing that the Flint Water Crisis could be addressed without them. In retrospect, \*845 this was objectively the wrong decision. And the Governor undoubtedly was within his authority to declare a state of emergency at an earlier time. But the Court cannot inquire further under rational basis review. As a result, plaintiffs’ second theory also fails to state a claim.

Plaintiffs’ third theory runs into a different problem. Plaintiffs allege that the MDEQ defendants Wyant, Shekter-Smith, Prysby, and Busch treated them differently by:

- (1) granting a fraudulent [ACO] to allow Flint to borrow funds to participate in the KWA;
- (2) issuing the [FWTP] a permit pursuant to the Michigan Safe Drinking Water Act without observing the statutorily mandated 45-day notice and comment period;
- (3) failing to comply with sampling and optimized corrosion control protocols as required under the State and Federal Lead and Copper

Rule; and (4) lacking any nondiscrimination policy for more than 30 years and ignoring EPA requirements to update its policy for years.

(*Id.* at 183–84.) However, they fail to explain how this treatment differed from that of a similarly situated class of persons.

[32] [33] [34] Class-based discrimination is the essence of an equal protection claim. See *Herron v. Harrison*, 203 F.3d 410, 417 (6th Cir. 2000) (citing cases). In limited situations, a plaintiff does not need to identify a specific group of persons who were treated differently. For instance, if government conduct was premised on a protected classification such as race or gender, a showing of discriminatory purpose may suffice. See, e.g., *Vill. of Arlington Heights v. Metro. Hous. Dev. Corp.*, 429 U.S. 252, 264–68, 97 S.Ct. 555, 50 L.Ed.2d 450 (1977) (explaining that a single act, if motivated by a desire to treat persons differently on the basis of race, can result in a violation of the Equal Protection Clause). However, outside of that narrow range of cases, plaintiffs must plead sufficient facts from which it can be inferred that defendants treated similarly situated individuals differently. *Braun v. Ann Arbor Charter Twp.*, 519 F.3d 564, 574–75 (6th Cir. 2008); see also *Klinger v. Dep't of Corr.*, 31 F.3d 727, 731 (8th Cir. 1994) (“Absent a threshold showing that she is similarly situated to those who allegedly receive favorable treatment, the plaintiff does not have a viable equal protection claim.”).

[35] Here, plaintiffs highlight several instances in which defendants failed to enforce either a law or a policy, but the allegations do not explain in anything but conclusory terms how defendants acted differently in other situations. For example, to the extent that defendants failed to observe the statutory forty-five day notice and comment period before issuing the FWTP an operating permit, it may be that they normally dispensed with this requirement. Likewise, although plaintiffs plead that defendants did not comply with state and federal lead and copper testing requirements, the complaint reveals nothing about the possibility that defendants failed to enforce these laws on a statewide basis. Accordingly, plaintiffs' third and final theory also fails to state a claim.

In some of their briefing, plaintiffs argue that rational basis review should not apply to their wealth-based equal protection claim because the claim should be construed as one involving discrimination implicating the fundamental right to bodily integrity. (Dkt. 379 at 88.) However, plaintiffs have not pleaded the claim this way in the fourth amended complaint. And in fact, plaintiffs do not raise this argument in their most

recent briefing. The Court therefore continues to view the claim as one involving discrimination on the basis of wealth. For these reasons, leave to amend \*846 the complaint to include this claim would be futile.

#### b. Race-Based Discrimination

[36] [37] Plaintiffs also fail to state a claim that defendants violated their right to equal protection on the basis of race discrimination. When state action is premised on a racial classification, courts strictly scrutinize the challenged conduct. *Cleburne*, 473 U.S. at 440, 105 S.Ct. 3249; *Mass. Bd. of Ret. v. Murgia*, 427 U.S. 307, 312, 96 S.Ct. 2562, 49 L.Ed.2d 520 (1976); see also *United States v. Carolene Prod. Co.*, 304 U.S. 144, 152 n.4, 58 S.Ct. 778, 82 L.Ed. 1234 (1938) (noting that courts act with greater vigilance when equal protection claims affect the politically powerless). Conduct subject to strict scrutiny is presumptively invalid; only official action that is narrowly tailored to meet a compelling state interest will survive. *Lac Vieux Desert Band of Lake Superior Chippewa Indians v. Mich. Gaming Control Bd.*, 276 F.3d 876, 879 (6th Cir. 2002).

[38] [39] [40] Yet “ ‘proof of ... discriminatory intent or purpose is required’ to show a violation of the Equal Protection Clause” on the basis of race discrimination. *City of Cuyahoga Falls v. Buckeye Cmty. Hope Found.*, 538 U.S. 188, 194, 123 S.Ct. 1389, 155 L.Ed.2d 349 (2003) (quoting *Arlington Heights*, 429 U.S. at 265, 97 S.Ct. 555); *Washington v. Davis*, 426 U.S. 229, 239–41, 96 S.Ct. 2040, 48 L.Ed.2d 597 (1976). If discriminatory intent is missing, claims are analyzed under rational basis. See *Radvansky*, 395 F.3d at 312. And the facts must offer more than “intent as volition or intent as awareness of consequences.” *Pers. Adm'r v. Feeney*, 442 U.S. 256, 279, 99 S.Ct. 2282, 60 L.Ed.2d 870 (1979). Rather, they must demonstrate that a decisionmaker “selected or reaffirmed a particular course of action at least in part ‘because of,’ not merely ‘in spite of,’ its adverse effects upon a particular racial group.” *Id.*; *Bennett v. City of Eastpointe*, 410 F.3d 810, 818 (6th Cir. 2005) (quoting *King v. City of Eastpointe*, 86 F. App'x 790, 802 (6th Cir. 2003)).

[41] [42] [43] [44] [45] At this stage in the case, plaintiffs need only raise an inference of discriminatory purpose. To do so, they must demonstrate that the application of a facially neutral law or policy had a discriminatory impact, and sufficient evidence exists to suggest an invidious motive. *Arlington Heights*, 429 U.S. at 265–66, 97 S.Ct. 555; *Ne*

*Ohio Coal. for the Homeless v. Husted*, 837 F.3d 612, 636–37 (6th Cir. 2016).<sup>10</sup> The challenged conduct does not need to rest “solely on racially discriminatory purposes,” but this must have been a “motivating factor.” *Arlington Heights*, 429 U.S. at 265, 97 S.Ct. 555. And although discriminatory impact is an important starting point, it is rarely enough on its own. *Id.* Instead, courts must conduct “a sensitive inquiry into such circumstantial and direct evidence of intent as may be available.” *Id.* at 266, 97 S.Ct. 555. Discriminatory impact alone is only sufficient in the rarest case where “a clear pattern, unexplainable on grounds other than race, emerges from the effect of the state action[.]” *Id.* at 266, 97 S.Ct. 555 (citations omitted).

[46] Several non-exhaustive factors guide this inquiry: (1) “[t]he historical background of the decision is one evidentiary source, particularly if it reveals a series of official actions taken for invidious purposes,” *id.* at 267, 97 S.Ct. 555; (2) “the \*847 specific sequence of events leading up to the challenged decision ... may shed ... light on the decisionmaker’s purposes,” *id.*; (3) “[d]epartures from the normal procedural sequence ... particularly if the factors usually considered important by the decisionmaker strongly favor a decision contrary to the one reached,” *id.*; and (4) “[t]he legislative or administrative history ... especially where there are contemporary statements by members of the decisionmaking body, minutes of its meetings, or reports,” *id.* at 268, 97 S.Ct. 555.<sup>11</sup>

As a starting point, plaintiffs plead discriminatory impact for all three theories. Under each theory, they allege that defendants’ conduct negatively impacted Flint. And Flint is majority African American. However, this is not the “rarest case” where the discriminatory impact is so stark as to immediately warrant an inference of discriminatory purpose. See *Gomillion v. Lightfoot*, 364 U.S. 339, 81 S.Ct. 125, 5 L.Ed.2d 110 (1960) (invalidating state action where redrawing of city boundaries disenfranchised all but four or five of the municipality’s 400 African American voters); *Yick Wo v. Hopkins*, 118 U.S. 356, 6 S.Ct. 1064, 30 L.Ed. 220 (1886) (finding a violation of equal protection where an ordinance was exclusively applied against Chinese-owned laundries). After all, Flint is 40.4% white. (Dkt. 620-3 at 128.)

Plaintiffs also point to the historical background for all three theories. They identify a long history of race discrimination and segregation and argue that this should factor into the Court’s analysis. (Dkt. 620-1 at 18.) But plaintiffs do not connect Flint’s history of systemic racism to defendants’

conduct. They imply that the legacy effects of historical racism should be imputed to defendants because they were state actors carrying out official business. But this alone is not enough to warrant an inference of discriminatory purpose. It will be considered with the other evidence.

[47] Plaintiffs’ first theory, the decision to switch Flint’s water supply to the Flint River while providing DWSD water to the remainder of Genesee County, lacks sufficient facts to warrant an inference of discriminatory purpose. Little about the sequence of events indicates that a racial bias was driving defendants. In the months leading up to the switch, cost studies suggested that DWSD water was the more economic mid-term option. But the KWA would only be viable if the Flint River was used as an interim water source. And defendants were concerned that DWSD water would become increasingly expensive.

Likewise, defendants’ contemporary statements do not change the outcome. Defendant Wright expressed the view that DWSD is a corrupt entity.<sup>12</sup> But this does not indicate racial animus and the fourth amended complaint offers nothing further. Therefore, when all the facts are taken into consideration, the allegations fail to show that race motivated defendants’ decision. At most they show that defendants \*848 acted in spite of the risk of harm that plaintiffs faced, not that they were driven by it. Plaintiffs’ first theory thus fails to state a claim.

[48] With their second theory that the Governor treated the emergency situation in Flint differently, plaintiffs also fail to allege sufficient facts to warrant an inference of discriminatory purpose. Governor Snyder allegedly knew that Flint’s water supply was contaminated months before publicly acknowledging it, but he did not alert the public until October 2015, when it was impossible to deny. The Governor also took many months more to declare a state of emergency. And presumably the conditions that gave rise to the eventual emergency declaration existed the whole time. Similarly, a departure from past practice works in plaintiffs’ favor. Governor Snyder’s conduct in Flint differed from that in some majority white communities, where he promptly issued states of emergency.

Nonetheless, these facts taken as a whole do not support an inference of discriminatory intent. The comparative states of emergency identified in the fourth amended complaint involved drastically different situations, such as several wildfires and floods, meaning plaintiffs’ comparison is less

apples-to-apples than it initially appears. And in the one instance where plaintiffs cite to an emergency involving water contamination, they identify an incident that occurred several years after the facts pertinent to this present case. Accordingly, it is hard to know whether the Governor's prompt response was a reaction to the criticism about his handling of Flint, rather than evidence of a desire to harm African Americans. Moreover, plaintiffs do not point to a clear pattern of discrimination where Governor Snyder consistently delayed declaring states of emergency in mostly African American areas. In fact, a close inspection of the analogous emergencies suggests the opposite was almost true. During a flood in Wayne County, which is 45.4% non-white, the Governor declared an emergency within two days. (Dkt. 620-3 at 152.)<sup>13</sup> The departure from practice is less salient.

Plaintiffs point to no other facts sufficient to support a finding of discriminatory purpose. During the crisis, a senior member of the Governor's administration dismissed complaints from Flint activists as the product of "old time negative racial experiences." (*Id.* at 161.) But even if the same thoughts are attributed to Governor Snyder, it only shows that he acted in spite of the fact that Flint was majority African American, not because of this fact. When the allegations are collectively considered, they do not warrant an inference of invidious intent. And as such, plaintiffs' second theory fails to state a claim.

[49] Finally, plaintiffs' third theory that the MDEQ defendants failed to enforce certain laws and policies also fails to allege sufficient facts to warrant an inference of discriminatory purpose. As discussed above, plaintiffs generally point to Flint's history of racial discrimination, and this alone is insufficient to show invidious intent. However, here, plaintiffs also note that the EPA had concluded earlier that the MDEQ had discriminated against Flint's African Americans when issuing an operating permit for a local power station. In particular, the EPA found that the MDEQ did not have a sufficient non-discrimination \*849 policy in place. And this lack of policy persisted during the Flint Water Crisis. As recently as 2017, the EPA was still raising concerns that the MDEQ did not take its non-discrimination obligations seriously.

However, the MDEQ's failure to develop a sufficient non-discrimination policy does not demonstrate discriminatory intent. Plaintiffs do not allege that Shekter-Smith, Prysby, and Busch were responsible for the MDEQ's internal policies. Nor is there any sign that they obstructed or otherwise

hindered the development of other procedural safeguards. Defendant Wyant, as MDEQ director, was presumably ultimately responsible for the non-discrimination policy, but plaintiffs do not plead facts that suggest his failure to develop such a policy was motivated by a nefarious purpose.

Neither the specific sequence of events nor any departure from standard procedures suggest a race-based motive. Defendants Shekter-Smith and Busch were allegedly involved in helping Flint secure a fraudulent ACO. Yet there is no suggestion that a desire to harm African Americans motivated their conduct. The same is true of the decision to grant the FWTP an operating permit without sufficient public participation, and the MDEQ's failure to enforce lead and copper testing requirements. The allegations do not provide any way to link these decisions to a discriminatory intent. Plaintiffs allege that these types of nonconformities with law and policy never occurred in majority white communities, but these are conclusory accusations. These defendants also made no contemporary statements indicating that race motivated their actions. And there is nothing else to connect their conduct to a discriminatory purpose. As such, when the facts are considered together, plaintiffs' third theory fails to state a claim.

\*

Plaintiffs' equal protection allegations fail to state a claim upon which relief can be granted. The wealth-based claim fails on grounds that rational justifications conceivably exist that explain the challenged conduct. And the race-based claim fails because plaintiffs plead insufficient facts to infer that a discriminatory purpose motivated defendants' decisions. That is not to say that race and poverty did not play a role in the Flint Water Crisis. As plaintiffs explain, multiple sources indicate that historical patterns of discrimination created the conditions for what happened. But under current caselaw, the Equal Protection Clause does not provide redress for the harms as alleged. Granting plaintiffs' leave to amend the complaint to include them would be futile.

### 3. Elliott Larsen Civil Rights Act

Plaintiffs next seek to revise their claim under Article 3 of Michigan's Elliott Larsen Civil Rights Act (ELCRA), which addresses discrimination in public services and accommodations. [Mich. Comp. Laws §§ 37.2301–37.2304 \(2017\)](#). Plaintiffs' ELCRA allegations mirror their equal

protection claims. Only the African American plaintiffs bring the claim on behalf of an African American class. (Dkt. 620-3 at 190.) And plaintiffs advance similar theories of liability: that (1) defendants Snyder, Dillon, Wright, Ambrose, Kurtz, Earley, and the City of Flint provided Flint's predominantly African American residents with inferior water when compared to the mostly white residents of Genesee County (*id.* at 192–93); (2) Governor Snyder failed to promptly declare a state of emergency in Flint compared to other emergencies in predominantly white communities, (*id.* at 193–94); and (3) the MDEQ defendants Wyant, Shekter-Smith, Prysby, and Busch failed to enforce certain laws and regulations. (*Id.* at 194–95.) For the following \*850 reasons, plaintiffs' ELCRA claim could not withstand a motion to dismiss if leave to amend were granted, and so granting leave would be futile.

\*

[50] [51] The ELCRA “is aimed at ‘the prejudices and biases’ borne against persons because of their membership in a certain class, and seeks to eliminate the effects of offensive or demeaning stereotypes, prejudices, and biases.” *Radtke v. Everett*, 442 Mich. 368, 379, 501 N.W.2d 155 (1993) (quoting *Miller v. C.A. Muer Corp.*, 420 Mich. 355, 363, 362 N.W.2d 650 (1984)). To state a claim under Article 3, plaintiffs must allege: “(1) discrimination based on a protected characteristic (2) by a person, (3) resulting in denial of the full and equal enjoyment of [a public service].” See *Haynes v. Neshewat*, 477 Mich. 29, 35, 729 N.W.2d 488 (2007); *Clarke v. K Mart Corp.*, 197 Mich. App. 541, 545, 495 N.W.2d 820 (1992). The ELCRA defines public service as “a public facility ... owned, operated, or managed by or on behalf of ... a political subdivision ... established to provide service to the public.” § 37.2301. For the purposes of this analysis, the Court assumes that Flint's municipal water supply is a public service under the ELCRA.

[52] [53] [54] [55] [56] The public service provision of the ELCRA uses the same framework to establish discrimination as that used generally under other provisions of the ELCRA. See *Schellenberg v. Rochester Mich. Lodge No. 2225 of the Benev. & Prot. Order of Elks*, 228 Mich. App. 20, 32, 577 N.W.2d 163 (1998); *Clarke*, 197 Mich. App. at 545, 495 N.W.2d 820. Plaintiffs must show either intentional discrimination directly or raise an inference of discrimination based on a disparate treatment theory. *Hazle v. Ford Motor Co.*, 464 Mich. 456, 462–63, 628 N.W.2d 515 (2001); *Clarke*, 197 Mich. App. at 545, 495 N.W.2d

820. In a case like this involving allegations of race-based discrimination, plaintiffs can plead intentional discrimination by pointing to direct evidence that defendants were predisposed to discriminate against African Americans, and that they acted on that pre-disposition. See *Reisman v. Regents of Wayne State Univ.*, 188 Mich. App. 526, 538, 470 N.W.2d 678 (1991). Direct evidence is “evidence which, if believed, requires the conclusion that unlawful discrimination was at least a motivating factor in [defendants'] actions.” *Hazle*, 464 Mich. at 462, 628 N.W.2d 515 (quoting *Jacklyn v. Schering-Plough Healthcare Prods. Sales Corp.*, 176 F.3d 921, 926 (6th Cir. 1999)). Alternatively, plaintiffs can raise an inference of discrimination by pleading that defendants treated them differently from non-protected individuals under the same or similar circumstances. See *Reisman*, 188 Mich. App. at 538, 470 N.W.2d 678 (citing *Singal v. Gen. Motors Corp.*, 179 Mich. App. 497, 502–03, 447 N.W.2d 152 (1989)); *Schellenberg*, 228 Mich. App. at 33, 577 N.W.2d 163. But here, they must also point to sufficient indirect evidence from which it can be inferred that race was a motivating factor, even if not “the sole factor.” See *Reisman*, 188 Mich. App. at 539, 470 N.W.2d 678; see also Mich. M Civ. II 108.04 (2018) (identifying intentional discrimination as an element in an Article 3 ELCRA claim).

It is unclear whether plaintiffs are relying on direct evidence or evidence of disparate treatment to prove this claim. Plaintiffs do not offer direct evidence to show defendants were predisposed to discriminate on the basis of race, nor that they acted on that predisposition. However, they have pleaded facts consistent with a disparate treatment theory and so the Court proceeds on this basis.

[57] Under a disparate treatment approach, plaintiffs fail to plead sufficient \*851 facts to raise an inference of racial discrimination. This is for the same reasons as set forth above with respect to plaintiffs' equal protection claims. See *supra* Section II.d.ii.2.b. Plaintiffs have not explained why their ELCRA claim should be evaluated under a different standard. Therefore, plaintiffs' ELCRA claim could not survive a motion to dismiss, and so granting leave to amend the complaint to include it would be futile.

#### 4. Conspiracy

Plaintiffs next seek leave to amend their conspiracy claim under § 1985(3). (Dkt. 620-3 at 186–90.) This claim is also only brought by the African American plaintiffs on behalf

of an African American class. (*Id.* at 186.) Plaintiffs argue that defendants Snyder, Dillon, Wright, Ambrose, Kurtz, and Earley conspired to expose them to contaminated water from the Flint River (*id.* at 186–87),<sup>14</sup> and the means by which they did this is familiar: defendants developed an interim plan to provide safe water to the predominately white population of Genesee County while supplying unsafe water to Flint residents. (*Id.* at 187–88.) In plaintiffs' view, there was no rational reason to treat these two groups differently. (*Id.* at 188.) Defendants' conduct was based on invidious discrimination, akin to imposing a badge, vestige, or symbol of slavery, as prohibited by the Thirteenth Amendment. (*Id.* at 188–89.)<sup>15</sup>

[58] [59] [60] In the context of § 1985(3), plaintiffs shoulder a heavy pleading burden. “Conspiracy claims must be pled with some degree of specificity[.]” *Gutierrez v. Lynch*, 826 F.2d 1534, 1538–39 (6th Cir. 1987). “[V]ague and conclusory allegations unsupported by material facts will not be sufficient to state such a claim[.]” *Id.* To state a claim under § 1985(3), plaintiffs must plead facts consistent with (1) a conspiracy between two or more persons, (2) conceived for the purpose of depriving a person or class of people of the equal protection of the laws, (3) an act committed in furtherance of the conspiracy, and (4) that a person was either injured in his or her person or property, or deprived of a right guaranteed by the Constitution. *Peters v. Fair*, 427 F.3d 1035, 1038 (6th Cir. 2005) (citing *Johnson v. Hills & Dales Gen. Hosp.*, 40 F.3d 837, 839 (6th Cir. 1994)).<sup>16</sup> In so doing, plaintiffs must demonstrate that the conspiracy was motivated by racial or other constitutionally suspect class-based animus. *Bartell v. Lohiser*, 215 F.3d 550, 559–60 (6th Cir. 2000) (citing *United Bhd. of Carpenters & Joiners of Am. v. Scott*, 463 U.S. 825, 829, 103 S.Ct. 3352, 77 L.Ed.2d 1049 (1983)).

\*852 [61] [62] Pleading invidious class-based animus is important. Section 1985(3) is not a “general federal tort law,” providing a federal cause of action for every assault and battery. *Bray v. Alexandria Women's Health Clinic*, 506 U.S. 263, 299, 113 S.Ct. 753, 122 L.Ed.2d 34 (1993) (citing *Griffin v. Breckenridge*, 403 U.S. 88, 102, 91 S.Ct. 1790, 29 L.Ed.2d 338 (1971)). The intent requirement ensures that only those conspiracies that “aim at a deprivation of the equal enjoyment of rights secured by the law to all” are actionable under the statute. *Griffin*, 403 U.S. at 102, 91 S.Ct. 1790.

[63] Plaintiffs fail to plausibly allege that defendants were motivated by racial or any other invidious class-based animus.

Plaintiffs possibly show that the impact of historical race discrimination played a role in the Flint Water Crisis, but not that it was a motivating factor. For example, plaintiffs repeatedly assert that the interim plan provided safe water to predominantly white Genesee County residents and unsafe water to the mostly African American Flint residents. But this only demonstrates a disparate impact resulting from defendants' decisions. It does not show that they were motivated by the kind of discriminatory animus necessary to state a § 1985(3) claim. Similarly, plaintiffs contend that early complaints from Flint residents would have been taken into account faster had they been affluent and predominantly white. This allegation suffers from the same flaw.

Many of the facts contained in the fourth amended complaint set forth the historic impact of racism in Flint, but not specific instances of racially motivated conduct by the defendants. This history is important to understanding patterns of segregation, poverty, and other conditions that may have left plaintiffs vulnerable to the Flint Water Crisis. Yet such theories do not show invidious class-based animus by the named defendants.

Plaintiffs raise many serious and challenging issues, but they fail to plausibly allege that race discrimination animated defendants' conduct. This is especially so considering the heightened pleading standard. Therefore, their revised § 1985(3) claim could not withstand a motion to dismiss. As such, granting leave to amend the complaint to include it would be futile.

## 5. Gross Negligence

Finally, plaintiffs seek leave to add a gross negligence claim against defendants Snyder, Dillon, Lyon, Shekter-Smith, Rosenthal, Busch, Cook, Prysby, Wurfel, Wright, Kurtz, Earley, Ambrose, Croft, Johnson, and Glasgow. (Dkt. 620-3 at 217.) Plaintiffs allege that these government defendants owed them a duty not to conduct their official responsibilities recklessly (*id.*), but that they did so by playing a role in Flint's transition to the Flint River and downplaying the resulting harm. (*Id.* at 217–18.) Plaintiffs argue that defendants' conduct was grossly negligent and caused their injuries, and that defendants are not entitled to immunity under Michigan's Government Tort Liability Act (GTLA), *Mich. Comp. Laws* § 691.1401–1419 (2014). (Dkt. 620-3 at 218.) However, this claim also could not withstand a motion

to dismiss and amending the complaint to include it would be futile.

\*

Defendants are immune from tort liability. The GTLA premises immunity on various theories. Pertinently, “the elective or highest appointive executive official of all levels of government are immune from tort liability for injuries to persons or damages to property if he or she is acting within the scope of his or her ... executive authority.” § 691.1407(5). Under this theory, defendants Snyder, Dillon, and Lyon are absolutely immune. See \*853 *Guertin*, 2017 WL 2418007, at \*25, 2017 U.S. Dist. LEXIS 85544, at \*77–78. The same is true of defendants Kurtz, Earley, Ambrose, and Croft, and defendants Shekter-Smith and Wurfel. See *id.*

[64] This leaves the remaining defendants, lower-level government employees. Lower-level employees are “immune from tort liability for an injury to a person or damage to property caused by the ... employee ... while in the course of employment” if the employee is “acting or reasonably believes he or she is acting within the scope of his or her authority,” unless the employees’ conduct amounts to “gross negligence that is the proximate cause of the injury or damage.” § 691.1407(2)(a)–(c). To identify whether a lower-level employee was the proximate cause of an injury, courts must first evaluate “the conduct and any legal responsibility” of the various parties to an accident, *Ray v. Swager*, 501 Mich. 52, 74, 903 N.W.2d 366 (2017), where legal responsibility is assessed by determining whether the accident was a foreseeable consequence of an individual’s actions, see *id.* at 69, 903 N.W.2d 366. And second, courts must jointly consider the actions of those legally responsible to determine whose conduct was the “one most immediate, efficient, and direct cause” of any injury. *Id.* at 83, 903 N.W.2d 366 (quoting *Robinson v. City of Detroit*, 462 Mich. 439, 462, 613 N.W.2d 307 (2000)). If the answer is anyone but the employee, the employee can claim immunity.

[65] Plaintiffs do not address *Ray*’s causation requirement. The fourth amended complaint states that defendants’ conduct was a direct and proximate cause of plaintiffs’ injuries, but fails to explain, first, why they were legally responsible for this harm in anything but conclusory terms, and second, why such conduct was the “one most immediate, efficient, and direct cause” preceding plaintiffs’ injuries. Instead, plaintiffs argue that they need only demonstrate that “it was foreseeable that the defendant’s conduct could result in harm to the

victim.” (Dkt. 620-1 at 22 (quoting *Ray*, 501 Mich. at 65, 903 N.W.2d 366).) But this is a misinterpretation of *Ray*, a case this Court is bound to follow. And because plaintiffs ask the Court to do something it cannot, amending the fourth amended complaint to include this claim would be futile.

Plaintiffs raise several counter arguments, but none are persuasive. First, plaintiffs argue that defendants were not acting within the scope of their authority, and, as such, they cannot claim immunity. (Dkt. 663 at 50–52.) For instance, plaintiffs contend that Governor Snyder did not have the power to discriminate against African American communities in his handling of emergencies. (*Id.* at 51.) Similarly, plaintiffs argue that Lyon and Wurfel did not have the authority to downplay the risks of harm posed by the contaminated water. (*Id.* at 52.) But these arguments are circular. Under plaintiffs’ view, tortious conduct is sufficient to deprive a government defendant of immunity because tortious conduct is not within the defendant’s scope of authority. This is not how the GTLA functions.

Second, plaintiffs claim that even if their interpretation of caselaw is wrong, they have adequately pleaded that defendants were the “most immediate, efficient, and direct cause” of plaintiffs’ injuries, because each defendants’ conduct served as “the proximate cause” for some discrete harm that, taken in the aggregate, forms a piece of the Flint Water Crisis. (*Id.* at 54–55.) For example, defendant Busch falsely informed an EPA official that Flint was using corrosion control, which proximately caused the State to slowly address the dangers of lead in the water. (*Id.* at 55.) \*854 However, the focus is on the injury claimed. See *Robinson*, 462 Mich. at 462, 613 N.W.2d 307. In other words, it is not enough that plaintiffs are able to point to some cause and effect relationship within the Flint Water Crisis. They must demonstrate how a defendant’s action was the “one most immediate, efficient, and direct cause” of their injuries. For this reason, this argument also fails.

[66] The sheer size and scale of the Flint Water Crisis makes it difficult for plaintiffs—or anyone—to identify any defendant most legally responsible for the resulting injuries. As such, “the more governmental actors that are involved in causing a massive tort in Michigan, the less likely it is that state tort claims can proceed against the individual government actors[.]” *Guertin*, 2017 WL 2418007, at \*27, 2017 U.S. Dist. LEXIS 85544, at \*81. Ultimately, the Court is required to apply the GTLA as interpreted by the Michigan

Supreme Court. It would therefore be futile to amend the complaint to include plaintiffs' gross negligence claim.

## 6. Res Judicata and Statute of Limitations

Defendants Wyant and Wurfel argue that the doctrine of res judicata or, alternatively, the relevant statutes of limitations prohibit plaintiffs from amending the complaint. (Dkt. 653 at 29–34; Dkt. 657 at 26–28.) Because the Court found plaintiffs' amended claims with respect to defendants Wyant and Wurfel futile, these issues need not be addressed.

### e. Conclusion

Plaintiffs' motion for leave to amend the complaint regarding the bodily integrity claim against Governor Snyder is granted, and plaintiffs' motion regarding the remaining claims is denied. Furthermore, the Court finds no reason to deny leave to amend to include the fourth amended complaint's new factual allegations, including the proposed class definitions, the certification of which will be addressed at a later date. Plaintiffs' motion as it relates to these facts is therefore also granted. These conclusions will again be summarized at the end of Part III, *infra*.

### III. Motions to Dismiss

As previously stated in Part I, *supra*, the Court adopts the fourth amended complaint as the operative complaint for the purpose of adjudicating defendants' motions to dismiss. If the Court denied leave to amend the complaint to include a particular claim in Part II, that claim will be dismissed with no further discussion.

#### a. Background

The facts, parties, and proposed classes remain unchanged from those set forth above in Part II. The fourth amended complaint contains the following counts:

Count	Claim	Defendants
I	§ 1983 Bodily Integrity	All government defendants
II-III	§ 1983 - Equal Protection	Snyder, Dillon, Wright, Ambrose, Kurtz, Earley, Wyant, Shekter-Smith, Prysby, and Busch
IV	§ 1985(3) - Conspiracy	Snyder, Dillon, Wright, Ambrose, Kurtz, and Earley
V	ELCRA	Snyder, Dillon, Wright, Ambrose, Kurtz, Earley, The City of Flint, Wyant, Shekter-Smith, Prysby, and Busch
VI	<i>Monell</i> Liability	The City of Flint
VII-VIII	Professional Negligence	LAN and Veolia
IX	§ 1983 – State-Created Danger	All government defendants
X	Fraud	Veolia
XI	Negligent Infliction of Emotional Distress	LAN and Veolia
XII	Negligence	LAN and Veolia
XIII-XIV	Gross Negligence	LAN, Veolia, Snyder, Dillon, Lyon, Shekter-Smith, Rosenthal, Busch, Cook, Prysby, Wurfel, Wright, Kurtz, Earley, Ambrose, Croft, Johnson, and Glasgow.

#### b. Standard of Review

[67] [68] A motion to dismiss pursuant to [Federal Rule of Civil Procedure 12\(b\)\(1\)](#) challenges the Court's subject matter jurisdiction. When ruling on a [Rule 12\(b\)\(1\)](#) motion “the court must take the material allegations of the [complaint] as true and construed in the light most favorable to the nonmoving party.” *United States v. Ritchie*, 15 F.3d 592, 598 (6th Cir. 1994). Plaintiffs need only show “that the complaint alleges a claim under federal law, and that the claim is substantial.” *Musson Theatrical, Inc. v. Fed. Express Corp.*, 89 F.3d 1244, 1248 (6th Cir. 1996) (citing *Transcon. Leasing, Inc. v. Mich. Nat'l Bank*, 738 F.2d 163, 166 (6th Cir. 1984)). This is a relatively light burden. *Id.* “Dismissal for lack of subject-matter jurisdiction ... is proper only when the claim is ‘so insubstantial, implausible, foreclosed by prior decisions of [the Supreme Court], or otherwise completely devoid of merit as not to involve a federal controversy.’” *Steel Co. v. Citizens for a Better Env't*, 523 U.S. 83, 89, 118 S.Ct. 1003, 140 L.Ed.2d 210 (1998) (quoting *Oneida Indian Nation of N.Y. v. Cty. of Oneida*, 414 U.S. 661, 666, 94 S.Ct. 772, 39 L.Ed.2d 73 (1974)).

A motion that challenges the legal sufficiency of a complaint is instead properly brought under [Federal Rule of Civil Procedure 12\(b\)\(6\)](#). *See supra* Section II.b (setting forth the motion to dismiss standard).

#### c. Threshold Issues

\*855

### i. Sovereign Immunity

The state and city defendants argue that sovereign immunity deprives the Court of subject matter jurisdiction to adjudicate certain claims. They therefore move to dismiss under Rule 12(b)(1). The Court \*856 grants the state defendants' motion in part as it relates to the State of Michigan, but denies the motions in all other respects.

First, the state defendants argue that sovereign immunity bars plaintiffs' claims against the State of Michigan and their claim for injunctive relief against the Governor in his official capacity. (Dkt. 279 at 29–32; Dkt. 739 at 16–18.) The state defendants are correct that sovereign immunity bars claims against the State of Michigan. *Boler v. Earley*, 865 F.3d 391, 413 (6th Cir. 2017). But for the following reasons, the Court has subject matter jurisdiction over claims against Governor Snyder in his official capacity.

[69] [70] In *Boler*, the Sixth Circuit explained that there are three exceptions to sovereign immunity, the relevant one here being “when the doctrine set forth in *Ex Parte Young*, 209 U.S. 123, 28 S.Ct. 441, 52 L.Ed. 714 (1908) applies.”<sup>17</sup> 865 F.3d at 410 (citations omitted). “*Ex Parte Young* allows plaintiffs to bring claims for prospective [injunctive] relief against state officials sued in their official capacity to prevent future federal constitutional or statutory violations.” *Id.* at 412. In this case, plaintiffs only seek injunctive relief against Governor Snyder in his official capacity.

[71] *Boler* is a Sixth Circuit decision that forms part of the Flint Water Cases litigation. It held that the *Ex Parte Young* exception applied to the injunctive relief sought by the plaintiffs against Governor Snyder in his official capacity. There, the plaintiffs sought an “injunctive order to remediate the harm caused by Defendants' unconstitutional conduct including, but not limited to: repairs of private property and establishment of medical monitoring to provide healthcare and other appropriate services to Class members for a period of time deemed appropriate by the Court.” *Id.* at 413. (quotations omitted). They also requested a “monitor who will assist in the development of remedial plans including, but not limited to: early education, education intervention programs, [and] criminal and juvenile justice evaluations.” *Id.* (quotations omitted).

Here, plaintiffs similarly seek an order “to remediate the harm caused by the Government Defendants' unconstitutional

conduct.” (Dkt. 620-3 at 219.) And this includes the exact same relief as that set forth in *Boler*. *Boler* therefore controls and requires the same outcome. The claim for injunctive relief against Governor Snyder in his official capacity may go forward.

Second, the city defendants argue that sovereign immunity deprives the Court of jurisdiction to adjudicate plaintiffs' claims against the City of Flint because it was an arm of the State in the period leading up to and including the Flint Water Crisis. (Dkt. 276 at 43–55.) According to the city defendants, Flint is therefore entitled to the same cloak of sovereign immunity as that afforded the State. (*Id.*) However, the Sixth Circuit recently rejected this argument in *Guertin v. Michigan*, 912 F.3d 907, 941 (6th Cir. 2019). This argument is therefore denied.

In sum, although immunity with regards to the State of Michigan is granted, it is denied as to Governor Snyder in his official capacity and the City of Flint. The Court has subject matter jurisdiction to adjudicate claims against these latter defendants.

### \*857 ii. Absolute Immunity

[72] Defendants Wyant and Wurfel claim absolute immunity from plaintiffs' lawsuit. They rely on the immunity awarded to federal officials carrying out discretionary prosecutorial actions, arguing that they were functionally acting as federal officials despite working for a state agency. (Dkt. 281 at 38–40; Dkt. 282 at 33–34.) However, the Sixth Circuit rejected this argument in *Mays v. City of Flint*, 871 F.3d 437, 444–47 (6th Cir. 2017), *cert. denied*, — U.S. —, 138 S.Ct. 1557, 200 L.Ed.2d 743 (2018). Moreover, defendants do not explain how their claim of immunity interacts with plaintiffs' allegations against them, instead speculating that absolute immunity would apply if “[p]laintiffs' claims ... ultimately prove to be an alleged failure ... to sufficiently enforce the [Safe Drinking Water Act] and/or initiate enforcement proceedings against Flint.” (Dkt. 281 at 39; Dkt. 282 at 34.) For both reasons, defendants' claims are denied.

### iii. Preemption

Several defendants argue that plaintiffs' § 1983 claims are preempted by the Safe Drinking Water Act, 42 U.S.C. §

300f–300j (2016). But this Court and the Sixth Circuit has rejected this argument. *Boler*, 865 F.3d at 409.

#### d. Main Analysis

##### i. Federal Claims

##### 1. Bodily Integrity

In Count I, plaintiffs allege that the government defendants violated their substantive due process right to bodily integrity under the Fourteenth Amendment. (Dkt. 620-3 at 172.) According to plaintiffs, they did this by acting with deliberate indifference to the risk of harm plaintiffs faced, creating and perpetuating their exposure to contaminated water. (*Id.* at 172–73.) Defendants move to dismiss. (Dkt. 294 at 3–10; Dkt. 282 at 17–23; Dkt. 281 at 17–28; Dkt. 279 at 37–43; Dkt. 277 at 18–22; Dkt. 276 at 20–23; Dkt. 273 at 32–42.)

As set forth earlier in this opinion, plaintiffs unknowingly drank and bathed in contaminated water, encroaching upon their right to bodily integrity. *See supra* Section II.d.ii.1. Therefore, to state a bodily integrity claim, plaintiffs must demonstrate that (1) the government defendants knew of facts from which they could infer a substantial risk of serious harm, (2) they did infer it, and (3) they nonetheless acted with indifference, demonstrating a callous disregard towards the rights of those affected. *See supra* Section II.d.ii.1.a.

The Court will address the allegations against each group of government defendants in turn. Because the right to bodily integrity is clearly established, defendants cannot rely on qualified immunity if plaintiffs state a valid claim against them. *See supra* Section II.d.ii.1.b.

##### a. Property Owners

[73] In its August 1, 2018 opinion and order, the Court stated that:

Numerous plaintiffs in this matter are not individuals, but instead businesses. Bodily integrity claims are premised on “the right of every individual to the possession and control of his own person, free from all restraint or interference of others, unless by clear and unquestionable authority of law.” The Court can find no case that extends the

fundamental right of bodily integrity to a business or business relationship, or to the property owned or used in a business's operations.

329 F.Supp.3d at 395 (citations omitted). For the same reason, plaintiffs Frances Gilcreast, EpcO Sales, LLC, and Angelo's Coney Island Palace, Inc. fail to state a bodily integrity claim.

##### \*858 b. State Defendants

The remaining plaintiffs allege that defendants Governor Snyder, Andrew Dillon, Nick Lyon, and Nancy Peeler violated their right to bodily integrity. For the following reasons, plaintiffs state a claim against defendants Governor Snyder and Dillon, but not against Lyon or Peeler.

\*

[74] Plaintiffs state a bodily integrity claim against Dillon. He allegedly knew that the Flint River had been rejected as a water source as recently as 2011, and that the FWTP would require substantial improvements to safely process the river's water. From this, it is reasonable to believe that Dillon was aware of the risks associated with using the Flint River as a water source. Yet despite this knowledge, Dillon helped to develop an interim plan that saw Flint transition to the Flint River. And importantly, he rejected a final bid from DWSD that could have obviated the need to use water from the Flint River until the FWTP had the capacity to treat it safely. This demonstrated an indifference to the risk of serious harm plaintiffs faced, made all the more inexplicable given that he knew DWSD presented the most cost effective mid-term option.<sup>18</sup>

[75] Conversely, plaintiffs do not state a bodily integrity claim against Lyon. It is reasonable to conclude that Lyon was aware of the risk of harm plaintiffs faced. As the crisis unfolded, he received materials showing an outbreak of [Legionnaires' disease](#) in Flint. He also received emails from senior government officials raising concerns about possible lead contamination in Flint's water. Moreover, he was surely aware that these incidents coincided with the transition to the Flint River. However, plaintiffs fail to show how Lyon was deliberately indifferent. It is true that he did not make the information he received public, nor did he alert other government departments. But he directed his team to investigate the reports and emails, which shows his concern. And plaintiffs do not plead that Lyon attempted to cover up what was happening. Therefore, without more, the

claim against Lyon does not rise to the level of deliberate indifference.<sup>19</sup>

Finally, plaintiffs' claim against Governor Snyder is successful for the reasons set forth in Section II.d.ii.1. On the other hand, plaintiffs' claim against Peeler fails because the complaint contains no factual allegations against her.

### c. MDEQ Defendants

Plaintiffs next allege that defendants Bradley Wurfel, Daniel Wyant, Liane Shekter-Smith, Adam Rosenthal, Stephen Busch, Patrick Cook, and Michael Prysby violated their right to bodily integrity. For \*859 the following reasons, plaintiffs state a bodily integrity claim against Wurfel, Shekter-Smith, Rosenthal, Busch, Cook, and Prysby. They do not state a claim against Wyant.

\*

[76] Plaintiffs state a bodily integrity claim against Wurfel. He knew of ample facts from which to infer that plaintiffs were facing a substantial risk of harm, and it is reasonable to conclude that he did infer it. For example, Wurfel knew about the outbreak of [Legionnaires' disease](#). And he was also well aware that something was wrong with Flint's water. Moreover, plaintiffs demonstrate that Wurfel acted with deliberate indifference. On several occasions as the crisis unfolded, he publicly denied that there was a problem with Flint's water. He appeared on radio and television to advise listeners that the water was safe to consume and bathe in, and he discredited others who suggested that lead was leaching into Flint's water. Such indifference showed a callous disregard for plaintiffs' right to bodily integrity.

[77] Plaintiffs also state a claim against Shekter-Smith, Rosenthal, Busch, Cook, and Prysby. It is reasonable to assume that they were aware of the substantial risk of harm plaintiffs faced. Before Flint's transition to the Flint River, Shekter-Smith and Busch knew of the risks associated with the Flint River. In addition, Busch, Rosenthal, and Prysby recognized that the FWTP was not ready to begin operations. After the transition, Rosenthal learned that the FWTP was not practicing corrosion control, and he and Shekter-Smith both knew that no legitimate lead and [copper](#) testing was occurring. Moreover, Busch, Shekter-Smith, and Prysby also knew that the transition had created the conditions for legionella bacteria to flourish. Not to mention the fact that the EPA and civic

leaders were raising concerns about the quality of Flint's water.

Yet despite knowing of these serious risks, these defendants were indifferent to them. Shekter-Smith ensured that Flint received the ACO that allowed it to transition to the Flint River; Cook signed the final permit necessary for the FWTP to begin operations; and Busch resolved the regulatory hurdles associated with Flint's use of the Flint River. Furthermore, these defendants took steps to deceive Flint's residents into continuing to drink and bathe in the contaminated water. Busch and Cook misled the EPA by falsely suggesting that the proper corrosion control was in use at the FWTP;<sup>20</sup> and Busch, Rosenthal, and Prysby directly or indirectly altered reports to remove results showing high lead concentrations in Flint's water. These actions exhibited a callous disregard for plaintiffs' right to bodily integrity.

[78] In contrast, plaintiffs do not state a claim against Wyant because the allegations do not demonstrate deliberate indifference. Wyant was likely aware of the health risks posed by using the Flint River as a water source. There is also some indication that he knew the FWTP was not utilizing the proper corrosion control techniques and that Flint's water was contaminated. However, the fourth amended complaint contains nothing to suggest that Wyant either publicly denied there was a problem with Flint's water, or that he otherwise encouraged Flint residents to use the contaminated water. Plaintiffs \*860 therefore do not plead that Wyant was deliberately indifferent.

### d. City Defendants and Defendant Wright

Finally, plaintiffs allege that defendants Darnell Earley, Gerald Ambrose, Howard Croft, Daugherty Johnson, Michael Glasgow, Jeffrey Wright, Edward Kurtz, and Dayne Walling violated their right to bodily integrity.<sup>21</sup> For the following reasons, plaintiffs state a claim against Earley, Ambrose, Croft, Johnson, and Glasgow. They fail to state a claim against Wright, Kurtz, and Walling.

\*

[79] Plaintiffs state a bodily integrity claim against Earley and Ambrose. It is reasonable to infer that Earley and Ambrose were aware of the substantial risk of harm plaintiffs faced. After Flint transitioned to the Flint River, they knew about the outbreak of [Legionnaires' disease](#); General Motors

stopped using Flint water at its Flint factory because of its corrosive nature; and test results revealed high lead levels in two locations on the University of Michigan-Flint's campus. There were even growing calls from senior government officials that Flint “should try to get back on the Detroit system as a stopgap ASAP before this thing gets too far out of control.” (Dkt. 630-3 at 66.) Additionally, plaintiffs plead that Earley and Ambrose were indifferent to this risk. Earley publicly denied any connection between the [Legionnaires' disease](#) outbreak and Flint's water, despite knowing that other branches of government concluded that there was a link. And he repeatedly refused to consider returning to DWSD water. Having replaced Earley as the Emergency Manager, Ambrose also refused to return to DWSD. He even went so far as rejecting a Flint City Council vote to reconnect to DWSD. In both cases, Earley and Ambrose's conduct thus showed a callous disregard for plaintiffs' right to bodily integrity.

[80] Similarly, plaintiffs state a claim against Croft, Glasgow, and Johnson. As with Early and Ambrose, it is reasonable to conclude that these defendants were aware of the substantial risk of harm facing plaintiffs. As the transition to the Flint River loomed, all three knew that the FWTP was not ready to process the raw water. And Croft, in particular, was aware of the lead and [Legionnaires' disease](#) issues that followed the transition. Glasgow tested for and found high concentrations of lead in the water. He also recognized that Flint was not using corrosion control treatment and had no legitimate lead and [copper](#) testing in place. Moreover, these defendants acted with a callous disregard for plaintiffs' right to bodily integrity. Despite knowing that the FWTP was not ready to process the Flint River water, Croft and Johnson pressured Glasgow to give the green light to the transition. Johnson later blocked the Genesee County Health Department from scrutinizing Flint's water testing process. And Glasgow altered reports to hide high lead concentrations in Flint's water. Croft, Glasgow, and Johnson were thus deliberately indifferent by deceiving plaintiffs into thinking that there was no problem with Flint's water.

\*861 [81] In contrast, plaintiffs fail to state a claim against Wright because they do not show how he either caused or prolonged their exposure to the contaminated water. First, plaintiffs do not plausibly allege that Wright caused their exposure because he had no oversight over Flint's transition to the Flint River. Plaintiffs argue that Flint and Genesee County's water systems were unified, suggesting that Wright's position as Genesee County's Drain Commissioner gave him the means to affect the choice of Flint's water. (*Id.* at 53–

54 (“[B]ecause of the joint operation of the combined water systems, each of [the defendants] played a role in the decision to provide ... Flint ... with the high risk water[.]”).) But the fourth amended complaint reveals that the arrangement between Flint and Genesee County was a standard contractual relationship. Those in charge of Flint's system purchased water and then sold it to Genesee County. And although Genesee County was required to buy it, the County had no say in where it came from. (*Id.* at 39–40 (“[GCDC] agreed to ‘accept water as delivered from the water system of the City [of Flint.]’ ”).) In other words, Wright was in charge of Genesee County's water system, but not Flint's. (*Id.* at 53 (“Wright was in control of the County side of the jointly operated water systems[.]”).)

Second, Wright did not prolong plaintiffs' exposure to the contaminated water. Plaintiffs do not plead that Wright took steps to deceive Flint residents about the safety of Flint's water following the transition, or that he otherwise played a role in any coverup. Although Wright may have been aware of the risk of harm plaintiffs faced, he did not cause their injuries.<sup>22</sup>

[82] The same goes for Kurtz and Walling. Here too, plaintiffs fail to state a claim against these defendants because they do not show how they caused or prolonged plaintiffs' exposure to the contaminated water. Although Kurtz may have set in motion the chain of events that led to the transition to the Flint River, he resigned as Flint's Emergency Manager before the transition and therefore lacked control over the final decision. Additionally, Walling was involved in the decision to use the Flint River as an interim source of water but he was stripped of virtually all authority over Flint's operations during emergency management. Plaintiffs also do not allege that either of these defendants deceived plaintiffs about the safety of Flint's water or that defendants helped coverup the crisis. Thus, plaintiffs have failed to state a claim against these defendants.<sup>23</sup>

In summary, plaintiffs state a claim against Earley, Ambrose, Croft, Glasgow, and Johnson. Plaintiffs do not state a claim against Wright, Kurtz, and Walling.

## 2. Equal Protection

In Counts II and III, the African American plaintiffs allege that defendants Snyder, Dillon, Wright, Ambrose, Kurtz, Earley, Wyant, Shekter-Smith, Prysby, and Busch violated their right to equal protection under the Fourteenth

Amendment. For the reasons set forth above, plaintiffs fail to state a claim. *See supra* Section II.d.ii.2.

### \*862 3. Conspiracy

In Count IV, plaintiffs bring suit under § 1985(3) alleging that defendants Snyder, Dillon, Wright, Ambrose, Kurtz, and Earley conspired to violate their rights. For the reasons set forth above, plaintiffs fail to state a claim. *See supra* Section II.d.ii.4.

### 4. State-Created Danger

In Count IX, plaintiffs allege that the government defendants violated their right to be free from a state-created danger. (Dkt. 620-3 at 208.) Plaintiffs plead that the government defendants created the conditions that led to the Flint Water Crisis and then attempted to cover up the resulting risk of harm. (*Id.*) In their view, because the government defendants knew or should have known of the danger they created, they violated the Due Process Clause of the Fourteenth Amendment. (*Id.* at 208–09.) Defendants move to dismiss. (Dkt. 294 at 13–16; Dkt. 282 at 23–25; Dkt. 281 at 28–30; Dkt. 279 at 35–37; Dkt. 277 at 18–22; Dkt. 276 at 19–20; Dkt. 273 at 24–32.)

[83] [84] In its vacated August 1, 2018 opinion and order, the Court granted defendants' motions to dismiss an identical count. Nothing in the fourth amended complaint affects the Court's earlier analysis. Moreover, plaintiffs have not subsequently challenged this part of the August 1 ruling.<sup>24</sup> There, the Court stated that:

To bring a state[-]created danger claim, the individual must show: (1) an affirmative act by the state which either created or increased the risk that the plaintiff would be exposed to an act of violence by a third party; (2) a special danger to the plaintiff wherein the state's actions placed the plaintiff specifically at risk, as distinguished from a risk that affects the public at large; and (3) the state knew or should have known that its actions specifically endangered the plaintiff.

329 F.Supp.3d at 392 (quoting *Jones v. Reynolds*, 438 F.3d 685, 690 (6th Cir. 2006)). With respect to (1), the Court explained that:

Plaintiffs do not allege that any defendant created or increased the risk that they would be exposed to an act of

violence by a third party. They argue, however, that they do not need to, based on *Schneider v. Franklin Cty.*, 288 F. App'x. 247 (6th Cir. 2008). In that case, the Sixth Circuit analyzed a state-created danger claim without referencing the third-party requirement of the test. *Id.* at 252.

However, it is clear that *Schneider* applied an incomplete version of this circuit's test for a state-created danger claim. The *Schneider* court cited *Kallstrom v. City of Columbus*, 136 F.3d 1055, 1066 (6th Cir. 1998) in setting forth the state-created danger standard. In doing so, however, the *Schneider* court omitted *Kallstrom*'s reference to the threat of violence by a private third party, and then proceeded to analyze the claim without that requirement.

In support of the argument that the *Schneider* standard is good law in the Sixth Circuit, plaintiffs cite *Stiles ex rel. D.S. v. Grainger Cty.*, 819 F.3d 834 (6th Cir. 2016) and *McQueen v. Beecher Cmty. Sch.*, 433 F.3d 460 (6th Cir. 2006), two cases that also analyzed state-created danger claims.

*Stiles* involved a state-created danger claim arising from the brutal emotional, psychological, and physical bullying of a \*863 junior high school student by other students. *Id.* at 840–46. The *Stiles* court stated:

As a general rule, the State has no obligation to protect the life, liberty, of property of its citizens against invasion by private actors. Two exceptions to this rule exist: 1) where the State enters into a “special relationship” with an individual by taking that person into its custody, and 2) where the State creates or increases the risk of harm to an individual. Because DS was harmed by students rather than school or government officials, there is no constitutional violation unless one of these two exceptions applies.

*Id.* at 853 (citing *DeShaney v. Winnebago Cty. Dep't of Soc. Servs.*, 489 U.S. 189, 109 S.Ct. 998, 103 L.Ed.2d 249 (1989)) (internal citations omitted) (emphasis added). The court then cited *McQueen, supra*, for the legal standard for a state-created danger claim. *Id.* at 854. The standard set forth was: “(1) an affirmative act that creates or increases the risk to the plaintiff, (2) a special danger to the plaintiff as distinguished from the public at large, and (3) the requisite degree of state culpability.” *Id.* at 854 (citing *McQueen*, 433 F.3d at 464).

In *McQueen*, the Sixth Circuit considered whether a grant of summary judgment on a state-created danger claim was

proper where a first-grader shot and killed his classmate, and the deceased child's parent sued the teacher, principal, and school district. *McQueen*, 433 F.3d at 462–63. The plaintiff brought a variety of claims, among them a state-created danger claim for failing to protect her daughter from her classmate. *Id.* at 463.

Quoting *Kallstrom*, the *McQueen* court stated that “[l]iability under the state-created danger theory is predicated upon affirmative acts by the state which either create or increase the risk that an individual will be exposed to private acts of violence.” *Id.* at 464 (quoting *Kallstrom*, 136 F.3d at 1066). The court also noted that a state-created danger claim is traditionally rejected where the act “did not create or increase the risk of private violence to the plaintiff.” *Id.* at 465 (collecting cases).

In most other circuits, the third-party requirement is also consistently applied. See *Rivera v. Rhode Island*, 402 F.3d 27, 34–35 (1st Cir. 2005); *Lombardi v. Whitman*, 485 F.3d 73, 80 (2d Cir. 2007); *Pinder v. Johnson*, 54 F.3d 1169, 1175 (4th Cir. 1995); *Doe ex rel. Magee v. Covington Cty. Sch. Dist. ex rel. Keys*, 675 F.3d 849, 857 (5th Cir. 2012); *Fields v. Abbott*, 652 F.3d 886, 889 (8th Cir. 2011); *Gray v. Univ. of Colo. Hosp. Auth.*, 672 F.3d 909, 917 (10th Cir. 2012); *Perez-Guerrero v. U.S. Atty. Gen.*, 717 F.3d 1224, 1233–34 (11th Cir. 2013); *Butera v. Dist. Of Columbia*, 235 F.3d 637, 651 (D.C. Cir. 2001); but see *Doe v. Village of Arlington Heights*, 782 F.3d 911, 916–17 (7th Cir. 2015) (omitting third-party requirement).

In *Kneipp v. Tedder*, 95 F.3d 1199, 1204–11 (3d Cir. 1996) the Third Circuit analyzed the third-party requirement for a state-created danger claim and declined to apply it to the claim in front of it, instead opting to apply a standard requiring only that an individual be placed in danger. However, the Third Circuit has inconsistently applied the third-party requirement to state-created danger claims since *Kneipp*. See, e.g., *LaGuardia v. Ross Twp.*, 705 F. App'x. 130, 133 (3d Cir. 2017) (applying the requirement); but see *Henry v. City of Erie* 728 F.3d 275 (3d Cir. 2013) (omitting the requirement).

**\*864** Because all events related to plaintiffs' claims occurred in Michigan, the Court must apply the clearly established state-created danger test set forth in *Kallstrom*, *McQueen*, *Stiles*, and *Jones*. The complaint does not plead that any act taken by any state actor created or increased the risk of private violence to the plaintiffs.

At oral argument, plaintiffs' counsel argued that the third-party requirement could be satisfied by, for instance, a situation where a mother fed her child formula mixed with tainted Flint water. The mother would be the private actor, and the child would be the individual harmed under the state-created danger theory. (Dkt. 532 at 212.)

The Court rejects this theory in its entirety. The residents of Flint were all made to use contaminated water that leached lead and bacteria from old lines. Parents, many of them struggling to even pay for the water the city provided, whether from the DWSD or the Flint River, used what resources they had available to them. For much of the time the Flint River was used as Flint's primary water source, residents did not and could not have known the danger the water posed to them or their families. To entertain plaintiffs' counsel's theory of harm, the Court would have to find that a loving parent, seeking only to provide their child with food or water, committed an intentional or at least negligent act of violence against his or her own child. According to counsel, every person who showered or washed their hands or made coffee or boiled pasta with bacteria-infected, lead-tainted water provided to them by their government committed repeated acts of violence against themselves, their families, their friends, and their guests. This is not what the state-created danger theory was developed to address.

Plaintiffs have failed to plead that the actions of the governmental actors named in this claim created or increased the risk of harm from a third party, and for this reason, this particular claim must be dismissed.

*Id.* at 392–94. And with respect to (2), the Court stated that:

Even if the Court could determine that the third-party harm requirement of plaintiffs' state-created danger claim had been met, such a claim will stand only where “the government could have specified whom it was putting at risk, nearly to the point of naming the possible victim or victims.” *Reynolds*, 438 F.3d at 696. The state-created danger must be a “special danger” to a “discrete class of individuals.” *Schroder v. City of Fort Thomas*, 412 F.3d 724, 729 (6th Cir. 2005). It is not sufficient for the purposes of this claim if the specific danger is “no more a danger to [the plaintiff] than to any other citizen on the City streets.” *Jones v. City of Carlisle*, 3 F.3d 945, 949–50 (6th Cir. 1993). The danger may not be one that “affects the public at large.” *Kallstrom*, 136 F.3d at 1066.

Plaintiffs argue that the entire population of Flint constitutes a discrete class of individuals. (Dkt. 379 at 82–84.) They argue that the “government could have specified whom it was putting at risk, nearly to the point of naming the possible victim or victims,” *Reynolds*, 438 F.3d at 696, because “identifying those at risk would have been as simple as looking up the names and addresses of residents and businesses serviced by Flint's water.” (Dkt. 379 at 83.)

The Sixth Circuit has routinely held that threats to any person on the street or to the public at large do not constitute risks that are specific enough for the purposes of a state-created danger \*865 claim. See, e.g., *City of Carlisle*, 3 F.3d at 950 (the city permitting an epileptic individual to maintain a driver's license posed a danger to any citizen on the streets); *Janan v. Trammell*, 785 F.2d 557, 560 (6th Cir. 1986) (a parolee's release endangered plaintiff as a member of the public at large); *Schroder*, 412 F.3d at 729 (government's creation of a street, and management of traffic conditions, posed a general risk to the public).

The largest groups the Sixth Circuit has determined were able to pursue a state-created danger claim were in *Kallstrom*, where a city's release of private information from the personnel files of three undercover officers “placed the personal safety of the officers and their family members, as distinguished from the public at large, in serious jeopardy,” *id.*; 136 F.3d at 1067, and in *McQueen*, where the risk of a shooter in a school posed a risk to the five students in the room with him and even those in the school building, but all those outside the school building constituted “the general public.” *Id.*; 433 F.3d at 468.

An entire city, plus all those who visit, work, or pass through that city is, by definition, “the general public.” Plaintiffs set the bar for the general public at “the general public of Michigan residents.” (Dkt. 379 at 84.) However, there is no case that supports this definition.

This claim must also be dismissed for failure to satisfy this element of the state-created danger test.

*Id.* at 394–95. The Court adopts this reasoning in full. Defendants motions to dismiss the state-created danger Count are therefore granted.

## 5. *Monell* Liability

In Count VI, plaintiffs plead a standalone claim against the City of Flint under *Monell v. Department of Social Services of the City of New York*, 436 U.S. 658, 98 S.Ct. 2018, 56 L.Ed.2d 611 (1978). Plaintiffs allege that Flint is responsible for the unconstitutional conduct of its employees because they committed unlawful acts pursuant to Flint's custom or policy. As set forth above, plaintiffs state a claim that the city defendants Earley, Ambrose, Croft, Glasgow, and Johnson violated their right to bodily integrity. See *supra* Section III.d.i.1.d. And their *Monell* claim can rely on these underlying constitutional violations. See *Robertson v. Lucas*, 753 F.3d 606, 622 (6th Cir. 2014). Flint moves to dismiss. (Dkt. 276 at 34–35.)

[85] Under *Monell*, a plaintiff can bring a § 1983 claim against a city for the unconstitutional conduct of its employees if the employees' conduct implemented an unofficial custom, or “a policy statement, ordinance, regulation, or decision officially adopted and promulgated by that body's officers.” 436 U.S. at 690, 98 S.Ct. 2018. In its vacated August 1, 2018 opinion and order, this Court held that the state-appointed emergency managers were final decisionmakers for Flint with respect to the decision to provide residents with contaminated water. 329 F.Supp.3d at 421–22. As such, their actions represented official policy and Flint could be held liable for their conduct insofar as it violated plaintiffs' rights. (*Id.* at 422.)

[86] Following the Court's August 1 ruling, Flint challenged the decision and requested that the Court certify the issue for interlocutory appeal. (Dkt. 565.) The Court denied that request, explaining that “well-established precedent” controlled its decision. (Dkt. 659.) And the Court now adopts the reasoning from its August 1, 2018 opinion and order and its subsequent order denying Flint's request to certify the question of *Monell* liability for interlocutory appeal. Because the emergency managers were state officials whose edicts or \*866 acts may fairly be said to represent official city policy, plaintiffs have stated a *Monell* claim with respect to the alleged bodily integrity violations. But plaintiffs fail to state a *Monell* claim for any other type of constitutional violation.

## ii. State Claims

### 1. ELCRA

In Count V, plaintiffs allege that defendants Snyder, Dillon, Wright, Ambrose, Kurtz, Earley, the City of Flint,

Wyant, Shekter-Smith, Prysby, and Busch violated the rights guaranteed them under Michigan's ELCRA. For the reasons set forth above, plaintiffs fail to state an ELCRA claim. *See supra* Section II.d.ii.3.

## 2. Professional Negligence

In Counts VII and VIII, plaintiffs allege that defendants LAN and Veolia committed professional negligence. (Dkt. 620-3 at 199–207.) Neither LAN nor Veolia have moved to dismiss these counts in their entirety. However, Veolia asks the Court to dismiss the claims of several named plaintiffs based on alleged pleading deficiencies. (Dkt. 274 at 26–35.) This includes Rhonda Kelso, individually and on behalf of her minor child; David Munoz; Amber Brown, on behalf of her minor child; Frances Gilcreast; EPCO Sales, LLC; and, Angelo's Coney Island Palace, Inc. (*Id.*)

In its vacated August 1, 2018 opinion and order, the Court rejected all but one of Veolia's requested dismissals. [329 F.Supp.3d at 424–25](#). The third amended complaint alleged that Veolia became involved in the Flint Water Crisis in February 2015. However, Kelso stated only that they “bathed, washed, and cooked with the water until at least January 2015.” (Dkt. 349 at 12–13.) Because other plaintiffs specifically pleaded that they used Flint River water for the entire time or did not otherwise mention a limited period of use, the Court inferred that Kelso and her minor child did not use the water after February 2015 and dismissed their claims against Veolia. [329 F.Supp.3d at 424](#).

[87] In the fourth amended complaint, Kelso revises her allegations. She and her daughter now allege that they “bathed, washed, and cooked with the water until at least November 2015.” (Dkt. 620-3 at 14 (emphasis added).) As a result, Veolia's motion to dismiss as to Kelso must be denied. And since the fourth amended complaint makes no changes with respect to the other plaintiffs, the Court adopts the remainder of its August 1 decision.

## 3. Fraud

In Count X, plaintiffs allege that defendant Veolia committed fraud by intentionally making false representations about the safety of Flint's water. (*Id.* at 210–12.) Veolia moves to dismiss, arguing that plaintiffs' complaint fails to identify any false representations (Dkt. 274 at 17–19), plaintiffs do not

adequately plead intent (*id.* at 19–20), and plaintiffs fail to explain how they detrimentally relied on any falsity. (*Id.* at 20–21.)

In its vacated August 1, 2018 opinion and order, the Court granted defendants' motions to dismiss an identical count. As with plaintiffs' state-created danger claim, nothing in the fourth amended complaint affects the Court's earlier analysis. Moreover, plaintiffs have not subsequently challenged this aspect of the August 1 ruling.<sup>25</sup>

[88] [89] [90] There, the Court explained that:

Unlike other claims at the motion to dismiss stage, fraud claims are subject to a higher pleading standard. The elements of fraud must be pleaded with \*867 particularity, except that malice, intent, knowledge, and other conditions of a person's mind may be alleged generally. *Fed. R. Civ. P. 9(b)*.

In Michigan, a claim for common law fraud requires a plaintiff to plead:

- (1) That defendant made a material representation; (2) that it was false; (3) that when he made it he knew that it was false, or made it recklessly, without any knowledge of its truth, and as a positive assertion; (4) that he made it with the intention that it should be acted upon by plaintiff; (5) that plaintiff acted in reliance upon it; and (6) that he thereby suffered injury.

*Hi-Way Motor Co. v. Intl. Harvester Co.*, 398 Mich. 330, 336, 247 N.W.2d 813 (1976) (internal citations and quotation marks omitted).

All plaintiffs allege that Veolia defrauded them, based on three statements in Veolia's 2015 Interim Report. Those statements are: 1) that Flint's water was “safe” and “in compliance with drinking water standards”; 2) that the observed discoloration was merely aesthetic and not indicative of water quality or health problems; and 3) that medical problems arose in Flint because “[s]ome people may be sensitive to any water.” (Dkt. 349 at 146.) Plaintiffs' complaint references no other specific statements made by Veolia, so these are the only three statements the Court may consider in evaluating the sufficiency of their pleadings. *See Republic Bank & Trust Co. v. Bear Stearns & Co.*, 683 F.3d 239, 247 (6th Cir. 2012) (holding that fraudulent statements must be specifically alleged, including the time and place the statements were made).

Veolia raises several arguments regarding the sufficiency of the fraud claim. Chief among those arguments are that the specific statements set forth above are inaccurately quoted, that plaintiffs fail to plead Veolia's intent and knowledge properly, and that plaintiffs fail to plead their own reliance properly.

At Veolia's request, the Court has reviewed the quoted statements in their full context in Veolia's 2015 Interim Report. The Court may review documents incorporated into the complaint by reference, such as documents relied on for the specific statements supporting a fraud claim. *Tellabs, Inc. v. Makor Issues & Rights, Ltd.*, 551 U.S. 308, 322, 127 S.Ct. 2499, 168 L.Ed.2d 179 (2007). The statements Veolia challenges are, for the purposes of the assertion of a fraud claim at the motion to dismiss stage, accurate and properly pleaded.

Further, the entirety of the pleading sets forth the basis for the plaintiffs' claim that these statements were false. The complaint alleges the tainted water in Flint in February 2015 was not safe, in compliance with drinking water standards, or merely aesthetically displeasing; that the tainted water did specifically cause medical problems apart from the standard problems that might be associated with a population's sensitivity to a clean water supply; and that this information was generally known absent Veolia's representations to the contrary.

Plaintiffs allege that “[u]pon information and belief, the Veolia Defendants knew the representations were made recklessly without any knowledge about their veracity” and that the representations were made “with the intention that Plaintiffs would act and rely on them.” (Dkt. 349 at 147.) Allegations of fraud “cannot be based upon information and belief, except where the relevant facts lie exclusively within knowledge and control of the opposing party, and even then, the plaintiff must plead a particular \*868 statement of facts upon which his belief is based.” *Craighead v. E.F. Hutton & Co.*, 899 F.2d 485, 489 (6th Cir. 1990).

At the motion to dismiss stage for a fraud claim, “the plaintiff ... must plead facts about the defendant's mental state, which, accepted as true, make the state-of-mind allegation plausible on its face.” *Republic Bank & Trust Co.*, 683 F.3d at 247 (internal quotation marks and citation omitted). Veolia argues that plaintiffs' allegation of recklessness is insufficiently pleaded. However, the allegation meets the fraud pleading standard. Information

about Veolia's recklessness in 2015 is solely within the knowledge of Veolia's decision-makers. On a theory of recklessness and ignorance about the veracity of a statement in a fraud claim, the plaintiffs' voluminous factual background about the information known to Flint, which retained Veolia, and to the public at large in 2015, satisfies this pleading requirement. To require further pleading regarding Veolia's alleged recklessness would be to ask the plaintiffs to plead facts they could not possibly know at this stage.

To sustain a fraud claim in Michigan, “the party claiming fraud must reasonably rely on the material representation.” *Zaremba Equip., Inc. v. Harco Nat'l Ins. Co.*, 280 Mich. App. 16, 39, 761 N.W.2d 151 (2008) (emphasis in original). As set forth above, this information cannot be pleaded based upon information and belief, because this information is solely within the knowledge of the plaintiffs, not the defendants.

Plaintiffs' complaint alleges only that “[u]pon information and belief, the Veolia Defendants made the representations with the intention that Plaintiffs would act and rely on them, which Plaintiffs did.” (Dkt. 349 at 147.) The phrase “which Plaintiffs did” is the sole factual allegation in the complaint stating that any plaintiff specifically relied on these statements in continuing to drink Flint River water after February 2015. That statement lacks any specificity as to when or how any plaintiff heard the allegedly fraudulent statements, which were set forth in a report purportedly on the city's website.

At oral argument, plaintiffs argued that specific reliance was pleaded with regard to at least one plaintiff: Tiantha Williams. The portion of the complaint introducing Williams states that “[p]rior to [December 2015], the family trusted previous reports that the condition of the water was not an immediate health emergency. They also relied on statements about the safety of the water that were made in public forums.” (*Id.* at 17.) These general allegations are insufficient to specifically plead that Williams heard and relied on Veolia's statements. Between April 2014 and December 2015, numerous parties, including Earley in October 2014, Busch and Shekter-Smith on March 13, 2015, and Wurfel on July 10 and July 24, 2015, issued allegedly false statements to the public. The general reference to “statements” may include any or all of these other false statements, and do not specifically implicate Veolia's 2015 Interim Report.

In the alternative, plaintiffs argue that reliance may be inferred because Veolia's report was available on Flint's public website. (Dkt. 379 at 123 n.47.) Plaintiffs failed to plead this information in the complaint, and the Court cannot rely on it to determine if reliance was properly pleaded. Even if the Court considers this additional information, the particular plaintiffs in this case do not plead that they specifically became aware of and relied on Veolia's statements in continuing \*869 to use Flint water after February 2015.

Plaintiffs argue that they need not show direct reliance on a fraudulent misrepresentation to assert a fraud claim, citing *Nernberg v. Pearce*, 35 F.3d 247 (6th Cir. 1994). In *Nernberg*, a plaintiff properly asserted a fraud claim where fraudulent misrepresentations were made to a third party, and the misrepresentations were repeated by that third party to induce the plaintiff's reliance. *Id.* at 251. However, the plaintiff still specifically demonstrated that he actually heard and relied on the misrepresentations. *Nernberg* does not mean that a plaintiff may allege a fraud claim where a third party heard and relied on a false statement, but the plaintiff does not allege with particularity that he or she also did so.

Finally, plaintiffs argue that questions as to reliance “pertain[s] to questions of fact, not sufficiency of the pleadings.” (Dkt. 379 at 124 (citing *State Farm Mut. Ins. Co. v. Elite Health Ctrs., Inc.*, No. 16-cv-13040, 2017 WL 2351744, at \*9, 2017 U.S. Dist. LEXIS 82736, at \*25 (E.D. Mich. May 31, 2017). *Elite Health* does not stand for the rule that a plaintiff does not need to plead reliance with particularity. That case did not concern a defendant's argument that the pleadings were insufficient as to reliance. Instead, the defendant in *Elite Health* argued that the plaintiff's allegations were “contradictory and/or self[-]serving,” and that contention pertained to questions of fact rather than sufficiency of the pleadings. *Id.* The *Elite Health* complaint contained a “116-page description of how the alleged scheme to defraud” worked, *id.* at \*8, 2017 U.S. Dist. LEXIS 82736, at \*23, and determined that the plaintiff had properly “alleged that it justifiably relied on Defendants' misrepresentations,” *id.* at \*9, 2017 U.S. Dist. LEXIS 82736, at \*26.

This analysis does not foreclose a fraud claim against Veolia by other plaintiffs. However, those plaintiffs *must* plead the necessary elements of their fraud claim with particularity, including their reliance on Veolia's allegedly

fraudulent statements. Because these plaintiffs did not plead the reliance element of their fraud claim with sufficient particularity, their fraud claim must be dismissed. 329 F.Supp.3d at 417–20. The Court adopts this reasoning in full. Therefore, plaintiffs fail to state a fraud claim.

#### 4. Negligent Infliction of Emotional Distress

[91] In Count XI, plaintiffs allege that LAN and Veolia negligently caused them emotional distress. (Dkt. 620-3 at 212.) In its vacated August 1, 2018 opinion and order, the Court addressed the identical claim and decided that it was actually “a request for emotional distress damages arising from the negligence claims asserted against LAN and Veolia.” 329 F.Supp.3d at 421. Nothing in the fourth amended complaint affects the Court's earlier analysis, and again plaintiffs have not subsequently challenged this aspect of the August 1 ruling. Specifically, the Court stated that:

Plaintiffs assert what they term a negligent infliction of emotional distress (“NIED”) claim against LAN and Veolia. (Dkt. 349 at 148.) The elements of a claim for [NIED] under Michigan law are:

- (1) serious injury threatened or inflicted on a person, not the plaintiff, of a nature to cause severe mental disturbance to the plaintiff,
- (2) shock by the plaintiff from witnessing the event that results in the plaintiff's actual \*870 physical harm,
- (3) close relationship between the plaintiff and the injured person (parent, child, husband, or wife), and
- (4) presence of the plaintiff at the location of the accident at the time the accident occurred or, if not presence, at least shock “fairly contemporaneous” with the accident.

*Hesse v. Ashland Oil, Inc.*, 466 Mich. 21, 34, 642 N.W.2d 330 (2002).

Plaintiffs argue that they are bringing a “direct negligent infliction of emotional distress” claim, different from the NIED claim set forth in *Hesse*. Plaintiffs rely on *Daley v. LaCroix*, 384 Mich. 4, 179 N.W.2d 390 (1970), to argue that NIED does not require an injury to a third party for a plaintiff to pursue the claim. In *Daley*, the Michigan Supreme Court considered whether a plaintiff alleging “a definite and objective physical injury [ ] produced as a result of emotional distress proximately caused by defendant's negligent conduct ... may recover in damages for such physical consequences to himself notwithstanding

the absence of any physical impact upon plaintiff at the time of the mental shock.” *Id.* at 12–13, 179 N.W.2d 390[.]

*Daley* did not create a cause of action for [NIED] absent an injury to a closely related third party. “[R]ather than create a cause of action, [*Daley* and cases following it] merely allow damages for emotional distress when the plaintiff has prevailed on a negligence cause of action.” *McNeil ex rel. McNeil v. Metinko*, Nos. 194595, 194596, 1998 WL 2016585, at \*3, 1998 Mich. App. LEXIS 2506, at \*7–8 (Mich. Ct. App. Mar. 13, 1998).

Plaintiffs rely on two other cases, *Apostle v. Booth Newspapers, Inc.*, 572 F.Supp. 897, 900 (W.D. Mich. 1983) and *Maldonado v. Nat. Acme Co.*, 73 F.3d 642, 645–46 (6th Cir. 1996), to argue that *Daley* did establish a tort of negligent infliction of emotional distress that did not require an injury to a third party. First, both cases predate *Hesse*, in which the Michigan Supreme Court limited the tort as set forth above. Second, *Hesse* explicitly stated that “[t]he common-law cause of action for negligent infliction of emotional distress has been recognized and applied in Michigan, although this Court has never ruled on the issue.” *Hesse*, 466 Mich. at 34, 642 N.W.2d 330. It is impossible to read *Daley*, a Michigan Supreme Court decision, to create a type of NIED claim when Michigan courts have stated that *Daley* did not do so, and when as of 2002, the Michigan Supreme Court had not recognized NIED at all.

Further, an NIED claim “clearly contemplates a sudden, brief, and inherently shocking accidental event which causes the injury ..., which contemporaneously, and by its very nature, results in emotional and physical injury to the plaintiff.” *Brennan v. Chippewa Cty. War Mem’l Hosp., Inc.*, Nos. 318452, 318594, 2014 WL 5306621, at \*9, 2014 Mich. App. LEXIS 1912 at \*25 (Oct. 16, 2014) (further citation omitted). Plaintiffs do not allege that either LAN or Veolia committed an act that was sudden or brief, but instead allege that over a period of months for Veolia and even years for LAN, these defendants repeatedly failed to properly evaluate or treat Flint’s water, resulting in prolonged injury to all who used Flint River water after April 25, 2014.

On review of plaintiff’s complaint, this claim is a request for emotional distress damages arising from the negligence claims asserted against LAN and Veolia. Because the claim is presented as one for NIED, it is dismissed on the grounds that it fails to plead an NIED claim under Michigan law.

However, this ruling does not preclude plaintiffs from \*871 seeking emotional distress damages arising from their surviving negligence claims.

*Id.* at 420–21. The Court adopts this reasoning in full. Plaintiffs may still pursue damages for emotional suffering, where permitted.

## 5. Negligence

In Count XII, plaintiffs allege that defendants LAN and Veolia were negligent with respect to their conduct in Flint, causing plaintiffs injury. (Dkt. 620-3 at 212–14.) LAN and Veolia move to dismiss on the ground that plaintiffs’ negligence claim is preempted by plaintiffs’ claim for professional negligence.

[92] In the context of the Flint Water Cases, the Court has twice held that negligence claims against LAN and Veolia may only be brought as professional negligence claims. First, in *Guertin*, the Court held that the professional negligence claims against LAN and Veolia could proceed, but the ordinary negligence claims had to be dismissed. 2017 WL 2418007, at \*30, 2017 U.S. Dist. LEXIS 85544, at \*90–93. Second, in its vacated August 1, 2018 opinion and order, the Court determined that since “[a]n ordinary layperson would have little to no knowledge about the appropriate methods and techniques for remediating, containing, and eliminating lead and bacteria in a municipal water supply,” the claim was properly brought as a professional negligence claim and it dismissed the ordinary negligence claim. 329 F.Supp.3d at 423–24.

What was true in *Guertin* and the Court’s August 1 decision is true here. Plaintiffs have thus failed to state a claim to ordinary negligence.

## 6. Gross Negligence

In Count XIII plaintiffs allege that defendants LAN and Veolia committed gross negligence. (Dkt. 620-3 at 215–17.) In Count IV, plaintiffs allege the same against defendants Snyder, Dillon, Lyon, Shekter-Smith, Rosenthal, Busch, Cook, Prysby, Wurfel, Wright, Kurtz, Earley, Ambrose, Croft, Johnson, and Glasgow. (*Id.* at 217–19.) For the reasons set forth above in Section II.d.ii.5, plaintiffs fail to state a claim with respect to Count IV. And for the following reasons, plaintiffs also fail to state a claim with respect to Count XIII.

\*

[93] Gross negligence is not an independent cause of action in Michigan. See *Xu v. Gay*, 257 Mich. App. 263, 268–69, 668 N.W.2d 166 (2003). At common law in Michigan, gross negligence was not a higher degree of negligence; it was a device to escape contributory negligence. *Gibbard v. Cursan*, 225 Mich. 311, 319, 196 N.W. 398 (1923), overruled by *Jennings v. Southwood*, 446 Mich. 125, 131–132, 521 N.W.2d 230 (1994), abrogated on other grounds. However, Michigan replaced the rule of contributory negligence with comparative negligence. *Placek v. Sterling Heights*, 405 Mich. 638, 650, 275 N.W.2d 511 (1979). And the Michigan Supreme Court therefore discarded the doctrine of common law gross negligence, recognizing that it had outlived its practical usefulness. *Jennings*, 446 Mich. at 129, 521 N.W.2d 230.

Gross negligence has since received a new life in the statutory context. See *supra* Section II.d.ii.5. The GTLA confers various degrees of immunity on tortious government actors. § 691.1407. This includes lower-level government officials, unless their conduct amounted to *gross negligence* that was the proximate cause of a plaintiff's injuries. § 691.1407(2).

[94] But despite its reference to gross negligence, neither the GTLA nor any other immunity statute created a new tort. \*872 See *Rakowski v. Sarb*, 269 Mich. App. 619, 627, 713 N.W.2d 787 (2006); *Beaudrie v. Henderson*, 465 Mich. 124, 139 n.12, 631 N.W.2d 308 (2001). Statutory gross negligence is instead an affirmative defense to be raised by a defendant. *Odom v. Wayne Cty.*, 482 Mich. 459, 479, 760 N.W.2d 217 (2008). And plaintiffs bringing a tort claim must still plead the common law elements of ordinary negligence. *Rakowski*, 269 Mich. App. at 627, 713 N.W.2d 787.<sup>26</sup>

With this in mind, although plaintiffs style their proposed claim as one of gross negligence, the Court must treat it as one of ordinary negligence. And for the reasons set forth in Section III.d.ii.5., plaintiffs' claims of ordinary negligence against LAN and Veolia must be brought as claims of professional negligence. Thus, plaintiffs fail to state a claim to gross negligence.

## 7. Exemplary Damages

Plaintiffs seek exemplary damages against defendants Veolia and LAN, solely in connection with their alleged professional negligence. (Dkt. 620-3 at 199–207, 219.) In response, Veolia and LAN move to dismiss the requested relief. (Dkt. 283 22–23; Dkt. 274 at 22–26.)<sup>27</sup> In *Guertin*, under similar circumstances, this Court stated that “plaintiffs may be entitled to exemplary damages.” 2017 WL 2418007, at \*30, 2017 U.S. Dist. LEXIS 85544, at \*94. And the Court therefore permitted “[t]heir request for exemplary damages [to] proceed.” *Id.* However, for the reasons stated below, *Guertin* was at odds with Michigan precedent. And because Michigan law controls the question of damages in counts involving professional negligence, plaintiffs fail to state a claim to exemplary damages.

\*

[95] [96] In Michigan, exemplary damages are a special class of compensatory damages. They are available under limited circumstances to reimburse for a non-economic harm. *Veselenak v. Smith*, 414 Mich. 567, 573–74, 327 N.W.2d 261 (1982); *Unibar Maint. Servs., Inc. v. Saigh*, 283 Mich. App. 609, 630, 769 N.W.2d 911 (2009). And in the context of exemplary damages, this only includes losses for the “humiliation, sense of outrage, and indignity” that results from malicious, willful, and wanton conduct. *Kewin v. Mass. Mut. Life Ins. Co.*, 409 Mich. 401, 419, 295 N.W.2d 50 (1980); *B & B Inv. Grp. v. Gitler*, 229 Mich. App. 1, 9–10, 581 N.W.2d 17 (1998).

[97] [98] [99] The malicious, willful, and wanton element is equivalent to malice. See *Peisner v. Detroit Free Press, Inc.*, 421 Mich. 125, 136, 364 N.W.2d 600 (1984). Because damages for mental pain and anxiety are normally included under actual damages, only intentional actions that show a reckless disregard for a plaintiff's rights will suffice. See *Veselenak*, 414 Mich. at 574–75, 327 N.W.2d 261; *McPeak v. McPeak*, 233 Mich. App. 483, 487–88, 593 N.W.2d 180 (1999). In other words, mere negligence is insufficient. A defendant's conduct must amount to more than \*873 a lack of care. See *Veselenak*, 414 Mich. at 574–75, 327 N.W.2d 261.

[100] [101] Here, the fact that professional negligence is the only claim plaintiffs raise to support exemplary damages against LAN and Veolia negates the mental element required for the award. It is the reprehensibility of a defendant's conduct that intensifies the emotional injury and justifies exemplary damages not the magnitude of the harm caused. See *McPeak*, 233 Mich. App. at 488, 593 N.W.2d 180; *Gitler*,

229 Mich. App. at 10, 581 N.W.2d 17. Plaintiffs do not state a claim for allegedly malicious, willful, and wanton conduct. In fact, they do not state a claim involving exemplary damages for any intentional tort. Rather, they argue that LAN and Veolia were professionally negligent and that their negligence caused the Flint Water Crisis. As such, plaintiffs fail to state a claim for exemplary damages.

### iii. LAN's Motion for a More Definite Statement

[102] [103] Defendant LAN argues that plaintiffs must provide a more definite statement in their complaint. (Dkt. 283 at 24.) The Court addressed this identical motion in its August 1, 2018 opinion and order, and sees no reason to deviate from this prior ruling. Accordingly, for the reasons set forth below, the Court denies LAN's motion.

Motions for more definite statements are disfavored, and should be granted “only if there is a major ambiguity or omission in the complaint that renders it unanswerable.” *Farah v. Martin*, 122 F.R.D. 24, 25 (E.D. Mich. 1988).

LAN argues that the complaint does not distinguish between the Leo A. Daly Company (“LAD”), Lockwood, Andrews & Newnam, P.C. (“LAN P.C.”), and Lockwood, Andrews & Newnam, Inc. (“LAN, Inc.”), making it impossible to tell what each entity did. The Court has addressed the relationship between LAD, LAN P.C., and LAN, Inc. in a prior opinion. (Dkt. 437.) LAD is the parent company of LAN, Inc.; LAN P.C. is a corporation established to satisfy licensing requirements for LAN, Inc. to operate in the state of Michigan. (Id. at 4.) An agreement between LAD and LAN, Inc. establishes a relationship between the two companies in which all LAN, Inc. employees are LAD employees and all LAN, Inc. revenues go to a joint bank account over which LAD had full control. (Id. at 10–11.)

Because LAN P.C. was a legal entity created solely to permit LAN, Inc. to perform work in Michigan, all work LAN, Inc. performed can be attributed to LAN P.C. Because all employees of LAN, Inc. were actually employees of LAD, all work those employees performed, including the work at issue in this case, can be attributed to LAD. The complaint treats all three companies as a single entity for pleading purposes because, based on the corporate structure of the companies, they are indistinguishable for the purposes of this lawsuit.

LAN also objects to being “lumped in” with Veolia with regard to some allegations. (Dkt. 283 at 26.) The complaint clearly specifies the actions LAN and Veolia each took with respect to Flint's water supply, and sometimes refers to them jointly because either both sets of defendants had similar duties, if at different times, or because plaintiffs are asserting similar claims against both sets of defendants.

329 F.Supp.3d at 391–92.

### e. Conclusion and Order

IT IS ORDERED THAT,

\*874 Plaintiffs' motion for leave to amend the complaint (Dkt. 620) regarding the bodily integrity claim against Governor Snyder is **GRANTED**. Plaintiffs may include the fourth amended complaint's new factual allegations, including the proposed class definitions. Plaintiffs' motion as it relates to these facts is therefore **GRANTED**. In all other respects, the motion is denied.

Having adopted the fourth amended complaint in part, IT IS FURTHER ORDERED THAT,

Defendants' motions to dismiss count I (bodily integrity) are **DENIED** with respect to defendants Snyder, Dillon, Wurfel, Shekter-Smith, Rosenthal, Busch, Cook, Prysby, Earley, Ambrose, Croft, Glasgow, and Johnson, with the exception of claims relating to property damage. Additionally, the city defendants' motion to dismiss count VI (*Monell* liability) is **DENIED**. LAN and Veolia's motions to dismiss counts VII and VIII (professional negligence) are **DENIED**. And LAN's motion for a more definite statement is also **DENIED**.

IT IS FURTHER ORDERED THAT,

All remaining counts are dismissed for failure to state a claim. Specifically, defendants' motions to dismiss count I (bodily integrity) with respect to defendants Lyon, Peeler, Wyant, Kurtz, Wright, and Walling are **GRANTED**. And defendants' motions to dismiss counts II, III, IV, V, IX, X, XI, XII, XIII, and XIV are also **GRANTED** in their entirety. Additionally, LAN and Veolia's motions to dismiss plaintiffs' claim for exemplary and punitive damages are **GRANTED**.

IT IS SO ORDERED.

## All Citations

384 F.Supp.3d 802

## Footnotes

- 1 Other cases were subsequently added to the consolidated docket. (Dkts. 185, 232, 441, 453.)
- 2 This includes *Walters v. Flint*, No. 17-cv-10164, and *Sirls v. Michigan*, No. 17-cv-10342. Neither of these cases are consolidated with the present case.
- 3 Some defendants challenged the Court's authority to do so, but the Sixth Circuit upheld this decision. *Carthan v. Snyder*, No. 18-1967 (6th Cir. Feb. 19, 2019).
- 4 Plaintiffs sue former Governor Snyder in his official and individual capacities. For the sake of consistency with earlier Flint water decisions, former Governor Snyder will simply be referred to as Governor Snyder or the Governor where the claim is against him in his individual capacity. Where the claim is against him in his official capacity, the count is now against Governor Gretchen Whitmer. See *Fed. R. Civ. P. 25(d)*. But, again, for consistency, the Court will still refer to Governor Snyder.
- 5 According to the fourth amended complaint, Flint's population is 54.3% African American. (Dkt. 620-3 at 152.)
- 6 On remand from the Sixth Circuit, *Boyer* was consolidated with the present case. (Dkt. 453.)
- 7 Where, as here, leave is sought following a dispositive ruling, the moving party must normally have the ruling set aside. *Morse v. McWhorter*, 290 F.3d 795, 799 (6th Cir. 2002). But in this case, the ruling has already been vacated. (Dkt. 670.)
- 8 In response, Governor Snyder points out that the new allegations do not indicate that he knew of the risks posed by the Flint River prior to the decision to switch water sources. (Dkt. 654 at 19.) And because, in his view, the right to bodily integrity only limits the State's power to take affirmative action, Governor Snyder argues that what he knew after the switch is irrelevant information. (*Id.* at 20.) Moreover, according to the Governor, the Constitution does not guarantee that the State will remedy local water contamination issues. (*Id.*)  
However, the Governor misunderstands the nature of plaintiffs' claim. Plaintiffs do not argue that Governor Snyder violated their right to bodily integrity by authorizing the switch to the Flint River. Nor do they assert that Governor Snyder should have remediated a polluted water source. Rather, plaintiffs contend that the Governor was indifferent to their rights by concealing the risk of harm posed by Flint's contaminated water.
- 9 This theory is identical to that stated in the third amended complaint, with the omission of Dayne Walling as a named defendant. However, the proposed complaint is somewhat inconsistent on this point. Walling is omitted as a named defendant but is still referred to in subsequent allegations. (Dkt. 620-3 at 174–86.) The Court assumes that plaintiffs intended to omit Walling.
- 10 Alternatively, plaintiffs can show that a law or policy explicitly classifies on the basis of race. *Hunt v. Cromartie*, 526 U.S. 541, 546, 119 S.Ct. 1545, 143 L.Ed.2d 731 (1999). But plaintiffs agree that this case involves a facially neutral policy. (Dkt. 620-1 at 18, 19; Dkt. 663 at 37.)
- 11 The Sixth Circuit has repeatedly relied on the *Arlington Heights* factors when addressing equal protection claims involving facially neutral laws or policies. *E.g.*, *Ne. Ohio Coal. for the Homeless*, 837 F.3d at 636–37 (6th Cir. 2016); *Smith & Lee Assocs., Inc. v. City of Taylor*, 102 F.3d 781, 790–91 (6th Cir. 1996).
- 12 In fact, DWSD was at the center of highly publicized public corruption charges. In March of 2013, former Mayor of Detroit, Kwame Kilpatrick was found guilty of twenty-four of thirty counts brought against him, including conspiracy charges related to the DWSD. *United States v. Kilpatrick*, et al., No. 10-cr-20403. Former DWSD Director Victor Mercado had previously pled guilty in November of 2012.
- 13 Plaintiffs detail the Governor's response to emergencies in majority white communities. (Dkt. 620-3 at 152–53.) However, this only tells half the story. Without knowing how the Governor reacted to emergencies in majority African American communities, it is difficult to assess his motive in Flint. The flood in Wayne County is the closest non majority white jurisdiction that plaintiffs provide.
- 14 As with the proposed equal protection and ELCRA claims, plaintiffs inconsistently name defendants in the Count. The conspiracy Count lists the defendants mentioned here, but subsequent briefing includes additional individuals.
- 15 There are two ways to construe plaintiffs' § 1985(3) argument. First, it could be understood as stating a claim based on the theory that defendants' actions burdened plaintiffs' rights under the Thirteenth Amendment, denying them equal protection of the laws. Or it could be read as articulating a violation of the Equal Protection Clause on the basis of racial

discrimination, where defendants acted with racial animus similar to a badge of slavery. Because in plaintiffs' briefing they argue in support of the latter interpretation, the Court will analyze the claim under that theory.

- 16 In pertinent part, § 1985(3) states: "If two or more persons ... conspire ... for the purpose of depriving, either directly or indirectly, any person or class of persons of the equal protection of the laws ... [and] do, or cause to be done, any act in furtherance of the object of such conspiracy, whereby another is injured in his person or property ... the party so injured or deprived may have an action for the recovery of damages occasioned by such injury or deprivation, against any one or more of the conspirators."
- 17 Another exception is "when the state has waived immunity by consenting to the suit." *Boyer*, 865 F.3d at 410. However, this exception is not at issue here. Although the State of Michigan has participated in the proceedings to some extent, it has carefully and consistently appeared reluctantly, and without waiving immunity.
- 18 In his motion to reconsider the now vacated August 1, 2018 opinion and order, Dillon argued that he resigned his position as State Treasurer in 2013, before Flint transitioned to the Flint River in early 2014. In his view, he therefore lacked authority over the Flint water system and could not have caused the harm that resulted. (Dkt. 561 at 16–17.) If true, the Court concedes that plaintiffs' claim against Dillon may ultimately fail. However, plaintiffs dispute whether he truly left the State's employment. (Dkt. 601 at 9–12.) Discovery will resolve this disagreement. For now, the Court must read the allegations in a light favorable to plaintiffs.
- 19 In its vacated August 1, 2018 opinion and order, the Court denied defendant Lyon's motion to dismiss this count. 329 F.Supp.3d at 403–04. In supplemental briefing, the state defendants argue that the Sixth Circuit's decision in *Guertin* precludes the same result here. (Dkt. 739 at 15–16.) The Court agrees, as now reflected in this decision. Plaintiffs argue that the factual allegations against Lyon in this case are more substantial than in *Guertin*, but they are not so substantial as to warrant a different outcome.
- 20 Defendant Cook argues that he never misled the EPA. (Dkt. 735 at 24.) According to Cook, he informed the EPA that there was no corrosion control as soon as he was asked by the Agency. (*Id.*) That may be so, but the Court must take plaintiffs' allegations as true at this stage in the litigation.
- 21 Because it is important to the following analysis, the Court again notes that Kurtz, Early, and Ambrose were all at one time Flint's Emergency Manager. Edward Kurtz was the Emergency Manager from August 2012 through July 2013, Darnell Early from September 2013 until January 13, 2015, and Ambrose from January 13, 2015 through April 28, 2015. Ambrose was also Flint's Finance Director before he became Emergency Manager.
- 22 In its vacated August 1, 2018 opinion and order, the Court denied defendant Wrights' motion to dismiss plaintiffs' bodily integrity claim. 329 F.Supp.3d at 407. Having reviewed that analysis, the Court reverses its earlier decision.
- 23 Defendant Kurtz did not file a motion to dismiss prior to the Court's vacated August 1, 2018 opinion and order. However, the city defendants' supplemental brief argues that he should be dismissed from the case because plaintiffs have failed to state a claim against him. (Dkt. 738 at 11–12). The Court treats these arguments as Kurtz's motion to dismiss.
- 24 Plaintiffs have not waived the right to appeal this issue. In their original response to defendants' motions to dismiss, plaintiffs argued at length that their state-created danger claim should proceed. (Dkt. 379 at 78–86.)
- 25 Again, plaintiffs have not waived the right to appeal this issue.
- 26 Perhaps because a plaintiff must sometimes show gross negligence to overcome immunity, courts have permitted claims styled as gross negligence to go forward under ordinary tort principles. See, e.g., *Holland v. City of Highland Park*, No. 324312, 2016 WL 1072194, 2016 Mich. App. LEXIS 555 (Mar. 17, 2016); *FOLTS v. CIGNA Ins. Co.*, No. 210163, 1999 WL 33438012, at \*1, 1999 Mich. App. LEXIS 740 at \*1 (Aug. 6, 1999).
- 27 The fourth amended complaint also seeks punitive damages. However, plaintiffs' claims against Veolia and LAN sound in Michigan law, and plaintiffs concede that Michigan law prevents them from seeking such relief in this case. (Dkt. 379 at 136 n.54.) As such, plaintiffs fail to state a claim to punitive damages.

# M T A H O E MOUNTAIN NEWS

Serving Lake Tahoe's South Shore Since 1994

Vol. 27, No. 7 • January 2021



Photo by Taylor Flynn

South Lake Tahoe native Monica Eisenstecken has filed a lawsuit against TRPA and others over cell towers.

## Standing Tall

In this month's Cover Story, we look at the issues surrounding cell towers in the Tahoe Basin and the legal battle of one resident to turn the tide.



**inside:**

### Staying at Home?

- Keeping It Real with Peggy
- Mike's Mutterings
- Dave at the Movies
- Trish the Dish
- Spoke Junkie
- Dr. Catherine
- Tahoe Dad



Photo by Taylor Flynn

Monica Eisenstecken speaks out against a proposed cell tower near her Ski Run area home.

# Legal battle looms over cell tower

By Heather Gould

“I had never gone to one city council or TRPA meeting in my life. I was just minding my own business. You think ‘Oh, this is Tahoe, they’re going to take care of that stuff,’” said 47-year-old Tahoe native Monica Eisenstecken. Now, Eisenstecken finds herself headlining a federal lawsuit against the Tahoe Regional Planning Agency, its executive director, Joanne

Marchetta, TRPA board members Marsha Berkgigler (Washoe County Commissioner) and Sue Novasel (El Dorado County Supervisor), Eisenstecken’s neighbor Guillian Nel, the Tahoe Prosperity Center and Sacramento Valley Limited, a subsidiary of Verizon.

Eisenstecken is suing over the anticipated placement of a cell tower over 100 feet tall on the property adjacent to her house on Needle Peak Road. The lawsuit alleges 13 violations of the law,

from inadequate environmental review, to violations of the Americans with Disabilities Act, to assault. Eisenstecken has been joined in her suit by Tahoe Stewards, Tahoe for Safer Tech and the Environmental Health Trust.

The cell tower was approved by the South Lake Tahoe City Council in a 3-2 vote a year ago. Eisenstecken previously sued the city on similar grounds, but the lawsuit was deemed moot after she narrowly missed the statute of limitations. The installation has now gone to the TRPA for final review. The TRPA previously issued a permit to erect a cell tower at that site several years ago, but it has since expired, said TRPA spokesman, Jeff Cowen. A new application is on file and is currently under review, said Cowen. No hearing date has yet been set by TRPA, he said.

On the environmental front, the lawsuit alleges that TRPA has never required cell tower applicants to complete a full scale environmental assessment, but only a cursory checklist, and believes this project will be no different. The suit notes that TRPA has no standards for the amount or level of RFR (radiofrequency radiation) that can be emitted in the basin, nor does it measure how RFR affects the environment. In the lawsuit, Eisenstecken’s attorneys specifically refer to a study published in the International Journal of Forestry showing aspens exposed to RFR are more diseased and suffer more plant death than those that are not.

Aspens are a key species in Tahoe’s fragile stream environment zone, according to the suit, and further, strip nutrients and debris from groundwater, runoff and streams, preventing it from entering Lake Tahoe and contributing to the growth of algae. Algal growth is one of the main causes of the decline in Lake clarity. “We’re getting at the heart of TRPA’s mission and... Lake Tahoe’s clarity. If they want to build a cell tower that’s going to cause damage to a stream environment zone then we ought to consider that and take a look at that” before approving the project, said Tahoe City attorney Greg Lien, one of those representing Eisenstecken.

Eisenstecken’s home and the proposed site of the cell tower sit right next to the stream environment zone of the middle reaches of Bijou Creek, which, she says, is also home to the endangered yellow-legged frog. In 2014, the federal Department of the Interior sent a letter to the federal Department of Commerce, which oversees the Federal Communications Commission, which regulates cell phone service. The letter expressed concern regarding the impact of cell tower emissions on all wildlife, but especially with respect to migratory birds, protected by executive order, and stated, “the... radiation standards used by the Federal Communications Commission (FCC) today continue to be based on... a criterion now nearly 30 years out of date and inapplicable

today.” The Federal Communications Commission recently updated the allowable limits of RFR emissions, but they actually don’t differ much from the levels allowed since the industry – and research about possible effects—was in its infancy. Knowledge and technology have both evolved considerably since then.

One of the main violations of TRPA standards the proposed cell tower would pose is scenic, according to Lien. Though it will ostensibly be disguised as a tree, it will still stand out like a sore thumb, according to the suit, in a vicinity that has been deemed one of the most sensitive for scenic value.

Joel Moskowitz of the UC Berkeley School of Public Health, who has studied the issue of cell tower and cell phone emissions since 2009, told the *Mountain News* he believes cell tower technology is harmful to humans as well, especially children and adolescents, in whom it has been associated with memory and concentration deficits, sleep disruption, motor coordination delays, lower verbal expression and Type 2 diabetes. He said most studies on humans show a correlation rather than cause and effect relationship between exposure to cell tower emissions and detrimental health effects. Moskowitz added that studies in animals show a much stronger cause and effect association at levels far below the limits the government allows, lending

Continued on Page 18

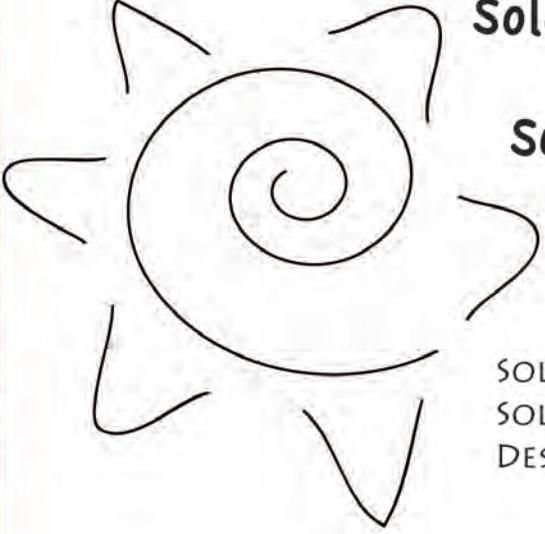


**TAHOE'S COFFEE.®**

alpensieracoffee.com

WHERE DOES YOUR POWER COME FROM? GO SOLAR! SAVE THE PLANET ONE WATT AT A TIME.

# Tahoe Solar Designs



**Solar Energy  
For a  
Sustainable  
Future**

SOLAR HOT WATER  
SOLAR ELECTRICITY  
DESIGN & INSTALL

THE COST OF SOLAR HAS NEVER BEEN SO LOW  
TAKE ADVANTAGE OF THE TAX CREDIT  
GO SOLAR

WWW.TAHOESOLARDESIGNS.COM 530.542.0780 CA#902208 NV#58631

YOUR POWER COME FROM? GO SOLAR! SAVE THE PLANET ONE WATT AT A TIME. WHERE DOES YOUR POWER COME FROM? GO SOLAR! SAVE THE PLANET ONE WATT AT A TIME.

## “Cell tower lawsuit”

Continued from Page 16

credibility to that type of a parallel in humans. He said cell tower emissions impact DNA and antioxidants in the blood.

Eisenstecken, who suffers from a variety of maladies, such as degenerative disc disease, spinal stenosis, arthritis and more is considered disabled under the Americans with Disabilities Act. It is the belief of her doctors that cell tower emissions within 50 yards of her home will aggravate and magnify her symptoms, according to the suit. Therefore, the placement of a cell tower next to her home is a violation of the ADA, the suit contends. One of her two elementary-aged children is also disabled and will be similarly affected, according to the suit.

Federal law prohibits jurisdictions from denying placement of a cell tower due to health impacts, but in this case, the federal law about cell towers and the federal law regarding people with disabilities come into conflict and must be balanced, according to Mark Pollock, another of Eisenstecken’s attorneys. “The preemption language (of the FCC regulations) does not preempt any other federal law which is what the ADA is,” said Pollock. “You have to make a reasonable accommodation under the ADA, which they did not do.”

Proponents of the cell tower often point to the American Cancer Society’s statement of no known adverse health impacts. A fuller reading of the ACS’s position states: “At this time, there’s no strong evidence that exposure to RF(R) waves from cell phone towers causes any noticeable health effects. However, this does not mean that the RF(R) waves from cell phone towers have been proven to be absolutely safe. Most expert organizations agree that more research is needed to help clarify this, especially for any possible long-term effects.”

In fact, according to Eisenstecken’s attorneys, a safer alternative for wireless and internet service exists that could be considered and recommended or required – fiber optic cable that is buried in the ground. A lawsuit in the courts right now, *Irregularators vs. FCC*, contends that wireless companies for years collected money from customers for the eventual rewiring of the country with fiber optic cable, but have failed to carry out their obligation.

Eisenstecken’s suit isn’t just about health or environmental impacts. She is also alleging violations of open meeting laws and conflicts of interest.

One of the defendants in the lawsuit is the Tahoe Prosperity Center, which has long advocated for better internet service in the basin. According to the suit, the TPC drew up a plan for the strategic placement of cell towers throughout the basin and that is now the blueprint for expanding internet and cell service. Sitting on the Tahoe Prosperity Center board as well as representing their respective jurisdictions on the TRPA board or as employees are Marchetta, Berkbigger and Novasel, and, at the city level, South Lake Tahoe Councilmember Devin Middlebrook. The suit alleges these people cannot fulfill their independent, objective duties regarding the

evaluation and approval of cell towers in their public official capacities when they serve on the board of an organization that has a particular bias and agenda. “There’s a lot of incestuous relationships going on between TPC and TRPA,” said Lien.

The formulation of a cell tower plan privately by TPC is specifically a violation of California’s Brown Act or open meeting laws, according to the suit. Though TPC is a private organization, it formulated public policy with public money and publicly elected officials, the suit contends. According to the First Amendment Coalition, a private organization is subject to the Brown Act “when it shares a member (or members) with a governing body and shares funds with that same agency.” This would appear to apply at least with respect to Novasel and Middlebrook (who voted to approve the placement of the cell tower next to Eisenstecken’s home) as El Dorado County and the city of South Lake Tahoe have given funds to TPC in the past.

Finally, according to the suit, the placement of a cell tower on an adjacent property and the emissions that would penetrate Eisenstecken’s home and impact her body constitute trespass and assault on the part of her neighbor, Nel, according to the suit.

Lien said relying on the cell and internet service industry to vouch for the benign nature of the technology is like relying on Big Oil to disclose the harmful effects of fracking or Big Tobacco to expose the dangers of smoking. “People wouldn’t believe Big Oil if they said ‘hey, let’s frack in the Tahoe basin.’ They would look at it with a bit of skepticism and there’s not the skepticism in Tahoe (about cell towers) among agencies and officials that should be questioning these things,” he said.

The *Mountain News* reached out to the various defendants for comment. Novasel said she could not comment on pending litigation. Heidi Hill Drum, executive director of the Tahoe Prosperity Center, said she was precluded from commenting on pending litigation. Heidi Flato of Verizon said the company does not comment on pending litigation. Nel did not respond to a message left for him by press time.

TRPA’s Cowen offered the following statement: “TRPA understands the pressing concerns in the community about the health and environmental impacts of cell towers. The agency’s role in cell tower permitting is to ensure the project is in compliance with Lake Tahoe’s environmental thresholds and zoning requirements. Federal regulators set the emission standards from cell towers and review the environmental impacts of those standards. The agency is not charged with implementing those federal regulations regarding communications equipment.

“Regarding the allegation against TRPA Executive Director Joanne S. Marchetta and individual governing board members, TRPA takes financial conflicts of interest very seriously. Since the Tahoe Prosperity Center is a not-for-profit organization, the allegation does not apply and we see no conflict of interest in this matter.”



## \$599 vehicle incentive program for El Dorado County residents

El Dorado County Air Quality Management District is accepting applications for the Drive Clean! program which provides \$599 towards the purchase/lease of a new eligible electric or plug-in hybrid vehicle.

- Funds are first-come first-served and available to El Dorado County residents only.
  - Pre-approval is required prior to vehicle purchase/lease.
  - You must own the vehicle in El Dorado County for at least three years.
  - Vehicles purchased/leased prior to AQMD pre-approval are not eligible.
  - Vehicle must be on the Air Resources Board CVRP eligible vehicle list: [cleanvehiclerebate.org/eng/eligible-vehicles](http://cleanvehiclerebate.org/eng/eligible-vehicles)

For a pre-approval application and more information  
visit [EDCCleanAir.org](http://EDCCleanAir.org)  
or call AQMD at (530) 621-7501

To date, the program has logged over 10  
million emissions-free miles and a  
reduction of 17.6 tons of VOC and NOx and  
3,977 tons of greenhouse gas emissions!





# The bad and ugly with cell towers

The following column was submitted by a local group calling themselves TRAP (Tahoe Residents for Actual Prosperity)

*"You can fool some of the people all of the time and those are the ones you should concentrate on."*  
G.W. Bush

Ever get the feeling that's what people think of us? The following is a brief overview of several issues regarding the expansion of cellular infrastructure in South Lake Tahoe. They include real estate values, fire safety, aesthetics, prosperity, ethics and health.

## Real Estate Values

The U.S. Census values owner-occupied homes in Tahoe at a median price of \$375,000, and with a conservative 20 percent drop, many residents will suffer considerable losses; some very little when a tower is out of sight,

but when it's 15 feet from your bedroom, it will be difficult to sell at any price. According to the National Association of Realtors, "An overwhelming 94 percent of home buyers and renters surveyed by the National Institute for Science, Law & Public Policy (NISLAPP) say they are less interested and would pay less for a property located near a cell tower or antenna."

## Fire Safety

Sometimes fire knocks out cell towers, causing communications disasters like the 2016 Gatlinburg Fire in Tennessee where firefighters were not able to order residents to evacuate for several days. In a 2004 *Philadelphia Enquirer* article, interference from cell phone signals following a new tower installation caused firefighters to complain "about radio problems at fires in Center City, Grays Ferry and University City." Far from enhancing

communication and public safety, we may experience our own catastrophic wildfire, especially in dry conditions.

## Aesthetics

This issue is difficult because it goes against the notion of informed consent. Of course, all of these structures are unsightly, but camouflaging them is problematic because "hiding them greatly complicates society's ability to monitor for safety," according to a paper in the *Environmental Review*. If one of them is nearby, people should know so they can turn around or at least cross the street. The ground level portions of these devices are painted a nondescript green. The lower portion should be painted a bright color, perhaps in stripes along with clear legible warning signs.

## Health

The simple high school physics assumption that radiation can only cause cancer by being of a high enough photon energy (UV/X-ray) to dislodge electrons and break chemical bonds is wrong. A preponderance of scientific evidence clearly indicates that radio frequency (RF) radiation causes oxidative stress in living cells and free radical production. Microwave radiation alters the antioxidant repair mechanism resulting in a buildup of reactive stress. Free radical DNA damage results, as well as reproductive harm and

some electro-hypersensitivity effects.

Laboratory toxicology experiments show DNA damage directly resulting from microwave RF exposure, and epidemiology has found cancer rates near cell towers are upwards of three to four times higher than background rates. Despite long-emerged science, the FCC continues to apply an outdated standard it imported from the "National Council on Radiation Protection" in 1996 before cell phones were widely adopted or any direct science existed to expose actual health effects.

The FCC exposure standards are now 10,000 times higher than the 0.1  $\mu\text{W}/\text{cm}^2$  recommended by current science. Cell towers should not be located less than 1,500 feet (~500 m) from the public. Telecommunications are a trillion-dollar industry, and their corporate lobbying has been tremendous. However, there's currently a bill on the Senate floor to allow cities to consider environmental effects in the installation of cell facilities and overturn the FCC shot-clock for approving applications.

## Prosperity

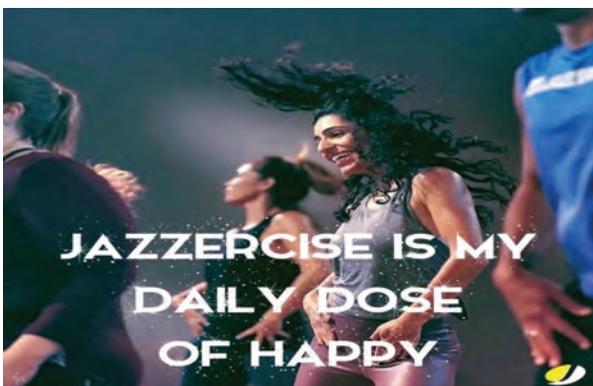
Arbitrary cell tower installations add uncertainty to real estate values, cost homeowner equity, unexpectedly ruin a family's nest egg, and generate large health expenses that we all pay for one way or another. A single cancer treatment

regimen costs between \$100,000 and \$1 million and human life, itself, is invaluable. Even small risks which result in grave consequences must be taken very seriously. Because of the large numbers of residents exposed to this risk, the cost of doing nothing would result in an increasing number of people, many of them young, developing cancer and suffering other health effects.

## Ethics

We have long proudly held a constitutional liberty in this country to personally make informed choices over the risks we exclusively take against our own health and bodily integrity. Regarding cancer, these ethos appear in California law through Proposition 65. Cell tower radiation is far worse than purchasing a cup of coffee, processed meat, BPA plastics, MTBE gasoline. Such purchases are all informed choices. Unlike the latter, cell towers incessantly and non-consensually intrude radiation into our bodies with harmful cumulative exposure.

Moreover, carcinogenic risk is not simply additive; there are synergistic effects because when cellular repair is consumed by one genotoxin, DNA is far less protected against additional mutagenic threats such as radon gas, UV light, or "recreational splurges." Callous infliction of bodily harm and disregard for home equity is un-American. We can do better.



**FALL INTO FITNESS**  
**Introductory offer :**  
**\$68 for 2 months**

<b>CSLT REC. Center</b>	<b>Kahle Park C.C. NV.</b>
<b>M/W/F/Sa 9:30am</b>	<b>T/TH 5:30 PM</b>
<b>T/TH 8:00 am</b>	<b>Saturday 9:00 am</b>
<b>M/W 4:30 pm</b>	
<b>Marcia Sarosik Dance Sunday 9:45 am</b>	

 **South Lake Tahoe jazzercise**  
Jazzercise .com

**FREE**  
**CLASSIFIEDS!**

(Up to 10 per private party)

WEBSITE  
[www.mountainnews.net](http://www.mountainnews.net)

EMAIL:  
[tahomountainnews@gmail.com](mailto:tahomountainnews@gmail.com)

**RICH'S HAULING**  
**SERVICE**



TRASH HAULING • SNOW PLOWING  
Dump Runs • Construction Debris  
Yard Clean-Up • Trailer Drop-Offs  
Appliance Removal • Junk Vehicles

"Quality  
Always  
Wins!"

**FREE ESTIMATES!**  
**Call Anytime 530.208.9293**

## Ex-Governor of Michigan Charged With Neglect in Flint Water Crisis

Rick Snyder, the former governor, faces two misdemeanor counts in the crisis, which left thousands of Flint residents drinking tainted water.



By **Julie Bosman**

Jan. 13, 2021

Rick Snyder, the former governor of Michigan who oversaw the state when a water crisis devastated the city of Flint, has been charged with two counts of willful neglect of duty, according to court records.

The charges are misdemeanors punishable by imprisonment of up to one year or a maximum fine of \$1,000.

Prosecutors in Michigan will report their findings in a wide-ranging investigation into the water crisis on Thursday, officials said, a long-awaited announcement that is also expected to include charges against several other officials and top advisers to Mr. Snyder.

The findings will be announced by Dana Nessel, the Michigan attorney general, Fadwa Hammoud, the state's solicitor general, and Kym L. Worthy, Wayne County's top prosecutor.

Charges had previously been filed in connection to the crisis, which began in 2014, but in June 2019, prosecutors stunned Flint by dropping all pending charges.

Fifteen state and local officials, including emergency managers who ran the city and a member of the governor's cabinet, had been accused by state prosecutors of crimes as serious as involuntary manslaughter. Seven had already taken plea deals. Eight more, including most of the highest-ranking officials, were awaiting trial.

**CALIFORNIA TODAY:** *The news and stories that matter to Californians (and anyone else interested in the state).*

[Sign Up](#)

Brian Lennon, a lawyer for Mr. Snyder, said on Wednesday evening, "We believe there is no evidence to support any criminal charges against Gov. Snyder."



Gov. Rick Snyder drank tap water from Flint, Mich., during a news conference in the city in 2016. Jake May/The Flint Journal-MLive.com, via AP

He added that lawyers for the former governor have sought a confirmation of charges — or a copy of them — but have yet to receive them from prosecutors.

Randall Levine, a lawyer for Richard L. Baird, a former top adviser to Mr. Snyder, said on Tuesday that he was informed this week that Mr. Baird would be among the people facing charges connected to the water crisis.

"At this time, we have not been made aware of what the charges are, or how they are related to his position with former Michigan Governor Rick Snyder's administration," Mr. Levine said. "Rich's relationship with the Flint community has always been strong. When the Flint water crisis hit, he wasn't assigned by Governor Snyder to go to Flint, but rather he raised his hand and volunteered."

In 2016, Mr. Snyder offered an apology for what had happened, but for many residents in Flint, it did not go far enough.

"He pushed this whole thing to the side, and he pushed people to the side," said Floyd Bell, a Flint resident whose two small grandchildren were poisoned by lead when they were babies and still struggle developmentally. "If he was truly aware of what was going on, he should be held accountable."

Dr. Mona Hanna-Attisha, a Flint pediatrician who warned officials about lead in the drinking supply, said that the prospect of new charges was a reminder that "accountability and justice are critical to health and recovery."

"This news is a salve, but it isn't the end of the story," she said in an email. "Healing wounds and restoring trust will take decades and long-term resources."

Melissa Mays, one of the first people in Flint to draw attention to the problems with the city's water, said that given the silence from the attorney general's office for more than 18 months, she was apprehensive that the charges would go far enough.

"We in Flint have been living in prison for the past almost 7 years and being forced to pay for water that's still being piped through corroded and damaged

infrastructure in the streets and in our homes while the people responsible have been walking free,” she wrote in an email. “We in Flint deserve REAL justice and that means wealthy, white politicians and agency heads going to jail for their actions and inaction that’s caused so much harm and loss to us.”

60 Cal.App.5th 1092  
Court of Appeal, Second  
District, Division 8, California.

Laurie BROWN, Plaintiff and Appellant,  
v.  
LOS ANGELES UNIFIED SCHOOL  
DISTRICT, Defendant and Respondent.

B294240  
|  
Filed 2/18/2021

### Synopsis

**Background:** Former teacher, who was alleged to have “electromagnet hypersensitivity,” filed a complaint against school district alleging district discriminated against her, failed to accommodate her disability, and retaliated against her in violation of the California Fair Employment and Housing Act (FEHA). The Superior Court, Los Angeles County, No. BC697060, [Richard E. Rico, J.](#), sustained school district's demurrer and dismissed the action. Teacher appealed.

**Holdings:** The Court of Appeal, [Stratton, J.](#), held that:

[1] former teacher adequately pled that she had a physical disability under the FEHA;

[2] former teacher failed to allege an adverse employment action was taken against her with retaliatory motive;

[3] former teacher adequately pled a cause of action for failure to provide a reasonable accommodation for her physical disability; and

[4] former teacher's allegations were insufficient to state a claim against school district for failure to engage in the interactive process.

Affirmed in part and reversed in part.

[Wiley, J.](#), filed a concurring opinion.

West Headnotes (26)

[1] **Pleading** 🔑 Nature and office of demurrer, and pleadings demurrable

A “demurrer” tests the legal sufficiency of the challenged pleading.

[2] **Appeal and Error** 🔑 Objections and exceptions; demurrer

Court of Appeal reviews de novo a trial court's ruling on a demurrer.

[3] **Appeal and Error** 🔑 Objections and exceptions; demurrer

In reviewing a trial court's ruling on a demurrer, Court of Appeal accepts as true all material facts properly pleaded in the complaint, but does not assume the truth of contentions, deductions, or conclusions of fact and law.

[4] **Appeal and Error** 🔑 Objections and exceptions; demurrer

**Pleading** 🔑 Insufficiency of facts to constitute cause of action

In reviewing a trial court's ruling on a demurrer, the question of a plaintiff's ability to prove the allegations, or the possible difficulty in making such proof, does not concern the reviewing court and plaintiffs need only plead facts showing that they may be entitled to some relief.

[5] **Appeal and Error** 🔑 Objections and exceptions; demurrer

When demurrer is sustained without leave to amend, Court of Appeal decides whether there is reasonable possibility that defect can be cured by amendment: if it can be, trial court has abused its discretion and Court of Appeal reverses; if not, there has been no abuse of discretion and Court of Appeal affirms.

[6] **Civil Rights** ➡ Impairments in general;  
major life activities

**Civil Rights** ➡ Particular conditions,  
limitations, and impairments

Former teacher adequately pled that she had a physical disability under the California Fair Employment and Housing Act (FEHA); the complaint alleged teacher could not work because she experienced chronic pain, headaches, nausea, itching, burning sensations on her skin, ear issues, shortness of breath, inflammation, heart palpitations, respiratory complications, foggy headedness, and fatigue, which were all symptoms of electromagnetic hypersensitivity (EHS), and the described symptoms affected one or more of the body systems listed in the Act and limited teacher's major life activity of working as a teacher. Cal. Code Regs. tit. 2, §§ 11065(d)(2)(A), (B), 11065(d)(8), 11065 subds. (d)(4)-(6).

[7] **Civil Rights** ➡ Public Employment

**Education** ➡ Protected activities in general  
**Public Employment** ➡ Causal connection;  
temporal proximity

Former teacher failed to allege an adverse employment action was taken against her with retaliatory motive, as required to state a claim against school district for retaliation under California Fair Employment and Housing Act (FEHA); teacher complained that school's WiFi system was adversely affecting her health, the parties engaged in the interactive process to arrive at a reasonable accommodation, school district made promises to take certain actions to reasonably accommodate teacher's complaints, school district later reneged on its promises because it decided to rely on the findings of its consultant that the campus was "safe," and teacher alleged no retaliatory actions were taken against her precisely because she engaged in protected activity. Cal. Gov't Code § 12940(a).

[8] **Civil Rights** ➡ Particular cases

Former teacher failed to allege an adverse employment action was taken against her by school district with discriminatory motive, as required to state a claim under the Fair Employment and Housing Act (FEHA) against school district for discrimination based on a physical disability; teacher did not alleged that she was the target of disparate treatment, or that school district had a policy or practice that had a disproportionate effect on employees suffering from a disability, and she merely alleged that school district failed to engage meaningfully with her in the interactive process and would not accommodate her disability. Cal. Gov't Code § 12940(a).

[9] **Civil Rights** ➡ Employment practices

Evidence of discriminatory motive must be examined carefully in disability discrimination cases to determine whether there is direct evidence that the motive for the employer's conduct was related to the employee's physical or mental condition.

[10] **Civil Rights** ➡ Disparate treatment

**Civil Rights** ➡ Disparate impact

The Fair Employment and Housing Act (FEHA) proscribes two types of disability discrimination: discrimination arising from an employer's intentionally discriminatory act against an employee because of his or her disability, referred to as disparate treatment discrimination, and discrimination resulting from an employer's facially neutral practice or policy that has a disproportionate effect on employees suffering from a disability, referred to as disparate impact discrimination. Cal. Gov't Code §§ 12900 et al.

[11] **Civil Rights** ➡ Adverse actions in general

What constitutes adverse employment action under Fair Employment and Housing Act (FEHA) is not, by its nature, susceptible to mathematically precise test, and, as result, significance of particular types of adverse actions must be evaluated by taking into

account legitimate interests of both employer and employee. [Cal. Gov't Code § 12940\(a\)](#).

**[12] Civil Rights** ➡ Adverse actions in general

For the purpose of discrimination statute, which indicated it was an unlawful employment practice for an employer to discriminate against an employee on the basis of race, sex, or the other enumerated characteristics “in compensation or in the terms, conditions, and privileges of employment,” the phrase “terms, conditions or privileges” of employment must be interpreted liberally and with reasonable appreciation of realities of workplace in order to afford employees appropriate and generous protection against employment discrimination that Fair Employment and Housing Act (FEHA) was intended to provide. [Cal. Gov't Code § 12940\(a\)](#).

**[13] Civil Rights** ➡ Practices prohibited or required in general; elements

It is appropriate to consider plaintiff's allegations of discrimination based on a physical disability collectively under totality-of-the-circumstances approach in a Fair Employment and Housing Act (FEHA) action. [Cal. Gov't Code § 12940\(a\)](#).

**[14] Civil Rights** ➡ Motive or intent; pretext

Even if former teacher's allegations were sufficient to establish an adverse employment action, she failed to allege any facts from which discriminatory motive could be inferred, as required to state a claim for discrimination based on physical disability against school district; teacher alleged no facts from which it could be inferred that school district clung to its belief that school campus was safe and refused to accommodate her because it was biased against her as a person with a disability. [Cal. Gov't Code § 12940\(a\), \(k\)](#).

**[15] Civil Rights** ➡ Particular cases

Former teacher adequately pled a cause of action for failure to provide a reasonable

accommodation for her physical disability due to electromagnetic hypersensitivity (EHS), in action against school district; teacher's complaint alleged she suffered from a physical disability, but could perform the essential functions of the position with the accommodation “to which [school district] initially agreed to but subsequently refused to honor and/or other reasonable accommodations, such as use of paints, fabrics and/or other shielding materials to block or minimize exposure to electromagnetic frequencies.” [Cal. Gov't Code § 12940 \(k\), \(m\) \(1\)](#); [Cal. Code Regs. tit. 2, § 11068\(a\)](#).

**[16] Civil Rights** ➡ In general; elements of accommodation claims

To establish a failure-to-accommodate claim, a plaintiff must show (1) she has a disability covered by Fair Employment and Housing Act (FEHA); (2) she can perform the essential functions of the position; and (3) defendant failed reasonably to accommodate her disability. [Cal. Gov't Code § 12940\(k\)](#).

**[17] Civil Rights** ➡ What are reasonable accommodations; factors considered

A reasonable accommodation under Fair Employment and Housing Act (FEHA) means a modification or adjustment to the workplace that enables the employee to perform the essential functions of the job held or desired. [Cal. Gov't Code § 12940\(k\)](#).

**[18] Civil Rights** ➡ Employment practices

Although an accommodation is not reasonable under Fair Employment and Housing Act (FEHA) if it produces an undue hardship to the employer, a plaintiff need not initially plead or produce evidence showing that the accommodation would not impose such an undue hardship. [Cal. Gov't Code § 12940\(k\)](#).

**[19] Civil Rights** ➡ Pleading

Whether a plaintiff's requested accommodation under Fair Employment and Housing Act (FEHA) is reasonable cannot be determined on demurrer. [Cal. Gov't Code § 12940\(k\)](#).

**[20] Civil Rights** 🔑 Requesting and choosing accommodations; interactive process; cooperation

Once notified of disability, employer's burden is to take positive steps to accommodate employee's limitations, and employee also retains duty to cooperate with employer's effort by explaining his or her disability and qualifications; reasonable accommodation under Fair Employment and Housing Act (FEHA) thus envisions exchange between employer and employee where each seeks and shares information to achieve best match between employee's capabilities and available positions. [Cal. Gov't Code § 12940\(k\)](#).

**[21] Civil Rights** 🔑 Requesting and choosing accommodations; interactive process; cooperation

If reasonable accommodation under Fair Employment and Housing Act (FEHA) does not work, employee must notify employer, who has duty to provide further accommodation. [Cal. Gov't Code § 12940\(k\)](#).

**[22] Civil Rights** 🔑 Requesting and choosing accommodations; interactive process; cooperation

Former teacher's allegations that school district agreed to hire an independent consultant to determine where on school campus exposure to the electromagnetic frequencies was most minimal, then changed its mind and decided school campus was "safe" were insufficient to state a claim under California Fair Employment and Housing Act (FEHA) against school district for failure to engage in the interactive process; teacher's allegations did not amount to failure to engage in the interactive process, instead, it was

failure to follow up with an accommodation. [Cal. Gov't Code §§ 12940\(n\), 12926.1\(e\)](#).

**[23] Civil Rights** 🔑 Requesting and choosing accommodations; interactive process; cooperation

Under Fair Employment and Housing Act (FEHA), it is an unlawful practice for an employer to fail to engage in a good faith interactive process with the employee to determine an effective reasonable accommodation if an employee with a known physical disability requests one. [Cal. Gov't Code §§ 12940\(n\), 12926.1\(e\)](#).

**[24] Civil Rights** 🔑 In general; elements of accommodation claims

**Civil Rights** 🔑 Requesting and choosing accommodations; interactive process; cooperation

Under Fair Employment and Housing Act (FEHA), failure to accommodate and failure to engage in the interactive process are separate, independent claims involving different proof of facts; the purpose of the interactive process is to determine what accommodations is required, and once a reasonable accommodation has been granted, then the employer has a duty to provide that reasonable accommodation. [Cal. Gov't Code §§ 12940\(n\), 12926.1\(e\)](#).

**[25] Pleading** 🔑 Authority and discretion of court

The trial court's action in sustaining school district's demurrer without leave to amend was not erroneous, in former teacher's action against school district for discrimination based on physical disability, where teacher failed to propose new facts and state in her reply brief that she "need not specify additional details for an amended complaint because she already alleged more than sufficient ultimate facts to support her claims and any additional allegations would be superfluous evidentiary facts."

**[26] Pleading** ➔ **Amendment or Further Pleading After Demurrer Sustained**

Generally, leave to amend after sustaining demurrer is warranted when the complaint is in some way defective, but plaintiff has shown in what manner the complaint can be amended and how that amendment will change the legal effect of the pleading.

**Witkin Library Reference:** 8 Witkin, *Summary of Cal. Law* (11th ed. 2017) *Constitutional Law*, § 1045 [Discrimination Against Disabled Persons; Statutory Protections.]

**\*\*326** APPEAL from a judgment of the Superior Court of Los Angeles County, [Richard E. Rico](#), Judge. Reversed in part and affirmed in part. (Los Angeles County Super. Ct. No. BC697060)

**Attorneys and Law Firms**

JML Law, [Joseph M. Lovretovich](#) and [Jennifer A. Lipski](#), Woodland Hills, for Plaintiff and Appellant.

[Anthony J. Bejarano](#), Los Angeles, and [David V. Greco](#) for Defendant and Respondent.

[STRATTON, J.](#)

**\*1097 INTRODUCTION**

Appellant Laurie Brown (Brown) has been a teacher employed by the Los Angeles Unified School District (LAUSD) since 1989. In 2015, LAUSD installed an updated Wi-Fi system at the school where Brown taught. She soon began to experience headaches and nausea, and believed the electromagnetic frequency of the new wireless system was the cause. She requested **\*1098** various accommodations from LAUSD, but ultimately sued, alleging LAUSD discriminated against her based on her “electromagnetic hypersensitivity,” failed to accommodate her condition, and retaliated against her—in violation of the California Fair Employment and Housing Act (FEHA) ([Gov. Code](#), § 12900 et seq.).

Brown appeals from a judgment of dismissal entered after the trial court sustained LAUSD's demurrer to her first amended

complaint (FAC) without leave to amend. She contends the trial court erred in sustaining the demurrer because she pled sufficient facts in support of each of her claims. She further contends the trial court abused its discretion by not granting her leave to amend the FAC.

We conclude Brown adequately pled her cause of action for failure to provide reasonable accommodation for her disability. We reverse on this cause of action only. Otherwise, the judgment is affirmed.

**FACTUAL AND PROCEDURAL BACKGROUND**

*A. Relevant Factual Background*

In 2012, LAUSD commissioned URS Corporation (URS) to consult with LAUSD about replacing the existing Wi-Fi system at Millikan Middle School (Millikan) with one that would accommodate iPads, Chromebooks, and tablets LAUSD intended to provide its students.

LAUSD requested public comment on the proposed new Wi-Fi system. Cindy Sage, an environmental scientist and expert on electromagnetic frequency (EMF), stated she could not support URS's conclusions about the safety of the new Wi-Fi system.

During a May 28, 2014, school board hearing, LAUSD's “medical personnel” presented a power point presentation indicating they were uncertain about any long-term effects the Wi-Fi system may have on students and staff. LAUSD promised to continue actively monitoring any developments.

In 2015, Brown began teaching at Millikan. Later that year, in April 2015, LAUSD installed and began operating the upgraded Wi-Fi system at Millikan. Brown **\*\*327** thereafter experienced chronic pain, which she alleged was caused by the new Wi-Fi.

**\*1099 B. Brown's First Amended Complaint**

On March 7, 2018, Brown filed a civil complaint against LAUSD. On June 6, 2018, the trial court sustained a demurrer to the complaint with leave to amend.

On June 26, 2018, Brown filed the FAC which alleged five causes of action pursuant to FEHA:

- 1) Discrimination based on physical disability;

- 2) Failure to accommodate;
- 3) Failure to engage in the interactive process;
- 4) Retaliation; and
- 5) Failure to prevent discrimination and retaliation.

The FAC alleged:

Following activation of the new Wi-Fi system on April 23, 2015, Brown began to experience chronic pain, headaches, nausea, itching, burning sensations on her skin, ear issues, shortness of breath, inflammation, heart palpitations, respiratory complications, foggy headedness, and fatigue. She reported the symptoms to her superiors at Millikan and was granted leave from work “due to these symptoms, on an intermittent basis, for several days thereafter.”

She returned to campus the following week and fell ill again “[w]ithin 2 to 3 hours.” Her “medical provider subsequently diagnosed her” with electromagnetic hypersensitivity (EHS), also referred to as “microwave sickness.”

On May 22, 2015, Brown filed her first formal request for accommodation with LAUSD.

On July 15, 2015, LAUSD held its first interactive process meeting with Brown. Following the meeting, LAUSD agreed to disconnect the Wi-Fi access points in Brown's assigned classroom and in an adjacent classroom. LAUSD also agreed to use “a hardwired computer lab with Wi-Fi turned off while testing for Common Core.”

On August 4, 2015, “Dr. Huy Hoang, internist, wrote that emerging EMF sensitivity was disabling” Brown.

**\*1100** Brown returned to work in August 2015. She was assigned to Room 22 at the Millikan campus. Brown alleged LAUSD's accommodations were “not reasonable” and “did not work.” While LAUSD disconnected the routers in Brown's classroom and one adjoining classroom, “multiple other classrooms in front and to the side of [Brown]’s classroom continued to have their routers active.”

On September 3, 2015, Brown's physician, Dr. Jody Levy, placed her on a medical leave of absence through November 16, 2015, due to her “migraines, headaches, and nausea.

Restrictions upon returning to work were for [Brown] to work with minimal Wi-Fi exposure.”

On September 8, 2015, Brown filed a second request for accommodation “on the grounds her symptoms persisted due to Wi-Fi and radio frequencies to which she was continuously exposed.” She requested LAUSD reduce her exposure and consider “using paints and other forms of shielding materials to block Wi-Fi and radio frequencies in her classroom.”

On October 22, 2015, LAUSD held its second interactive process meeting with Brown. Brown requested LAUSD authorize “further studies to evaluate and determine the best location on the Millikan campus where [Brown] would encounter minimal exposure to Wi-Fi and radio frequencies, along with consideration of using paints and other shielding materials.”

**\*\*328** On November 13, 2015, LAUSD denied Brown's second request for accommodation, relying on testing performed by URS that indicated the Wi-Fi system was “safe.” Brown appealed LAUSD's denial.

Meanwhile, Brown's medical leave was extended from November 2015 through June 14, 2016 by Dr. Michael Hirt, “citing migraines and nausea. Restrictions include minimal EMF exposure and writes patient could return to work if EMF exposure [or] measurement were reduced.”

The appeal hearing took place in February 2016. LAUSD “reversed course” and agreed to provide a “neutral expert EMF inspection for further microwave measurements.” Brown was notified that LAUSD will provide Brown “with the test results, but is not required to provide [her] advance information regarding the logistics of the testing.”

On April 18, 2016, LAUSD provided Brown with three options for neutral EMF testing: 1) allow LAUSD's retained consultant URS to conduct the requested testing; 2) choose another consultant “which might delay the process”; or 3) advise LAUSD she no longer desired additional EMF testing.

**\*1101** On April 26, 2016, Brown indicated she wanted a different consultant—not URS—to conduct the additional EMF testing/inspection. She alleged “a new analysis by URS, LAUSD's own consultant, would be inherently biased due to URS’ relationship with LAUSD.” Brown alleged, however, that LAUSD failed to inform her that “selecting another

consultant would require the consultant to submit to LAUSD's bidding process for a contract to do the inspection.”<sup>2</sup>

On June 19, 2016, LAUSD notified Brown it did not agree with her selected consultant and that URS's “prior evaluation of Wi-Fi and radio frequencies at Millikan evidenced a safe and non-hazardous working environment.”

In November 2016, Brown followed up with LAUSD about what “reasonable accommodation” LAUSD would provide. In January 2017, Brown sent LAUSD another follow-up email and expressed “frustration and concerns about LAUSD appearing to retract the accommodation it had promised ... a year earlier.”

Brown alleged she could not return to work “without being overcome with crippling pain.” She was “forced to go out on a disability leave from her job, which exhausted her approximately 800 hours of accrued paid time off and sick leave.” As a result, she experienced “an economic loss of earnings due to not receiving her full income.”

Based on the foregoing, Brown argued LAUSD “engaged in a course or pattern of conduct that, taken as a whole, materially and adversely affected the terms, conditions, or privileges” of Brown's employment. She believed she “could have continued performing all essential duties and functions of her job” had she been provided reasonable accommodations from LAUSD. She argued LAUSD failed to “engage in an interactive process” with Brown and “explore all reasonable accommodation for her physical disability.” Brown also characterized the foregoing as “adverse employment action” and “discriminatory and retaliatory conduct.”

She requested general damages, special damages, loss of earnings and benefits, attorney fees and costs, injunctive relief, equitable relief, and any other relief the trial court deemed just and proper.

### **\*\*329** C. LAUSD's Demurrer and Brown's Opposition

On July 31, 2018, LAUSD filed a demurrer to the FAC pursuant to [Code of Civil Procedure section 430.10, subdivision \(e\)](#). LAUSD argued Brown failed **\*1102** to allege with particularity sufficient facts in support of her causes of action. Brown's FAC did not include any facts that demonstrated LAUSD's decision not to provide additional testing created adverse work conditions such that a reasonable person would have felt compelled to resign. LAUSD next

pointed out that Brown had not pled facts that would establish the original testing by URS was “unreliable or faulty” and instead merely concluded “URS is biased.”

LAUSD argued Brown did not suffer any adverse employment action, “much less an adverse action *because* of her alleged medical condition.” Per LAUSD, Brown “voluntarily chose” to go on leave; she was never dismissed. LAUSD argued it “went above and beyond to accommodate” Brown's alleged disability and provided examples of accommodations it had granted. LAUSD noted Brown's symptoms mysteriously persisted “despite being away from Millikan's campus and being on a lengthy approved leave of absence.”

LAUSD requested the court sustain the demurrer without leave to amend, as Brown could not identify any adverse employment action taken by LAUSD *because* of her disability.

On August 14, 2018, Brown filed her opposition to LAUSD's demurrer. She argued the FAC alleged sufficient facts to establish all five causes of action. She further argued that while LAUSD “*proposed* multiple efforts, [it] *never implemented* any of them fully.” (Boldface omitted.)

### D. Hearing and Ruling

On August 27, 2018, the trial court entertained brief oral argument and took the matter under submission.

The next day, on August 28, 2018, the court sustained the demurrer without leave to amend as to all five causes of action.

On September 20, 2018, the court signed the judgment of dismissal.

Brown timely appealed from the judgment.

## DISCUSSION

As a preliminary matter, we disagree with LAUSD that Brown failed to include a complete record. The record does not include a copy of the original complaint, first demurrer, and the court's June 6, 2018 ruling. However, the absence of these pleadings does not foreclose our review of **\*1103** Brown's contentions on appeal. Where, as here, Brown amended

the original complaint, the FAC supersedes the original complaint. (See *Alfaro v. Community Housing Improvement System & Planning Assn., Inc.* (2009) 171 Cal.App.4th 1356, 1372, 124 Cal.Rptr.3d 271.) The record on appeal contains the operative FAC and LAUSD's demurrer; these are the pleadings necessary for our review.

#### A. Standard of Review

[1] [2] [3] [4] A demurrer tests the legal sufficiency of the challenged pleading. (*Milligan v. Golden Gate Bridge Highway & Transportation Dist.* (2004) 120 Cal.App.4th 1, 5, 15 Cal.Rptr.3d 25.) We review de novo a trial court's ruling on a demurrer. (*Dudek v. Dudek* (2019) 34 Cal.App.5th 154, 163, 246 Cal.Rptr.3d 27 (*Dudek*.) We accept as true all material facts properly pleaded in the complaint, but do not assume the truth of contentions, deductions, or conclusions of fact and law. (*Ibid.*; *Estate of Holdaway* (2019) 40 Cal.App.5th 1049, 1052, 253 Cal.Rptr.3d 659.) \*\*330 The question of a plaintiff's ability to prove the allegations, or the possible difficulty in making such proof, does not concern the reviewing court and plaintiffs need only plead facts showing that they may be entitled to some relief. (*Alcorn v. Anbro Engineering, Inc.* (1970) 2 Cal.3d 493, 496, 86 Cal.Rptr. 88, 468 P.2d 216.)

[5] In addition, “ ‘[w]hen a demurrer is sustained without leave to amend, “we decide whether there is a reasonable possibility that the defect can be cured by amendment: if it can be, the trial court has abused its discretion and we reverse; if not, there has been no abuse of discretion and we affirm.” ’ ” (*Dudek, supra*, 34 Cal.App.5th at p. 163, 246 Cal.Rptr.3d 27.) Brown shoulders the burden to show a reasonable possibility the FAC can be amended to state a cause of action. (*Id.* at pp. 163–164, 246 Cal.Rptr.3d 27.)

#### B. Brown Adequately Pled a Physical Disability.

[6] In an argument it makes as to all five causes of action, LAUSD contends Brown's alleged disability, electromagnetic sensitivity, is not a “recognized” disability. In support of this contention, LAUSD relies on a federal case from the Seventh Circuit and a federal district court case from the District of Massachusetts, both interpreting the Americans with Disabilities Act of 1990 (ADA): *Hirmiz v. New Harrison Hotel Corp.* (7th Cir. 2017) 865 F.3d 475 and *G v. Fay Sch., Inc.* (D. Mass. 2017) 282 F.Supp.3d 381.

LAUSD's reliance on ADA cases is misplaced. The FEHA protections against torts based on disability are independent

of those under the ADA. “The law of this state in the area of disabilities provides protections \*1104 independent from those in the federal Americans with Disabilities Act of 1990 .... Although the federal act provides a floor of protection, this state's law has always, even prior to passage of the federal act, afforded additional protections.” (§ 12926.1, subd. (a); Cal. Code Regs., tit. 2, § 11065, subd. (d) (8).) The Legislature has stated its intent that “physical disability” be construed so that employees are protected from discrimination due to actual or perceived physical impairment that is disabling, potentially disabling, or perceived as disabling or potentially disabling. (§ 12926.1, subd. (b); Cal. Code Regs., tit. 2, § 11065, subds. (d)(4)–(6).) And the Legislature has specifically stated its intent that the FEHA provide broader protection than under the ADA. (§ 12926.1, subd. (c); Cal. Code Regs., tit. 2, § 11065, subd. (d)(8).)

FEHA states a “physical disability” includes, but is not limited to, “any physiological disease, disorder, condition, cosmetic disfigurement, or anatomical loss that does both of the following: [¶] (A) Affects one or more of the following body systems: neurological, immunological, musculoskeletal, special sense organs, respiratory, including speech organs, cardiovascular, reproductive, digestive, genitourinary, hemic and lymphatic, skin and endocrine. [¶] (B) Limits a major life activity. For purposes of this action: [¶] ... [¶] (ii) A ... condition ... limits a major life activity if it makes the achievement of the major life activity difficult. [¶] (iii) ‘Major life activities’ shall be broadly construed and includes physical, mental, and social activities and working.” (§ 12926, subd. (m)(1); see also Cal. Code Regs., tit. 2, § 11065, subd. (d)(2)(A), (B).)

The FAC alleges that Brown could not work because she experienced “the various symptoms of which LAUSD had been warned could occur, namely, chronic pain, headaches, nausea, itching, burning sensations on her skin, ear issues, shortness of breath, inflammation, heart palpitations, respiratory complications, foggy headedness, \*\*331 and fatigue, all symptoms of Microwave Sickness or EHS.” These described symptoms affect one or more of the body systems listed in the statute and limited Brown's major life activity of working as a teacher at Millikan. That the ADA may not “recognize” EHS is immaterial to our interpretation of FEHA. Brown adequately pled physical disability within the four corners of the statute.

#### C. Brown Failed to Allege Adverse Employment Action Taken Against Her with Discriminatory or Retaliatory Motive

LAUSD next argues that the first cause of action for discrimination based on physical disability and the fourth cause of action for retaliation fail for lack of specificity and are insufficient to withstand the demurrer. We agree.

**\*1105** 1. Retaliation

[7] The elements of a cause of action for retaliation in violation of section 12940, subdivision (h) are: “(1) the employee's engagement in a protected activity ...; (2) retaliatory animus on the part of the employer; (3) an adverse action by the employer; (4) a causal link between the retaliatory animus and the adverse action; (5) damages; and (6) causation.” (*Mamou v. Trendwest Resorts, Inc.* (2008) 165 Cal.App.4th 686, 713, 81 Cal.Rptr.3d 406; *Le Mere v. Los Angeles Unified School Dist.* (2019) 35 Cal.App.5th 237, 243, 247 Cal.Rptr.3d 76.)

Here, the FAC alleges no facts coming close to retaliatory actions or motive. According to the FAC, Brown made her complaints that the Wi-Fi system was adversely affecting her health; the parties engaged in the interactive process to arrive at a reasonable accommodation; LAUSD made promises to take certain actions to reasonably accommodate her complaints; LAUSD later reneged on its promises because it decided to rely on the findings of its consultant URS that the campus was “safe.” She alleges no retaliatory actions taken against her precisely because she engaged in protected activity, that is, because she made her initial complaint. Brown conflates actions taken by LAUSD in response to the complaint with actions taken by LAUSD to harm her because of her complaint. None of the alleged facts implicate retaliation.

2. Discrimination

[8] [9] Under section 12940, it is unlawful for an employer, because of physical disability, to “refuse to hire or employ the person or to refuse to select the person for a training program leading to employment, or to bar or to discharge the person from employment or from a training program leading to employment, or to discriminate against the person in compensation or in terms, conditions, or privileges of employment.” (§ 12940, subd. (a).) The elements of a prima facie case of discrimination vary depending on the particular facts. Generally, the plaintiff must provide evidence that he or she (1) was a member of a protected class; (2) was qualified for the position sought or was performing competently in the position already held; (3) suffered an adverse employment action, such as termination, demotion,

or denial of an available job; and (4) some other circumstance suggests discriminatory motive. (*Guz v. Bechtel National, Inc.* (2000) 24 Cal.4th 317, 355, 100 Cal.Rptr.2d 352, 8 P.3d 1089.) Evidence of discriminatory motive must be examined carefully in disability discrimination cases to determine “whether there is direct evidence that the motive for the employer's conduct was related to the employee's physical or mental condition.” ( **\*\*332** *Wallace v. County of Stanislaus* (2016) 245 Cal.App.4th 109, 123, 199 Cal.Rptr.3d 462.)

**\*1106** [10] FEHA proscribes two types of disability discrimination: (1) discrimination arising from an employer's intentionally discriminatory act against an employee because of his or her disability (referred to as disparate treatment discrimination) and discrimination resulting from an employer's facially neutral practice or policy that has a disproportionate effect on employees suffering from a disability (referred to as disparate impact discrimination). (*Knight v. Hayward Unified School Dist.* (2005) 132 Cal.App.4th 121, 128–129, 33 Cal.Rptr.3d 287, disapproved on other grounds in *Williams v. Chino Valley Independent Fire Dist.* (2015) 61 Cal.4th 97, 115, 186 Cal.Rptr.3d 826, 347 P.3d 976.)

Here, just as with the retaliation cause of action, there are two issues as to the discrimination cause of action: whether Brown sufficiently alleged that LAUSD took any adverse employment actions and whether Brown sufficiently alleged facts to support the allegation of discriminatory motive. Brown contends LAUSD refused to participate in the interactive process in good faith and refused to put in place reasonable accommodations to which it has previously agreed. While these allegations against LAUSD support other causes of action as discussed below, we conclude they do not constitute “adverse employment actions” in the context of a claim of discrimination.

[11] [12] [13] Our Supreme Court has recognized that what constitutes an adverse employment action “is not, by its nature, susceptible to a mathematically precise test,” and, as a result, “the significance of particular types of adverse actions must be evaluated by taking into account the legitimate interests of both the employer and the employee.” (*Yanowitz v. L'Oreal USA, Inc.* (2005) 36 Cal.4th 1028, 1054, 32 Cal.Rptr.3d 436, 116 P.3d 1123.) *Yanowitz* defined an adverse employment action generally as one that materially affects the terms and conditions of employment. (*Id.* at p. 1051, fn. 10, 32 Cal.Rptr.3d 436, 116 P.3d 1123.) The phrase “terms, conditions or privileges” of employment must be interpreted

liberally and with a reasonable appreciation of the realities of the workplace in order to afford employees the appropriate and generous protection against employment discrimination that the FEHA was intended to provide.” (*Id.* at p. 1054, 32 Cal.Rptr.3d 436, 116 P.3d 1123.) It is appropriate to consider plaintiff’s allegations collectively under a totality-of-the-circumstances approach. (*Id.* at p. 1052, fn. 11 & pp. 1055–1058, 32 Cal.Rptr.3d 436, 116 P.3d 1123.)

However, we note the FEHA scheme prohibits specific unlawful employment practices by covered employers, e.g., discrimination, retaliation, failure to make reasonable accommodation, failure to engage in the interactive process with the employee. We conclude that the commission of one specific prohibited employment practice does not, in and of itself, constitute commission of all other prohibited employment practices under the broad rubric of policies or practices affecting the “terms, conditions or privileges of employment.” Such an interpretation would be contrary to the whole point of **\*1107** specifically separating conduct into individual unlawful employment practices. Brown has not alleged she was the target of disparate treatment. Nor has she alleged a policy or practice that had a disproportionate effect on employees suffering from a disability. She simply alleged that LAUSD failed to engage meaningfully with her in the interactive process and would not reasonably accommodate her disability. Those allegations pertain to her remaining causes of action, but we decline to construe them, **\*\*333** without more, as adverse employment actions sufficient to support a claim of discrimination in the terms and conditions of employment. We agree with the trial court that Brown has conflated “ ‘adverse employment action’ with the failure to accommodate and failure to engage claims.”

[14] Moreover, even if the allegations are deemed sufficient to constitute adverse employment actions, Brown has alleged no facts from which discriminatory intent be inferred. In other words, she has alleged no facts from which we can infer LAUSD clung to its belief that the campus was safe and refused to accommodate her because it was biased against her as a person with a disability. At most, the FAC alleged facts showing a disagreement between the parties as to whether the Wi-Fi was causing her disability. We conclude she has failed to allege discrimination in employment.

Because we find Brown has failed to allege discrimination or retaliation in employment, we also conclude she has failed to sufficiently allege, in her fifth cause of action, failure

to prevent discrimination and retaliation in employment, in violation of section 12940, subdivision (k).

*D. Brown Adequately Pled a Cause of Action for Failure to Provide Reasonable Accommodation for a Physical Disability*

[15] An employer must provide a reasonable accommodation for an applicant or employee with a known mental or physical disability unless the accommodation would cause undue hardship. Failure to do so is an unlawful employment practice. (§ 12940, subd. (m)(1); *Cal. Code Regs.*, tit. 2, § 11068 subd. (a).) Failure to do so is an unlawful employment practice.

[16] [17] [18] [19] To establish a failure to accommodate claim, Brown must show (1) she has a disability covered by FEHA; 2) she can perform the essential functions of the position; and 3) LAUSD failed reasonably to accommodate her disability. (*Jensen v. Wells Fargo Bank* (2000) 85 Cal.App.4th 245, 256–257, 102 Cal.Rptr.2d 55.) A “reasonable accommodation” means a modification or adjustment to the workplace that enables the employee to perform the essential functions of the job held or desired. (*Scotch v. Art Institute of California* (2009) 173 Cal.App.4th 986, 1010, 93 Cal.Rptr.3d 338.) Although an accommodation is not reasonable if it produces an undue **\*1108** hardship to the employer, a plaintiff need not initially plead or produce evidence showing that the accommodation would not impose such an undue hardship. (*Bagatti v. Department of Rehabilitation* (2002) 97 Cal.App.4th 344, 356, 118 Cal.Rptr.2d 443.) Importantly, whether plaintiff’s requested accommodation is reasonable cannot be determined on demurrer. (*Id.* at p. 368–369, 118 Cal.Rptr.2d 443.)

[20] [21] Once notified of a disability, the employer’s burden is to take positive steps to accommodate the employee’s limitations. The employee also retains a duty to cooperate with the employer’s effort by explaining his or her disability and qualifications. Reasonable accommodation thus envisions an exchange between employer and employee where each seeks and shares information to achieve the best match between the employee’s capabilities and available positions. (*Spitzer v. Good Guys, Inc.* (2000) 80 Cal.App.4th 1376, 1385, 96 Cal.Rptr.2d 236 (*Spitzer*).) If a reasonable accommodation does not work, the employee must notify the employer, who has a duty to provide further accommodation. (See *id.* at p. 1384, 96 Cal.Rptr.2d 236 [if employer did not know a reasonable accommodation was not working, a duty to provide further accommodation never arose].)

**\*\*334** Brown has adequately pled failure to accommodate. The FAC alleges that she suffers from a physical disability, but can perform the essential functions of the position with the accommodation “to which LAUSD initially agreed to but subsequently refused to honor and/or other reasonable accommodations, such as use of paints, fabrics and/or other shielding materials to block or minimize exposure to electromagnetic frequencies.” Further, although LAUSD provided Brown with three options to choose from for neutral EMF testing, including the option to choose a consultant other than URS to conduct the testing (which Brown opted for), LAUSD reneged on its agreement, concluded that URS's prior evaluation evidenced a safe, non-hazardous working environment, and took no further action. As mentioned above, “reasonable accommodation” envisions an exchange between employer and employee in good faith; based on our reading of Brown's FAC, LAUSD's actions here do not align with those of an employer taking positive steps to accommodate the employee's limitations (*Spitzer, supra*, 80 Cal.App.4th at p. 1385, 96 Cal.Rptr.2d 236).

On appeal LAUSD argues that it attempted to accommodate her multiple times to no avail. It also argues that because Brown alleged that she suffered symptoms at her home, there was nothing LAUSD could do to ameliorate her disability. These are questions for the ultimate finder of fact and not questions properly resolved by demurrer. Brown's allegations were sufficient.

**\*1109** E. Brown Failed to Allege Failure to Engage in the Interactive Process.

[22] [23] [24] Under FEHA, it is an unlawful practice for an employer to fail to engage in a good faith interactive process with the employee to determine an effective reasonable accommodation if an employee with a known physical disability requests one. (§ 12940, subd. (n); see § 12926.1, subd. (e); *A.M. v. Albertsons, LLC* (2009) 178 Cal.App.4th 455, 463, 100 Cal.Rptr.3d 449 (*Albertsons*)). Failure to accommodate and failure to engage in the interactive process are separate, independent claims involving different proof of facts. The purpose of the interactive process is to determine what accommodations is required. Once a reasonable accommodation has been granted, then the employer has a duty to provide that reasonable accommodation. (*Albertsons*, at pp. 463–464, 100 Cal.Rptr.3d 449.)

Here, Brown's FAC alleges LAUSD did agree on a reasonable accommodation (to hire an independent consultant to determine where on campus exposure to the electromagnetic frequencies was most minimal) and then changed its mind, deciding that the campus was “safe.” This is not a failure to engage in the interactive process; it is a failure to follow up with an accommodation to which it had agreed. (*Albertsons, supra*, 178 Cal.App.4th at pp. 463–464, 100 Cal.Rptr.3d 449.)

*Albertsons* is instructive in this regard. In that case, employer Albertsons agreed to reasonable accommodations and then failed to advise plaintiff's supervisors about the agreement. As a result, when plaintiff sought to take advantage of the accommodations, her supervisors did not allow her to do so. Plaintiff employee sued for failure to accommodate. Albertsons argued the plaintiff employee had a personal responsibility to advise her supervisors of her disability and of the agreed-upon accommodations. Albertsons argued plaintiff's failure to so advise her supervisors constituted a failure by the employee to continue the interactive process and vitiated her cause of action for failure to accommodate. (*Albertsons, supra*, 178 Cal.App.4th at p. 464, 100 Cal.Rptr.3d 449.)

**\*\*335** The Court of Appeal disagreed. It held that the Legislature did not intend that “after a reasonable accommodation is granted, the interactive process continues to apply in a failure to accommodate context.” (*Albertsons, supra*, 178 Cal.App.4th at p. 464, 100 Cal.Rptr.3d 449.) The court held that to “graft an interactive process intended to apply to the determination of a reasonable accommodation onto a situation in which an employer failed to provide a reasonable, agreed-upon accommodation is contrary to the apparent intent of the FEHA and would not support the public policies behind that provision.” (*Ibid.*) Thus, a failure to engage in the interactive process cannot be used to support a failure to accommodate cause of action.

**\*1110** Here we have the inverse of *Albertsons*: the employee using a failure to accommodate in support of a claim of failure to engage in the interactive process. Brown alleged LAUSD agreed upon a reasonable accommodation (to hire a neutral expert to determine locations of minimal exposure) and then failed to follow through. We conclude Brown's allegations fit the logic of *Albertsons* holding. Without more, the allegations are insufficient under *Albertsons* to constitute a failure to engage in the interactive process.

*F. The Trial Court Did Not Err in Sustaining the Demurrer Without Leave to Amend*

[25] [26] The trial court sustained the demurrer without granting Brown leave to amend the FAC. Generally, leave to amend is warranted when the complaint is in some way defective, but plaintiff has shown in what manner the complaint can be amended and “ ‘how that amendment will change the legal effect of [the] pleading.’ ” (*Goodman v. Kennedy* (1976) 18 Cal.3d 335, 349, 134 Cal.Rptr. 375, 556 P.2d 737.) In her reply brief, Brown announced that she “need not specify additional details for an amended complaint because she already alleged more than sufficient ultimate facts to support her claims and any additional allegations would be superfluous evidentiary facts.” In the absence of proposed new facts, we find no error in the trial court’s decision not to grant leave to amend.

**DISPOSITION**

We reverse as to the cause of action for failure to accommodate. The judgment is affirmed in all other respects. Parties are to bear their own costs on appeal.

I concur:

**GRIMES**, Acting P. J.

**WILEY**, J., Concurring.

I join the court’s decision, which rejects a pleading challenge. For good reason, California state civil procedure makes complaints easy to write and hard to attack: experience shows litigation effort devoted solely to attacking pleadings is costly and time consuming and rarely yields much helpful information for litigants about the true value of their case. (Cf. Clermont & Yeazell, *Inventing Tests, Destabilizing Systems* (2010) 95 Iowa L.Rev. 821, 829–859 [critique of contrary federal practice that devotes much effort to testing litigation at the complaint stage].)

The consequence of this relatively lax state attitude is relatively easier access to discovery. But California trial judges have the tools and training to curb weaponized discovery.

\*1111 Instead of encouraging attacks at the pleading stage, ordinarily it is wiser for a procedural system to save the big

litigation investments for stages where judicial rulings can provide the parties with information \*\*336 that helps them agree on the case’s settlement value.

Yet even with our state’s healthy attitudes about easy pleading, I worry about giving any sort of green light to this unprecedented and unorthodox disability claim. Plaintiff’s counsel was most reluctant at oral argument to admit it, but it seems clear we are the first court in the United States of America—a nation of over 300 million people—to allow a claim that “Wi-Fi can make you sick.” Up till now, the main published appellate opinion seems to have been the one where Judge Posner wrote that a “great deal of psychological distress is trivial—fear of black cats, for example.” (*Hirmiz v. New Harrison Hotel Corp.* (7th Cir. 2017) 865 F.3d 475, 476.)

Millions use Wi-Fi. Merchants, employers, cafes, hotels—indeed, commercial concerns of every kind throughout the land have been installing Wi-Fi at an impressive pace. Nearly everyone wants the phenomenal convenience of the virtual world in your hand, everywhere you go, and the faster the better. All the potential defendants responding to this popular demand may take solemn note of news that, as of today, their Wi-Fi systems now may possibly invite costly litigation from members of the public who say that Wi-Fi made them sick. And potential plaintiffs and their counsel will have an interest too.

The law worries about junk science in the courtroom. One concern is that a partisan expert witness can bamboozle a jury with a commanding bearing, an engaging manner, and a theory that lacks respectable scientific support. (E.g., *Daubert v. Merrell Dow Pharmaceuticals, Inc.* (1993) 509 U.S. 579, 595, 113 S.Ct. 2786, 125 L.Ed.2d 469 (*Daubert*) [“ ‘Expert evidence can be both powerful and quite misleading because of the difficulty in evaluating it.’ ”].)

This concern is nothing new. The old fear is that “[e]xperience has shown that opposite opinions of persons professing to be experts may be obtained to any amount ....” (*Winans v. New York & Erie Railroad Co.* (1859) 62 U.S. (21 How.) 88, 101, 16 L.Ed. 68.)

“ ‘It is often surprising to see with what facility and to what an extent [experts’] views can be made to correspond with the wishes or interests of the parties who call them .... [T]heir judgment becomes so warped by regarding the subject in one point of view that even when conscientiously disposed, they are incapable of expressing a candid opinion.... They

are selected on account of their ability to express a favorable opinion, which, there is great reason to believe, is in many instances the result alone of employment and the bias growing out of it.’” (Foster, *Expert Testimony,—Prevalent Complaints and Proposed Remedies* (1897) 11 Harv. L.Rev. 169, 170–171; see **\*1112** Learned Hand, *Historical and Practical Considerations Regarding Expert Testimony* (1901) 15 Harv. L.Rev. 40, 53 (Learned Hand) [“the expert becomes a hired champion of one side”]; *id.* at pp. 54–55 [describing the “absurdity” and “evil” of the “present system”]; *id.* at p. 46 [recounting 1665 case where “Dr. Brown, of Norwich, was desired to state his opinion of the accused persons, and he was clearly of opinion that they were witches”].)

It does not take much experience as a trial judge in Los Angeles to realize the use of expert witnesses has run riot. To get a feel for the situation, try an internet search on “expert witness los angeles.” If your client has the budget, the available inventory is remarkable. Surprising numbers of these experts also happen to be lawyers—or perhaps, after reflection, this is not so surprising.

**\*\*337** The partisan expert witness has enormous potential as a weapon of pure advocacy. Excellent trial lawyers know this potential. They risk disadvantage and even defeat if they do not wring every drop of advocacy power from their retained experts. In this process, the search for truth can suffer. (E.g., Rubinfeld & Cecil, *Scientists as Experts Serving the Court* (Fall 2018) 147 *Daedalus* 152, 153 (Rubinfeld & Cecil).)

An expert witness can be the advocate's strongest ally. Mid-trial, after the opening statement and before the closing argument, the expert can argue the client's position in the most forceful terms, speaking directly to the judge and jury with a demeanor chosen for its fluent and compelling sincerity.

The expert's motivation can be prompted by ample compensation and guaranteed through careful selection. For the advocate, finding and selecting experts can be a momentous event in the litigation process. Resume horsepower is useful, but better yet is a captivating communication style married to the proper attitude.

What is the proper attitude? It can be a subtle thing, perhaps detected through give-and-take on casual and seemingly irrelevant issues during a private telephone call or a relaxed interview in a comfortable office. For the trial lawyer puzzling over whether to retain this expert, a core question is whether

the expert will become a team player. At some deep level, will the expert come to embrace the cause of the client?

Experts with the proper attitude willingly deploy their potentially awesome experience and intelligence in the advocate's service. The result is unlikely to involve lying or deception, if for no other reason than such conduct rarely survives cross-examination. The result is, however, likely to be highly partisan. And the highly partisan character of expert testimony can imperil the search for truth.

**\*1113** When one trial lawyer tells a colleague in an unguarded moment that the lawyer is “shopping for an expert,” we should reflect on how accurate this phrase truly is.

Our highest courts responded to these concerns by empowering trial judges to be gatekeepers and to sort the reliable from the speculative. (*Daubert, supra*, 509 U.S. at pp. 589–597, 113 S.Ct. 2786; *Sargon Enterprises, Inc. v. University of Southern California* (2012) 55 Cal.4th 747, 753, 149 Cal.Rptr.3d 614, 288 P.3d 1237.) Gatekeeping may be vital to the integrity of this particular case. And rulings on *Sargon* motions can give the parties information that is highly pertinent to the settlement value of a case.

Trial judges also have another tool in their kit: court-appointed experts. (See *Evid. Code*, §§ 730–732.) Preferably in consultation with counsel and avoiding ex parte contacts, the trial court can select and appoint an independent expert of unquestioned stature. The parties foot the bill. The expert can write a report, be deposed, testify, and be cross-examined, like any other expert. Crucially, the jury can learn this expert has been appointed by the court rather than hired by the parties.

The option of a court-appointed expert has been available in California for generations. Few judges have tried this option, though, because the parties *never* suggest it. The last thing trial lawyers want is another source of uncertainty in the case: something powerful and beyond their control. But the hard-working judges with experience “reported a high degree of satisfaction with the services provided by the expert ....” (Cecil & Willging, **\*\*338** *The Use of Court-Appointed Experts in Federal Court* (1994) 78 *Judicature* 41, 42; cf. Learned Hand, *supra*, 15 Harv. L.Rev. at p. 56 [advocating “a board of experts or a single expert, not called by either side, who shall advise the jury of the general propositions applicable to the case ....”].)

The trial court may want to consider this option in this case. It is more effort to go off the beaten path, but scholarly literature can help by surveying some practical aspects. (See generally, Rubinfeld & Cecil, *supra* [citing and discussing sources].)

This nation has a vast wealth of genuine scientific expertise, and the pandemic has been forcing our scientists to become familiar with video communication. The internet has reduced the significance of geographic distance.

You don't need a Nobel prize winner: excellent junior faculty and even graduate students can be vastly knowledgeable, motivated, and hungry to boot. After all, few scholars are accustomed to the rates at which California \*1114 lawyers

bill. Authentic and objective experts thus may be surprisingly affordable, given the scholarly world's commitment to public service and the prestige and satisfaction that can flow from a judicial appointment like this. And once you appoint that expert, it can be startling how fast the case settles.

With concern and hope, I join the majority opinion.

#### **All Citations**

60 Cal.App.5th 1092, 275 Cal.Rptr.3d 322, 387 Ed. Law Rep. 297, 2021 A.D. Cases 56,294, 21 Cal. Daily Op. Serv. 1690, 2021 Daily Journal D.A.R. 1624

#### **Footnotes**

- 1 All further statutory references are to the Government Code unless otherwise designated.
- 2 We gather from LAUSD's demurrer that Brown was unaware of LAUSD's "statutory obligation to undergo competitive bidding for any contracts until January 2017."

---

End of Document

© 2021 Thomson Reuters. No claim to original U.S. Government Works.

2021 WL 3573769

Only the Westlaw citation is currently available.

United States Court of Appeals,  
District of Columbia Circuit.

ENVIRONMENTAL HEALTH  
TRUST, et al., Petitioners  
v.  
FEDERAL COMMUNICATIONS  
COMMISSION and United  
States of America, Respondents

No. 20-1025 Consolidated with 20-1138

|  
Argued January 25, 2021

|  
Decided August 13, 2021

On Petitions for Review of an Order of the Federal  
Communications Commission

**Attorneys and Law Firms**

[W. Scott McCollough](#) argued the cause for petitioners. With him on the joint briefs were [Edward B. Myers](#) and Robert F. Kennedy, Jr.

[Sharon Buccino](#) was on the brief for amici curiae Natural Resources Defense Council and Local Elected Officials in support of petitioners.

Dan Kleiber and Catherine Kleiber, pro se, were on the brief for amici curiae Dan and Catherine Kleiber in support of petitioners.

[James S. Turner](#) was on the brief for amicus curiae Building Biology Institute in support of petitioners.

[Stephen L. Goodman](#) was on the brief for amicus curiae Joseph Sandri in support of petitioners.

[Ashley S. Boizelle](#), Deputy General Counsel, Federal Communications Commission, argued the cause for respondents. With her on the brief were [Jonathan D. Brightbill](#), Principal Deputy Assistant Attorney General at the time the brief was filed, U.S. Department of Justice, [Eric Grant](#), Deputy Assistant Attorney General at the time the brief was filed, [Jeffrey Beelaert](#) and [Justin Heminger](#), Attorneys,

[Thomas M. Johnson, Jr.](#), General Counsel at the time the brief was filed, Federal Communications Commission, [Jacob M. Lewis](#), Associate General Counsel, and William J. Scher and [Rachel Proctor May](#), Counsel. [Richard K. Welch](#), Deputy Associate General Counsel, entered an appearance.

Before: [HENDERSON](#), [MILLETT](#) and [WILKINS](#), Circuit Judges.

**Opinion**

Opinion dissenting in part filed by Circuit Judge [Henderson](#).

[Wilkins](#), Circuit Judge:

\*1 Environmental Health Trust and several other groups and individuals petition for review of an order of the Federal Communications Commission (“the Commission”) terminating a notice of inquiry regarding the adequacy of the Commission's guidelines for exposure to radiofrequency radiation. The notice of inquiry requested comment on whether the Commission should initiate a rulemaking to modify its guidelines. The Commission concluded that no rulemaking was necessary. Petitioners argue that the Commission violated the requirements of the Administrative Procedure Act by failing to respond to significant comments. Petitioners also argue that the National Environmental Policy Act required the Commission to issue an environmental assessment or environmental impact statement regarding its decision to terminate its notice of inquiry.

We grant the petitions in part and remand to the Commission. The Commission failed to provide a reasoned explanation for its determination that its guidelines adequately protect against the harmful effects of exposure to radiofrequency radiation unrelated to [cancer](#).

**I.**

The Federal Communications Commission regulates various facilities and devices that transmit radio waves and microwaves, including cell phones and facilities for radio, TV, and cell phone communications. [47 U.S.C. §§ 301, 302a\(a\)](#); see *EMR Network v. FCC*, 391 F.3d 269, 271 (D.C. Cir. 2004). Radio waves and microwaves are forms of electromagnetic energy that are collectively described by the term “radiofrequency” (“RF”). Office of Eng'g & Tech., Fed. Comm'ns Comm'n, *OET Bulletin No. 56, Questions and Answers about Biological Effects and Potential Hazards*

of *Radiofrequency Electromagnetic Fields* 1 (4th ed. Aug. 1999). The phenomenon of radio waves and microwaves moving through space is described as “RF radiation.” *Id.*

We often associate the term “radiation” with the term “radioactivity.” “Radioactivity,” however, refers only to the emission of radiation with enough energy to strip electrons from atoms. *Id.* at 5. That kind of radiation is called “ionizing radiation.” *Id.* It can produce molecular changes and damage biological tissue and DNA. *Id.* Fortunately, RF radiation is “non-ionizing,” meaning that it is not sufficiently energetic to strip electrons from atoms. *Id.* It can, however, heat certain kinds of materials, like food in your microwave oven or, at sufficiently high levels, human body tissue. *Id.* at 6–7. Biological effects that result from the heating of body tissue by RF energy are referred to as “thermal” effects, and are known to be harmful. *Id.* Exposure to lower levels of RF radiation might also cause other, “non-thermal” biological effects. *Id.* at 8. Whether it does, and whether such effects are harmful, are subjects of debate. *Id.*

The National Environmental Policy Act (“NEPA”) and its implementing regulations require federal agencies to “establish procedures to account for the environmental effects of [their] proposed actions.” *Am. Bird Conservancy, Inc. v. FCC*, 516 F.3d 1027, 1032 (D.C. Cir. 2008) (per curiam). If an agency proposes a “major Federal action[ ]” that stands to “significantly affect[ ] the quality of the human environment,” the agency must prepare an environmental impact statement (“EIS”) that examines the adverse environmental effects of the proposed action and potential alternatives. 42 U.S.C. § 4332(C). Not every agency action, however, requires the preparation of a full EIS. *Theodore Roosevelt Conservation P'ship v. Salazar*, 616 F.3d 497, 503 (D.C. Cir. 2010). If it is unclear whether a proposed action will “significantly affect[ ] the quality of the human environment,” 42 U.S.C. § 4332(C), the responsible agency may prepare a more limited environmental assessment (“EA”). See 40 C.F.R. § 1501.5(a). An EA serves to “[b]riefly provide sufficient evidence and analysis for determining whether to prepare an [EIS] or a finding of no significant impact.” 40 C.F.R. § 1501.5(c)(1). Additionally, an agency may use “categorical exclusions” to “define categories of actions that normally do not have a significant effect on the human environment and therefore do not require preparation of an environmental impact statement.” 40 C.F.R. § 1500.4(a); see also 40 C.F.R. § 1501.4(a).

\*2 To fulfill its obligations under NEPA, the Commission has promulgated guidelines for human exposure to RF radiation. *Cellular Phone Taskforce v. FCC*, 205 F.3d 82, 87 (2d Cir. 2000). The guidelines set limits for RF exposure. Before the Commission authorizes the construction or use of any wireless facility or device, the applicant for authorization must determine whether the facility or device is likely to expose people to RF radiation in excess of the limits set by the guidelines. 47 C.F.R. § 1.1307(b). If the answer is yes, the applicant must prepare an EA regarding the likely effects of the Commission's authorization of the facility or device. *Id.* Depending on the contents of the EA, the Commission may require the preparation of an EIS, and may subject approval of the application to a full vote by the Commission. Office of Eng'g & Tech., Fed. Comm'n's Comm'n, *OET Bulletin No. 65, Evaluating Compliance with FCC Guidelines for Human Exposure to Radiofrequency Electromagnetic Fields* 6 (ed. 97-01, Aug. 1997). If the answer is no, the applicant is generally not required to prepare an EA. 47 C.F.R. § 1.1306(a).

The Commission last updated its limits for RF exposure in 1996. *Resolution of Notice of Inquiry, Second Report and Order, Notice of Proposed Rulemaking, and Memorandum Opinion and Order*, 34 FCC Rcd. 11,687, 11,689–90 (2019) (“2019 Order”); see also Telecommunications Act of 1996, Pub. L. No. 104-104, § 704(b), 110 Stat. 56, 152 (directing the Commission to “prescribe and make effective rules regarding the environmental effects of radio frequency emissions” within 180 days). The limits are based on standards for RF exposure issued by the American National Standards Institute Committee (“ANSI”), the Institute of Electrical and Electronic Engineers, Inc. (“IEEE”), and the National Council on Radiation Protection and Measurements (“NCRP”). *In re Guidelines for Evaluating the Environmental Effects of Radiofrequency Radiation*, 11 FCC Rcd. 15,123, 15,134–35, 15,146–47 (1996). The limits are designed to protect against “thermal effects” of exposure to RF radiation, but not “non-thermal” effects. *EMR Network*, 391 F.3d at 271.

In March 2013, the Commission issued a notice of inquiry regarding the adequacy of its 1996 guidelines. See *Reassessment of Radiofrequency Exposure Limits & Policies, Notice of Inquiry*, 28 FCC Rcd. 3,498 (2013) (“2013 Notice of Inquiry”). The Commission divided its notice of inquiry into five sections. In the first section, it sought comment on the propriety of its exposure limits for RF radiation, particularly as they relate to device use by children. *Id.* at 3,575–80. In the second section, the Commission sought

comment on how to better provide information to consumers and the public about exposure to RF radiation and methods for reducing exposure. *Id.* at 3,580–82. In the third section, the Commission sought comment on whether it should impose additional precautionary restrictions on devices and facilities that are unlikely to expose people to RF radiation in excess of the limits set by the Commission's guidelines. *Id.* at 3,582–85. In the fourth and fifth sections, the Commission sought comment on whether it should change its methods for determining whether devices and facilities comply with the Commission's guidelines. *Id.* at 3,585–89.

The Commission explained that it was issuing the notice of inquiry in response to changes in the ubiquity of wireless devices and in scientific standards and research since 1996. *Id.* at 3,570. Specifically, the Commission noted that the IEEE had “published a major revision to its RF exposure standard in 2006.” *Id.* at 3,572. The Commission also noted that the International Commission on Non-Ionizing Radiation Protection had published RF exposure guidelines in 1998 that differed somewhat from the Commission's 1996 guidelines, and was likely to release a revision of those guidelines “in the near future.” *Id.* at 3,573. And the Commission noted that the International Agency for Research on Cancer (“IARC”) had classified RF radiation as possibly carcinogenic to humans, and was likely to release a detailed monograph regarding that classification prior to the resolution of the notice of inquiry. *Id.* at 3,575 & n.385. The Commission invited public comment on all of these developments, but underscored that it would “work closely with and rely heavily—but not exclusively—on the guidance of other federal agencies with expertise in the health field.” *Id.* at 3,571.

\*3 In December 2019, the Commission issued a final order resolving its 2013 notice of inquiry by declining to undertake any of the changes contemplated in the notice of inquiry. *See 2019 Order*, 34 FCC Rcd. at 11,692–97.

In January 2020, Petitioners Environmental Health Trust, Consumers for Safe Cell Phones, Elizabeth Barris, and Theodora Scarato timely petitioned this Court for review of the Commission's 2019 final order. In February 2020, Petitioners Children's Health Defense, Michele Hertz, Petra Brokken, Dr. David O. Carpenter, Dr. Paul Dart, Dr. Toril H. Jelter, Dr. Ann Lee, Virginia Farver, Jennifer Baran, and Paul Stanley, M.Ed., timely petitioned the Ninth Circuit for review of the same order, and the Ninth Circuit transferred their petition to this Court pursuant to 28 U.S.C. § 2112. This

Court consolidated the petitions. We have jurisdiction under 47 U.S.C. § 402(a) and 28 U.S.C. § 2342(1).

## II.

Petitioners challenge the 2019 final order under NEPA and the Administrative Procedure Act (“APA”). We begin with the APA.

### A.

Petitioners argue that the order is arbitrary and capricious and therefore must be set aside under 5 U.S.C. § 706(2) (A) for the following reasons: (1) the order fails to acknowledge evidence of negative health effects caused by exposure to RF radiation at levels below the limits set by the Commission's 1996 guidelines, including evidence of cancer, radiation sickness, and adverse effects on sleep, memory, learning, perception, motor abilities, prenatal and reproductive health, and children's health; (2) the order fails to respond to comments concerning environmental harm caused by RF radiation; (3) the order fails to discuss the implications of long-term exposure to RF radiation, exposure to RF pulsation or modulation (two methods of imbuing radio waves with information), and the implications of technological developments that have occurred since 1996, including the ubiquity of wireless devices and Wi-Fi, and the emergence of “5G” technology; (4) the order fails to adequately explain the Commission's refusal to modify its procedures for determining whether cell phones comply with its RF limits; and (5) the order fails to respond to various “additional legal considerations,” Pet'rs' Br. at 84.

Before discussing these arguments, and the Commission's responses to them, we clarify our standard of review. The arbitrary and capricious standard of the Administrative Procedure Act “encompasses a range of levels of deference to the agency.” *Am. Horse Prot. Ass'n v. Lyng*, 812 F.2d 1, 4 (D.C. Cir. 1987). We completely agree with the dissenting opinion that the Commission's order is entitled to a high degree of deference, both because it is akin to a refusal to initiate a rulemaking, *see id.* at 4–5, and because it concerns highly technical determinations of the kind courts are ill-equipped to second-guess, *see Am. Radio Relay League, Inc., v. FCC*, 524 F.3d 227, 233 (D.C. Cir. 2008). So as to the governing law, the dissenting opinion and we are on the same page. Nevertheless, the Commission's decision to terminate

its notice of inquiry must be “reasoned” if it is to survive arbitrary and capricious review. *See Am. Horse*, 812 F.2d at 5; *Am. Radio*, 524 F.3d at 241. As with other agency decisions not to engage in rulemaking, we will overturn the Commission's decision if there is “compelling cause, such as plain error of law or a fundamental change in the factual premises previously considered by the agency[.]” *Flyers Rights Educ. Fund, Inc. v. Fed. Aviation Admin.*, 864 F.3d 738, 743 (D.C. Cir. 2017) (quoting *WildEarth Guardians v. EPA*, 751 F.3d 649, 653 (D.C. Cir. 2014)). When an agency in the Commission's position is confronted with evidence that its current regulations are inadequate or the factual premises underlying its prior judgment have eroded, it must offer more to justify its decision to retain its regulations than mere conclusory statements. *See Am. Horse*, 812 F.2d at 6; *Am. Radio*, 524 F.3d at 241. Rather, the agency must provide “assurance that [it] considered the relevant factors,” and it must provide analysis that follows “a discernable path to which the court may defer.” *Am. Radio*, 524 F.3d at 241.

i.

\*4 Under this highly deferential standard of review, we find the Commission's order arbitrary and capricious in its failure to respond to record evidence that exposure to RF radiation at levels below the Commission's current limits may cause negative health effects unrelated to [cancer](#). (As we explain below, we find that the Commission offered an adequate explanation for its determination that exposure to RF radiation at levels below the Commission's current limits does not cause [cancer](#).) That failure undermines the Commission's conclusions regarding the adequacy of its testing procedures, particularly as they relate to children, and its conclusions regarding the implications of long-term exposure to RF radiation, exposure to RF pulsation or modulation, and the implications of technological developments that have occurred since 1996, all of which depend on the premise that exposure to RF radiation at levels below its current limits causes no negative health effects. Accordingly, we find those conclusions arbitrary and capricious as well. Finally, we find the Commission's order arbitrary and capricious in its complete failure to respond to comments concerning environmental harm caused by RF radiation.

Petitioners point to multiple studies and reports, which were published after 1996 and are in the administrative record, purporting to show that RF radiation at levels below the Commission's current limits causes negative health effects

unrelated to [cancer](#), such as reproductive problems and neurological problems that span from effects on memory to motor abilities. *See, e.g.*, J.A. 3,068 (BioInitiative Working Group, BioInitiative Report (Cindy Sage & David O. Carpenter eds., 2012) (describing evidence that human sperm and their DNA are damaged by low levels of RF radiation)); J.A. 5,243 (Igor Yakymenko et al., *Oxidative Mechanisms of Biological Activity of Low-Intensity Radiofrequency Radiation*, *Electromagnetic Biology & Med.*, Early Online, 1–16 (2015)); J.A. 5,259–69 (Henrietta Nittby et al., *Increased Blood-Brain Barrier Permeability in Mammalian Brian 7 Days After Exposure to the Radiation from a GSM-900 Mobile Phone*, 16 *Pathophysiology* 103 (2009)); J.A. 5,320–68 (Henry Lai, *A Summary of Recent Literature on Neurobiological Effects of Radiofrequency Radiation*, in *Mobile Communications and Public Health* 187–222 (M. Markov ed., 2018)); J.A. 5,994–6,007 (Milena Foerster et al., *A Prospective Cohort Study of Adolescents' Memory Performance and Individual Brain Dose of Microwave Radiation from Wireless Communication*, 126 *Env't Health Persps.* 077007 (July 2018)). Petitioners also point to approximately 200 comments submitted by individuals who advised the Commission that either they or their family members suffer from [radiation sickness](#), “a constellation of mainly neurological symptoms that manifest as a result of RF[ ] exposure.” Pet'rs' Br. at 30–31, 30 n.99.

The Commission argues that its order adequately responded to this evidence by citing the Food and Drug Administration (“FDA”)’s determination that exposure to RF radiation at levels below the Commission's current limits does not cause negative health effects. The order cites three statements from the FDA. First, the order cites an FDA webpage titled “Do cell phones pose a health hazard?” that, as of December 4, 2017, stated that “[t]he weight of scientific evidence has not linked cell phones with any health problems.” *2019 Order*, 34 *FCC Rcd.* at 11,692–93, 11,693 n.31. Second, the order cites a February 2018 statement from the Director of the FDA's Center for Devices and Radiological Health advising the public that

As part of our commitment to protecting the public health, the FDA has reviewed, and will continue to review, many sources of scientific and medical evidence related to the possibility of adverse health effects from radiofrequency energy exposure in both humans and animals and will continue to do so as new scientific data are published. Based on our ongoing evaluation of the issue, the totality of the available scientific evidence continues to not support

adverse health effects in humans caused by exposures at or under the current radiofrequency energy exposure limits. *Id.* at 11,695 n.42. Third, the order cites an April 2019 letter from the Director of the FDA's Center for Devices and Radiological Health that does not discuss non-cancer-related health effects but instead addresses a 2018 study by the National Toxicology Program that found that exposure to RF radiation emitted by cell phones may cause cancer in rodents. 2019 Order, 34 FCC Rcd. at 11,692 & n.28. The letter explains that “[a]s a part of our ongoing monitoring activities, we have reviewed the results and conclusions of the recently published rodent study from the National Toxicology Program in the context of all available scientific information, including epidemiological studies, and concluded that no changes to the current standards are warranted at this time.” Letter from Jeffrey Shuren, M.D., J.D., Dir., Ctr. for Devices & Radiological Health, Food & Drug Admin., Dep't of Health & Hum. Servs., to Julius Knapp, Chief, Off. Of Eng'g & Tech., FCC (April 24, 2019).

\*5 We do not agree that these statements provide a reasoned explanation for the Commission's decision to terminate its notice of inquiry. Rather, we find them to be of the conclusory variety that we have previously rejected as insufficient to sustain an agency's refusal to initiate a rulemaking. In *American Horse*, this Court considered whether the Secretary of Agriculture had offered a satisfactory explanation under the APA of his refusal to institute rulemaking proceedings regarding the practice of deliberately injuring show horses by fastening heavy chains or similar equipment—referred to as “action devices”—to the horses’ front limbs. 812 F.2d at 2. In response to the argument that a certain study presented facts that merited a new rulemaking, the Secretary offered the following two-sentence explanation:

6. I have reviewed studies and other materials, relating to action devices, presented by humane groups, Walking Horse industry groups, and independent institutions, including the study referred to in the Complaint.

7. On the basis of this information, I believe that the most effective method of enforcing the Act is to continue the current regulations.

*Id.* at 5. This Court found these “two conclusory sentences ... insufficient to assure a reviewing court that the agency's refusal to act was the product of reasoned decisionmaking.” *Id.* at 6. *American Horse* explained that the study at issue “may or may not remove a ‘significant factual predicate’ of the original rules’ gaps[.]” and remanded to the Secretary to make that determination. *Id.* at 7.

Similarly, in *American Radio*, this Court considered whether the Commission had offered a satisfactory explanation for its decision to retain in its regulations a particular “extrapolation factor”—an estimate of the projected rate at which radio frequency strength decreases from a radiation-emitting source—despite studies submitted in a petition for reconsideration indicating that a different extrapolation factor would be more appropriate. 524 F.3d at 240–41. The Commission explained its decision by asserting that “[n]o new information has been submitted that would provide a convincing argument for modifying the extrapolation factor ... at this time.” *Id.* (internal alterations omitted). We rejected that explanation as conclusory and unreasoned. *Id.*

The statements from the FDA on which the Commission's order relies are practically identical to the Secretary's statement in *American Horse* and the Commission's statement in *American Radio*. They explain that the FDA has reviewed certain information—here, “all,” “the weight,” or “the totality” of “scientific evidence.” And they state the FDA's conclusion that, in light of that information, exposure to RF radiation at levels below the Commission's current limits does not cause harmful health effects. But they offer “no articulation of the factual ... bases” for the FDA's conclusion. *Am. Horse*, 812 F.2d at 6 (internal quotation marks omitted). In other words, they do not explain why the FDA determined, despite the studies and comments that Petitioners cite, that exposure to RF radiation at levels below the Commission's current limits does not cause harmful health effects. Such conclusory statements “cannot substitute for a reasoned explanation,” for they provide “neither assurance that the [FDA] considered the relevant factors nor [do they reveal] a discernable path to which the court may defer.” *Am. Radio*, 524 F.3d at 241. They instead represent a failure by the FDA to address the implication of Petitioners’ studies: The factual premise—the non-existence of non-thermal biological effects—underlying the current RF guidelines may no longer be accurate.

When repeated by the Commission, the FDA's conclusory statements still do not substitute for the reasoned explanation that the APA requires. It is the Commission's responsibility to regulate radio communications, 47 U.S.C. § 301, and devices that emit RF radiation and interfere with radio communications, *id.* § 302a(a), and to do so in the public interest, including in regard to public health, *Banzhaf v. FCC*, 405 F.2d 1082, 1096 (D.C. Cir. 1968). Even the Commission itself recognizes this. *See* 2019 Order, 34 FCC Rcd. at 11,689

(“The Commission has the responsibility to set standards for RF emissions”); *2013 Notice of Inquiry*, 28 FCC Rcd. at 3,571 (explaining that the Commission opened the notice of inquiry “to ensure [it] [was] meeting [its] regulatory responsibilities” and that it would “work closely with and rely heavily—but not exclusively—on the guidance of other federal agencies with expertise in the health field” in order to “fully discharge[ ] [its] regulatory responsibility”) (emphasis added). And the APA requires that Commission’s decisions concerning the regulation of radio communications and devices be reasoned. The Commission’s purported reasoning in this case is that it chose to rely on the FDA’s evaluation of the studies in the record. Absent explanation from the FDA as to how and why it reached its conclusions regarding those studies, however, we have no basis on which to review the reasonableness of the Commission’s decision to adopt the FDA’s conclusions. Ultimately, the Commission’s order remains bereft of any explanation as to *why*, in light of the studies in the record, its guidelines remain adequate. The Commission may turn to the FDA to provide such an explanation, but if the FDA fails to do so, as it did in this case, the Commission must turn elsewhere or provide its own explanation. Were the APA to require less, our very deferential review would become nothing more than a rubber stamp.

\*6 The Commission also argues that its order provided a reasoned explanation for its decision to terminate the notice of inquiry, despite Petitioners’ evidence, by observing that “no expert health agency expressed concern about the Commission’s RF exposure limits,” and that “no evidence has moved our sister health and safety agencies to issue substantive policy recommendations for strengthening RF exposure regulation.” *2019 Order*, 34 FCC Rcd. at 11,692. The silence of other expert agencies, however, does not constitute a reasoned explanation for the Commission’s decision to terminate its notice of inquiry for the same reason that the FDA’s conclusory statements do not constitute a reasoned explanation: silence does not indicate why the expert agencies determined, in light of evidence suggesting to the contrary, that exposure to RF radiation at levels below the Commission’s current limits does not cause negative health effects unrelated to [cancer](#). Silence does not even indicate whether the expert agencies made any such determination, or whether they considered any of the evidence in the record.

Our decision in *EMR Network* is not to the contrary. There, we rejected the argument that the Commission improperly delegated its NEPA duties by relying on input from other government agencies and non-governmental expert

organizations in deciding whether to initiate a rulemaking to modify its RF radiation guidelines. 391 F.3d at 273. We found the Commission “not to have abdicated its responsibilities, but rather to have properly credited outside experts,” and noted that “the FCC’s decision not to leap in, at a time when the EPA (and other agencies) saw no compelling case for action, appears to represent the sort of priority-setting in the use of agency resources that is least subject to second-guessing by courts.” *Id.* (citing *Am. Horse*, 812 F.2d at 4). We agree with the dissenting opinion that the Commission may credit outside experts in deciding whether to initiate a rulemaking to modify its RF radiation guidelines. To be sure, “[a]gencies can be expected to respect the views of such other agencies as to those problems for which those other agencies are more directly responsible and more competent.” *City of Boston Delegation v. FERC*, 897 F.3d 241, 255 (D.C. Cir. 2018) (internal alteration and quotation marks omitted). What the Commission may not do, however, is rely on an outside expert’s silence or conclusory statements in lieu of some reasoned explanation for its decision. And while it is certainly true that an agency’s decision not to initiate a rulemaking at a time when other agencies see no compelling case for action may represent “the sort of priority-setting in the use of agency resources that is least subject to second-guessing by courts,” *EMR Network*, 391 F.3d at 273, the same is true of most agency decisions not to initiate a rulemaking, *see Am. Horse*, 812 F.2d at 4–5. Nevertheless, an agency’s decision not to initiate a rulemaking must have some reasoned basis, and an agency cannot simply ignore evidence suggesting that a major factual predicate of its position may no longer be accurate. *Id.* at 5.

Nor does *Cellular Phone Taskforce* help the Commission. There, the Second Circuit rejected the argument that the Commission was required to consult with the Environmental Protection Agency (“EPA”) or other outside agencies before declining to modify its RF radiation guidelines in the face of new evidence regarding non-thermal effects caused by RF radiation. 205 F.3d at 90–91. In so holding, the Second Circuit found that “[i]t was fully reasonable for the FCC to expect the agency with primacy in evaluating environmental impacts to monitor all relevant scientific input into the FCC’s reconsideration, particularly because the EPA had been assigned the lead role in RF radiation health effects since 1970,” and that the Commission was not required to “supply the new evidence to the other federal agencies with expertise in the area.” *Id.* at 91. But the Second Circuit did not hold that the Commission could rely solely on the silence or unexplained conclusions of other federal agencies to justify

its own inaction. It merely held that the Commission was not required to consult with outside agencies before declining to modify its RF radiation guidelines. No party before us today questions the propriety of that holding.

\*7 Finally, the Commission argues that the Commission itself addressed the major studies in the record in its order terminating the notice of inquiry. Specifically, the Commission points to its statement that “[t]he vast majority of filings were unscientific.” *2019 Order*, 34 FCC Rcd. at 11,694. Elsewhere, however, the order acknowledges that “the record include[d] some research information” and “filings that sought to present scientific evidence.” *Id.* The order dismisses that research and evidence as “fail[ing] to make a persuasive case for revisiting our existing RF limits,” *id.*, but again, such a conclusory statement cannot substitute for the minimal reasoning required at this stage, *Am. Radio*, 524 F.3d at 241. And while “[a]n agency is not obliged to respond to every comment, only those that can be thought to challenge a fundamental premise,” *MCI WorldCom, Inc. v. FCC*, 209 F.3d 760, 765 (D.C. Cir. 2000), the studies in the record to which Petitioners point *do* challenge a fundamental premise of the Commission's decision to terminate its notice of inquiry—namely, the premise that exposure to RF radiation at levels below the Commission's current limits does not cause negative health effects. But the Commission said nothing at all in its order about any specific health effects unrelated to [cancer](#).

The Commission also points to its statement that “the record [does not] include actionable alternatives or modifications to the current RF limits supported by scientifically rigorous data or analysis.” *2019 Order*, 34 FCC Rcd. at 11,692; *see also id.* at 11,694. Had the notice of inquiry focused exclusively on whether the Commission should modify its RF exposure limits, we might agree that the failure of any commenter to propose actionable modifications to the RF limits would have justified the Commission's decision to terminate the notice of inquiry. But the notice of inquiry did not focus exclusively on whether the Commission should modify its RF exposure limits. Instead, it also sought comment on how to better provide information to consumers and the public about exposure to RF radiation and methods for reducing exposure, and whether the Commission should impose additional precautionary restrictions on devices and facilities that are unlikely to expose people to RF radiation in excess of the Commission's limits. The Commission needed no actionable alternative to its current limits in order to provide additional information to the public or to impose

precautionary restrictions in addition to its current limits. The failure of any commenter to propose actionable modifications to the Commission's RF exposure limits therefore does not justify the Commission's decision to terminate the notice of inquiry.

ii.

The Commission's failure to provide a reasoned explanation for its determination that exposure to RF radiation at levels below its current limits does not cause negative health effects unrelated to [cancer](#) renders the order arbitrary and capricious in three additional respects. First, it undermines the Commission's explanation for retaining its procedures for determining whether cell phones and other portable electronic devices comply with its RF limits. These procedures consist of testing the device against the head of a specialized mannequin, *2013 Notice of Inquiry*, 28 FCC Rcd. at 3,586 n.434, and no more than 2.5 centimeters away from the body of the mannequin, *id.* at 3,588 n.447. Petitioners claim that the testing is inaccurate because of the space between the device and the mannequin's body. On this point, the Commission's order cites the “large safety margin” incorporated in its existing RF exposure limits as a justification for its refusal to modify these procedures to include testing against the body. *2019 Order*, 34 FCC Rcd. at 11,696. Because the Commission's existing RF limits are overprotective, the order explains, the Commission need not worry about whether its testing procedures accurately detect devices that are likely to expose people to RF emissions in excess of the Commission's limits. *See id.* (“[E]ven if certified or otherwise authorized devices produce RF exposure levels in excess of Commission limits under normal use, such exposure would still be well below levels considered to be dangerous, and therefore phones legally sold in the United States pose no health risks.”). As the Commission itself recognizes, this explanation depends on the premise that RF radiation does not cause harmful effects at levels below its current limits. *See id.* at 11,696 n.49 (“We note that any claim as to the adequacy of the FCC required testing, certification, and authorization regime is no different than a challenge to the adequacy of the federal RF exposure limits themselves. Both types of claims would undermine the FCC's substantive policy determinations.”). The Commission's failure to provide a reasoned explanation for its determination that exposure to RF radiation at levels below its current limits does not cause negative health effects therefore renders inadequate the

Commission's explanation for its refusal to modify its testing procedures.

\*8 Second, the Commission equally failed to provide a reasoned explanation for brushing off record evidence addressing non-cancer-related health effects arising from the impact of RF radiation on children. Many commenters, including the American Academy of Pediatrics, urged the Commission to adopt limits that account for the use of RF-emitting devices by vulnerable children and pregnant women. *See, e.g.*, J.A. 4,533–34. In dismissing those concerns, the Commission again relied on a conclusory statement from the FDA that “[t]he scientific evidence does not show a danger to any users of cell phones from RF exposure, including children and teenagers.” *2019 Order*, 34 FCC Rcd. at 11,696. But, as we have already explained, such a conclusory and unexplained statement is not the “reasoned” explanation required by the APA. In addition, the Commission noted that the testing to determine compliance with its limits “represents a conservative case” for both adults and children. *Id.* at 11,696 n.50. Whether the testing of compliance with existing limits was conservative is not the point. The unanswered question remains whether low levels of RF radiation allowed by those existing limits cause negative health effects. So once again, the Commission's failure to provide a reasoned or even relevant explanation of its position that RF radiation below the current limits does not cause health problems unrelated to cancer renders its explanation as to the effect of RF radiation on children arbitrary and capricious.

Third, the Commission's failure to provide a reasoned explanation for its determination that exposure to RF radiation at levels below its current limits does not cause negative health effects unrelated to cancer renders inadequate the Commission's explanation for its failure to discuss the implications of long-term exposure to RF radiation, exposure to RF pulsation or modulation, or the implications of technological developments that have occurred since 1996, including the ubiquity of wireless devices and Wi-Fi, and the emergence of “5G” technology. In its brief, the Commission responds that it was not required to address these topics in its order because it “rationally concluded that the weight of scientific evidence does not support the existence of adverse health effects from radiofrequency exposure below the FCC's limits, regardless of the service or equipment at issue.” Resp't's Br. at 45–46. (The Commission points out that “5G” cell towers, unlike traditional cell towers, are subject to its RF exposure limits.) Again, this explanation depends on the premise that RF radiation does not cause harmful

health effects at levels below the Commission's current limits, and will not suffice absent a reasoned explanation for the Commission's determination that that premise is correct.

### iii.

In addition to the Commission's inadequate response to the non-cancer-related effects of RF radiation on human health, the Commission also completely failed even to acknowledge, let alone respond to, comments concerning the impact of RF radiation on the environment. That utter lack of a response does not meet the Commission's obligation to provide a reasoned explanation for terminating the notice of inquiry. The record contains substantive evidence of potential environmental harms. Most relevantly, the record included a letter from the Department of the Interior voicing concern about the impact of RF radiation from communication towers on migratory birds, *see* J.A. 8,379, 8,383–86. In the Department of the Interior's expert view, the Commission's RF radiation limits “continue to be based on thermal heating, a criterion now nearly 30 years out of date and inapplicable today.” J.A. 8,383. “The [current environmental] problem,” according to the Department of the Interior, “appears to focus on very low-level, non-thermal electromagnetic radiation.” *Id.* Although the Commission has repeatedly claimed that it considered “inputs from [its] sister federal agencies[.]” *2019 Order*, 34 FCC Rcd. at 11,689, the Commission entirely failed to address the environmental harm concerns raised by the Department of the Interior. To be sure, the Commission could conclude that the link between RF radiation and environmental harms is too weak to warrant an amendment to its RF radiation limits. All we hold now is that the Commission should have said something about its sister agency's view rather than ignore it altogether. That lack of any reasoned explanation as to environmental harms does not satisfy the requirements of the APA.

### iv.

\*9 The dissenting opinion portrays this case as about the Commission's disregard of just five articles and one Department of Interior letter. Not so. The record contained substantial information and material from, for example, the American Academy of Pediatrics, J.A. 4,533; the Council of Europe, J.A. 4,242–44, 4,247–57; the Cities of Boston and Philadelphia, J.A. 4,592–99; medical associations, *see, e.g.*, J.A. 4,536–40 (California Medical Association); thousands

of physicians and scientists from around the world, *see, e.g.*, J.A. 4,197–4,206 (letter to United Nations); J.A. 4,208–17 (letter to European Union); J.A. 5,173–86 (Frieburger Appeal by over one thousand German physicians); and hundreds of people who were themselves or who had loved ones suffering from the alleged effects of RF radiation, *see, e.g.*, J.A. 8,774–9,940; *see also* J.A. 4,218–39 (collecting statements from physicians and health organizations expressing concern about health effects of RF radiation).

The dissenting opinion then offers its own explanation as to why those select sources were not worth being addressed by the agency. This in-the-weeds assessment of scientific studies and assessments falls “outside our bailiwick[.]” Dissenting Op. at ——. More to the point, the Commission said none of what the dissenting opinion does. If it had and if those six sources fairly represented the credible record evidence seeking a change in Commission policy, that discussion likely would have sufficed. But just as *post hoc* rationales offered by counsel cannot fill in the holes left by an agency in its decision, neither can a dissenting opinion. *See Grace v. Barr*, 965 F.3d 883, 903 (D.C. Cir. 2020) (“[W]hen ‘assessing the reasonableness of [an agency’s action], we look only to what the agency said at the time of the [action]—not to its lawyers’ post-hoc rationalizations.’”) (second and third alterations in original) (quoting *Good Fortune Shipping SA v. Commissioner*, 897 F.3d 256, 263 (D.C. Cir. 2018)).

Instead, the Commission chose to hitch its wagon to the FDA’s unexplained disinterest in some similar information. Importantly, the dissenting opinion does not dispute that the FDA’s conclusory dismissal of that evidence ran afoul of our precedent in *American Horse* and *American Radio*. It just says that the deficiency in the FDA’s analysis cannot be imputed to a second agency, and so the dissenting opinion would hold dispositive “the fact that the Commission and the FDA are, to state the obvious, distinct agencies.” Dissenting Op. at ——.

They certainly are. But that does not amount to a legal difference here. While imitation may be the highest form of flattery, it does not meet even the low threshold of reasoned analysis required by the APA under the deferential standard of review that governs here. One agency’s unexplained adoption of an unreasoned analysis just compounds rather than vitiates the analytical void. Said another way, two wrongs do not make a right. *Compare City of Tacoma v. FERC*, 460 F.3d 53, 76 (D.C. Cir. 2006) (“[T]he action agency must not blindly adopt the conclusions of the consultant agency, citing that agency’s expertise. Rather, the ultimate responsibility

for compliance with the [Endangered Species Act] falls on the action agency.”), and *Ergon-West Virginia, Inc. v. EPA*, 896 F.3d 600, 612 (4th Cir. 2018) (“Although the EPA is statutorily required to consider the [Department of Energy]’s recommendation, it may not turn a blind eye to errors and omissions apparent on the face of the report, which [petitioner] pointed out and the EPA did not address in any meaningful way. In doing so, the EPA ‘ignore[d] important aspects of the problem.’”) (internal citations omitted), with *Bellion Spirits, LLC v. United States*, No. 19-5252, — F.4th —, — — —, slip op. at 13-14 (D.C. Cir. Aug. 6, 2021) (approving consultation by the Alcohol and Tobacco Tax and Trade Bureau (“TTB”) with the FDA where the TTB “did not rubberstamp FDA’s analysis of the scientific evidence or delegate final decisionmaking authority to FDA,” but instead “systematically evaluated and explained its reasons for agreeing with FDA’s analysis of each scientific study” and “then made its own determinations” about the claims at hand).

## B.

\*10 Petitioners’ remaining challenges under the APA are unavailing.

Petitioners first argue that the Commission failed to respond to record evidence that exposure to RF radiation at levels below the Commission’s current limits may cause **cancer**. Specifically, Petitioners argue the Commission failed to mention the IARC’s classification of RF radiation as possibly carcinogenic to humans, and its 2013 monograph regarding that classification, on which the Commission’s notice of inquiry specifically sought comment. Petitioners also argue that the Commission failed to adequately respond to two 2018 studies—the National Toxicology Program (“NTP”) study and the Ramazzini Institute study—that found increases in the incidences of certain types of **cancer** in rodents exposed to RF radiation. Had these 2018 studies been available prior to the IARC’s publication of its monograph, Petitioners assert, the IARC would have likely classified RF radiation as “probably carcinogenic,” rather than “possibly carcinogenic.” This is so, according to Petitioners, because the IARC will classify an agent as “possibly carcinogenic” if there is “limited evidence” that it causes **cancer** in humans and animals, and as “probably carcinogenic” if there is “limited evidence” that it causes **cancer** in humans and “sufficient evidence” that it causes **cancer** in animals. In its 2013 monograph, the IARC found “limited evidence” that RF radiation causes **cancer** in humans and animals, and therefore classified RF

radiation as “possibly carcinogenic.” Int’l Agency for Resch. on Cancer, *Non-Ionizing Radiation, Part 2: Radiofrequency Electromagnetic Fields*, 102 IARC Monographs on the Evaluation of Carcinogenic Risks to Humans 419 (2013) (emphases omitted). Petitioners assert that the NTP and Ramazzini Institute studies provide “sufficient evidence” that RF radiation causes **cancer** in animals. Therefore, according to Petitioners, had those studies been available prior to the IARC’s publication of its monograph, the IARC would have found “limited evidence” that RF radiation causes **cancer** in humans and “sufficient evidence” that it causes **cancer** in animals, and would have accordingly classified RF radiation as “probably carcinogenic.”

Although the Commission’s failure to make any mention of the IARC monograph does not epitomize reasoned decision making, we find that the Commission’s order adequately responds to the record evidence that exposure to RF radiation at levels below the Commission’s current limits may cause **cancer**. In contrast to its silence regarding non-cancerous effects, the order provides a reasoned response to the NTP and Ramazzini Institute studies. It explains that the results of the NTP study “cannot be extrapolated to humans because (1) the rats and mice received RF radiation across their whole bodies; (2) the exposure levels were higher than what people receive under the current rules; (3) the duration of exposure was longer than what people receive; and (4) the studies were based on 2G and 3G phones and did not study WiFi or 5G.” *2019 Order*, 34 FCC Rcd. at 11,693 n.33. And the order cites a response to both studies published by the International Commission on Non-Ionizing Radiation Protection that provides a detailed explanation of various inconsistencies and limitations in the studies and concludes that “consideration of their findings does not provide evidence that radiofrequency EMF is carcinogenic.” Int’l Comm’n on Non-Ionizing Radiation Prot., ICNIRP Note on Recent Animal Carcinogenesis Studies 6 (2018), <https://www.icnirp.org/cms/upload/publications/ICNIRPnote2018.pdf>; *see also 2019 Order*, 34 FCC Rcd. at 11,693 n.34. Petitioners’ contention that the IARC would have classified RF radiation as “probably carcinogenic” had the NTP and Ramazzini Institute studies been published earlier is speculative, particularly in light of the International Commission on Non-Ionizing Radiation Protection’s evaluation of those studies. And the IARC monograph’s classification of RF radiation as “possibly carcinogenic” is not so contrary to the Commission’s determination that exposure to RF radiation at levels below

its current limits does not cause **cancer** as to render that determination arbitrary or capricious.

\*11 Petitioners also argue that the Commission’s order impermissibly fails to respond to various “additional legal considerations.” Specifically, Petitioners argue that the order (i) ignores “express invocations of constitutional, statutory and common law based individual rights,” including property rights and the rights of “bodily autonomy and informed consent”; (ii) fails to explain whether FCC regulation preempts rights and remedies under the Americans with Disabilities Act and the Fair Housing Act; (iii) does not assess the costs and benefits associated with maintaining the Commission’s current limits; (iv) does not resolve the question of whether “those advocating more protective limits have to prove the existing limits are inadequate,” or whether the Commission carries the burden of proving that its existing limits are adequate; and (v) overlooks that the Supreme Court’s decision in *Jacobson v. Massachusetts*, 197 U.S. 11, 25 S.Ct. 358, 49 L.Ed. 643 (1905), “flatly requires that the Commission allow for some remedy for those who suffer from exposure.” Pet’rs’ Br. at 84–101.

These arguments are not properly before us. The Communications Act provides that a petition for reconsideration is a “condition precedent to judicial review” of “questions of fact or law upon which the Commission ... has been afforded no opportunity to pass.” 47 U.S.C. § 405(a). We will accordingly only consider a question raised before us if “a reasonable Commission *necessarily* would have seen the question ... as part of the case presented to it.” *NTCH, Inc. v. FCC*, 841 F.3d 497, 508 (D.C. Cir. 2016) (quoting *Time Warner Ent. Co. v. FCC*, 144 F.3d 75, 81 (D.C. Cir. 1998)). Petitioners did not submit a petition for reconsideration to the Commission, and they point to no comments raising their “additional legal considerations” in such a manner as to necessarily indicate to the Commission that they were part of the case presented to it.

Although Petitioners assert that the “Cities of Boston and Philadelphia specifically flagged [the issue of whether FCC regulation preempts rights and remedies under the Americans with Disabilities Act and the Fair Housing Act] and sought clarification,” Pet’rs’ Br. at 86, they are incorrect. The Cities of Boston and Philadelphia merely observed that the Second Circuit’s decision in *Cellular Phone Taskforce* did not address whether “ ‘electrosensitivity’ [is] a cognizable disability under the Americans with Disabilities Act,” J.A. 4,598. And the Cities noted that “the FCC and its sister

regulatory agencies share responsibility for adherence to the ADA,” J.A. 4,598–99, and urged the Commission to “lead in advice to electrosensitive persons about prudent avoidance,” J.A. 4,599. This did not put the Commission on notice that the question whether FCC regulation preempts rights and remedies under the Americans with Disabilities Act and the Fair Housing Act was part of the case presented to it. Nor did a comment asserting that “[t]he telecommunications Act should not be interpreted to injure an identifiable segment of the population, exile them from their homes and their city, leave them no place where they can survive, and allow them no remedy under City, State or Federal laws or constitutions.” J.A. 10,190. And Petitioners point to no comments that did a better job of flagging their other “additional legal considerations” for the Commission. The Commission therefore did not have an opportunity to pass on these arguments, so we may not review them. 47 U.S.C. § 405(a).

### C.

Petitioners also argue that NEPA required the Commission to issue an EA or EIS regarding its decision to terminate its notice of inquiry.

Petitioners are wrong. The Commission was not required to issue an EA or EIS because there was no ongoing federal action regarding its RF limits. The Commission already published an assessment of its existing RF limits that “‘functionally’ satisfied NEPA’s requirements ‘in form and substance.’” *EMR Network*, 391 F.3d at 272 (quoting *Cellular Phone Taskforce*, 205 F.3d at 94–95). NEPA obligations attach only to “proposals” for major federal action. See 42 U.S.C. § 4332(c); see also 40 C.F.R. § 1502.5. Once an agency has satisfied NEPA’s requirements, it is only required to issue a supplemental assessment when “there remains major federal action to occur.” *W. Org. of Res. Councils v. Zinke*, 892 F.3d 1234, 1242 (D.C. Cir. 2018) (internal quotation marks omitted) (quoting *Marsh v. Ore. Nat’l Res. Council*, 490 U.S. 360, 374, 109 S.Ct. 1851, 104 L.Ed.2d 377 (1989)). An agency’s promulgation of regulations constitutes a final agency action that is not ongoing. *Id.* at 1243. Once an agency promulgates a regulation and complies with NEPA’s requirements regarding that regulation, it is not required to conduct any supplemental environmental assessment, even if its original assessment is outdated. *Id.* at 1242. Such is the case here. As we explained in *EMR Network* in response to the argument that new data required the Commission to issue a

supplemental environmental assessment of its RF guidelines under NEPA, “the regulations having been adopted, there is at the moment no ongoing federal action, and no duty to supplement the agency’s prior environmental inquiries.” 391 F.3d at 272 (internal quotation marks and citation omitted).

\*12 That the Commission voluntarily initiated an inquiry to “determine whether there is a need for reassessment of the Commission radiofrequency (RF) exposure limits and policies” does not change the analysis. *2013 Notice of Inquiry*, 28 FCC Rcd. at 3,501. As the Supreme Court explained long ago, “the mere contemplation of certain action is not sufficient to require an impact statement” under NEPA, *Kleppe v. Sierra Club*, 427 U.S. 390, 404, 96 S.Ct. 2718, 49 L.Ed.2d 576 (1976) (internal quotation marks omitted), because, as in this case, “the contemplation of a project and the accompanying study thereof do not necessarily result in a proposal for major federal action,” *id.* at 406, 96 S.Ct. 2718. See also *Pub. Citizen v. Off. of U.S. Trade Representatives*, 970 F.2d 916, 920 (D.C. Cir. 1992) (“In accord with *Kleppe*, courts routinely dismiss NEPA claims in cases where agencies are merely contemplating a particular course of action but have not actually taken any final action at the time of suit.”) (collecting cases). Were the Commission to propose revising its RF exposure guidelines, it might be required to prepare NEPA documentation. But since the Commission for now has not proposed to alter its guidelines, it need not yet conduct any new environmental review.

### III.

For the reasons given above, we grant the petitions in part and remand to the Commission to provide a reasoned explanation for its determination that its guidelines adequately protect against harmful effects of exposure to radiofrequency radiation unrelated to cancer. It must, in particular, (i) provide a reasoned explanation for its decision to retain its testing procedures for determining whether cell phones and other portable electronic devices comply with its guidelines, (ii) address the impacts of RF radiation on children, the health implications of long-term exposure to RF radiation, the ubiquity of wireless devices, and other technological developments that have occurred since the Commission last updated its guidelines, and (iii) address the impacts of RF radiation on the environment. To be clear, we take no position in the scientific debate regarding the health and environmental effects of RF radiation—we merely conclude that the Commission’s cursory analysis of material record

evidence was insufficient as a matter of law. As the dissenting opinion indicates, there may be good reasons why the various studies in the record, only some of which we have cited here, do not warrant changes to the Commission's guidelines. But we cannot supply reasoning in the agency's stead, *see SEC v. Chenery Corp.*, 318 U.S. 80, 87–88, 63 S.Ct. 454, 87 L.Ed. 626 (1943), and here the Commission has failed to provide any reasoning to which we may defer.

*So ordered.*

Karen LeCraft Henderson, Circuit Judge, dissenting in part: “[A] court is not to substitute its judgment for that of the agency.” *Motor Vehicle Mfrs. Ass'n v. State Farm Mut. Auto. Ins. Co.*, 463 U.S. 29, 43, 103 S.Ct. 2856, 77 L.Ed.2d 443 (1983). We thus must “uphold a decision of less than ideal clarity if the agency's path may reasonably be discerned.” *Id.* (quoting *Bowman Transp., Inc. v. Arkansas-Best Freight Sys., Inc.*, 419 U.S. 281, 286, 95 S.Ct. 438, 42 L.Ed.2d 447 (1974)). I believe my colleagues' limited remand contravenes these first principles of administrative law. Because I would deny the petitions in full, I respectfully dissent from Part II.A.i.–iv. and Part III of the majority opinion.

## I.

It is important to emphasize how deferential our standard of review is here—where, first, an agency's decision to terminate a notice of inquiry without initiating a rulemaking occurred after the agency opened the inquiry on its own and, second, the inquiry involves a highly technical subject matter at the frontier of science. As the majority recognizes, “[t]he arbitrary and capricious standard of the Administrative Procedure Act ‘encompasses a range of levels of deference to the agency.’ ” Maj. Op. — (quoting *Am. Horse Prot. Ass'n v. Lyng*, 812 F.2d 1, 4 (D.C. Cir. 1987)). The majority further acknowledges that the Federal Communications Commission's (Commission or FCC) “order is entitled to a high degree of deference.” *Id.* at —. And our precedent also makes plain that “[i]t is only in the rarest and most compelling of circumstances that this court has acted to overturn an agency judgment not to institute rulemaking.” *WWHT, Inc. v. FCC*, 656 F.2d 807, 818 (D.C. Cir. 1981); *see also Cellnet Commc'n, Inc. v. FCC*, 965 F.2d 1106, 1111 (D.C. Cir. 1992) (“an agency's refusal to initiate a rulemaking is evaluated with a deference so broad as to make the process akin to non-reviewability”). For the reasons that follow, I believe

the Commission's order does not fit those rarest and most compelling circumstances.

## A.

\*13 We have held that research articles containing tentative conclusions do not provide a basis for disturbing an agency's decision not to initiate rulemaking. *See EMR Network v. FCC*, 391 F.3d 269, 274 (D.C. Cir. 2004). Nevertheless, the majority rejects reaching the same conclusion here regarding the petitioners' assertion that radiofrequency (RF) radiation exposure below the Commission's limits can cause negative health effects unrelated to cancer. To do so, it relies on five research articles in an over 10,500-page record. *See* Maj. Op. at — — —. <sup>1</sup>

A close inspection of the five research articles confirms that they also “are nothing if not tentative.” *EMR Network*, 391 F.3d at 274. The Foerster article concludes “[o]ur findings do not provide conclusive evidence of causal effects and should be interpreted with caution until confirmed in other populations.” Joint Appendix (J.A.) 6,006 (Milena Foerster et al., *A Prospective Cohort Study of Adolescents' Memory Performance and Individual Brain Dose of Microwave Radiation from Wireless Communication*, 126 *Env't Health Persp.* 077007 (July 2018)) (emphases added). <sup>2</sup> The Lai article provides a similarly murky picture of the current science. *See* J.A. 5,320–68 (Henry Lai, *A Summary of Recent Literature (2007–2017) on Neurological Effects of Radiofrequency Radiation*, in *Mobile Commc'ns & Pub. Health* 187–222 (M. Markov ed., 2018)). In summarizing the results of human studies on the behavioral effects of RF radiation, the Lai article lists 31 studies that showed *no significant* behavioral effects compared to 20 studies that showed behavioral effects. *See* J.A. 5,327–32. Moreover, of the 20 studies that showed a behavioral effect, at least four found behavioral *improvements*, not negative health effects.

Even the Yakymenko article, which asserts that 93 of 100 peer-reviewed studies found low-intensity RF radiation induces oxidative effects in biological systems, fails to address the critical issue—whether RF radiation below the Commission's current limits can cause negative health effects. *See* J.A. 5,243–58 (Igor Yakymenko et al., *Oxidative Mechanisms of Biological Activity of Low-Intensity Radiofrequency Radiation*, *Electromagnetic Biology & Med.*, Early Online, 1–16 (2015)). Specifically, the Yakymenko article discusses the International Commission on Non-

Ionizing Radiation Protection's (ICNIRP) recommended RF exposure limit—a specific absorption rate of 2 W/kg. *See* J.A. 5,243–44. But the ICNIRP's recommended RF exposure limit is significantly higher than the Commission's current limit—0.08 W/kg averaged over the whole body and a peak spatial-average of 1.6 W/kg over any 1 gram of tissue. *See* 47 C.F.R. § 1.1310(c). Accordingly, it is uncertain how many, if any, of the referenced peer-reviewed studies were conducted at RF radiation levels below the Commission's current limits.<sup>3</sup>

\*14 Given this record, I believe we should have arrived at the same conclusion we did in *EMR Network*—“nothing in th[e]se studies so strongly evidenc[es] risk as to call into question the Commission's decision to maintain a stance of what appears to be watchful waiting.” *EMR Network*, 391 F.3d at 274. “An agency is not obliged to respond to every comment, only those that can be thought to challenge a fundamental premise.” *MCI WorldCom, Inc. v. FCC*, 209 F.3d 760, 765 (D.C. Cir. 2000). A review of the five articles on which the majority opinion relies makes plain that the articles do not challenge a fundamental premise of the Commission's order. Instead, it “cherry-pick[s] the factual record to reach [its] conclusion.” *Ortiz-Diaz v. U.S. Dep't of Hous. & Urb. Dev.*, 867 F.3d 70, 79 (D.C. Cir. 2017) (Henderson, J., concurring in the judgment).

My colleagues assert that “[t]he dissenting opinion portrays this case as about the Commission's disregard of just five articles.” Maj. Op. ——. But their attempt to “turn the tables” plainly fails. It is they who chose the five articles, *see* Maj. Op. ——, to rely on as the basis for their remand, *see id.* at — (“the Commission's order remains bereft of any explanation as to why, *in light of the studies in the record*, its guidelines remain adequate”) (emphasis altered); *id.* at — (“*the studies in the record* to which Petitioners point *do* challenge a fundamental premise of the Commission's decision to terminate its notice of inquiry”) (first emphasis added). I discuss the five articles *only* to demonstrate that the studies “are nothing if not tentative.” *EMR Network*, 391 F.3d at 274. Because the studies on which the majority relies plainly are tentative, they do not challenge a fundamental premise of the Commission's decision and therefore cannot provide the basis for the majority's limited remand under our precedent.<sup>4</sup>

B.

I reach the same conclusion regarding the majority's remand of the petitioners' environmental harm argument. *See* Maj. Op. ——. The majority relies on a 2014 letter from the U.S. Department of the Interior (Interior) to the U.S. Department of Commerce about, *inter alia*, the impact of communications towers on migratory birds. But the Interior letter itself concedes that “[t]o date, no independent, third-party field studies have been conducted in North America on impacts of tower electromagnetic radiation on migratory birds.” J.A. 8,383.

Moreover, the petitioners did not raise the Interior letter in the environmental harm section of their briefs. “We apply forfeiture to unarticulated [legal and] evidentiary theories not only because judges are not like pigs, hunting for truffles buried in briefs or the record, but also because such a rule ensures fairness to both parties.” *Jones v. Kirchner*, 835 F.3d 74, 83 (D.C. Cir. 2016) (alteration in original) (citation omitted). And finally, the environmental harm studies on which the petitioners *did* rely “are nothing if not tentative.” *EMR Network*, 391 F.3d at 274.<sup>5</sup>

C.

\*15 More importantly, the majority's limited remand runs afoul of our precedent on this precise subject matter. In *EMR Network*, the petitioner asked “the Commission to initiate an inquiry on the need to revise [its] regulations to address the non-thermal effects” of RF radiation. 391 F.3d at 271. In denying the petition, we concluded “the [Commission]'s decision not to leap in, at a time when the [Environmental Protection Agency (EPA)] (and other agencies) saw no compelling case for action, appears to represent the sort of priority-setting in the use of agency resources that is least subject to second-guessing by courts.” *Id.* at 273.

This time around, the majority faults the Commission for the U.S. Food and Drug Administration's (FDA) allegedly “conclusory statements” in response to the Commission's 2013 notice of inquiry. *See* Maj. Op. ——. The crux of the majority's position is that “[t]he statements from the FDA on which the Commission's order relies are practically identical to the Secretary's statement in *American Horse* and the Commission's statement in *American Radio*.” *Id.*<sup>6</sup> But the analogy to *American Horse* and *American Radio* does not hold water. The majority's Achilles' heel is the fact that the Commission and the FDA are, to state the obvious, distinct agencies.

In *American Horse*, the appellant relied on the results of a study commissioned by the U.S. Department of Agriculture (Agriculture) to support its request for revised Agriculture regulations. *Am. Horse*, 812 F.2d at 2–3. The study found that devices Agriculture had declined to prohibit caused effects falling within the statutory definition of the condition known as “sore”;<sup>7</sup> and the Congress had charged Agriculture to eliminate the practice of soring show horses. *Am. Horse*, 812 F.2d at 2–3. Against this backdrop, we found the Agriculture Secretary’s “two conclusory sentences [dismissing the need to revise agency regulations] ... insufficient to assure a reviewing court that the agency’s refusal to act was the product of reasoned decisionmaking.” *Id.* at 6. But an agency head’s terse dismissal of his own agency’s study is not the case here. First, as noted *supra*, there is no conclusive study in the record, much less one commissioned by the agency whose regulations are being considered for revision. Instead, the record contains dozens of highly technical studies from various sources—the credibility and findings of which we are ill-equipped to evaluate. And crucially, unlike in *American Horse*, the Commission requested the opinion of the FDA—the agency charged with “establish[ing] and carry[ing] out an electronic product radiation control program,” 21 U.S.C. § 360ii(a)—studied that opinion and explained why it relied thereon in making its decision.

Similarly, in *American Radio*, the studies summarily dismissed by the FCC were studies the FCC sought to evaluate *itself*; we remanded for the FCC to explain why it failed to do so. *See Am. Radio*, 524 F.3d at 241. Moreover, *American Radio* addressed the reasoning underlying the FCC’s *promulgation* of a rule, an action subjected to far less deference than an agency’s decision not to initiate a rulemaking.<sup>8</sup>

\*16 I believe the Commission reasonably relied on the conclusions of the FDA, the agency statutorily charged with protecting the public from RF radiation. *See* 21 U.S.C. § 360ii(a) (FDA “shall establish and carry out an electronic product radiation control program designed to protect the public health and safety from electronic product radiation”).<sup>9</sup> Our precedent is well-settled that “[a]gencies can be expected to ‘respect [the] views of such other agencies as to those problems’ for which those ‘other agencies are more directly responsible and more competent.’ ” *City of Bos. Delegation v. FERC*, 897 F.3d 241, 255 (D.C. Cir. 2018) (second alteration in original) (quoting *City of Pittsburgh v. Fed.*

*Power Comm’n*, 237 F.2d 741, 754 (D.C. Cir. 1956)). That is precisely what the Commission did here.

The Commission’s 2013 *Notice of Inquiry* explained that the Commission intended to rely on, *inter alia*, the FDA to determine whether to reassess its own RF exposure limits. *See In re Reassessment of Fed. Commc’ns Comm’n Radiofrequency Exposure Limits & Policies*, 28 FCC Rcd. 3,498, 3,501 ¶ 6 (2013) (2013 *Notice of Inquiry*) (“Since the Commission is not a health and safety agency, we defer to other organizations and agencies with respect to interpreting the biological research necessary to determine what [RF radiation] levels are safe.”). And the Commission has consistently deferred to expert health and safety agencies in this context. *See id.* at 3,572 ¶ 211 (RF exposure limits adopted in 1996 “followed recommendations received from the [EPA], the [FDA], and other federal health and safety agencies”).<sup>10</sup>

The Commission was true to its word. On March 22, 2019, it asked the FDA if changes to the RF exposure limits were warranted by the current scientific research.<sup>11</sup> On April 24, 2019, the FDA responded:

FDA is responsible for the collection and analysis of scientific information that may relate to the safety of cellphones and other electronic products. ... As we have stated publicly, ... the available scientific evidence to date does not support adverse health effects in humans due to exposures at or under the current limits, and ... the FDA is committed to protecting public health and continues its review of the many sources of scientific literature on this topic.

J.A. 8,187 (Letter from Jeffrey Shuren, M.D., J.D., Dir., Ctr. for Devices and Radiological Health, U.S. Food & Drug Admin., Dep’t of Health & Hum. Servs., to Julius Knapp, Chief, Off. of Eng’g & Tech., U.S. Fed. Commc’ns Comm’n (April 24, 2019)).<sup>12</sup> In my view, the Commission, relying on the FDA, reasonably concluded no changes to the current RF exposure limits were warranted at the time. *See In re Reassessment of Fed. Commc’ns Comm’n Radiofrequency Exposure Limits & Policies*, 34 FCC Rcd. 11,687, 11,691 ¶ 10 (2019) (2019 *Order*).

\*17 Simply put, the Commission’s reliance on the FDA is reasonable “[i]n the face of conflicting evidence at the frontiers of science.” *See Cellular Phone Taskforce v. FCC*, 205 F.3d 82, 90 (2d Cir. 2000). The majority takes issue with what it categorizes as “conclusory statements.” Maj.

Op. ——. But the Supreme Court's "*State Farm* [decision] does not require a word count; a short explanation can be a reasoned explanation." *Am. Radio*, 524 F.3d at 247 (Kavanaugh, J., dissenting in part). Brevity is even more understandable if the agency whose rationale is challenged relies on the agency the Congress has charged with regulating the matter.

Granted, "[w]hen an agency in the Commission's position is confronted with evidence that its current regulations are inadequate or the factual premises underlying its prior judgment have eroded, it must offer more to justify its decision to retain its regulations than mere conclusory statements." Maj. Op. ——. But the majority opinion rests on an inaccurate premise—the Commission was not confronted with evidence that its regulations are inadequate nor have the factual premises underlying its RF exposure limits eroded. Sifting through the record's technical complexity is outside our bailiwick. If the record here establishes one point, however, it is that there is no scientific consensus regarding the "non-thermal" effects, if any, of RF radiation on humans. More importantly, the FDA, not the Commission, made the allegedly "conclusory statements" with which the majority takes issue and I believe the Commission adequately explained why it relied on the FDA's expertise.<sup>13</sup>

\*18 As in *EMR Network*, the record does not "call into question the Commission's decision to maintain a stance of what appears to be watchful waiting." 391 F.3d at 274. To hold otherwise begs the question: what was the Commission supposed to do? It has no authority over the level of detail the FDA provides in response to the Commission's inquiry. It admits that it does not have the expertise "to interpret[ ] the biological research necessary to determine what [RF radiation] levels are safe." 2013 Notice of Inquiry, 28 FCC Rcd. at 3,501 ¶ 6. The Commission opened the 2013 Notice of Inquiry "as a matter of good government" despite its "continue[d] ... confidence in the current [RF] exposure limits." *Id.* at 3,570 ¶ 205. If it had reached a conclusion contrary to the FDA's, it most likely would have been attacked as *ultra vires*. For us to require the Commission to, in effect, "nudge" the FDA stretches both our jurisdiction as well as its authority beyond recognized limits.

Accordingly, I respectfully dissent from the limited remand set forth in Part II.A.i.–iv. and Part III of the majority opinion.<sup>14</sup>

#### All Citations

--- F.4th ----, 2021 WL 3573769

#### Footnotes

- 1 "The record in an informal rulemaking proceeding is 'a less than fertile ground for judicial review' and has been described as a 'sump in which the parties have deposited a sundry mass of materials.'" *Pro. Drivers Council v. Bureau of Motor Carrier Safety*, 706 F.2d 1216, 1220–21 (D.C. Cir. 1983) (quoting *Nat'l Res. Def. Council, Inc. v. SEC*, 606 F.2d 1031, 1052 (D.C. Cir. 1979)).
- 2 See also J.A. 5,995 ("[T]he health effects of [exposure to radiofrequency electromagnetic fields (RF-EMFs)] are still unknown. ... [T]o date studies addressing this topic have produced inconsistent results."); J.A. 6,005 ("Although we found decreases in figural memory, some experimental and epidemiological studies on RF-EMF found *improvements* in working memory performance.") (emphasis added).
- 3 The BioInitiative Report the majority opinion cites is hardly worth discussing because the self-published report has been widely discredited as a biased review of the science.
- 4 The majority's hand wave to other record information, see Maj. Op. —— – ———, does not carry the day. Rather than provide "substantial information," *id.* at ———, the cited material consists primarily of letters expressing generalized concerns about RF limits worldwide.
- 5 See, e.g., J.A. 5,231 (Albert Manville, II, *A Briefing Memorandum: What We Know, Can Infer, and Don't Yet Know about Impacts from Thermal and Non-Thermal Non-Ionizing Radiation to Birds and Other Wildlife* 2 (2016)) ("the direct relationship between electromagnetic radiation and wildlife health continues to be complicated and in cases involving non-thermal effects, still unclear"); J.A. 6,174 (Ministry of Env't & Forest, Gov't of India, *Report on Possible Impacts of Communication Towers on Wildlife Including Birds and Bees* 4 (2011)) ("exact correlation between radiation of communication towers and wildlife, are not yet very well established").
- 6 See *Am. Radio Relay League, Inc. v. FCC*, 524 F.3d 227 (D.C. Cir. 2008).

- 7 See 15 U.S.C. § 1821(3) (“The term ‘sore’ when used to describe a horse means that [as a result of any substance or device used on a horse’s limb] such horse suffers, or can reasonably be expected to suffer, physical pain or distress, inflammation, or lameness when walking, trotting, or otherwise moving ....”).
- 8 See, e.g., *ITT World Commc'ns, Inc. v. FCC*, 699 F.2d 1219, 1245–46 (D.C. Cir. 1983), *rev'd on other grounds*, 466 U.S. 463, 104 S.Ct. 1936, 80 L.Ed.2d 480 (1984) (“Where an agency promulgates rules, our standard of review is diffident and deferential, but nevertheless requires a searching and careful examination of the administrative record to ensure that the agency has fairly considered the issues and arrived at a rational result. Where, as here, an agency chooses *not* to engage in rulemaking, our level of scrutiny is even more deferential ...” (emphasis in original) (footnotes and internal quotations omitted)).
- 9 See also *In re Guidelines for Evaluating the Env't Effects of Radiofrequency Radiation*, 11 FCC Rcd. 15,123, 15,130 ¶ 18 (1996) (“The FDA has general jurisdiction for protecting the public from potentially harmful radiation from consumer and industrial devices and in that capacity is expert in RF exposures that would result from consumer or industrial use of hand-held devices such as cellular telephones.”).
- 10 See also *In re Guidelines for Evaluating the Env't Effects of Radiofrequency Radiation*, 12 FCC Rcd. 13,494, 13,505 ¶ 31 (1997) (“It would be impracticable for us to independently evaluate the significance of studies purporting to show biological effects, determine if such effects constitute a safety hazard, and then adopt stricter standards that [sic] those advocated by federal health and safety agencies. This is especially true for such controversial issues as non-thermal effects and whether certain individuals might be ‘hypersensitive’ or ‘electrosensitive.’ ”).
- 11 See J.A. 8,184 (Letter from Julius Knapp, Chief, Off. of Eng'g & Tech., U.S. Fed. Commc'ns Comm'n, to Jeffrey Shuren, M.D., J.D., Dir., Ctr. for Devices and Radiological Health, U.S. Food & Drug Admin. (March 22, 2019)) (“Given that existing studies are continually being evaluated as new research is published, and that the work of key organizations such as [the Institute of Electrical and Electronics Engineers] and ICNIRP is continuing, we ask FDA’s guidance as to whether any changes to the standards are appropriate at this time.”).
- 12 See also *Statement from Jeffrey Shuren, M.D., J.D., director of the FDA’s Center for Devices and Radiological Health on the recent National Toxicology Program draft report on radiofrequency energy exposure*, Food & Drug Admin. (Feb. 2, 2018), <https://www.fda.gov/news-events/press-announcements/statementjeffrey-shuren-md-jd-director-fdas-center-devices-and-radiological-health-recent-national> (Since 1999, “there have been hundreds of studies from which to draw a wealth of information about these technologies which have come to play an important role in our everyday lives. Taken together, all of this research provides a more complete picture regarding radiofrequency energy exposure that has informed the FDA’s assessment of this important public health issue, and given us the confidence that the current safety limits for cell phone radiation remain acceptable for protecting the public health. ... I want to underscore that based on our ongoing evaluation of this issue and taking into account all available scientific evidence we have received, we have not found sufficient evidence that there are adverse health effects in humans caused by exposures at or under the current radiofrequency energy exposure limits.”).
- 13 The majority asserts that “[o]ne agency’s unexplained adoption of an unreasoned analysis just compounds rather than vitiates the analytical void.” Maj. Op. —. As set out *supra*, however, the Commission adequately explained its reliance—for the past 25 years—on the FDA’s RF exposure expertise. Plus, after a review of “hundreds of studies,” the FDA’s conclusion is far from unreasoned. See *supra* note 12. And the two cases to which the majority points are inapposite. See Maj. Op. — (citing *City of Tacoma v. FERC*, 460 F.3d 53, 76 (D.C. Cir. 2006), and *Ergon-West Virginia, Inc. v. EPA*, 896 F.3d 600, 612 (4th Cir. 2018)). Importantly, unlike these petitions, neither case involves a decision not to initiate a rulemaking. As noted, inaction is reviewed under an especially deferential standard. It would be inappropriate to apply precedent using a less deferential standard to modify the standard applicable here. And finally, the Commission did not “blindly adopt the conclusions” of the FDA. See *City of Tacoma*, 460 F.3d at 76. Nor did it “turn a blind eye to errors and omissions apparent on the face of” the FDA’s conclusions. See *Ergon-West Virginia*, 896 F.3d at 612.
- The majority’s citation to *Bellion Spirits, LLC v. United States*, No. 19-5252, — F.4th — (D.C. Cir. Aug. 6, 2021), is even further afield. First, *Bellion Spirits* addressed a “statutory authority” question—it did not apply arbitrary and capricious review, much less the especially deferential standard applicable to a decision not to initiate a rulemaking. See *Bellion Spirits*, — F.4th at —, *slip op.* at 13. Second, to the extent *Bellion Spirits* is remotely relevant, I believe it supports my position. There, the Alcohol and Tobacco Tax and Trade Bureau “consulted with [the] FDA on a matter implicating [the] FDA’s expertise and then considered that expertise in reaching its own final decision.” *Id.* at —, *slip op.* at 14. Again, in my view, the Commission did the same thing.
- 14 Although I join Part II.B. of the majority opinion, I do not agree with the majority’s aside, contrasting the Commission’s purported silence regarding non-cancerous effects and its otherwise reasoned response. See Maj. Op. —. As

explained *supra*, I believe the Commission reasonably relied on the FDA's conclusion that RF radiation exposure below the Commission's limits does not cause negative health effects—cancerous or non-cancerous.

---

End of Document

© 2021 Thomson Reuters. No claim to original U.S. Government Works.

969 F.3d 1020

United States Court of Appeals, Ninth Circuit.

CITY OF PORTLAND, Petitioner,

v.

UNITED STATES of America;

Federal Communications

Commission, Respondents,

City and County of San Francisco;

City of Arcadia; City of Bellevue; City

of Brookhaven; City of Burien; City

of Burlingame; City of Chicago; City

of Culver City; City of Dubuque; City

of Gig Harbor; City of Kirkland; City

of Las Vegas; City of Lincoln; City of

Monterey; City of Philadelphia; City of

Piedmont; City of Plano; City of San Bruno;

City of San Jacinto; City of San Jose;

City of Santa Monica; City of Shafter;

County of Los Angeles; Howard County;

Michigan Municipal League; [CTIA - The](#)

[Wireless Association](#); Town of Fairfax;

Town of Hillsborough, Intervenors.

American Electric Power Service

Corporation; CenterPoint Energy Houston

Electric, LLC; Duke Energy Corporation;

Entergy Corporation; Oncor Electric

Delivery Company, LLC; Southern

Company; Tampa Electric Company;

Virginia Electric and Power Company;

Xcel Energy Services Inc., Petitioners,

v.

Federal Communications Commission;

United States of America, Respondents,

Verizon; US Telecom—The Broadband

Association, Respondents-Intervenors.

Sprint Corporation, Petitioner,

v.

Federal Communications Commission;

United States of America, Respondents,

City of Bowie, Maryland; City of Eugene,

Oregon; City of Huntsville, Alabama;

[City of Westminster, Maryland](#); [County](#)

[of Marin, California](#); City of Arcadia,

California; Culver City, California;

City of Bellevue, California; [City of](#)

[Burien, Washington](#); [City of Burlingame,](#)

[California](#); [City of Gig Harbor, Washington](#);

[City of Issaquah, Washington](#); [City of](#)

[Kirkland, Washington](#); [City of Las Vegas,](#)

[Nevada](#); City of Los Angeles, California;

City of Monterey, California; [City of](#)

[Ontario, California](#); [City of Piedmont,](#)

[California](#); City of Portland, Oregon;

City of San Jacinto, California; City of

San Jose, California; [City of Shafter,](#)

[California](#); [City of Yuma, Arizona](#);

County of Los Angeles, California;

Town of Fairfax, California; City of

New York, New York, Intervenors.

Verizon Communications, Inc., Petitioner,

v.

Federal Communications Commission;

United States of America, Respondents,

City of Arcadia, California; City of Bellevue,

California; [City of Burien, Washington](#);

[City of Burlingame, California](#); [City of Gig](#)

[Harbor, Washington](#); [City of Issaquah,](#)

[Washington](#); [City of Kirkland, Washington](#);

[City of Las Vegas, Nevada](#); City of Los

Angeles, California; City of Monterey,

California; [City of Ontario, California](#); [City](#)

[of Piedmont, California](#); City of Portland,

Oregon; City of San Jacinto, California;

City of San Jose, California; [City of Shafter,](#)

California; [City of Yuma, Arizona](#); County of Los Angeles, California; Culver City, California; City of New York, New York; Town of Fairfax, California, Intervenor.

Puerto Rico Telephone Company, Inc., Petitioner,

v.

Federal Communications Commission; United States of America, Respondents, City of Arcadia, California; City of Bellevue, California; [City of Burien, Washington](#); [City of Burlingame, California](#); [City of Gig Harbor, Washington](#); [City of Issaquah, Washington](#); [City of Kirkland, Washington](#); [City of Las Vegas, Nevada](#); City of Los Angeles, California; City of Monterey, California; [City of Ontario, California](#); [City of Piedmont, California](#); City of Portland, Oregon; City of San Jacinto, California; City of San Jose, California; [City of Shafter, California](#); [City of Yuma, Arizona](#); County of Los Angeles, California; Culver City, California; Town of Fairfax, California; City of New York, New York, Intervenor.

City of Seattle, Washington; [City of Tacoma, Washington](#); King County, Washington; League of Oregon Cities; League of California Cities; [League of Arizona Cities and Towns](#), Petitioners,

v.

Federal Communications Commission; United States of America, Respondents, [City of Bakersfield, California](#); City of Coconut Creek, Florida; [City of Lacey, Washington](#); [City of Olympia, Washington](#); City of Rancho Palos Verdes, California; [City of Tumwater, Washington](#); Colorado Communications and Utility Alliance;

Rainier Communications Commission; County of Thurston, Washington; City of Arcadia, California; City of Bellevue, Washington; [City of Burien, Washington](#); [City of Burlingame, California](#); [City of Gig Harbor, Washington](#); [City of Issaquah, Washington](#); [City of Kirkland, Washington](#); [City of Las Vegas, Nevada](#); City of Los Angeles, California; City of Monterey, California; [City of Ontario, California](#); [City of Piedmont, California](#); City of Portland, Oregon; City of San Jacinto, California; City of San Jose, California; [City of Shafter, California](#); [City of Yuma, Arizona](#); County of Los Angeles, California; Culver City, California; Town of Fairfax, California; City of New York, New York, Intervenor. City of San Jose, California; City of Arcadia, California; City of Bellevue, Washington; [City of Burien, Washington](#); [City of Burlingame, California](#); Culver City, California; Town of Fairfax, California; [City of Gig Harbor, Washington](#); [City of Issaquah, Washington](#); [City of Kirkland, Washington](#); [City of Las Vegas, Nevada](#); City of Los Angeles, California; County of Los Angeles, California; City of Monterey, California; [City of Ontario, California](#); [City of Piedmont, California](#); City of Portland, Oregon; City of San Jacinto, California; [City of Shafter, California](#); [City of Yuma, Arizona](#), Petitioners,

v.

Federal Communications Commission; United States of America, Respondents, [CTIA - The Wireless Association](#); Competitive Carriers Association; Sprint Corporation; Verizon Communications,

Inc.; City of New York, New York; [Wireless Infrastructure Association](#), Intervenor.

City and County of San Francisco, Petitioner,

v.

Federal Communications Commission; United States of America, Respondents.

City of Huntington Beach, Petitioner,

v.

Federal Communications Commission; United States of America, Respondents, City of Arcadia, California; City of Bellevue, Washington; [City of Burien, Washington](#); [City of Burlingame, California](#); [City of Gig Harbor, Washington](#); [City of Issaquah, Washington](#); [City of Kirkland, Washington](#);

[City of Las Vegas, Nevada](#); City of Los Angeles, California; City of Monterey, California; [City of Ontario, California](#); [City of Piedmont, California](#); City of Portland, Oregon; City of San Jacinto, California; City of San Jose, California; [City of Shafter, California](#); [City of Yuma, Arizona](#); County of Los Angeles, California; Culver City, California; Town of Fairfax, California; City of New York, New York, Intervenor.

Montgomery County, Maryland, Petitioner,

v.

Federal Communications Commission; United States of America, Respondents.

AT&T Services, Inc., Petitioner,

v.

Federal Communications Commission; United States of America, Respondents,

[City of Baltimore, Maryland](#); [City and County of San Francisco, California](#); Michigan Municipal League; [City of Albuquerque, New Mexico](#); National

League of Cities; [City of Bakersfield, California](#); Town of Ocean City, Maryland;

City of Brookhaven, Georgia; City of Coconut Creek, Florida; [City of Dubuque, Iowa](#); City of Emeryville, California; City

of Fresno, California; [City of La Vista, Nebraska](#); [City of Lacey, Washington](#); City

of Medina, Washington; [City of Olympia, Washington](#); [City of Papillion, Nebraska](#);

[City of Plano, Texas](#); City of Rancho Palos Verdes, California; City of Rockville,

Maryland; City of San Bruno, California;

[City of Santa Monica, California](#); [City of Sugarland, Texas](#); [City of Tumwater, Washington](#); [City of Westminster, Maryland](#); Colorado Communications

and Utility Alliance; [Contra Costa County, California](#); [County of Marin, California](#);

[International City/County Management Association](#); International Municipal

Lawyers Association; League of Nebraska Municipalities; National Association of

Telecommunications Officers and Advisors;

Rainier Communications Commission;

[Thurston County, Washington](#); [Town of Corte Madera, California](#); [Town of Hillsborough, California](#); Town of

Yarrow Point, Washington; City of Arcadia, California; City of Bellevue,

Washington; [City of Burien, Washington](#);

[City of Burlingame, California](#); City of Culver City, California; [City of Gig Harbor, Washington](#); [City of Issaquah, Washington](#); [City of Kirkland, Washington](#);

[City of Las Vegas, Nevada](#); City of Los Angeles, California; City of Monterey,

California; [City of Ontario, California](#);

[City of Piedmont, California](#); City of

Portland, Oregon; City of San Jacinto, California; City of San Jose, California; [City of Shafter, California](#); [City of Yuma, Arizona](#); County of Los Angeles, California; Town of Fairfax, California, Intervenors.

American Public Power Association, Petitioner,

v.

Federal Communications Commission; United States of America, Respondents, [City of Albuquerque, New Mexico](#); National League of Cities; City of Brookhaven, Georgia; [City of Baltimore, Maryland](#); [City of Dubuque, Iowa](#); Town of Ocean City, Maryland; City of Emeryville, California; Michigan Municipal League; [Town of Hillsborough, California](#); [City of La Vista, Nebraska](#); City of Medina, Washington; [City of Papillion, Nebraska](#); [City of Plano, Texas](#); City of Rockville, Maryland; City of San Bruno, California; [City of Santa Monica, California](#); [City of Sugarland, Texas](#); League of Nebraska Municipalities; National Association of Telecommunications Officers and Advisors; [City of Bakersfield, California](#); City of Fresno, California; City of Rancho Palos Verdes, California; City of Coconut Creek, Florida; [City of Lacey, Washington](#); [City of Olympia, Washington](#); [City of Tumwater, Washington](#); Town of Yarrow Point, Washington; [Thurston County, Washington](#); Colorado Communications and Utility Alliance; Rainier Communications Commission; [City and County of San Francisco, California](#); [County of Marin, California](#); [Contra Costa County, California](#); Town

[of Corte Madera, California](#); [City of Westminster, Maryland](#), Intervenors. City of Austin, Texas; [City of Ann Arbor, Michigan](#); County of Anne Arundel, Maryland; City of Atlanta, Georgia; [City of Boston, Massachusetts](#); City of Chicago, Illinois; Clark County, Nevada; City of College Park, Maryland; City of Dallas, Texas; District of Columbia; [City of Gaithersburg, Maryland](#); Howard County, Maryland; City of Lincoln, Nebraska; Montgomery County, Maryland; [City of Myrtle Beach, South Carolina](#); [City of Omaha, Nebraska](#); City of Philadelphia, Pennsylvania; [City of Rye, New York](#); City of Scarsdale, New York; City of Seat Pleasant, Maryland; City of Takoma Park, Maryland; Texas Coalition of Cities for Utility Issues; Meridian Township, Michigan; Bloomfield Township, Michigan; [Michigan Townships Association](#); Michigan Coalition To Protect Public Rights-of-way, Petitioners,

v.

Federal Communications Commission; United States of America, Respondents, [City of Albuquerque, New Mexico](#); National League of Cities; City of Brookhaven, Georgia; [City of Baltimore, Maryland](#); [City of Dubuque, Iowa](#); Town of Ocean City, Maryland; City of Emeryville, California; Michigan Municipal League; [Town of Hillsborough, California](#); [City of La Vista, Nebraska](#); City of Medina, Washington; [City of Papillion, Nebraska](#); [City of Plano, Texas](#); City of Rockville, Maryland; City of San Bruno, California; [City of Santa Monica, California](#); [City of](#)

[Sugarland, Texas](#); League of Nebraska Municipalities; National Association of Telecommunications Officers and Advisors; [City of Bakersfield, California](#); City of Fresno, California; City of Rancho Palos Verdes, California; City of Coconut Creek, Florida; [City of Lacey, Washington](#); [City of Olympia, Washington](#); [City of Tumwater, Washington](#); Town of Yarrow Point, Washington; [Thurston County, Washington](#); Colorado Communications and Utility Alliance; Rainier Communications Commission; [City and County of San Francisco, California](#); [County of Marin, California](#); [Contra Costa County, California](#); [Town of Corte Madera, California](#); [City of Westminster, Maryland](#), Intervenors.

City of Eugene, Oregon; City of Huntsville, Alabama; City of Bowie, Maryland, Petitioners,

v.

Federal Communications Commission; United States of America, Respondents, [City of Albuquerque, New Mexico](#); National League of Cities; City of Brookhaven, Georgia; [City of Baltimore, Maryland](#); [City of Dubuque, Iowa](#); Town of Ocean City, Maryland; City of Emeryville, California; Michigan Municipal League; [Town of Hillsborough, California](#); [City of La Vista, Nebraska](#); City of Medina, Washington; [City of Papillion, Nebraska](#); [City of Plano, Texas](#); City of Rockville, Maryland; City of San Bruno, California; [City of Santa Monica, California](#); [City of Sugarland, Texas](#); League of Nebraska Municipalities; National Association

of Telecommunications Officers and Advisors; [City of Bakersfield, California](#); City of Fresno, California; City of Rancho Palos Verdes, California; City of Coconut Creek, Florida; [City of Lacey, Washington](#); [City of Olympia, Washington](#); [City of Tumwater, Washington](#); Town of Yarrow Point, Washington; [Thurston County, Washington](#); Colorado Communications and Utility Alliance; Rainier Communications Commission; [City and County of San Francisco, California](#); [County of Marin, California](#); [Contra Costa County, California](#); [Town of Corte Madera, California](#); [City of Westminster, Maryland](#), Intervenors.

No. 18-72689, No. 19-70490, No. 19-70123, No. 19-70124, No. 19-70125, No. 19-70136, No. 19-70144, No. 19-70145, No. 19-70146, No. 19-70147, No. 19-70326, No. 19-70339, No. 19-70341, No. 19-70344

|  
Argued and Submitted February  
10, 2020 Pasadena, California

|  
Filed August 12, 2020

#### Synopsis

**Background:** Local governments, public and private utilities, and wireless service providers petitioned for review of orders of the Federal Communications Commission (FCC), 33 FCC Rcd. 7705, [2018 WL 3738326](#), 33 FCC Rcd. 9088, [2018 WL 4678555](#), relating to installation and management of small cell wireless facilities.

**Holdings:** The Court of Appeals, [Schroeder](#), Senior Circuit Judge, held that:

[1] limitation on fees that state and local governments could charge providers to deploy small cell wireless facilities was in accord with congressional directive in Telecommunications Act;

[2] requirement that aesthetic requirements imposed on small cell wireless facilities be no more burdensome than those imposed on providers of equivalent services exceeded scope of FCC's authority;

[3] requirement that aesthetic requirements imposed on small cell wireless facilities be "objective" was unduly vague;

[4] requirement that aesthetic requirements be "reasonable" was not unduly vague;

[5] time limits within which local governments had to act on applications constituted reasonable interpretation of Telecommunication Act;

[6] FCC did not act arbitrarily in refusing to deem applications to be granted if local governments failed to timely act;

[7] FCC reasonably determined that municipalities, in controlling providers' access to rights-of-way, were therefore subject to preemption under Telecommunications Act;

[8] FCC had authority under Telecommunications Act to remove barriers preventing wireless service providers from accessing existing utility poles owned by public power utilities; and

[9] FCC did not act arbitrarily or capriciously in imposing moratoria on state and local ordinances and practices that were either explicitly or having effect of barring small cell deployment.

Petitions granted in part, denied in part, and dismissed in part.

Bress, Circuit Judge, dissented in part and filed opinion.

West Headnotes (20)

- [1] **Administrative Law and Procedure** 🔑 Telecommunications  
**Telecommunications** 🔑 Standard and scope of review

Where terms of Telecommunications Act are ambiguous, court will defer to Federal Communications Commission's (FCC)

reasonable interpretations. Communications Act of 1934 § 1 et seq., 47 U.S.C.A. § 151 et seq.

- [2] **Administrative Law and Procedure** 🔑 Change of policy; reason or explanation

Where agency is departing from prior policy, court must look to see if it acknowledged that it was changing positions and gave good reasons for new policy. 5 U.S.C.A. § 706(2)(A), (C).

- [3] **Municipal Corporations** 🔑 Political Status and Relations

**States** 🔑 Telecommunications; wiretap  
**Telecommunications** 🔑 Preemption; interplay of federal, state and local laws

Federal Communications Commission (FCC) order limiting fees that state and local governments could charge wireless service providers to deploy small cell wireless facilities was in accord with congressional directive in Telecommunications Act, and not otherwise arbitrary, capricious, or contrary to law, even though FCC did not examine prohibitive effect of fees in each jurisdiction, where record supported FCC's factual determination that above-cost fees, in aggregate, were having prohibitive effect on national basis, FCC adopted presumptively permissible fee levels, and order permitted localities to charge fees above those levels where they could demonstrate that their actual costs exceeded presumptive levels. 5 U.S.C.A. § 706(2)(A), (C); Communications Act of 1934 § 253, 47 U.S.C.A. § 253.

- [4] **Zoning and Planning** 🔑 Telecommunications towers and facilities

Federal Communications Commission (FCC) order requiring that aesthetic requirements imposed on small cell wireless facilities be no more burdensome than those imposed on providers of functionally equivalent services exceeded scope of its authority under Telecommunications Act; Act permitted some

difference in treatment of different providers, so long as treatment was reasonable, and FCC failed to take into account differences among functionally equivalent, but physically different services. Communications Act of 1934 § 332, 47 U.S.C.A. § 332(c)(7)(B)(i)(I).

[5] **Administrative Law and Procedure** ➡ Statutory limitation

Agency may not rewrite clear statutory terms.

[6] **Zoning and Planning** ➡ Telecommunications towers and facilities

Federal Communications Commission (FCC) order requiring that aesthetic requirements imposed on small cell wireless facilities be “objective”—defined to mean that local regulation “must incorporate clearly-defined and ascertainable standards, applied in a principled manner”—was unduly vague; FCC failed to explain its conclusion that all subjective standards were without public benefit and addressed no public harm. Communications Act of 1934 §§ 253, 332, 47 U.S.C.A. §§ 253, 332.

[7] **Zoning and Planning** ➡ Telecommunications towers and facilities

Federal Communications Commission's (FCC) requirement that aesthetic requirements imposed on small cell wireless facilities be “reasonable” was not unduly vague or overbroad; FCC explained that reasonableness requirement resulted in preemption only if aesthetic regulations were not “technically feasible and reasonably directed” at remedying aesthetic harms.

[8] **Zoning and Planning** ➡ Time for determination

Federal Communications Commission's (FCC) requirement that local authorities act within 60 days to decide applications for installation

on existing wireless infrastructure and 90 days for all other applications—such as building, electric, road closure or other permits—was reasonable interpretation of Telecommunication Act provision requiring that such decisions be made within “reasonable period of time”; limiting time limits to zoning permits could lead states and localities to delay their consideration of other permits to thwart proposed deployment. Communications Act of 1934 § 332, 47 U.S.C.A. § 332(c)(7)(B)(ii).

1 Cases that cite this headnote

[9] **Zoning and Planning** ➡ Time for determination

**Zoning and Planning** ➡ Injunctive relief

Federal Communications Commission (FCC) did not act arbitrarily or capriciously in refusing to deem applications for small cell wireless facilities to be granted if local governments failed to act on applications within specified time limits, but instead requiring applicants to seek injunctive relief. Communications Act of 1934 § 332, 47 U.S.C.A. § 332(c)(7)(B)(ii).

1 Cases that cite this headnote

[10] **Municipal Corporations** ➡ Political Status and Relations

**Telecommunications** ➡ Preemption; interplay of federal, state and local laws

Federal Communications Commission (FCC) reasonably determined that municipalities, in controlling wireless service providers' access to rights-of-way, were not acting as owners of property, but instead were acting in regulatory manner, and were therefore subject to preemption under Telecommunications Act; rights-of-way, and manner in which municipalities exercised control over them, served public purpose, and they were regulated in public interest, not in cities' financial interests. Communications Act of 1934 § 253, 47 U.S.C.A. § 253.

**[11] Electricity** ⚡ Permit or consent by public authorities**Telecommunications** ⚡ Construction, Equipment and Maintenance; Towers

Federal Communications Commission (FCC) had authority under Telecommunications Act to remove barriers that would prevent wireless service providers from accessing existing utility poles owned by public power utilities for installation of small cell wireless facilities, despite Act provision governing utility pole attachment rates that contained express exclusion for government-owned utilities; regulation in question did not regulate rates, but instead was promulgated to ensure that state and local statutes did not have prohibitory effect on telecommunications services. Communications Act of 1934 §§ 224, 253, 47 U.S.C.A. §§ 224, 253.

**[12] Municipal Corporations** ⚡ Political Status and Relations**States** ⚡ Telecommunications; wiretap**Telecommunications** ⚡ Preemption; interplay of federal, state and local laws

Federal Communications Commission (FCC) did not act arbitrarily or capriciously in imposing moratoria on state and local ordinances and practices that were either explicitly or having effect of barring small cell deployment; FCC provided that municipal ordinances of general applicability would qualify as de facto moratoria only where delay caused by ordinances “continues for an unreasonably long or indefinite amount of time.” Communications Act of 1934 § 253, 47 U.S.C.A. § 253.

**[13] Eminent Domain** ⚡ Telecommunications

Federal Communications Commission (FCC) order limiting fees that state and local governments could charge wireless service providers to deploy small cell wireless facilities did not constitute physical taking without just compensation; order precluded local governments from charging unreasonable fees

when granting applications and limited cost recovery to actual costs, but continued to allow municipalities to deny access to property for number of reasons. U.S. Const. Amend. 5; Communications Act of 1934 § 253, 47 U.S.C.A. § 253.

**[14] Municipal Corporations** ⚡ Political Status and Relations**Telecommunications** ⚡ Preemption; interplay of federal, state and local laws

Federal Communications Commission (FCC) order requiring municipalities to respond to applications for use from small cell wireless installers within prescribed period of time or risk immediate control of its property did not violate Tenth Amendment; FCC was interpreting and enforcing Telecommunications Act, adopted by Congress pursuant to its delegated authority under Commerce Clause, to ensure that municipalities were not charging small cell providers unreasonable fees. U.S. Const. art. 1, § 8, cl. 3; U.S. Const. Amend. 10; Communications Act of 1934 §§ 253, 332, 47 U.S.C.A. §§ 253, 332.

**[15] States** ⚡ Powers Reserved to States

If power is delegated to Congress in Constitution, Tenth Amendment expressly disclaims any reservation of that power to states. U.S. Const. art. 1, § 8, cl. 3; U.S. Const. Amend. 10.

**[16] Telecommunications** ⚡ Pole attachments

Federal Communications Commission (FCC) rule prohibiting utility from requiring overlashers to conduct pre-overlashing engineering studies or to pay utility's cost of conducting such studies was reasonable attempt to prevent unnecessary costs for attachers, despite utilities' contention that rule undermined their authority to deny pole access; rule allowed overlashers and utilities to negotiate details of overlashing arrangement, and authorized utilities to require that overlashers give 15 days' notice to utilities prior to overlashing so that safety

concerns could be addressed. Communications Act of 1934 § 224, [47 U.S.C.A. § 224](#).

**[17] Telecommunications** 🔑 Pole attachments

Federal Communications Commission (FCC) rule prohibiting utilities from denying utility pole access to new attacher solely because of preexisting safety violation that attacher did not cause did not conflict with Telecommunications Act provision allowing utilities to deny access for “reasons of safety.” Communications Act of 1934 § 224, [47 U.S.C.A. § 224\(f\)\(2\)](#).

**[18] Telecommunications** 🔑 Pole attachments

Federal Communications Commission (FCC) rule permitting utility-approved contractors to prepare entire pole for attachment was not arbitrary and capricious, despite utilities' contention that permitting attachers to hire contractors to work on upper portion of poles jeopardized safety; rule contained provisions designed to mitigate any increased safety risks, and reasonably addressed delays caused by prior rule. Communications Act of 1934 § 224, [47 U.S.C.A. § 224](#).

**[19] Telecommunications** 🔑 Pole attachments

Federal Communications Commission (FCC) had authority under Telecommunications Act to regulate utility-owned pole attachments; FCC could not ensure nondiscriminatory access to poles without allowing make-ready work that would reposition utility attachments, as utilities could otherwise deny access to attachers based on pretextual reasons of insufficient capacity. Communications Act of 1934 § 224, [47 U.S.C.A. § 224](#).

**[20] Telecommunications** 🔑 Pole attachments

Federal Communications Commission's (FCC) rate reform rule, which established presumption that all telecommunication carriers were similarly situated and thus entitled to same utility pole attachment rates, was appropriate

exercise of FCC's regulatory authority under Telecommunications Act. Communications Act of 1934 § 224, [47 U.S.C.A. § 224](#).

**Attorneys and Law Firms**

\***1028** Petitioners/Intervenors [Joseph Van Eaton](#) (argued) and [John Gasparini](#), Best Best & Krieger LLP, Washington, D.C.; [Gail A. Karish](#), Best Best & Krieger LLP, Los Angeles, California; [Michael J. Watza](#), Kitch Drutchas Wagner Valitutti & Sherbrook, Detroit, Michigan; for Petitioners/Intervenors Cities of San Jose, Arcadia, Bellevue, Burien, Burlingame, Culver City, Gig Harbor, Issaquah, Kirkland, Las Vegas, Los Angeles, Monterey, Ontario, Piedmont, Portland, San Jacinto, Shafter, Yuma, Albuquerque, Brookhaven, Baltimore, Dubuque, Emeryville, La Vista, Medina, Papillion, Plano, Rockville, San Bruno, Santa Monica, Sugarland, Austin, Ann Arbor, Atlanta, Boston, Chicago, College Park, Dallas, Gaithersburg, Lincoln, Myrtle Beach, Omaha, Philadelphia, Rye, Scarsdale, Seat Pleasant, and Takoma Park; Los Angeles, Anne Arundel, Clark, Howard, and Montgomery Counties; Towns of Fairfax, Ocean City, and Hillsborough; Townships of Meridian and Bloomfield, Michigan Townships Association; District of Columbia; Michigan Coalition to Protect Public Rights-of-Way, National League of Cities, Michigan Municipal League, League of Nebraska Municipalities, and Texas Coalition of Cities for Utility Issues.

[Sean A. Stokes](#) (argued) and [James Baller](#), Baller Stokes & Lide PC, Washington, D.C., for Petitioner American Public Power Association.

[Eric P. Gotting](#) (argued), Keller and Heckman LLP, Washington, D.C., for Petitioners/Intervenors Montgomery County, Maryland; and International Municipal Lawyers Association; International City/County Management Association.

[Eric B. Langley](#) (argued) and [Robin F. Bromberg](#), Langley & Bromberg LLC, Birmingham, Alabama, for Petitioners American Electric Power Service Corporation, Duke Energy Corporation, Entergy Corporation, Oncor Electric Delivery Company, Southern Company, and Tampa Electric Company.

[Joshua S. Turner](#) (argued), [Sara M. Baxenberg](#), and [Boyd Garriott](#), Wiley Rein LLP, Washington, D.C.; [Thomas Power](#), Senior Vice President and General Counsel, CTIA - The

Wireless Association, Washington, D.C.; for Intervenor CTIA - The Wireless Association.

[Claire J. Evans](#) (argued) and [Christopher S. Huther](#), Wiley Rein LLP, Washington, D.C., for Intervenor US Telecom—The Broadband Association.

[Kenneth S. Fellman](#) and Gabrielle A. Daley, Kissinger & Fellman PC, Denver, Colorado; [Robert C. May III](#) and Michael D. Johnston, Telecom Law Firm PC, San Diego, California; for Petitioners/Intervenors Cities of Bakersfield, Coconut Creek, Fresno, Lacey, Olympia, Rancho Palos Verdes, Seattle, Tacoma, Tumwater; Town of Yarrow Point; King and Thurston Counties; League of Oregon Cities, League of California Cities, League of Arizona Cities and Towns, Colorado Communications and Utility Alliance, and Rainier Communications Commission.

[Brett H. Freedson](#), [Charles A. Zdebski](#), and [Robert J. Gastner](#), Eckert Seamans Cherin & Mellott LLC, Washington, D.C., for Petitioners CenterPoint Energy Houston Electric and Virginia Electric and Power Company.

[David D. Rines](#) and [Kevin M. Cookler](#), Lerman Senter PLLC, Washington, D.C., for Petitioner Xcel Energy Services.

[Christopher J. Wright](#) and [E. Austin Bonner](#), Harris Wiltshire & Grannis LLP, Washington, D.C., for Petitioner/Intervenor Sprint Corporation.

[Sean A. Lev](#) and [Frederick Gaston Hall](#), Kellogg Hansen Todd Figel & Frederick P.L.L.C., Washington, D.C., for Petitioner AT&T Services.

[Henry Weissmann](#), Munger Tolles & Olson LLP, Los Angeles, California; [Jonathan Meltzer](#), Munger Tolles & Olson LLP, Washington, D.C.; for Petitioner/Intervenor Verizon Communications.

[Megan L. Brown](#) and [Jeremy J. Broggi](#), Wiley Rein LLP, Washington, D.C., for Petitioner Puerto Rico Telephone Company.

[Tillman L. Lay](#) and [Jeffrey M. Bayne](#), Spiegel & McDiarmid LLP, Washington, D.C., [Dennis J. Herrera](#), City Attorney; Theresa L. Mueller, Chief Energy and Telecommunications Deputy; [William K. Sanders](#), Deputy City Attorney; Office of the City Attorney, San Francisco, California; for Petitioners/Intervenors Cities of Eugene, Huntsville, Bowie, Westminster; Town of Corte Madera; and Counties of San Francisco, Marin, and Contra Costa.

[Michael E. Gates](#), City Attorney; Michael J. Vigliotta, Chief Assistant City Attorney; Office of the City Attorney, Huntington Beach, California; for Petitioner City of Huntington Beach.

[Nancy L. Werner](#), General Counsel, Alexandria, Virginia, as and for Intervenor National Association of Telecommunications Officers and Advisors.

[Zachary W. Carter](#), Corporation Counsel; [Richard Dearing](#), [Claude S. Platton](#), and [Elina Druker](#), Attorneys; Office of Corporation Counsel, New York, New York; for Intervenor City of New York.

Amanda Kellar and [Charles W. Thompson Jr.](#), Rockville, Maryland; for Intervenors International Municipal Lawyers Association and International City/County Management Association.

[Jennifer P. Bagg](#), Harris Wiltshire & Grannis LLP, Washington, D.C., for Intervenor Competitive Carriers Association.

[Thomas Scott Thompson](#) and [Patrick Curran](#), Davis Wright Tremaine LLP, Washington, D.C.; for Intervenor Wireless Infrastructure Association. Respondents

[Sarah E. Citrin](#) (argued), [Scott M. Noveck](#) (argued), and [James M. Carr](#) (argued), Counsel; [Richard K. Welch](#), Deputy Associate General Counsel; [Jacob M. Lewis](#), Associate General Counsel; Thomas M. Johnson Jr., General Counsel; Federal Communications Commission, Washington, D.C.; [Robert B. Nicholson](#), Adam D. Chandler and [Patrick M. Kuhlmann](#), Attorneys; [Michael F. Murray](#), Deputy Assistant Attorney General; [Andrew C. Finch](#), Principal Deputy Assistant Attorney General; [Makan Delrahim](#), Assistant Attorney General; United States Department of Justice, Washington, D.C.; for Respondents United States of America and Federal Communications Commission. Amici Curiae

[James E. Moore](#) and [Tim R. Shattuck](#), Woods Fuller Shultz & Smith P.C., Sioux Falls, South Dakota, for Amicus Curiae Missouri Basin Municipal Power Agency.

[Ellen F. Rosenblum](#), Attorney General; [Benjamin Gutman](#), Solicitor General; [Rolf C. Moan](#), Senior Assistant Attorney General; Office of the Attorney General, Salem, Oregon; for Amicus Curiae State of Oregon.

[Thomas E. Montgomery](#), County Counsel; [Jeffrey P. Michalowski](#), Senior Deputy; Office of County Counsel, San Diego, California; for Amicus Curiae County of San Diego.

Spencer Q. Parsons, Beery Elsner & Hammond LLP, Portland, Oregon, for Amici Curiae Nebraska Municipal Power Pool and Lincoln Electric System.

[Gerit F. Hull](#), Jennings Strouss & Salmon PLC, Washington, D.C.; [Lisa G. McAlister](#), SVP & General Counsel for Regulatory Affairs; American Municipal Power Inc., Columbus, Ohio; for Amicus Curiae American Municipal Power Inc.

[Emily Fisher](#), [Aryeh Fishman](#), and Amanda Aspatore, Edison Electric Institute, Washington, D.C.; [Brett Kilbourne](#), Vice President Policy and General Counsel, Utilities Technology Council, Arlington, Virginia; [Jeffrey L. Sheldon](#) and [Stephen J. Rosen](#), Levine Blaszak Block & Boothby LLP, Washington, D.C.; [Brian O'Hara](#), Senior Director Regulatory

Issues, National Rural Electric Cooperative Association, Arlington, Virginia; for Amici Curiae Edison Electric Institute, Utilities Technology Council, and National Rural Electric Cooperative Association.

[Matthew A. Love](#), Van Ness Feldman LLP, Seattle, Washington, for Amicus Curiae Northwest Public Power Association.

[Sblend A. Sblendorio](#), [Mallory L. Homewood](#), and Cara Mae Acibo, Hoge Fenton Jones & Appel Inc., Pleasanton, California, for Amicus Curiae Berkshire-Litchfield Environmental Council.

[Terry M. Jarrett](#), Healy Law Offices LLC, Jefferson City, Missouri, for Amici Curiae Iowa Association of Municipal Utilities Association, Missouri Association of Municipal Utilities, and Arkansas Municipal Power Association.

[W. Scott Snyder](#), Ogden Murphy Wallace, Seattle, Washington, for Amicus Curiae Association of Washington Cities.

David A. Rosenfeld, Weinberg Roger & Rosenfeld, Alameda, California, for Amici Curiae Communications Workers of America, National Digital Inclusion Alliance, and Public Knowledge.

[Jane Luckhardt](#), General Counsel, Northern California Power Agency, Roseville, California; Jody Lamar Finklea, General

Counsel & Chief Legal Officer; Dan O'Hagan, Assistant General Counsel & Regulatory Compliance Counsel; Florida Municipal Power Agency, Tallahassee, Florida; [James N. Horwood](#) and [Latif M. Nurani](#), Spiegel & McDiarmid LLP, Washington, D.C.; for Amici Curiae Northern California Power Agency; Municipal Electric Power Association of Virginia; Florida Municipal Electric Association, Inc.; City of Fort Meade; Fort Pierce Utilities Authority; City of Jacksonville Beach (Beaches Energy Services); Utility Board of the City of Key West, Florida (Keys Energy Services); Kissimmee Utility Authority; City of Lakeland (Lakeland Electric); City of Mount Dora; Utilities Commission, City of New Smyrna Beach; Orlando Utilities Commission; and City of Wauchula.

On Petitions for Review of Orders of the Federal Communications Commission, FCC No. 18-111, FCC Nos. 18-133, 83-fr-51867

Before: [Mary M. Schroeder](#), [Jay S. Bybee](#), and [Daniel A. Bress](#), Circuit Judges.

Partial Dissent by Judge [Bress](#)

## OPINION

[SCHROEDER](#), Circuit Judge:

### \*1031 I. INTRODUCTION

These matters arise out of the wireless revolution that has taken place since 1996 when Congress passed amendments to the Telecommunications Act to support the then nascent technology. The revolution now represents the triumph of cellular technology over just about everything else in telecommunications services.

The newest generation of wireless broadband technology is known as “5G” and requires the installation of thousands of “small cell” wireless facilities. These facilities have become subject to a wide variety of local regulations. The Federal Communications Commission (FCC) in 2018 therefore promulgated orders relating to the installation and management of small cell facilities, including the manner in which local governments can regulate them. The principal orders we review here thus constitute the FCC's contemporary \*1032 response to these technological and regulatory developments. These orders were promulgated

under the authority of a statute Congress enacted very early in the era of cellular communication, the Telecommunications Act of 1996, to encourage the expansion of wireless communications.

That expansion has been met with some resistance where 5G is concerned, however, particularly from local governments unhappy with the proliferation of cell towers and other 5G transmission facilities dotting our urban landscapes. Petitioners seeking review of the FCC orders thus include numerous local governments, the lead Petitioner being the City of Portland, Oregon. Also unhappy with the expanded installation of 5G technology contemplated by the FCC's orders are public and private power utilities, whose utility poles are often used for wireless facility deployment. Here as well are wireless service providers, who largely support the FCC's orders, but argue the FCC should have gone even further in restricting the authority of state and local governments.

Before us are three FCC orders, issued in 2018, that deal with myriad issues arising from the application of a twentieth century statute to twenty-first century technology. The two orders we deal with first are known as the Small Cell Order and the Moratoria Order. *Accelerating Wireless Broadband Deployment by Removing Barriers to Infrastructure Inv.*, 33 FCC Rcd. 9088 (2018) [hereinafter *Small Cell Order*]; *Accelerating Wireless Broadband Deployment by Removing Barriers to Infrastructure Inv.*, 33 FCC Rcd. 7705, 7775–91 (2018) [hereinafter *Moratoria Order*]. The Orders spell out the limits on local governments' authority to regulate telecommunications providers.

The FCC's statutory authority for limiting local regulation on the deployment of this technology is contained in Sections 253(a) and 332(c)(7) of the Act and reflects congressional intent in 1996 to expand deployment of wireless services. Those provisions authorize the FCC to preempt any state and local requirements that “prohibit or have the effect of prohibiting” any entity from providing telecommunications services. *See* 47 U.S.C. § 253(a), (d).

Many of the issues before us concern whether challenged provisions constitute excessive federal regulation outside the scope of that congressional preemption directive, as understood by our Circuit's leading case interpreting the statute, *Sprint Telephony PCS, L.P. v. County of San Diego*, 543 F.3d 571 (9th Cir. 2008) (en banc). We conclude that, given the deference owed to the agency in interpreting

and enforcing this important legislation, the Small Cell and Moratoria Orders are, with the exception of one provision, in accord with the congressional directive in the Act, and not otherwise arbitrary, capricious, or contrary to law. *See* 5 U.S.C. § 706(2)(A).

The exception is the Small Cell Order provision dealing with the authority of local governments in the area of aesthetic regulations. We hold that to the extent that provision requires small cell facilities to be treated in the same manner as other types of communications services, the regulation is contrary to the congressional directive that allows different regulatory treatment among types of providers, so long as such treatment does not “unreasonably discriminate among providers of functionally equivalent services.” 47 U.S.C. § 332(c)(7)(B)(i)(I). We also hold that the FCC's requirement that all aesthetic criteria must be “objective” lacks a reasoned explanation.

The third FCC order before us is intended to prevent owners and operators of utility poles from discriminatorily denying or delaying 5G and broadband service providers access to the poles. \*1033 *Accelerating Wireless Broadband Deployment by Removing Barriers to Infrastructure Inv.*, 33 FCC Rcd. 7705, 7705–91 (2018). Known as the “One-Touch Make-Ready Order,” it was issued pursuant to the Pole Attachment Act originally passed in 1978 and expanded in the wake of the Telecommunications Act of 1996. 47 U.S.C. § 224. Section 224 of that Act allows utilities to deny access to pole attachers under some circumstances. Several utilities object to discrete aspects of the One-Touch Make-Ready Order. We uphold the Order, concluding that the FCC reasonably interpreted Section 224 as a matter of law, and the Order is not otherwise arbitrary or capricious.

## II. STATUTORY AND INTERPRETIVE FRAMEWORK AND BACKGROUND

What we know as 5G technology is so named because it is the fifth generation of cellular wireless technology. It is seen as transformational because it provides increased bandwidth, allows more devices to be connected at the same time, and is so fast that connected devices receive near instantaneous responses from servers.

Although 5G transmits data at exceptionally fast speeds, it does so over relatively short distances. For this reason, wireless providers must use smaller power-base stations in

more locations, as opposed to the fewer, more powerful base stations used for 4G data transmission. These smaller base stations, known as “small cells,” are required in such numbers that 5G technology is currently being deployed on a city-by-city basis. *See generally* Brian X. Chen, *What You Need to Know About 5G in 2020*, N.Y. Times (Jan. 8, 2020), [https://www.nytimes.com/2020/01/08/technology/personaltech/5g-mobile-network.html?](https://www.nytimes.com/2020/01/08/technology/personaltech/5g-mobile-network.html?searchResultPosition=1)

[searchResultPosition=1](https://www.nytimes.com/2020/01/08/technology/personaltech/5g-mobile-network.html?searchResultPosition=1); Clare Duffy, *What Is 5G? Your Questions Answered*, CNN Business (Mar. 6, 2020), <https://www.cnn.com/interactive/2020/03/business/what-is-5g/index.html>; Sascha Segan, *What Is 5G?*, PCMag (Apr. 6, 2020), <https://www.pcmag.com/news/what-is-5g>. The prospective proliferation of “small cell” structures throughout our cities, coupled with the inevitable efforts of local governments to regulate their looks and location, gave rise to the FCC's Small Cell and Moratoria Orders—with which local governments are not entirely happy and which were issued under the general provisions of a decades-old statute.

The heart of these proceedings therefore lies in the early efforts of Congress, and now the FCC, to balance the respective roles of the federal government and local agencies in regulating telecommunications services for a rapidly changing technological world. A key statute in these proceedings is Section 253 of the Act. Entitled “Removal of Barriers to Entry,” it reflects Congress's intent to encourage expansion of telecommunication service. Section 253(a) provides that “[n]o state or local statute or regulation ... may prohibit or have the effect of prohibiting ... telecommunications service.” 47 U.S.C. § 253(a). At the same time Section 253(c) provides that state or local governments can manage public rights-of-way and require reasonable compensation for their use. 47 U.S.C. § 253(c).

In dealing with mobile services, Section 332(c)(7) similarly preserves local zoning authority while recognizing some specific limitations on traditional authority to regulate wireless facilities. 47 U.S.C. § 332(c)(7); *see City of Rancho Palos Verdes v. Abrams*, 544 U.S. 113, 115, 125 S.Ct. 1453, 161 L.Ed.2d 316 (2005) (explaining that section 332(c)(7) “imposes specific limitations on the traditional authority of state and local governments to regulate the location, construction, and modification of ... facilities”). Section 332(c)(7) also contains a limitation on local \*1034 authority nearly identical to Section 253(a). *See* 47 U.S.C. § 332(c)(7)(B)(i)(II) (“The regulation of the placement, construction, and modification of personal wireless service facilities by

any State or local government ... shall not prohibit or have the effect of prohibiting the provision of personal wireless services.”). The other major limitation on local authority relates to ensuring fair treatment of different services. *See* 47 U.S.C. § 332(c)(7)(B)(i)(I). Under that limitation, local governments “shall not unreasonably discriminate among providers of functionally equivalent services.” *Id.* Section 332(c)(7) further requires that state or local governments act on requests for placement of personal wireless service facilities “within a reasonable period of time.” 47 U.S.C. § 332(c)(7)(B)(ii). We deal with issues pertaining to all of these provisions in the challenges to the Small Cell and Moratoria Orders.

In the One-Touch Make-Ready Order, the FCC was concerned with facilitating attachment of new cellular facilities to existing utility poles. The FCC's authority to regulate pole attachments is found in Section 224 of the Act. That section provides that the FCC “shall regulate the rates, terms, and conditions” imposed upon pole attachments by utilities to ensure that such rates are “just and reasonable,” 47 U.S.C. § 224(b)(1), but expressly exempts entities “owned by the Federal Government or any State” from its definition of “utility,” *id.* § 224(a)(1). Section 224 also requires utilities to allow service providers “nondiscriminatory access” to its poles, *id.* § 224(f)(1), permitting utilities to deny access “on a non-discriminatory basis where there is insufficient capacity and for reasons of safety, reliability and generally applicable engineering purposes,” *id.* § 224(f)(2).

In their petitions, private utilities contend several provisions of the One-Touch Make-Ready Order violate Section 224 or are otherwise arbitrary or capricious in restricting a utility's ability to deny access to attachers. We uphold this Order in all respects.

As relevant to this litigation, the most disputed provision of the Act has been Section 253(a). The provision says that “[n]o State or local statute or regulation, or other State or local legal requirement, may prohibit or have the effect of prohibiting the ability of any entity to provide any interstate or intrastate telecommunications service.” 47 U.S.C. § 253(a). Soon after the Act's passage, the FCC decided *California Payphone Association*, concerning the location of the now antiquated, but formerly ubiquitous, payphone technology. 12 FCC Rcd. 14,191 (1997). The FCC considered a local regulation that prohibited the installation of payphones on private property outdoors, and held it was not an actual or effective prohibition of services, because phones could still be installed indoors on

public or private property, and outdoors on public property. *Id.* at 14,210. The FCC therefore held the requirement did not “materially inhibit[ ]” payphone service. *Id.* at 14,210.

This court's leading case interpreting Section 253 is our en banc decision in *Sprint*, 543 F.3d 571. We there straightened out an errant panel decision that had been concerned with the phrase “no State or local statute or regulation ... may prohibit ...” in Section 253. That decision read the phrase to mean that Section 253 preempted any state or local regulation that “might possibly” have the effect of prohibiting service. *Id.* at 578. We held in *Sprint* that more than “the mere possibility” of prohibition was required to trigger preemption. *Id.* There must be an actual effect, and we recognized the continuing validity of the material inhibition test from *California Payphone*. See *id.* (“[W]e note \*1035 that our interpretation is consistent with the FCC's.”).

Many of the issues we must decide here involve contentions by Petitioners that various provisions of the Small Cell and Moratoria Orders limit state and local regulatory authority to a greater degree than that contemplated in the Act, as interpreted by *California Payphone* and *Sprint*. The application of the FCC's “material inhibition” standard thus comes into play when we consider a number of the challenged provisions.

As a threshold issue, Local Government Petitioners argue that the FCC must demonstrate that an “actual prohibition” of services is occurring before preempting any municipal regulations, and that anything less than that showing is contrary to Section 253(a) and our decision in *Sprint*. We must reject this argument. The FCC's application of its standard in the Small Cell and Moratoria Orders is consistent with *Sprint*, which endorsed the material inhibition standard as a method of determining whether there has been an effective prohibition. The FCC here made factual findings, on the basis of the record before it, that certain municipal practices are materially inhibiting the deployment of 5G services. Nothing more is required of the FCC under *Sprint*.

Local Government Petitioners raise a corollary general objection to the Small Cell and Moratoria Orders, contending that the FCC, without a reasoned explanation, has departed from its prior approach in *California Payphone*, and has made it much easier to show an effective prohibition. *California Payphone*'s material inhibition standard remains controlling, however. The FCC has explained that it applies a little differently in the context of 5G, because state and local

regulation, particularly with respect to fees and aesthetics, is more likely to have a prohibitory effect on 5G technology than it does on older technology. The reason is that when compared with previous generations of wireless technology, 5G is different in that it requires rapid, widespread deployment of more facilities. See, e.g., *Small Cell Order* ¶ 53 (explaining that “even fees that might seem small in isolation have material and prohibitive effects on deployment, particularly when considered in the aggregate given the nature and volume of anticipated Small Wireless Facility deployment” (footnote omitted)). The differences in the FCC's new approach are therefore reasonably explained by the differences in 5G technology.

We therefore turn to Petitioners' challenges to specific provisions of the Orders. We deal with the Small Cell and Moratoria Orders together. Both Orders relate to the ways state and local governments can permissibly regulate small cell facilities.

### III. SMALL CELL AND MORATORIA ORDERS

The FCC initiated proceedings leading to the Small Cell and Moratoria Orders in response to complaints from wireless service providers. They reported that a variety of state and local regulations and practices were delaying and inhibiting small cell deployment nationwide in violation of Section 253. Those state and local governments now seek review of the Orders. We here summarize the challenged provisions of each Order.

The FCC issued the Moratoria Order in August 2018, and the Small Cell Order the following month. Two principal types of state and local regulation the agency considered relate to fees and aesthetic requirements. The FCC concluded such requirements frequently materially inhibit 5G deployment. The FCC found that when state and local governments charge excessive fees for wireless facility applications, \*1036 the cumulative impact of such charges amounts to an effective prohibition of deployment in other parts of the country. The FCC therefore limited the fees that a state or local government can assess, above a safe harbor amount, to the government's approximate costs. Specifically, the fee is permissible only if it is a “reasonable approximation of the state or local government's costs” of processing applications and managing the rights-of-way. *Small Cell Order* ¶ 50.

With respect to local aesthetic requirements, the FCC concluded such regulations were materially inhibiting small cell deployment within the meaning of the *California Payphone* standard. A key provision of the Small Cell Order sets out the applicable criteria: aesthetic restrictions are preempted unless they are (1) reasonable, (2) no more burdensome than requirements placed on other facilities, and (3) objective and published in advance. *Id.* ¶ 86. To qualify as a “reasonable” aesthetic requirement, an ordinance must be both “technically feasible and reasonably directed to avoiding or remedying the intangible public harm of unsightly or out-of-character deployments.” *Id.* ¶ 87.

Another important provision of the Small Cell Order modified the rules for when local jurisdictions have to act on wireless permitting requests, the so-called “shot clock” rules. Nearly a decade earlier, the FCC adopted the first shot clock rules, requiring zoning authorities to decide applications for wireless facility deployment on existing structures within ninety days, and all other applications for zoning permits within 150 days. *Petition for Declaratory Ruling*, 24 FCC Rcd. 13,994 (2009) [hereinafter *2009 Order*]; see *City of Arlington v. FCC*, 668 F.3d 229, 235–36 (5th Cir. 2012), *aff’d*, 569 U.S. 290, 133 S.Ct. 1863, 185 L.Ed.2d 941 (2013). Under the 2009 Order, when a local zoning authority exceeded a shot clock, it was presumed that the municipality violated the statutory requirement to respond within a reasonable time. *City of Arlington*, 668 F.3d at 236. When a local zoning authority failed to act within the proscribed time, the permit applicant could then file a lawsuit seeking a declaration that the city’s delay was unreasonable, and the city would have the opportunity to rebut the presumed statutory violation. *2009 Order* ¶¶ 37–38.

The 2018 Small Cell Order broadens the application of these shot clocks to include all telecommunications permits, not just zoning permits, and it shortens the shot clocks. State and local governments now have sixty days to decide applications for installations on existing infrastructure, and ninety days for all other applications. *Small Cell Order* ¶¶ 104–05, ¶ 132, ¶ 136. The Order does not add enforcement mechanisms. If a state or local government misses a permitting deadline, the applicant still must seek an injunction.

In the Moratoria Order, the FCC found that municipal actions that halt 5G deployment, deemed “moratoria,” violate Section 253(a) of the Act when they effectively prohibit the deployment of 5G technology. The FCC recognized two general moratoria categories: express and de facto. As

with the Small Cell Order, the Moratoria Order does not specifically preempt or invalidate any particular state or local requirement. See *Moratoria Order* ¶ 150. (“[W]e do not reach specific determinations on the numerous examples discussed by parties in our record....”). It lays out the applicable standards.

### A. Challenges to the Small Cell Order

[1] [2] Following the publication of the Small Cell Order, Local Government and Public Power Petitioners filed these petitions for review, asserting a number of legal challenges. We evaluate these challenges \*1037 under the Administrative Procedure Act by examining whether “an agency’s decreed result [is] within the scope of its lawful authority,” and whether “the process by which it reaches [a given] result [is] logical and rational.” *Michigan v. EPA*, 576 U.S. 743, 135 S. Ct. 2699, 2706, 192 L.Ed.2d 674 (2015) (internal quotation marks omitted); see 5 U.S.C. § 706(2) (A), (C). Where terms of the Telecommunications Act are ambiguous, we defer to the FCC’s reasonable interpretations. *City of Arlington*, 569 U.S. at 296–97, 133 S.Ct. 1863; see *Chevron v. Nat. Res. Def. Council*, 467 U.S. 837, 104 S.Ct. 2778, 81 L.Ed.2d 694 (1984). And where the FCC is departing from prior policy, we look to see if it acknowledged that it was changing positions, and gave “good reasons for the new policy.” *FCC v. Fox Television Stations, Inc.*, 556 U.S. 502, 515, 129 S.Ct. 1800, 173 L.Ed.2d 738 (2009).

To the extent that Petitioners challenge factual findings, we review them for substantial evidence, that is, evidence “a reasonable mind might accept as adequate to support a conclusion.” *Biestek v. Berryhill*, — U.S. —, 139 S. Ct. 1148, 1154, 203 L.Ed.2d 504 (2019) (internal quotation marks omitted). “[W]hatever the meaning of substantial in other contexts, the threshold for such evidentiary sufficiency is not high.” *Id.* (internal quotation marks omitted).

The Small Cell Order covers three major subjects and sets out the standards by which local regulations will be judged in determining whether they are preempted. Local Government Petitioners are not happy with any of them. The subjects are fees, aesthetics, and the time for approving permit applications (shot clocks). We deal with each of them in turn.

#### 1. Fees

State and local governments generally charge a wireless service provider fees to deploy facilities in their jurisdictions. These fees include one-time fees for new wireless facility deployment, as well as recurring annual fees on existing facilities in the public rights-of-way. The FCC concluded in the Small Cell Order that some of these fees were so excessive that they were effectively prohibiting the nationwide deployment of 5G technology and were therefore preempted. The Order places conditions on fees above a certain level to avoid preemption: fees must be: “(1) a reasonable approximation of the state or local government's costs, (2) [with] only objectively reasonable costs ... factored into those fees, and (3) ... no higher than the fees charged to similarly-situated competitors in similar situations.” *Small Cell Order* ¶ 50 (footnote omitted).

The Small Cell Order does not require a cost basis for all fees to avoid preemption. There is a safe harbor. Fees are presumptively lawful if, for each wireless facility, application fees are less than \$500, and recurring fees are less than \$270 per year. *Id.* ¶ 79. If fees exceed those levels, they are not automatically preempted, but can be justified. Localities may charge fees above these levels where they can demonstrate that their actual costs exceed the presumptive levels. *Id.* ¶ 80 & n.234.

The FCC offers two principal rationales for limiting fees above the safe harbor to costs. When local governments charge fees in excess of their costs, they take funds of wireless service providers that would otherwise be used for additional 5G deployment in other jurisdictions. Statements in the record from wireless service providers, and an empirical study, are cited to support the conclusion that limiting fees will lead to additional, faster deployment of 5G technology throughout the country. See *Small Cell Order* ¶¶ 61–64. The FCC explained that high fees also reduce the availability of service in the jurisdiction \*1038 charging the fee. *Id.* ¶ 53. The FCC points to numerous, geographically diverse cities, where excessive fees are delaying deployment of 5G services. In one example, deployment had to be completely halted when a city tried to charge a one-time fee of \$20,000 per small cell, with an additional recurring annual fee of \$6000.

Local Government Petitioners challenge the fee limitations on a number of grounds. Their primary argument is that there is no rational connection between whether a particular fee is higher than that particular city's costs, and whether that fee is prohibiting service.

[3] The FCC did not base its fee structure on a determination that there was a relationship between particular cities' fees and prohibition of services. The FCC instead found that above-cost fees, in the aggregate, were having a prohibitive effect on a national basis. See *id.* ¶ 53 (explaining that “even fees that might seem small in isolation have material and prohibitive effects on deployment, particularly when considered in the aggregate given the nature and volume of anticipated Small Wireless Facility deployment” (footnote omitted)).

The FCC found there was no readily-available alternative. See *id.* ¶ 65 n.199 (explaining that “the record does not reveal an alternative, administrable approach to evaluating fees without a cost-based focus”). Administrability is important. In *Mayo Foundation for Medical Education & Research v. United States*, 562 U.S. 44, 58–59, 131 S.Ct. 704, 178 L.Ed.2d 588 (2011), the Supreme Court explained that an agency's rule “easily” satisfies *Chevron*'s step two, reasonable interpretation requirement, when the agency concluded that its new approach would “improve administrability.” As the FCC explained here, its cost-based standard would prevent excessive fees and the effective prohibition of 5G services in many areas across the country.

Local Government Petitioners are implicitly suggesting an alternative approach that would require an examination of the prohibitive effect of fees in each of the 89,000 state and local governments under the FCC's jurisdiction, a nearly impossible administrative undertaking. Local Government Petitioners do not contend that this is required by statute, nor do they offer any other workable standard. The FCC here made the requisite “rational connection between the facts found and the choice made.” *Burlington Truck Lines v. United States*, 371 U.S. 156, 168, 83 S.Ct. 239, 9 L.Ed.2d 207 (1962).

Our colleague's partial dissent offers one legal objection to the fee regulation. The dissent quotes language from our decision in *Qwest Communications Inc. v. City of Berkeley*, 433 F.3d 1253, 1257 (9th Cir. 2006), overruled on other grounds by *Sprint Telephony*, 543 F.3d at 578, to suggest that the FCC's cost based fee regulation should be vacated because it contravenes our precedent. In *Qwest*, however, we considered a challenge to a particular city's fee that was not based on costs. On the basis of then-binding authority we held that city's fee was preempted, but cautioned that we were not holding that “all non-cost based fees are automatically preempted.” *Id.* at 1257. Instead we said that in reviewing a

particular city's ordinance “courts must consider substance of the particular regulation at issue.” *Id.*

The *Qwest* language has no relevance in this case where we review a nationwide administrative regulation the FCC has adopted, after careful study and notice and comment, that invokes [Section 253\(a\)](#) to preempt only those fees above the safe harbor that exceed municipalities’ costs. There has been no “automatic preemption” of “all non-cost based fees.”

\*1039 Local Government Petitioners also attack the FCC's key factual finding, that high fees were inhibiting deployment both within and outside the jurisdictions charging the fees. Yet, the FCC had statements from wireless service providers, which explained that the providers have been unable to deploy small cells in many cities because both original application and annually recurring fees were excessive. For example, AT&T reported it has been unable to deploy in Portland due to recurring annual fees ranging from \$3500 to \$7500 per node.

The record also supports the FCC's factual conclusion that high fees in one jurisdiction can prevent deployment in other jurisdictions. In addition to relying on firsthand reports of service providers, the FCC looked to an academic study, known as the Corning Study. A group of economists there estimated that limiting 5G fees could result in carriers reinvesting an additional \$2.4 billion in areas “previously not economically viable.” The FCC reasonably relied upon this study to support its conclusion that a nationwide reduction in fees in “must-serve,” heavily-populated areas, would result in significant additional deployment of 5G technology in other less lucrative areas of the country. The FCC therefore has easily met the standard of offering “more than a mere scintilla” of evidence to support its conclusions regarding the prohibitive effect of above-cost fees. See *Biestek*, 139 S. Ct. at 1154.

We also conclude that the FCC's fee limitation does not violate [Section 253\(c\)](#) of the Act, which ensures that cities receive “fair and reasonable” compensation for use of their rights-of-way. The FCC explained that the calculation of actual, direct costs is a well-accepted method of determining reasonable compensation, and further, that a standard lacking a cost anchor would “have left providers entirely at the mercy of effectively unconstrained requirements of state or local governments.” *Small Cell Order* ¶74. The statute requires that compensation be “fair and reasonable;” this does not mean that state and local governments should be permitted to make a profit by charging fees above costs. [47 U.S.C. § 253\(c\)](#). The

FCC's approach to fees is consistent with the language and intent of [Section 253\(c\)](#) and is reasonably explained.

Moreover, the FCC did not require local jurisdictions to justify all fees with costs. The FCC adopted presumptively permissible fee levels. In setting those levels, the FCC looked to a range of sources, including state laws that limit fees. See *Small Cell Order* ¶ 78, ¶ 79 n.233. Local Government Petitioners argue that the FCC was in effect, setting rates, and that it was arbitrary and capricious to do so, when it could reference only a few state laws. The FCC was not setting rates, however; it was determining a level at which fees would be so clearly reasonable that justification was not necessary, and litigation could be avoided. The presumptive levels are not arbitrary and capricious.

## 2. Aesthetics

Local governments have always been concerned about where utilities’ infrastructure is placed and what it looks like. When Congress enacted the 1996 Telecommunications Act, it wanted to ensure state and local governments grant fair access to new technologies, and not prefer incumbent service providers over new entrants. Congress recognized that state and local governments could effect such preferential treatment through a wide array of regulations, including regulations on aesthetics. An important provision to prevent this is [Section 332\(c\)\(7\)\(B\)\(i\)\(I\)](#). It requires that “[t]he regulation of ... personal wireless service facilities by any State or local government ... shall not unreasonably discriminate among providers of functionally \*1040 equivalent services.” [47 U.S.C. § 332\(c\)\(7\)\(B\)\(i\)\(I\)](#). The legislators who drafted this limitation on local regulation sought to ensure that state and local governments did not “unreasonably favor one competitor over another” in exercising their regulatory authority over facility deployments—including authority to regulate aesthetics. S. Rep. No. 104-230, at 209 (1996) (Conf. Rep.).

Because it recognized that state and local governments often have legitimate aesthetic reasons for accepting some deployments and rejecting others, Congress preempted only regulations that “unreasonably discriminate” among providers. [47 U.S.C. § 332\(c\)\(7\)\(B\)\(i\)\(I\)](#). Because there were differences among providers, those who crafted [Section 332\(c\)](#) sought to preserve state and local governments’ “flexibility to treat facilities that create different ... aesthetic ... concerns differently, ... even if those facilities provide

functionally equivalent services.” S. Rep. No. 104-230, at 209 (1996) (Conf. Rep.).

The provisions of the Small Cell Order dealing with aesthetics are among the most problematic. The Order says, “aesthetics requirements are not preempted if they are (1) reasonable, (2) no more burdensome than those applied to other types of infrastructure deployments, and (3) objective and published in advance.” *Small Cell Order* ¶ 86.

In the Small Cell Order, the FCC does not use [Section 332](#)’s unreasonable discrimination standard in describing the limits on local regulation of small cell infrastructure. The Small Cell Order says instead that small cell aesthetic requirements must be “no more burdensome” than those imposed on other providers. *Id.* For example, the FCC explained that its standard would prohibit a requirement that small cell carriers “paint small cell cabinets a particular color when like requirements were not imposed on similar equipment placed in the [right-of-way] by electric incumbents, competitive telephone companies, or cable companies.” *Id.* ¶ 84 n.241.

Local Government Petitioners point out that the FCC’s standard amounts to requiring similar treatment and does not take into account the differences among technologies. The FCC’s own justification for its provision bears this out. The FCC asserts that any application of different aesthetic standards to 5G small cells necessarily “evidences that the requirements are not, in fact, reasonable and directed at remedying the impact of the wireless infrastructure deployment.” *Id.* ¶ 87. Thus, in the FCC’s view, when a state or local government imposes different aesthetic requirements on 5G technology, those requirements are pretextual, unrelated to legitimate aesthetic goals, and must be preempted.

Yet the statute expressly permits some difference in the treatment of different providers, so long as the treatment is reasonable. Indeed, we have previously recognized that [Section 332\(c\)\(7\)\(B\)\(i\)\(I\)](#) of the Telecommunications Act “explicitly contemplates that some discrimination among providers ... is allowed.” *MetroPCS, Inc. v. City & Cty. of S.F.*, 400 F.3d 715, 727 (9th Cir. 2005) (internal quotation marks omitted), *abrogated on other grounds by T-Mobile S., LLC v. City of Roswell*, 574 U.S. 293, 135 S.Ct. 808, 190 L.Ed.2d 679 (2015). We explained that to establish unreasonable discrimination, providers “must show that they have been treated differently from other providers whose facilities are *similarly situated* in terms of the *structure, placement or cumulative impact* as the facilities in question.”

*Id.* (citation and internal quotation marks omitted). We explained that this “similarly-situated” standard is derived from the text of [Section 332](#), and “strike[s] an appropriate balance \*1041 between Congress’s twin goals of promoting robust competition and preserving local zoning authority.” *Id.* at 728.

[4] The FCC’s regulation here departs from the carefully crafted balance found in [Section 332](#) in at least two critical respects. Unlike [Section 332](#), the regulation does not permit even reasonable regulatory distinctions among functionally equivalent, but physically different services. Under this Order, any local regulation of 5G technology that creates additional costs is necessarily preempted. The FCC’s limitation on local zoning authority differs from [Section 332](#) in another respect. The Order requires the comparison of the challenged aesthetic regulation of 5G deployments to the regulation of any other infrastructure deployments, while the statute only requires a comparison with the regulation of functionally equivalent infrastructure deployments. *Small Cell Order* ¶ 87. The prohibition on local regulatory authority in the regulation is in that respect broader than that contemplated by Congress.

[5] The Supreme Court has told us that “an agency may not rewrite clear statutory terms” and that this is a “core administrative-law principle.” *Util. Air Regulatory Grp. v. EPA*, 573 U.S. 302, 328, 134 S.Ct. 2427, 189 L.Ed.2d 372 (2014). The FCC has contravened this principle here by placing a limitation on local zoning authority that departs from the explicit directive of Congress in [Section 332](#).

Congress prohibited unreasonable discrimination, but permitted state and local governments to differentiate in the regulation of functionally equivalent providers with very different physical infrastructure. Members of Congress, in writing [Section 332](#), recognized that applying different standards for physically different infrastructure deployments may, in some situations, be a reasonable use of local zoning authority. *See* S. Rep. No. 104-230, at 208 (1996) (Conf. Rep.) (“For example, the conferees do not intend that if a state or local government grants a permit in a commercial district, it must also grant a permit for a competitor’s 50-foot tower in a residential district.”). Requirements imposed on 5G technology are not always preempted as unrelated to legitimate aesthetic concerns just because they are “more burdensome” than regulations imposed on functionally equivalent services. We therefore conclude that the requirement in Paragraph 86 of the Small

Cell Order, that limitations on small cells be “no more burdensome” than those applied to other technologies, must be vacated.

[6] The other problematic limitation in the Small Cell Order is that locally-imposed aesthetic requirements be “objective and published in advance.” *Small Cell Order* ¶ 86. The Order defines “objective” to mean the local regulation “must incorporate clearly-defined and ascertainable standards, applied in a principled manner.” *Id.* ¶ 88.

The FCC explained that it adopted this requirement in response to wireless service providers’ complaints that they were being kept in the dark about what requirements they had to meet, and that those requirements were often so subjective that they had no readily ascertainable meaning. As the Order explained, the providers complained that they are unable to “design or implement rational plans for deploying Small Wireless Facilities if they cannot predict in advance what aesthetic requirements they will be obligated to satisfy to obtain permission to deploy a facility at any given site.” *Id.* The FCC responded by requiring aesthetic regulations to be “objective and published in advance.” *Id.* ¶ 86. The condition of advance publication is not seriously challenged, but the requirement that all local aesthetic regulation be “objective” gives rise to serious concerns.

\*1042 Although the FCC was apparently responding to complaints of vague standards, Local Government Petitioners point out that the provision the FCC adopted bars any regulation other than one related to color, size, shape, and placement. It targets for preemption regulations focused on legitimate local objectives, such as ordinances requiring installations to conform to the character of the neighborhood. We do not see how all such regulations, designed like traditional zoning regulations to preserve characteristics of particular neighborhoods, materially inhibit, materially limit, or effectively prohibit the deployment of 5G technology.

We have previously expressed considerable doubt about the view that “malleable and open-ended,” aesthetic criteria *per se* prohibit service. *Sprint*, 543 F.3d at 580. In *Sprint*, we recognized that “[a] certain level of discretion is involved in evaluating any application for a zoning permit,” and that while “[i]t is certainly true that a zoning board *could* exercise its discretion to effectively prohibit” service, “it is equally true (and more likely) that a zoning board would exercise its discretion only to balance the competing goals of an

ordinance,” including “valid public goals such as safety and aesthetics.” *Id.*

The FCC’s position that all subjective aesthetic regulations constitute a *per se* material inhibition must therefore be viewed with considerable skepticism. Its justification for this limitation is that all subjective aesthetic requirements “substantially increase providers’ costs without providing any public benefit or addressing any public harm.” *Small Cell Order* ¶ 88. This conclusion, that all subjective standards are without public benefit and address no public harm, is unexplained and unexplainable.

The FCC says that its objectivity requirement is “feasible” because some states have adopted laws that prevent cities from applying subjective aesthetic requirements. *See id.* nn.246–47. As the FCC itself recognizes in its brief, aesthetic regulation of small cells should be directed to preventing the “intangible public harm of unsightly or out-of-character deployments.” Such harm is, at least to some extent, necessarily subjective. The fact that certain states have prohibited municipalities from enacting subjective aesthetic standards does not demonstrate that such standards never serve a public purpose. We conclude that the FCC’s requirement that all aesthetic regulations be “objective” is arbitrary and capricious. At the very least, the agency must explain the harm that it is addressing, and the extent to which it intends to limit regulations meant to serve traditional zoning objectives of preventing deployments that are unsightly or out of neighborhood character.

[7] The only remaining argument of Local Government Petitioners with which we must deal is a challenge to the FCC’s requirement that aesthetic regulations be “reasonable.” Petitioners contend that it is unduly vague and overbroad. We read this requirement as the FCC does, however, and conclude that it should be upheld. The FCC explains that the reasonableness requirement results in preemption only if aesthetic regulations are not “technically feasible and reasonably directed” at remedying aesthetic harms. *Id.* ¶ 87. We recognized in *Sprint* that imposing an aesthetic requirement that is not technically feasible would constitute an effective prohibition of service under the Act. 543 F.3d at 580. The FCC’s justification for adopting this rule is therefore consistent with our case law, as well as congressional intent in enacting Sections 253 and 332, and is not unduly vague or overbroad.

In sum, the requirement that aesthetic regulations be “no more burdensome” than those imposed on other technologies is not \*1043 consistent with the more lenient statutory standard that regulations not “unreasonably discriminate.” The requirement that local aesthetic regulations be “objective” is neither adequately defined nor its purpose adequately explained. On its face, it preempts too broadly. We therefore hold those provisions of Paragraph 86 of the Small Cell Order must be vacated.

### 3. Shot Clocks

Since 2009, the FCC has set time limits, known as shot clocks, for local authorities to act on applications to deploy wireless facilities. In the Small Cell Order, the FCC made two major changes from the shot clocks provisions in the 2009 Order. It expanded the application of shot clock timing requirements from zoning applications to include all permitting decisions. It shortened the shot clock time. State and local governments now have sixty days to decide applications for installation on existing infrastructure, and ninety days for all other applications. *Small Cell Order* ¶¶ 104–05, ¶ 132, ¶ 136. The previous shot clocks were ninety days and 150 days respectively. *Id.* ¶ 104.

To remedy a violation of the 2009 requirements, the applicant had to seek an injunction. During this rulemaking, providers urged the FCC to adopt a “deemed granted” remedy, i.e. where, at the expiration of a shot clock, a permit would be “deemed granted” and the city would have to file a lawsuit to prevent the wireless service provider from beginning construction. The FCC ultimately did not change the remedy, so under the Small Cell Order, when a state or local government misses a shot clock deadline for deciding an application, the applicant must still seek injunctive relief. Wireless Service Provider Petitioners (Sprint et al.) now challenge the FCC's refusal to adopt a deemed granted remedy for shot clock violations.

Local Government Petitioners are unhappy with the shortened time limits for decisions on applications, and with the expansion of shot clocks beyond zoning applications to all applications for deployment of wireless services. We consider their challenges first.

Local Government Petitioners attack the shortened shot clock time frames, contending they arbitrarily restrict municipalities’ ability to conduct traditional zoning review

that may take longer than the prescribed shot clock requirements. Petitioners criticize the FCC's reliance on a limited survey of state and local laws, contending that those laws had unusual, shorter time frame requirements. Petitioners contend that most state and local governments will be unable to decide permits within the time limits prescribed under the Small Cell Order.

[8] The FCC's reliance on the survey of local laws and practices was reasonable, however, because it served only a limited purpose. The FCC used the survey only to support its unremarkable assertion that some municipalities “can complete reviews more quickly than was the case when the existing Section 332 shot clocks were adopted” in 2009. *Small Cell Order* ¶ 106. It must be remembered that the shot clock requirements create only presumptions. As under the 2009 Order, if permit applicants seek an injunction to force a faster decision, local officials can show that additional time is necessary under the circumstances. *Id.* ¶ 137; *see id.* ¶ 109, ¶ 127; *see also City of Arlington*, 668 F.3d at 259–61 (upholding previous FCC shot-clock presumptions).

The Telecommunications Act itself supports the expansion of shot clocks to all permitting decisions. Section 332(c)(7)(B)(ii) requires a decision to be made within a “reasonable period of time,” and applies both to applications “to place” \*1044 wireless facilities as well as requests to “construct, or modify” such facilities. 47 U.S.C. § 332(c)(7)(B)(ii). Together, these enumerations of the categories of applications can reasonably be interpreted to authorize the application of shot clocks to building and construction permits, as well as zoning permits.

The FCC also provided sound reasons for this expansion. It explained that limiting shot clocks to zoning permits could lead states and localities to “delay their consideration of other permits (e.g., building, electric, road closure or other permits) to thwart the proposed deployment.” *Small Cell Order* ¶ 134 n.390. Courts interpreting Section 332 have reached a similar conclusion for the same reason. *See, e.g., Ogden Fire Co. No. 1 v. Upper Chichester Twp.*, 504 F.3d 370, 395–96 (3d Cir. 2007) (rejecting the argument that the Act only applies to zoning permits, because the city could use other permits to delay construction of telecommunications infrastructure). The FCC acted well within its authority, and in accordance with the purpose of the Act, when it broadened the application of the shot clocks to encompass all permits, in order to prevent unreasonable delays.

For their part, Wireless Service Provider Petitioners contend that the FCC did not go far enough in modifying the shot clock requirements. Petitioners contend that the FCC should have adopted a deemed granted remedy for shot clock violations, and argue that the Small Cell Order's factual findings compel the adoption of such a remedy.

[9] This argument relies on a mischaracterization of the FCC's factual findings. It is true that the FCC found that delays under the old shot clock regime were so serious they would “virtually bar providers from deploying wireless facilities.” *Small Cell Order* ¶ 126. But the FCC concluded that under its new shot clock rules, which shorten the time frames and expand the applicability of the rules, there will be no similar bar to wireless deployment. *Id.* ¶ 129. Because the FCC reasonably explained it has taken measures to reduce delays that would otherwise have occurred under its old regime, the factual findings here do not compel the adoption of a deemed granted remedy.

Wireless Service Providers next argue that the failure to adopt a deemed granted remedy is arbitrary and capricious because the FCC adopted the remedy in a different statutory context, the Spectrum Act, *see* 47 U.S.C. §§ 1451–57, and never explained why it did not do so here. It is understandable that the FCC gave no explanation of the difference because no comments raised any such disparity during the regulatory process. *See Perez v. Mortg. Bankers Ass'n*, 575 U.S. 92, 96, 135 S.Ct. 1199, 191 L.Ed.2d 186 (2015) (explaining that an agency has an obligation to respond to significant comments received). There are critical differences between the language of the Telecommunications Act and the language of the Spectrum Act. The Telecommunications Act requires cities make a decision on applications within a reasonable period of time. *See* 47 U.S.C. § 332(c)(7)(B)(ii) (“A State or local government or instrumentality thereof *shall act* on any request for authorization to place, construct, or modify personal wireless service facilities within a reasonable period of time...” (emphasis added)). The Spectrum Act provides that the local government must grant all qualifying applications. 47 U.S.C. § 1455(a)(1) (“[A] State or local government may not deny, and *shall approve*, any eligible facilities request for a modification of an existing wireless tower or base station...” (emphasis added)). The deemed granted remedy in the FCC's Spectrum Act order was in accordance with the text of the statute. There is no similar language in the Telecommunications Act. The FCC's conclusion that a \*1045 different remedy was appropriate here was therefore not arbitrary and capricious.

#### 4. Regulation of Property in the Public Rights of Way

Local governments generally exercise control over public rights-of-way for purposes of determining where installations such as utility poles and traffic lights should be placed. Some of these installations are owned by the municipalities themselves and some are owned by other entities, such as public and private utilities. Local Government and Public Power Petitioners (American Public Power Association et al.) argue that under Supreme Court authority, the preemption provision of Section 253(a) cannot apply to the municipal regulation of access to municipally-owned installations.

The Supreme Court has considered whether a provision of the National Labor Relations Act that preempts local regulation of labor relations prevented a municipality that was running a construction project from enforcing an otherwise valid collective bargaining agreement. *Bldg. & Constr. Trades Council of Metro. Dist. v. Associated Builders & Contractors of Mass./R.I. Inc.*, 507 U.S. 218, 231–32, 113 S.Ct. 1190, 122 L.Ed.2d 565 (1993). The Court explained that when a municipality is acting like a private business, and not acting as a regulator or policymaker, there can be no preemption by the NLRA because the municipality was not engaged in regulation of labor relations. *Id.* It was acting as a property owner.

Local Government Petitioners and Public Power Petitioners here contend that the municipalities are acting like private property owners in controlling access to, and construction of, facilities in public rights-of-way and that the Act's preemption provision therefore does not apply. They thus contend the FCC lacks authority to regulate the fees they charge for access to the rights-of-way and to the property on the rights-of-way. They emphasize that the provisions of the Small Cell Order are intended to preempt not only regulation of installations owned by non-municipal entities but also regulation of installations owned by the municipalities themselves.

[10] The issue thus becomes whether the FCC reasonably concluded that local jurisdictions are acting like private property owners when the jurisdictions charge fees or otherwise control the access to public rights-of-way. The FCC's regulations in the Small Cell Order were premised on the agency's determination that municipalities, in controlling access to rights-of-way, are not acting as owners of the property; their actions are regulatory, not propriety, and

therefore subject to preemption. *Small Cell Order* ¶ 96. This is a reasonable conclusion based on the record. The rights-of-way, and manner in which the municipalities exercise control over them, serve a public purpose, and they are regulated in the public interest, not in the financial interests of the cities. As the FCC explained, the cities act in a regulatory capacity when they restrict access to the public rights-of-way because they are acting to fulfill regulatory objectives, such as maintaining aesthetic standards. *Id.*

This conclusion is supported by case law in this Circuit, where we have held that cities operate in a regulatory capacity when they manage access to public rights-of-way and public property thereon. See *Olympic Pipe Line Co. v. City of Seattle*, 437 F.3d 872, 881 (9th Cir. 2006). For example, in *Olympic Pipe Line*, we concluded that the City of Seattle operated in a regulatory capacity when it made certain demands of an oil pipeline that operated under city-owned streets in the public rights-of-way. *Id.*; see also \*1046 *Shell Oil Co. v. City of Santa Monica*, 830 F.2d 1052, 1057–58 (9th Cir. 1987) (holding that the City of Santa Monica does not act as a market participant when it sets franchise fees for pipelines that run under its streets).

The FCC's conclusions here about the Order's scope are reasonably explained, and do not violate any presumption against preemption of proprietary municipal conduct. Municipalities do not regulate rights-of-way in a proprietary capacity.

#### 5. Section 224

The FCC adopted the Small Cell Order to remove barriers that would prevent 5G providers from accessing existing facilities for installation of small cells. These existing facilities often include utility poles. Public Power Petitioners, representing the interests of public power utilities, contend the Order cannot affect poles owned by public utilities, because Section 224 of the Telecommunications Act, relating to regulation of utility pole attachment rates, contains an express exclusion for government-owned utilities. See 47 U.S.C. § 224(a)(1).

[11] The Small Cell Order is not a regulation of rates pursuant to Section 224, however. It is promulgated under the authority of Section 253 to ensure that state and local statutes do not have a prohibitory effect on telecommunications services. See 47 U.S.C. § 253(a); The FCC responded appropriately when it said, “[n]othing in Section 253 suggests

such a limited reading, nor does Section 224 indicate that other provisions of the Act do not apply. We conclude that our interpretation of effective prohibition extends to fees for all government-owned property in the [right-of-way], including utility poles.” See *Small Cell Order* ¶ 92 n.253. Because Section 253 does not exempt public power utilities from its terms, the FCC reasonably relied on Section 253 to regulate such utilities.

#### 6. Radiofrequency Exposure

More than twenty years ago, the FCC first adopted “radiofrequency standards,” (RF standards) which limit the amount of radiation that can be emitted from wireless transmitters. *Guidelines for Evaluating the Envtl. Effects of Radiofrequency Radiation*, 11 FCC Rcd. 15,123 (1996). The FCC is obligated to evaluate the potential impacts of human exposure to radiofrequency emissions under the National Environmental Policy Act. See Pub. L. 104-104, 110 Stat. 56 (1996); 47 C.F.R. § 1.1310. In the Telecommunications Act, Congress preempted all municipal regulation of radiofrequency emissions to the extent that such facilities comply with federal emissions standards. 47 U.S.C. § 332(e)(7)(B)(iv).

In 2013, the FCC opened a “Notice of Inquiry,” requesting comments on whether it should reassess its RF standards. See *Reassessment of Fed. Commc'ncs Comm'n Radiofrequency Exposure Limits and Policies*, 28 FCC Rcd. 3498 (2013). The agency did not take immediate action on that docket. During the later process leading up to the adoption of the Small Cell Order, Petitioner Montgomery County requested that the Commission complete its 2013 RF proceeding before adopting the Small Cell Order, and that it examine the potential effects of 5G technology on its RF standards. The FCC did not address its RF standards or close the 2013 docket before adopting the Small Cell Order.

Petitioner Montgomery County now challenges the FCC's Small Cell Order as unlawful because the FCC did not complete the 2013 docket review before adopting the Small Cell Order. After its petition was filed, however, the FCC adopted a new order examining radiofrequency exposure in the 5G environment, and concluded that it did not warrant changes to its 1996 standards. Challenges to the FCC's failure \*1047 to perform updated radiofrequency analysis, as contemplated by the 2013 docket, are therefore moot. See,

e.g., *Alliance for the Wild Rockies v. U.S. Dep't of Agr.*, 772 F.3d 592, 601 (9th Cir. 2014).

There is no merit to Montgomery County's further suggestion that we should penalize the FCC for what the County calls evasive litigation tactics in not acting earlier. The Supreme Court has emphasized that agencies have "significant latitude as to the manner, timing, content, and coordination of [their] regulations." *Massachusetts v. EPA*, 549 U.S. 497, 533, 127 S.Ct. 1438, 167 L.Ed.2d 248 (2007); see also *Mobil Oil Expl. & Producing Se. Inc. v. United Distrib. Cos.*, 498 U.S. 211, 230–31, 111 S.Ct. 615, 112 L.Ed.2d 636 (1991) ("An agency enjoys broad discretion in determining how best to handle related, yet discrete, issues in terms of procedures and priorities.... [A]n agency need not solve every problem before it in the same proceeding." (citations omitted)). More important, Montgomery County now has what it wanted; the FCC has examined the effects of 5G technology on its RF standards, and closed the 2013 docket. Any challenges to the adequacy of that final agency action must now be brought in a new proceeding.

### B. Challenges to the Moratoria Order

The FCC adopted the Moratoria Order in response to complaints from a "broad array of large and small ... wireless providers" that state and local ordinances and practices were either explicitly or having the effect of barring small cell deployment. *Moratoria Order* ¶ 143. In the Order, the FCC concluded that ordinances and practices were materially inhibiting small cell deployment, and the agency provided general standards to differentiate between permissible municipal regulations and impermissible "moratoria." The Moratoria Order describes two general categories of moratoria: express and de facto. See *id.* ¶ 144. It defined express moratoria as "statutes, regulations, or other written legal requirements" in which state or local governments "expressly ... prevent or suspend the acceptance, processing, or approval of applications or permits necessary for deploying telecommunications services." *Id.* ¶ 145. The Order provided such bars to 5G deployment qualify as moratoria even though they are of a limited duration. *Id.*

The FCC then defined de facto moratoria as "state or local actions that are not express moratoria, but that effectively halt or suspend the acceptance, processing, or approval of applications or permits for telecommunications services or facilities in a manner akin to an express moratorium." *Id.* ¶ 149. De facto moratoria violate Section 253 only when they unreasonably or indefinitely delay deployment. *Id.* ¶ 150.

The Order provides a new definition of Section 253(b)'s exemption for local regulations that protect "the public safety and welfare." The Order permits what it describes as "emergency" bans on the construction of 5G facilities to protect public safety and welfare, but only where those laws are (1) "competitively neutral", (2) necessary to address the emergency, disaster, or related public needs, and (3) target only those geographic areas affected by the disaster or emergency. *Id.* ¶ 157.

[12] The City of Portland, not joined by the other Local Government Petitioners, challenges the Order with a handful of criticisms. The City's primary contention is that the Order's definitions of moratoria are overly broad, and therefore unreasonable, because, in the City's view, the Moratoria Order preempts even benign seasonal restrictions on construction, such as freeze-and-frost laws. The City also contends that the Moratoria Order is an invalid application of Section 253, and self-contradictory \*1048 in its definitions. None of these contentions have merit.

As an initial matter, we do not read the Moratoria Order as broadly as the City does in arguing that it would preempt all restrictions on construction, even seasonal ones that cause some delay in small cell deployment. The FCC carefully explained in the Order that municipal ordinances of general applicability will qualify as de facto moratoria only where the delay caused by the ordinances "continues for an unreasonably long or indefinite amount of time such that providers are discouraged from filing applications." *Id.* ¶ 150. Municipal regulations on construction are therefore not preempted if they "simply entail some delay in deployment." *Id.* The explanation is supported by the FCC's assurance in the Order that municipalities retain authority over "construction schedul[ing]." *Id.* ¶ 160. The City's concerns about the breadth of the Moratoria Order are therefore unfounded. The Order does not preempt necessary and customary restrictions on construction.

The City argues that the Moratoria Order preempts laws of general applicability, while Section 253 preempts only those that specifically target the provision of telecommunications services. By its terms, however, Section 253(a) is not so limited. It looks to both the language and impact of local regulations. It preempts all "local statute[s] or regulation[s], or other ... legal requirement[s]" that prohibit or have the effect of prohibiting telecommunications services. 47 U.S.C. § 253(a).

Nor is the Moratoria Order contradictory in its definitions of express and de facto prohibitions. After examining the factual record, the FCC found that some localities had repeatedly re-authorized temporary bans on 5G installation to prohibit the installation of 5G cells indefinitely. *Moratoria Order* ¶ 148 n.546. The FCC therefore clarified that such explicit bans on 5G deployment qualify as express moratoria, even if they have a “limited, defined duration.” *Id.* ¶ 148. In a separate paragraph dealing with de facto prohibitions resulting from more general laws, the FCC explained that generally applicable laws, i.e. those that do not facially target small cells, are not preempted unless they cause a delay that “continues for an unreasonably long or indefinite amount of time.” *Id.* ¶ 150. There is nothing inconsistent or unexplained in the FCC's separate definitions of express and de facto moratoria.

Finally, the City challenges the FCC's purportedly narrow construction of Section 253(b)'s preemption exception for laws regulating safety and welfare. The FCC reasonably interpreted the phrase “public safety and welfare” in this context to permit emergency bans on 5G deployment where the regulations are competitively neutral and intended to remedy an ongoing public safety concern. The FCC explained such an interpretation was necessary to prevent the pretextual use of safety “as a guise for” preventing deployment. *Id.* ¶ 157. The Order is consistent with the FCC's earlier interpretations of Section 253(b). *See, e.g., New Eng. Pub. Commc'ns Council Petition for Preemption*, 11 FCC Red. 19,713 (1996) (rejecting a broad interpretation of Section 253(b)).

The Moratoria Order is not arbitrary, capricious, or contrary to law on a facial basis. As the FCC has recognized, objections to specific applications of the Moratoria Order may be made on a case-by-case basis.

### C. Constitutional Challenges to Both Orders

[13] Local Government Petitioners also argue that the Small Cell and Moratoria Orders violate the Fifth and Tenth Amendments. First, Petitioners argue that the Small Cell Order is a physical taking in violation of the Fifth Amendment because it requires municipalities to grant providers access to municipal property, including rights-of-way, thereby creating a physical taking without just compensation. Petitioners compare the Small Cell Order to the New York state law at issue in *Loretto v. Teleprompter Manhattan CATV Corp.*, 458 U.S. 419, 421, 102 S.Ct. 3164,

73 L.Ed.2d 868 (1982), which required landlords to permit cable television companies to install cables on their property. In *Loretto*, the Court held the law to be a physical taking because the installation resulted in “permanent occupations of land.” *Id.* at 430, 102 S.Ct. 3164. Here, on the other hand, the Small Cell Order precludes state and local governments from charging unreasonable fees when granting applications, and it continues to allow municipalities to deny access to property for a number of reasons. *See Small Cell Order* ¶ 73 n.217. It does not compel access to property in a manner akin to *Loretto*. *See id.* Once again, challenges to particular applications of the Small Cell Order must be made on an as-applied basis.

Petitioners also argue that the Small Cell Order constitutes a regulatory taking by limiting cost recovery. The Supreme Court rejected a similar argument in *FCC v. Florida Power Corp.*, 480 U.S. 245, 107 S.Ct. 1107, 94 L.Ed.2d 282 (1987), holding that limiting cost recovery to actual costs did not result in a regulatory taking. *Id.* at 254, 107 S.Ct. 1107. Because the Small Cell Order allows for the recovery of actual costs as well, the Order does not constitute a regulatory taking. *See Small Cell Order* ¶ 50 (explaining that the Small Cell Order continues to allow for fees that “are a reasonable approximation of the state or local government's costs”).

[14] [15] Finally, Local Government Petitioners argue that, by requiring municipalities to respond to applications for use from 5G and broadband installers within a prescribed period of time or risk immediate control of its property, the Small Cell and Moratoria Orders compel Petitioners to enforce federal law in violation of the Tenth Amendment. In support, they cite *National Federation of Independent Business v. Sebelius*, 567 U.S. 519, 579–80, 132 S.Ct. 2566, 183 L.Ed.2d 450 (2012) (plurality opinion), where the Court held that financial inducement had the effect of compelling states to enforce a federal program. Nothing like that is happening here. Instead, the FCC is interpreting and enforcing the 1996 Telecommunications Act, adopted by Congress pursuant to its delegated authority under the Commerce Clause, to ensure that municipalities are not charging small cell providers unreasonable fees. “If a power is delegated to Congress in the Constitution, the Tenth Amendment expressly disclaims any reservation of that power to the States.” *New York v. United States*, 505 U.S. 144, 156, 112 S.Ct. 2408, 120 L.Ed.2d 120 (1992). In addition, by preempting certain State and local policies, the FCC did not commandeer State and local officials in violation of the Tenth Amendment. Although their “language might appear to operate directly on the States,”

the Orders—as applications of the Telecommunications Act—simply “confer[ ] on private entities ... a federal right to engage in certain conduct subject only to certain (federal) constraints.” See *Murphy v. Nat'l Collegiate Athletic Ass'n*, — U.S. —, 138 S. Ct. 1461, 1480, 200 L.Ed.2d 854 (2018) (citing *Morales v. Trans World Airlines, Inc.*, 504 U.S. 374, 378, 112 S.Ct. 2031, 119 L.Ed.2d 157 (1992)). The Orders do not violate the Constitution.

#### IV. ONE-TOUCH MAKE-READY ORDER

In adopting the One-Touch Make-Ready Order, the FCC intended to make it faster and cheaper for broadband providers to \*1050 attach to already-existing utility poles. See *Accelerating Wireline Broadband Deployment by Removing Barriers to Infrastructure Inv.*, 33 FCC Red. 7705, ¶ 1 (2018) [hereinafter *One-Touch Make-Ready Order*]. Previously, only the pole owners could perform the preparatory work necessary for attachment. The main purpose of the Order is to create a new process, called one-touch make-ready, that allows new attachers themselves to do all the preparations. *Id.* ¶ 2.

Petitioners American Electric Power Service Corporation et al., a group of private utility companies, do not challenge the most important aspects of the One-Touch Make-Ready Order. Instead, they challenge four secondary aspects of the Order: rules for overloading, preexisting violations, self-help, and rate reform. For the following reasons, we uphold them all.

##### A. Overloading

Overloading is the process by which attachers affix additional cables or other wires to ones already attached to a pole. The overloading rule prohibits a utility from requiring overlashers to conduct pre-overloading engineering studies or to pay the utility's cost of conducting such studies. *Id.* ¶ 119 n.444.

[16] Petitioner utility companies first contend the overloading rule contradicts the text of Section 224(f)(2), because the rule does not expressly say that a utility can exercise its statutory authority to deny access to poles for safety, capacity, reliability, or engineering reasons. See 47 U.S.C. § 224(f)(2). But the overloading rule does not prevent utilities from exercising their statutory rights, nor has the FCC interpreted the overloading rule to do so. It is speculative to suggest that it might do so in the future. See *Texas v. United States*, 523 U.S. 296, 300, 118

S.Ct. 1257, 140 L.Ed.2d 406 (1998) (declining to consider claim because “it rests upon contingent future events that may not occur as anticipated, or indeed may not occur at all.” (internal quotation marks omitted)). The rule allows overlashers and utilities to negotiate the details of the overloading arrangement, and is thus consistent with FCC's longstanding policy. See *Amendment of Comm'n's Rules & Policies Governing Pole Attachments*, 16 FCC Red. 12,103, ¶ 74 (2001).

Petitioners also argue that the overloading rule undermines a utility's Section 224(f)(2) authority to deny pole access, because it prevents utilities from requiring overlashers to provide certain information. We conclude that the overloading rule does not impede a utility's exercise of its statutory authority to deny access to poles. The rule authorizes utilities to require that overlashers give fifteen days' notice to utilities prior to overloading so that safety concerns can be addressed. *One-Touch Make-Ready Order* ¶¶ 115–16. The record shows that such notice provisions were frequently negotiated in the past on a voluntary basis and supports the FCC's conclusion that such “an advance notice requirement has been sufficient to address safety and reliability concerns.” *Id.* ¶ 117. Indeed, in evaluating similar rules, the D.C. Circuit has already held that there is “no merit” to the claim that utilities cannot effectively exercise their rights under Section 224(f)(2) without “prior notice” of overloading. See *S. Co. Servs., Inc. v. FCC*, 313 F.3d 574, 582 (D.C. Cir. 2002).

Finally, Petitioners argue that by prohibiting the utilities from charging overlashers for the cost of conducting pre-overloading studies, the overloading rule contradicts Section 224(d)(1). That section ensures cost recovery, but it does so only for attachments by cable television providers. See 47 U.S.C. § 224(d)(1)–(3). It does not apply here. The overloading rule is \*1051 thus a reasonable attempt by the FCC to prevent unnecessary costs for attachers.

##### B. Preexisting Violation Rule

[17] The preexisting violation rule prohibits utilities from denying access to a new attacher solely because of a preexisting safety violation that the attacher did not cause. *One-Touch Make-Ready Order* ¶ 122. Petitioners contend that this is contrary to Section 224(f)(2), which allows utilities to deny access for “reasons of safety.” There is no conflict.

The rule defines the term “reasons of safety” as preventing a utility from denying access to a new attacher because of a safety hazard created by a third party. *Id.* ¶ 122. Such denials

have the effect of forcing an innocent would-be attacher to fix the violation. This rule prevents the utilities from passing the costs off on entities that did not cause the safety problem in the first place. The FCC confirmed at oral argument that the preexisting violation rule would not prevent utilities from rejecting proposed attachments that increase safety risks on a utility pole. The rule thus operates to prevent utilities from relying on preexisting violations pretextually to deny pole access to attachments that pose no greater safety risk than existing attachments. Because the preexisting violation rule reasonably defines the term “reasons of safety,” the FCC’s interpretation is reasonable.

### C. Self-Help Rule

[18] Prior to the One-Touch Make-Ready Order, attachers could hire contractors to perform preparatory work only on the lower portion of a pole. The self-help rule lets the utility-approved contractors prepare the entire pole for attachment. *Id.* ¶¶ 97–99. Petitioners argue that this expansion is contrary to Section 224(f)(2) because permitting attachers to hire contractors to work on the upper portion of poles jeopardizes safety. Yet, the rule has a number of provisions designed to mitigate any increased safety risks. For example, the rule gives a utility a ninety-day window to complete the pre-attachment work itself (thereby circumventing the rule’s contractor provisions entirely). *Id.* ¶ 99. The rule also requires new attachers to use a utility-approved contractor to perform the self-help work, and it requires the attacher to give the utility advanced notice of when the self-help work will occur so that the utility can be present if it wishes. *Id.* ¶¶ 99–106.

The rule represents a change from earlier rules on what self-help measures an attacher could perform, and the FCC explained that use of approved contractors would improve efficiency. *Id.* ¶ 97. A complaint process in the old self-help rule allowed new attachers to file complaints when a utility was not preparing the pole in a timely fashion. This did not encourage efficiency. It was an “insufficient tool for encouraging [a utility’s] compliance with [the FCC’s] deadlines.” *Id.* ¶ 98. The FCC reasonably views the deployment of new 5G technology to be a matter of “national importance,” justifying extension of the self-help rule to promote timely installations. *Id.* ¶ 97. The self-help rule is thus not arbitrary or capricious.

[19] Petitioners also argue that the FCC lacks authority to regulate utility-owned pole attachments, since Section 224 defines “pole attachments” to include attachments to a utility-owned or -controlled pole. But the FCC has authority

to promulgate “regulations to carry out the provisions of” Section 224, 47 U.S.C. § 224(b)(2), which includes regulations addressing “nondiscriminatory access” to utility poles, *id.* § 224(f)(1). It was reasonable for the FCC to conclude that it could not ensure nondiscriminatory access to poles without allowing make-ready work that would reposition utility attachments; \*1052 otherwise, utilities could simply deny access to attachers based on pretextual reasons of insufficient capacity. See *S. Co. v. FCC*, 293 F.3d 1338, 1348 (11th Cir. 2002) (“[T]he FCC must have some way of assessing whether these needs are bona fide; otherwise, a utility could arbitrarily reserve space on a pole ... and proceed to deny attachers space on the basis of ‘insufficient capacity.’”). Petitioners’ statutory challenge thus fails.

Petitioners mount a procedural challenge to the rule, arguing that the FCC did not comply with the APA’s notice requirement, 5 U.S.C. § 553, because it had not issued a proposed rule before announcing the final self-help rule. In raising the issue in a single footnote, petitioners have waived any challenge to the APA’s notice requirement. See *Idaho Conservation League v. Bonneville Power Admin.*, 826 F.3d 1173, 1178 (9th Cir. 2016). In any event, the FCC’s Notice of Proposed Rulemaking (NPRM) sought proposals to speed up access to poles by allowing new attachers to prepare poles for attachment, and several commenters proposed expanding an attacher’s ability to perform preparatory work on the entire pole. We conclude that, at the very least, the self-help rule is a logical outgrowth of the NPRM. See *Rybachek v. EPA*, 904 F.2d 1276, 1288 (9th Cir. 1990) (explaining that an agency need not provide a new NPRM as long as the final published rule is “a logical outgrowth of the notice and comments received”). There is no reason to force the agency to begin the self-help rulemaking process anew.

### D. Rate-Reform Rule

The rate reform rule continues regulatory efforts to remove rate disparities between telecommunications carriers who historically owned utility poles (so-called incumbent local exchange carriers, or ILECs) and telecommunications carriers who do not own utility poles (so-called competitive local exchange carriers, or CLECs). See *Am. Elec. Power Serv. Corp. v. FCC*, 708 F.3d 183, 185–86 (D.C. Cir. 2013). This rule establishes a presumption that all telecommunication carriers are similarly situated and thus entitled to the same rates. *One-Touch Make-Ready Order* ¶ 123. But if a utility successfully rebuts the presumption by showing that an ILEC continues to retain “net benefits” that other telecommunications providers do not enjoy, then the rate

reform rule imposes a maximum rate that ILECs and utilities may negotiate. *See id.* ¶¶ 128–29.

Section 224(e)(1) authorizes the FCC to prescribe rates for pole attachments used by CLECs, but not ILECs. *See* 47 U.S.C. § 224(e)(1); *see also id.* § 224(a)(5). Petitioners therefore argue that the FCC lacks the authority to prescribe the same rates for ILECs. Section 224(b)(1), however, requires the FCC to set just and reasonable rates for all telecommunications carriers, and the FCC interpreted that to include ILECs as well as CLECs. *See id.* § 224(b)(1). The FCC has interpreted Section 224(b)(1) this way since 2011, and the D.C. Circuit upheld this interpretation some years ago. *See Am. Elec. Power Serv. Corp.*, 708 F.3d at 188. And the Supreme Court has made clear that Section 224(e)(1) “work[s] no limitation” on the FCC’s more general ratemaking authority under Section 224(b)(1), which is the statutory provision that the agency invoked here. *See Nat’l Cable & Telecomm. Ass’n, Inc. v. Gulf Power Co.*, 534 U.S. 327, 335–36, 122 S.Ct. 782, 151 L.Ed.2d 794 (2002).

[20] This rule does, for the first time, set the same presumptive rates for ILECs and CLECs, and the FCC explained why its record supported such a rule. *See One-Touch Make-Ready Order* ¶ 126. A study by US Telecom showed that earlier efforts to decrease rate disparities between ILECs and CLECs had not been successful, and that historic differences between \*1053 ILECs and CLECs that supported different rates in the past are now disappearing. *See id.* ¶¶ 124–26. The FCC provided an adequate justification for setting the same presumptive rates for all telecommunications providers.

Finally, Petitioners argue that the rate reform rule may result in their incomplete recovery of costs, because if a utility successfully rebuts the presumption that an ILEC should have the same rates as CLECs, the rule imposes a maximum rate ILECs and utilities may negotiate. *See id.* ¶ 129. The maximum negotiable rate is not arbitrary or capricious, however, because FCC set the rate at a value that is higher than both CLEC and cable operator rates, and the FCC had previously determined those rates were just, reasonable, and allowed full cost recovery. *Id.* ¶ 129 n.483; *see also Implementation of Section 224 of the Act*, 26 FCC Rcd. 5240, ¶ 183 (2011).

The rate reform rule, like the overlashing, preexisting violations, and self-help rules, is an appropriate exercise of

the FCC’s regulatory authority under the Telecommunications Act.

## V. CONCLUSION

We therefore hold that the FCC’s requirement in the Small Cell Order that aesthetic regulations be “no more burdensome” than regulations applied to other infrastructure deployment is contrary to the controlling statutory provision. *See* 47 U.S.C. § 332(c)(7)(B)(i)(II). We also hold that the FCC’s requirement that all local aesthetic regulations be “objective” is not adequately explained and is therefore arbitrary and capricious. We therefore **GRANT** the petitions as to those requirements, **VACATE** those portions of the rule and **REMAND** them to the FCC. The petition of Montgomery County is **DISMISSED** as moot. As to all other challenges, the petitions are **DENIED**. Each party to bear its own costs.

**BRESS**, Circuit Judge, dissenting in part:

The majority opinion carefully addresses an array of legal challenges to a series of FCC *Orders* designed to accelerate the deployment of 5G service. I join the court’s fine opinion except as to Part III.A.1, which upholds the FCC’s decision to preempt any fees charged to wireless or telecommunications providers that exceed a locality’s costs for hosting communications equipment. In my view, the FCC on this record has not adequately explained how all above-cost fees amount to an “effective prohibition” on telecommunications or wireless service under 47 U.S.C. §§ 253(a) and 332(c)(7)(B)(i)(II).

The Telecommunications Act of 1996 provides that “[n]o State or local statute or regulation, or other State or local legal requirement, may prohibit or have the effect of prohibiting the ability of any entity to provide any interstate or intrastate telecommunications service.” 47 U.S.C. § 253(a). The Act contains a similar provision for wireless service. *See id.* § 332(c)(7)(B)(i)(II) (“The regulation of the placement, construction, and modification of personal wireless service facilities by any State or local government or instrumentality thereof ... shall not prohibit or have the effect of prohibiting the provision of personal wireless services.”).

The Act does not define what it means for a local policy to “have the effect of prohibiting” service. Since 1997, however, the FCC has interpreted the phrase to preempt local policies that “materially inhibit” the ability of providers “to compete

in a fair and balanced legal and regulatory environment.” See *Small Cell Order* ¶ 35 (quoting *Cal. Payphone Ass'n*, 12 FCC Rcd. 14191, 14206 (1997)). This standard does not require a “complete or insurmountable” barrier to service. *Id.* But it \*1054 does require that a local rule materially inhibit the ability to provide service based upon the “actual effects” “of a state or local ordinance,” “not [ ] what effects the ordinance might possibly allow.” *Sprint Telephony PCS, L.P. v. Cty. of San Diego*, 543 F.3d 571, 578 (9th Cir. 2008) (en banc) (emphasis in original); see also *id.* (the statute requires an “actual or effective prohibition, rather than the mere possibility of prohibition”) (quotations omitted).

In the *Small Cell Order*, the FCC concluded that state and local fees materially inhibit telecommunications and wireless service when they exceed a locality's reasonable cost of accommodating communications facilities. *Small Cell Order* ¶¶ 50, 53. The FCC cited evidence that certain exorbitant fees have stopped providers from offering service in certain locales. See, e.g., AT&T Aug. 10, 2018 *Ex Parte* Letter (AT&T “has not deployed any small cell sites in Portland, Oregon” due to the city's \$7,500 attachment fee and recurring fee of \$3,500 to \$5,500). The agency also found that “even fees that might seem small in isolation have material and prohibitive effects on deployment particularly considered in the aggregate.” *Small Cell Order* ¶ 53. This latter finding was based on the FCC's determination that reduced fees generate cost-savings for providers, which enables them to use the newfound savings to expand wireless and telecommunications coverage. See *id.* ¶ 50, 55–56, 64–65 & nn.194–95. The agency estimated aggregate cost-savings from a reduction in fees to be over \$2 billion, relying on a 2018 study by Corning, Inc. *Id.* ¶¶ 7, 60 & n.169.

The FCC carved out a safe harbor from the *Order*'s broad preemption rule for pole construction fees up to \$1,000, attachment fees up to \$500 (or \$100 after a provider's first five 5G facilities), and recurring fees up to \$270. *Id.* ¶ 79. Fees may exceed the levels in the *Small Cell Order*'s safe harbor only if they reasonably approximate a locality's costs, which include expenses “related to processing an application,” street closures, issuing “building or construction permits,” and access to and maintenance of public rights of way. *Id.* ¶¶ 32 n.71, 50 n.131, 79.<sup>1</sup>

No one doubts that exorbitant fees can impede the deployment of communications infrastructure. See, e.g., *P.R. Tel. Co. v. Mun. of Guayanilla*, 450 F.3d 9, 17–19 (1st Cir. 2006). But fees are prohibitive because of their financial effect on service

providers, not because they happen to exceed a state or local government's costs. Consider a \$500 fee in Small Town A that exceeds the town's costs by 1¢, and a \$2,000 cost-based fee in Big City B. By the *Small Cell Order*'s logic, the lower fee is preempted, but the higher fee is not. It is hard to rationalize the former under the statute, which requires an actual and material inhibition of telecommunications or wireless service. *Sprint Telephony*, 543 F.3d at 578.

Perhaps for this reason, this court over a decade ago “declin[ed]” to hold “that all non-cost based fees are automatically preempted” under the Telecommunications Act. See *Qwest Commc'ns Inc. v. City of Berkeley*, 433 F.3d 1253, 1257 (9th Cir. 2006), *overruled on other grounds by Sprint Telephony*, 543 F.3d at 578.<sup>2</sup> The \*1055 FCC was aware of this precedent when it issued the *Small Cell Order*, but expressly “reject[ed] the view of those courts that have concluded that [§] 253(a) necessarily requires some additional showing beyond the fact that a particular fee is not cost-based.” See *Small Cell Order* ¶ 53 n.143 (citing *Qwest*, 433 F.3d at 1257).

On this record, the FCC has not adequately explained its basis for concluding, contra our precedent, that there is an intrinsic relationship between a fee's approximation of costs and its prohibitive effect on service providers. The FCC's reliance on individual fees it considers “excessive” tells us that fees can work effective prohibitions. But this does not on its own justify a blanket prohibition on all above-cost fees. A \$7,500 fee in Portland may well prohibit service, but that is because of the financial toll it inflicts, not because it exceeds the city's costs. And the FCC has not identified in the administrative record the frequency of above-cost fees or the amounts that localities have generally charged above cost.

The FCC has instead determined that a prohibition on all above-cost fees is justified because all above-cost fees, in the aggregate, effectively prohibit 5G deployment. The linchpin of the agency's aggregation theory is a 2018 study by Corning, Inc., which estimates at over \$2 billion the cost-savings and reinvestment from reduced fees. *Small Cell Order* ¶¶ 7, 60 & n.169. But the Corning Study is not about fees above costs. And the FCC has not explained how this study tells us about the prevalence of above-cost fees or the burden such fees place on service providers.

Instead, the Corning Study calculated “the cost savings from capping fees at a level in line with the median of recent state regulations,” estimating that amount at over \$2 billion.

Because this is not a measure of fees above costs, the Corning Study does not say whether the caps it used to measure savings approximate costs. Indeed, the Corning Study notes that “[t]here is still significant uncertainty around what ‘typical’ rates are.” The study further states that “attachment and application fees” are “lesser drivers” of 5G deployment economics, raising questions about the extent to which all fees above costs necessarily effectively prohibit service.

At bottom, what the Corning Study conveys is that if fees are reduced, it will produce cost savings to those who pay the fees. *Small Cell Order* ¶¶ 50, 53, 55–56, 60 & n.169, 64–65 & nn.194–95. But that commonsense observation would be true of any fee considered in the aggregate. And it would seemingly mean that any fee in any amount could qualify as an effective prohibition, once aggregated. The same would be true of the aggregate effects of any form of regulation that localities would apply outside the fee context. I am therefore concerned that on the record as it stands, the FCC's approach lacks a limiting principle. At least absent some estimated quantification of above-cost fees in the aggregate (which the Corning Study does not provide) or some further estimate tied to the rule it adopted, the FCC's logic would appear to justify the preemption of any state or local rule.

The FCC's “reinvestment” theory invites similar concerns. It may be true that every fee imposes some cost that, if avoided, could potentially be reinvested to expand 5G coverage. But it does not follow that every type of fee rises to the level of an “effective prohibition,” which is the line Congress drew in the Telecommunications Act. See \*1056 Cal. Payphone, 12 F.C.C. Rcd. at 14209 (stating that, “standing alone,” the fact that providers “would generate less revenue ... does not necessarily mean that [services] are impractical and uneconomic”) (quotations omitted); cf. *AT&T Corp. v. Iowa Utils. Bd.*, 525 U.S. 366, 390 n.11, 119 S.Ct. 721, 142 L.Ed.2d 835 (1999) (disagreeing “that a business can be impaired in its ability to provide services—even impaired in that ability in an ordinary, weak sense of impairment—when the business receives a handsome profit but is denied an even handsomer one”). A provider reinvestment theory, without more, would similarly appear to justify the preemption of any local policy that imposes costs on providers.

On this record, the FCC thus has not shown that above-cost fees effectively prohibit service in many, most, or a plurality of cases. I therefore cannot conclude that the agency has articulated “a rational connection between the facts found and

the choice made.” *Motor Vehicle Mfrs. Ass'n v. State Farm Mut. Auto. Ins. Co.*, 463 U.S. 29, 43, 103 S.Ct. 2856, 77 L.Ed.2d 443 (1983) (quotations omitted).

The FCC itself recognizes that “in theory, a sufficiently small departure from actual and reasonable costs might not have the effect of prohibiting service,” but concludes its cost-based standard is still appropriate because “the record does not reveal an alternative, administrable approach to evaluating fees.” *Small Cell Order* ¶ 65 n.199. Concerns about administrability, though important as a policy matter, must still be operationalized under the statute's effective prohibition standard. A rule prohibiting fees that exceed cost by \$1 would be equally administrable, but that does not mean such fees are invariably effective prohibitions on service, which is the relevant question under §§ 253(a) and 332(c)(7).

The *Order*'s safe harbors underscore my concerns. The FCC concedes that its safe harbors, which are not based on estimated costs, tolerate fee levels “in excess of costs in many cases.” *Small Cell Order* ¶ 79 n.233. That makes it more difficult to credit the agency's finding that above-cost fees are *per se* effective prohibitions on service. The safe harbor also allows local governments to charge recurring fees of \$270, which is substantially greater than the \$150 cap on recurring fees used to calculate cost-savings in the Corning Study. There are also discrepancies between the FCC's safe harbors for application fees and the Corning Study's caps. The FCC does not estimate how much of the over \$2 billion in cost-savings from the Corning Study would be left over under its more expansive safe harbors. Nor has the agency explained what portion of that figure can be attributed to above-cost fees.

I would have vacated and remanded the *Small Cell Order*'s prohibition on above-cost fees. See 5 U.S.C. § 706(2)(A), (E). While the FCC's objective of advancing 5G service is undoubtedly an important one, Congress set limits on when local actions can be preempted. While a prohibition on all above-cost fees may well be justifiable, I do not believe the FCC has sufficiently justified it on the present record. With the exception to its references to legislative history, I otherwise join the court's opinion in full.

#### All Citations

969 F.3d 1020, 20 Cal. Daily Op. Serv. 8306, 2020 Daily Journal D.A.R. 8518

Footnotes

- 1 The *Small Cell Order* also interpreted the phrase “fair and reasonable compensation” in 47 U.S.C. § 253(c) to limit state and local fees to cost-recovery. *Small Cell Order* ¶ 55. But the agency declined to use this savings clause “as an independent prohibition on conduct that is not itself prohibited by [§] 253(a).” *Id.* ¶ 53 n.143; see also *id.* ¶ 50 n.132.
- 2 Qwest applied a lenient standard that more easily allowed the FCC to show an effective prohibition, 433 F.3d at 1256, a standard our en banc court later rejected. See *Sprint Telephony*, 543 F.3d at 576–78. If above-cost fees were not *per se* prohibitions under the less stringent *Qwest* standard, it is hard to see how they would be under the stricter approach of *Sprint Telephony*. I do not suggest that *Qwest* imposes a “legal” bar to the FCC’s contrary determination, Maj. Op. 1038, but rather that the FCC has not adequately explained the basis for its conclusion here.

---

End of Document

© 2021 Thomson Reuters. No claim to original U.S. Government Works.

6 Cal.5th 1107  
Supreme Court of California.

T-MOBILE WEST LLC et  
al., Plaintiffs and Appellants,

v.

CITY AND COUNTY OF SAN FRANCISCO  
et al., Defendants and Respondents.

S238001

|  
April 4, 2019

### Synopsis

**Background:** Wireless telephone service providers brought action against city for declaratory and injunctive relief challenging city's wireless facility site permit ordinance. The Superior Court, City and County of San Francisco, No. CGC-11-510703, [James McBride](#), J., granted summary adjudication for city on providers' claims that the ordinance violated a state wireless facility permit statute and California Environmental Quality Act (CEQA), but ruled in providers' favor in part, after bench trial, on claims that the ordinance violated the Public Utilities Code and the Middle Class Tax Relief and Job Creation Act. Providers appealed. The Court of Appeal, [208 Cal.Rptr.3d 248](#), affirmed. Providers sought review.

**Holdings:** After grant of review, the Supreme Court, [Corrigan](#), J., held that:

[1] state statute providing that telephone corporations may construct lines and erect equipment along public roads in ways and locations that do not “incommode the public use of the road” did not preempt local regulation allowing city to condition permit approval for telephone line construction on aesthetic considerations, and

[2] statute roads are “accessed” by wireless telephone corporations only applies to temporary access during construction and installation of telephone lines and equipment.

Affirmed.

West Headnotes (22)

[1] **Counties**  [Governmental powers in general](#)  
**Municipal Corporations**  [Conformity to constitutional and statutory provisions in general](#)

General laws, for purposes of constitutional provision allowing cities and counties to make and enforce ordinances not in conflict with general laws, are those that apply statewide and deal with matters of statewide concern. [Cal. Const. art. 11, § 7](#).

[2] **Zoning and Planning**  [Police power](#)

The inherent local police power includes broad authority to determine, for purposes of the public health, safety, and welfare, the appropriate uses of land within a local jurisdiction's borders.

[3] **Zoning and Planning**  [Police power](#)

The local police power generally includes the authority to establish aesthetic conditions for land use.

[4] **Municipal Corporations**  [Conformity to constitutional and statutory provisions in general](#)

Municipal legislation that conflicts with state law is void.

[1 Cases that cite this headnote](#)

[5] **Municipal Corporations**  [Conformity to constitutional and statutory provisions in general](#)

A conflict exists between local legislation and state law, rendering local legislation void, when the local legislation duplicates, contradicts, or enters an area fully occupied by general law, either expressly or by legislative implication.

[3 Cases that cite this headnote](#)

[6] **Municipal Corporations** ➔ Conformity to constitutional and statutory provisions in general

Local legislation duplicates general law, as could render local legislation void, if both enactments are coextensive.

1 Cases that cite this headnote

[7] **Municipal Corporations** ➔ Conformity to constitutional and statutory provisions in general

Local legislation is contradictory to general law, as could render the local legislation void, when it is inimical to general law.

[11] **Municipal Corporations** ➔ Proceedings concerning construction and validity of ordinances

Ruling on a facial challenge to a local ordinance, the court considers the text of the measure itself, not its application to any particular circumstances or individual.

2 Cases that cite this headnote

[8] **Municipal Corporations** ➔ Conformity to constitutional and statutory provisions in general

State law fully occupies a field, as could render local legislation in same field void, when the Legislature expressly manifests its intent to occupy the legal area or when the Legislature impliedly occupies the field.

[12] **Appeal and Error** ➔ De novo review

Questions of law are subject to de novo review.

4 Cases that cite this headnote

[9] **Municipal Corporations** ➔ Presumptions and burden of proof

The party claiming preemption of local legislation by state law has the burden of proof.

1 Cases that cite this headnote

[13] **Municipal Corporations** ➔ Concurrent and Conflicting Exercise of Power by State and Municipality

**Telecommunications** ➔ Franchises or licenses and rights of way regulation

State statute providing that telephone corporations could construct lines and erect equipment along public roads in ways and locations that did not “incommode the public use of the road” did not preempt local regulation allowing city to condition permit approval for telephone line construction on aesthetic considerations; city's inherent police power to determine appropriate uses of land included authority to establish aesthetic conditions for land use, to “incommode” could include various impacts disturbing quiet enjoyment, ordinance did not require a company to obtain any local franchise, and statute said nothing about aesthetics or appearance of telephone lines. Cal. Pub. Util. Code § 7901.

[10] **Municipal Corporations** ➔ Presumptions and burden of proof

**Zoning and Planning** ➔ Validity of regulations in general

When local government regulates in an area over which it traditionally has exercised control, such as the location of particular land uses, courts will presume the regulation is not preempted by state law unless there is a clear indication of preemptive intent.

[14] **Municipal Corporations** ➔ Concurrent and Conflicting Exercise of Power by State and Municipality

**Telecommunications** ➔ Franchises or licenses and rights of way regulation

Because state law grants telephone corporations a statewide franchise to engage in the telecommunications business, a local government cannot insist that a telephone corporation obtain a local franchise to operate within its jurisdiction. [Cal. Pub. Util. Code § 7901](#).

**[15] Telecommunications**  [Local Franchise or Regulation; Use of Streets, Roads or Public Places](#)

The right of telephone corporations to construct telephone lines in public rights-of-way is not absolute; instead, it is a limited right to use the highways only to the extent necessary for the furnishing of services to the public.

**[16] Municipal Corporations**  [Concurrent and Conflicting Exercise of Power by State and Municipality](#)

**Municipal Corporations**  [Ordinances permitting acts which state law prohibits](#)

The “contradictory and inimical” form of preemption of an ordinance does not apply unless the ordinance directly requires what the state statute forbids or prohibits what the state enactment demands.

**[17] Municipal Corporations**  [Concurrent and Conflicting Exercise of Power by State and Municipality](#)

Field preemption generally exists over an ordinance where the Legislature has comprehensively regulated in an area, leaving no room for additional local action.

[2 Cases that cite this headnote](#)

**[18] Telecommunications**  [Regulation in general](#)

The power to regulate the location and manner of telephone line installation is generally a matter left to local regulation.

**[19] Administrative Law and Procedure**  [Public utilities](#)

**Public Utilities**  [Review and determination in general](#)

The Public Utility Commission's (PUC) interpretation of the Public Utility Code should not be disturbed unless it fails to bear a reasonable relation to statutory purposes and language. [Cal. Pub. Util. Code § 1 et seq.](#)

**[20] Telecommunications**  [Judicial review or intervention](#)

Default policy of Public Utilities Commission (PUC) is one of deference to municipalities in matters concerning the design and location of wireless facilities. [Cal. Const. art. 12, § 8](#).

**[21] Public Utilities**  [Nature and status](#)

Role of Public Utilities Commission (PUC) is that of the agency of last resort, intervening only when a utility contends that local actions impede statewide goals. [Cal. Const. art. 12, § 8](#).

**[22] Telecommunications**  [Local government regulation; proceedings](#)

Statute allowing cities to control time, place, and manner in which roads are “accessed” by wireless telephone corporations only applies to temporary access during construction and installation of telephone lines and equipment. [Cal. Pub. Util. Code § 7901.1](#).

**Witkin Library Reference:** [8 Witkin, Summary of Cal. Law \(11th ed. 2017\) Constitutional Law, § 1108 \[Test of Valid Regulation\]](#)

**\*\*241 \*\*\*414** First Appellate District, Division Five, A144252, San Francisco City and County Superior Court, CGC-11-510703, [James J. McBride](#), Judge

## Attorneys and Law Firms

Wiley Rein, [Joshua S. Turner](#), Matthew J. Gardner, [Megan L. Brown](#), [Meredith G. Singer](#); Davis Wright Tremaine, [Martin L. Fineman](#), San Francisco, [T. Scott Thompson](#) and [Daniel P. Reing](#) for Plaintiffs and Appellants.

[Janet Galeria](#); Jenner & Block, [Scott B. Wilkens](#), [Matthew S. Hellman](#), [Adam G. Unikowsky](#), [Erica L. Ross](#) and [Leonard R. Powell](#) for the Chamber of Commerce of the United States of America, the California Chamber of Commerce, the San Francisco Chamber of Commerce, the Bay Area Council and the Silicon Valley Leadership Group as Amici Curiae on behalf of Plaintiffs and Appellants.

Mayer Brown, [Hans J. Germann](#), [Donald M. Falk](#), Palo Alto, and [Samantha Booth](#) for Pacific Bell Telephone Company and AT&T Mobility, LLC, as Amici Curiae on behalf of Plaintiffs and Appellants.

Crowell & Moring, [Emily T. Kuwahara](#), Los Angeles, and [Colin Proksel](#) for American Consumer Institute Center for Citizen Research as Amicus Curiae on behalf of Plaintiffs and Appellants.

Wilkinson Barker Knauer, [Christine M. Crowe](#) and [Craig E. Gilmore](#) for CTIA-The Wireless Association and the Wireless Infrastructure Association as Amici Curiae on behalf of Plaintiffs and Appellants.

[Dennis J. Herrera](#), City Attorney, Yvonne R. Meré, Chief of Complex and Affirmative Litigation, [Christine Van Aken](#), Chief of Appellate Litigation, [William K. Sanders](#), Erin B. Bernstein and [Jeremy M. Goldman](#), Deputy City Attorneys, for Defendants and Respondents.

Rutan & Tucker, [Jeffrey T. Melching](#), Costa Mesa, and [Ajit Singh Thind](#) for League of California Cities, California State Association of Counties, International Municipal Lawyers Association and the States of California and Nevada Chapter of the National Association of Telecommunications Officers and Advisors as Amici Curiae on behalf of Defendants and Respondents.

## Opinion

Opinion of the Court by [Corrigan](#), J.

\*1113 By ordinance the City and County of San Francisco (the City) requires wireless telephone service companies to obtain permits to \*1114 install and maintain lines

and equipment in public rights-of-way. Some permits will not issue unless the application conforms to the City's established \*\*\*415 aesthetic guidelines. Plaintiffs assert a facial challenge urging that (1) the ordinance is preempted by state law and (2) even if not preempted, the ordinance violates a state statute. The trial court and the Court of Appeal rejected both arguments. We do likewise.

## I. BACKGROUND

Plaintiffs are telecommunications companies. They install and operate wireless equipment throughout the City, including on utility poles located along public roads and highways.<sup>1</sup> In January 2011, the City adopted ordinance No. 12-11 (the Ordinance),<sup>2</sup> which requires “any Person seeking to construct, install, or maintain a Personal Wireless Service Facility in the Public Rights-of-Way to obtain” a permit. (S.F. Pub. Works Code, art. \*\*242 25, § 1500, subd. (a).) In adopting the Ordinance, the board of supervisors noted that the City “is widely recognized to be one of the world's most beautiful cities,” which is vital to its tourist industry and an important reason that residents and businesses locate there. Due to growing demand, requests from the wireless industry to place equipment on utility poles had increased. The board opined that the City needed to regulate the placement of this equipment to prevent installation in ways or locations “that will diminish the City's beauty.” The board acknowledged that telephone corporations have a right, under state law, “to use the public rights-of-way to install and maintain ‘telephone lines’ and related facilities required to provide telephone service.” But it asserted that local governments may “enact laws that limit the intrusive effect of these lines and facilities.”

The Ordinance specifies areas designated for heightened aesthetic review. (See S.F. Pub. Works Code, art. 25, § 1502.) These include historic districts and areas that have “ ‘good’ ” or “ ‘excellent’ ” views or are adjacent to parks or open spaces. (*Ibid.*) The Ordinance establishes various standards of aesthetic compatibility for wireless equipment. In historic districts, for example, installation may only be approved if the City's planning department \*1115 determines that it would not “significantly degrade the aesthetic attributes that were the basis for the special designation” of the building or district. (S.F. Pub. Works Code, art. 25, § 1502; see also *id.*, §§ 1508, 1509, 1510.) In “view” districts, proposed installation may not “significantly impair” the protected views.<sup>3</sup> (S.F. Pub. Works Code, art. 25, § 1502.)



application to any particular circumstances or individual. (*San Francisco Apartment Assn. v. City and County of San Francisco* (2016) 3 Cal.App.5th 463, 487, 207 Cal.Rptr.3d 684, citing *Pieri v. City and County of San Francisco* (2006) 137 Cal.App.4th 886, 894, 40 Cal.Rptr.3d 629, which in turn cites *Tobe v. City of Santa Ana* (1995) 9 Cal.4th 1069, 1084, 40 Cal.Rptr.2d 402, 892 P.2d 1145.)<sup>6</sup>

## 2. Analysis

[12] [13] Section 7901 provides that telephone corporations may construct lines and erect equipment along public roads in ways and locations that do not “incommode the public use of the road.” We review the statute’s language to determine the scope of the rights it grants to telephone corporations and whether, by granting those rights, the Legislature **\*\*244** intended to preempt local regulation based on aesthetic considerations. These questions of law are subject to de novo review. (*Bruns v. E-Commerce Exchange, Inc.* (2011) 51 Cal.4th 717, 724, 122 Cal.Rptr.3d 331, 248 P.3d 1185; *Farm Raised Salmon Cases* (2008) 42 Cal.4th 1077, 1089, fn. 10, 72 Cal.Rptr.3d 112, 175 P.3d 1170.)

[14] The parties agree that section 7901 grants telephone corporations a statewide **\*\*\*418** franchise to engage in the telecommunications business.<sup>7</sup> (See *Western Union Tel. Co. v. Visalia* (1906) 149 Cal. 744, 750, 87 P. 1023 (*Visalia*)). Thus, a local government cannot insist that a telephone corporation obtain a *local* franchise to operate within its jurisdiction. (See *Visalia*, at p. 751, 87 P. 1023; see also *Pac. Tel. & Tel. Co. v. City & County of S. F.* (1959) 51 Cal.2d 766, 771, 336 P.2d 514 (*Pacific Telephone I*)). The parties also agree that the franchise rights conferred are limited by the prohibition against incommoding the public use of roads, and that local governments have authority to prevent those impacts.

**\*1118** Plaintiffs argue section 7901 grants them more than the mere right to operate. In their view, section 7901 grants them the right to construct lines and erect equipment along public roads so long as they do not obstruct the path of travel. The necessary corollary to this right is that local governments cannot prevent the construction of lines and equipment unless the installation of the facilities will obstruct the path of travel. Plaintiffs urge that the Legislature enacted section 7901 to promote technological advancement and ensure a functioning, statewide telecommunications system. In light of those objectives, they contend that their right to construct

telephone lines must be construed broadly, and local authority limited to preventing roadway obstructions.

Preliminarily, plaintiffs’ argument appears to rest on the premise that the City only has the power to regulate telephone line construction based on aesthetic considerations if section 7901’s incommode clause can be read to accommodate that power. That premise is flawed. As mentioned, the City has inherent local police power to determine the appropriate uses of land within its jurisdiction. That power includes the authority to establish aesthetic conditions for land use. Under our preemption cases, the question is not whether the incommode clause can be read to permit the City’s exercise of power under the Ordinance. Rather, it is whether section 7901 divests the City of that power.

We also disagree with plaintiffs’ contention that section 7901’s incommode clause limits their right to construct lines only if the installed lines and equipment would obstruct the path of travel. Contrary to plaintiffs’ argument, the incommode clause need not be read so narrowly. As the Court of Appeal noted, the word “‘incommode’ ” means “‘to give inconvenience or distress to: disturb.’ ” (*T-Mobile West, supra*, 3 Cal.App.5th at p. 351, 208 Cal.Rptr.3d 248, citing Merriam-Webster Online Dict. <<http://www.merriam-webster.com/dictionary/incommode>> [as of April 3, 2019].)<sup>8</sup> The Court of Appeal also quoted the definition of “incommode” from the 1828 version of Webster’s Dictionary. Under that definition, “incommode” means “‘[t]o give inconvenience to; to give trouble to; to disturb or molest in the quiet enjoyment of something, or in the facility of acquisition.’ ” (*T-Mobile West, supra*, 3 Cal.App.5th at p. 351, 208 Cal.Rptr.3d 248, citing Webster’s Dict. 1828—online ed., available at <<http://www.webstersdictionary1828.com/Dictionary/incommode>> [as of April 3, 2019].) For our purposes, it is sufficient to state that the meaning of incommode has not changed meaningfully since section **\*\*\*419** 7901’s enactment.<sup>9</sup> Obstructing the **\*1119** path of **\*\*245** travel is one way that telephone lines could disturb or give inconvenience to public road use. But travel is not the sole use of public roads; other uses may be incommoded beyond the obstruction of travel. (*T-Mobile West, at pp. 355-356*, 208 Cal.Rptr.3d 248.) For example, lines or equipment might generate noise, cause negative health consequences, or create safety concerns. All these impacts could disturb public road use, or disturb its quiet enjoyment.

Plaintiffs assert the case law supports their statutory construction. For example, *City of Petaluma v. Pac. Tel. &*

*Tel. Co.* (1955) 44 Cal.2d 284, 282 P.2d 43 (*Petaluma*) stated that the “franchise tendered by [section 7901] ... [is] superior to and free from any grant made by a subordinate legislative body.” (*Id.* at p. 287, 282 P.2d 43; see also *Pacific Telephone I, supra*, 51 Cal.2d at p. 770, 336 P.2d 514; *County of Inyo v. Hess* (1921) 53 Cal.App. 415, 425, 200 P. 373 (*County of Inyo* ).) Similarly, *Pac. Tel. & Tel. Co. v. City of Los Angeles* (1955) 44 Cal.2d 272, 282 P.2d 36 (*City of Los Angeles* ), held that the “authority to grant a franchise to engage in the telephone business resides in the state, and the city is without power to require a telephone company to obtain such a franchise unless the right to do so has been delegated to it by the state.” (*Id.* at pp. 279-280, 282 P.2d 36.)

But these cases do not go as far as plaintiffs suggest. Each addressed the question whether a telephone corporation can be required to obtain a local franchise to operate. (See *Pacific Telephone I, supra*, 51 Cal.2d at p. 767, 336 P.2d 514; *Petaluma, supra*, 44 Cal.2d at p. 285, 282 P.2d 43; *City of Los Angeles, supra*, 44 Cal. 2d at p. 276, 282 P.2d 36; *County of Inyo, supra*, 53 Cal.App. at p. 425, 200 P. 373.) None considered the distinct question whether a local government can condition permit approval on aesthetic or other considerations that arise under the local police power. A permit is, of course, different from a franchise. The distinction may be best understood by considering the effect of the denial of either. The denial of a franchise would completely bar a telephone corporation from operating within a city. The denial of a permit, on the other hand, would simply prevent construction of lines in the proposed manner at the proposed location.

A few published decisions have tangentially addressed the scope of the inherent local police power to regulate the manner and location of telephone line installations. Those cases cut against plaintiffs’ proposed construction.

In *Pacific Tel. & Tel. Co. v. City & County of San Francisco* (1961) 197 Cal.App.2d 133, 17 Cal.Rptr. 687 (*Pacific Telephone II* ), the City argued \*1120 it could require a telephone corporation to obtain a local franchise to operate within its jurisdiction because the power to grant franchises fell within its police power. (*Id.* at p. 152, 17 Cal.Rptr. 687.) The court rejected the City’s argument, reasoning that the phrase “ ‘police power’ has two meanings, ‘a comprehensive one embracing in substance the whole field of state authority and the other a narrower one including only state power to deal with the health, safety and morals of the people.’ ” (*Ibid.*) “Where a corporation has a state franchise to use a city’s

\*\*\*420 streets, the city derives its rights to regulate the particular location and manner of installation of the franchise holder’s facilities from the narrower sense of the police power. Thus, because of the state concern in communications, the state has retained to itself the broader police *power of granting franchises*, leaving to the municipalities the narrower police *power of controlling location and manner of installation.*” (*Ibid.*, italics added.)

This court, too, has distinguished the power to grant franchises from the power to regulate the location and manner of installation by permit. In *Visalia, supra*, 149 Cal. 744, 87 P. 1023, the city adopted an ordinance that (i) authorized a telephone company to erect telegraph poles and wires on city streets, (ii) approved the location of poles and wires then in use, (iii) prohibited poles and wires from interfering with travel on city streets, and (iv) required all poles to be of a uniform height. (*Id.* at pp. 747-748, 87 P. 1023.) The city asserted its ordinance operated to grant the company a “ ‘franchise,’ ” and \*\*246 then attempted to assess a tax on the franchise. (*Id.* at p. 745, 87 P. 1023.) The company challenged the assessment. It argued that, because the ordinance did not create a franchise, the tax assessment was invalid. (*Id.* at pp. 745-746, 87 P. 1023.) We concluded the ordinance did not create a local franchise. (*Id.* at p. 750, 87 P. 1023.) By virtue of its state franchise, “the appellant had the right, of which the city could not deprive it, to construct and operate its lines along the streets of the city.” (*Ibid.*) “[N]evertheless it could not maintain its poles and wires in such a manner as to unreasonably obstruct and interfere with ordinary travel; and the city had the authority, under its police power, to so regulate the manner of plaintiff’s placing and maintaining its poles and wires *as to prevent unreasonable obstruction of travel.*” (*Id.* at pp. 750-751, 87 P. 1023, italics added.) “[T]he ordinance in question was not intended to be anything more ... than the exercise of this authority to regulate.” (*Id.* at p. 751, 87 P. 1023)<sup>10</sup>

[15] Plaintiffs argue the italicized language above shows that local regulatory authority is limited to preventing travel obstructions. But the quoted language is merely descriptive, not prescriptive. *Visalia* involved an ordinance that specifically prohibited interference with travel on city streets, and \*1121 the court was simply describing the ordinance before it, not establishing the bounds of local government regulatory authority. Moreover, the *Visalia* court did not question the propriety of the ordinance’s requirement that all poles be a uniform height, nor suggest that requirement was related to preventing obstructions to travel. Thus, *Visalia*

does not support the conclusion that [section 7901](#) was meant to restrict local government power in the manner plaintiffs suggest. The “right of telephone corporations to construct telephone lines in public rights-of-way is not absolute.” (*City of Huntington Beach v. Public Utilities Com.* (2013) 214 Cal.App.4th 566, 590, 154 Cal.Rptr.3d 241 (*City of Huntington Beach*)). Instead, it is a “ ‘limited right to use the highways ... only to the extent necessary for the furnishing of services to the public.’ ” (*Ibid.*, quoting *County of L. A. v. Southern Cal. Tel. Co.* (1948) 32 Cal.2d 378, 387, 196 P.2d 773; see also *Pacific Tel. & Tel. Co. v. Redevelopment Agency* (1977) 75 Cal.App.3d 957, 963, 142 Cal.Rptr. 584.)<sup>11</sup>

\*\*\*421 Having delineated the right granted by [section 7901](#), we now turn to its preemptive sweep. Because the location and manner of line installation are areas over which local governments traditionally exercise control (*Visalia, supra*, 149 Cal. at pp. 750-751, 87 P. 1023), we presume the ordinance is not preempted absent a clear indication of preemptive intent. (*Big Creek Lumber, supra*, 38 Cal.4th at p. 1149, 45 Cal.Rptr.3d 21, 136 P.3d 821.) Plaintiffs put forth a number of preemption theories. They argue the Ordinance is contradictory to [section 7901](#). At oral argument, they asserted the Legislature occupied the field with [section 7901](#), the terms of which indicate that a paramount state concern will not tolerate additional local action. And in their briefs, many of plaintiffs’ arguments were focused on what has been labeled, in the federal context, as obstacle preemption.

[16] “The ‘contradictory and inimical’ form of preemption does not apply unless the ordinance directly requires what the state statute forbids or prohibits what the state enactment demands.” (*City of Riverside, supra*, 56 Cal.4th at p. 743, 156 Cal.Rptr.3d 409, 300 P.3d 494, citing \*\*247 *Big Creek Lumber, supra*, 38 Cal.4th at p. 1161, 45 Cal.Rptr.3d 21, 136 P.3d 821.) “[N]o inimical conflict will be found where it is reasonably possible to comply with both the state and local laws.” (*City of Riverside, supra*, at p. 743, 156 Cal.Rptr.3d 409, 300 P.3d 494.) As noted, [section 7901](#) grants telephone corporations the right to install lines on \*1122 public roads without obtaining a local franchise. The Ordinance does not require plaintiffs to obtain a local franchise to operate within the City. Nor does it allow certain companies to use public roads while excluding others. Any wireless provider may construct telephone lines on the City’s public roads so long as it obtains a permit, which may sometimes be conditioned on aesthetic approval. Because [section 7901](#) says nothing about the aesthetics or appearance of telephone lines, the Ordinance is not inimical to the statute.

[17] [18] The argument that the Legislature occupied the field by implication likewise fails. Field preemption generally exists where the Legislature has comprehensively regulated in an area, leaving no room for additional local action. (See, e.g., *American Financial Services Assn. v. City of Oakland* (2005) 34 Cal.4th 1239, 1252-1257, 23 Cal.Rptr.3d 453, 104 P.3d 813; *O’Connell, supra*, 41 Cal.4th 1061, 1068-1074, 63 Cal.Rptr.3d 67, 162 P.3d 583.) Unlike the statutory schemes addressed in *American Financial* and *O’Connell*, [section 7901](#) does not comprehensively regulate telephone line installation or provide a general regulatory scheme. On the contrary, [section 7901](#) consists of a single sentence. Moreover, although the granting of telephone franchises has been deemed a matter of statewide concern (*Pacific Telephone I, supra*, 51 Cal.2d at p. 774, 336 P.2d 514; *Pacific Telephone II, supra*, 197 Cal.App.2d at p. 152, 17 Cal.Rptr. 687), the power to regulate the location and manner of line installation is generally a matter left to local regulation. The City is not \*\*\*422 attempting to regulate in an area over which the state has traditionally exercised control. Instead, this is an area of regulation in which there are “ ‘significant local interest[s] to be served that may differ from one locality to another.’ ” (*Big Creek Lumber, supra*, 38 Cal.4th at p. 1149, 45 Cal.Rptr.3d 21, 136 P.3d 821.)

*City of Riverside, supra*, 56 Cal.4th 729, 156 Cal.Rptr.3d 409, 300 P.3d 494, is instructive. There, the question was whether state statutes designed to enhance patient and caregiver access to medical marijuana preempted a local zoning law banning dispensaries within a city’s limits. (*Id.* at pp. 737, 739-740, 156 Cal.Rptr.3d 409, 300 P.3d 494.) An early enactment had declared that physicians could not be punished for recommending medical marijuana and that state statutes prohibiting possession and cultivation of marijuana would not apply to patients or caregivers. (*Id.* at p. 744, 156 Cal.Rptr.3d 409, 300 P.3d 494.) A subsequent enactment established a program for issuing medical marijuana identification cards and provided that a cardholder could not be arrested for possession or cultivation in permitted amounts. (*Id.* at p. 745, 156 Cal.Rptr.3d 409, 300 P.3d 494.) We concluded that the “narrow reach of these statutes” (*ibid.*) showed they did not “expressly or impliedly preempt [the city’s] zoning provisions” (*id.* at p. 752, 156 Cal.Rptr.3d 409, 300 P.3d 494).

Preemption was not implied because the Legislature had not tried “to fully occupy the field of medical marijuana regulation as a matter of statewide concern, or to partially occupy this field under circumstances indicating that further

local regulation will not be tolerated.” ( \*1123 *City of Riverside, supra*, 56 Cal.4th at p. 755, 156 Cal.Rptr.3d 409, 300 P.3d 494.) While state statutes took “limited steps toward recognizing marijuana as a medicine,” they described “no comprehensive scheme or system for authorizing, controlling, or regulating the processing and distribution of marijuana for medical purposes, such that no room remains for local action.” (*Ibid.*) Moreover, there were significant local interests that could vary by jurisdiction, giving rise to a presumption against preemption. (*Ibid.*)

Similarly, here, the Legislature has not adopted a comprehensive regulatory scheme. Instead, it has taken the limited step of guaranteeing that telephone corporations need not secure a local franchise to operate in the state or to construct local lines and equipment. Moreover, the statute leaves room for additional local action and there are \*\*248 significant local interests relating to road use that may vary by jurisdiction.

Finally, plaintiffs’ briefing raises arguments that sound in the theory of obstacle preemption. Under that theory, a local law would be displaced if it hinders the accomplishment of the purposes behind a state law. This court has never said explicitly whether state preemption principles are coextensive with the developed federal conception of obstacle preemption. (See, e.g., *Great Western Shows, Inc. v. County of Los Angeles* (2002) 27 Cal.4th 853, 867-868, 118 Cal.Rptr.2d 746, 44 P.3d 120; cf. *City of Riverside, supra*, 56 Cal.4th at pp. 763-765, 156 Cal.Rptr.3d 409, 300 P.3d 494 (conc. opn. of Liu, J.)) But assuming for the sake of argument that the theory applies, we conclude there is no obstacle preemption here.

The gist of plaintiffs’ argument is that section 7901’s purpose is to encourage technological advancement in the state’s telecommunications networks and that, because enforcement of the Ordinance *could* hinder that purpose, the Ordinance is preempted. But no legislation pursues its objectives at all costs. (*Pension Ben. Guar. Corp. v. LTV Corp.* (1990) 496 U.S. 633, 646-647, 110 S.Ct. 2668, 110 L.Ed.2d 579.) \*\*\*423 Moreover, the Legislature made clear that the goal of technological advancement is not paramount to all others by including the incommode clause in section 7901, thereby leaving room for local regulation of telephone line installation.

[19] Finally, we think it appropriate to consider the Public Utilities Commission’s (PUC) understanding of the statutory

scheme. In recognition of its expertise, we have consistently accorded deference to the PUC’s views concerning utilities regulation. The PUC’s “interpretation of the Public Utility Code ‘should not be disturbed unless it fails to bear a reasonable relation to statutory purposes and language.’” (*Southern California Edison Co. v. Peevey* (2003) 31 Cal.4th 781, 796, 3 Cal.Rptr.3d 703, 74 P.3d 795, quoting *Greyhound Lines, Inc. v. Public Utilities Com.* (1968) 68 Cal.2d 406, 410-411, 67 Cal.Rptr. 97, 438 P.2d 801.) Here, the PUC has made determinations about the scope of permissible regulation that are on point.

\*1124 The state Constitution vests principal regulatory authority over utilities with the PUC, but carves out an ongoing area of municipal control. (Cal. Const., art. XII, § 8.) A company seeking to build under section 7901 must approach the PUC and obtain a certificate of public necessity. (§ 1001; see *City of Huntington Beach, supra*, 214 Cal.App.4th at p. 585, 154 Cal.Rptr.3d 241.) The certificate is not alone sufficient; a utility will still be subject to local control in carrying out the construction. Municipalities may surrender to the PUC regulation of a utility’s relations with its customers (§ 2901), but they are forbidden from yielding to the PUC their police powers to protect the public from the adverse impacts of utilities operations (§ 2902).

[20] [21] Consistent with these statutes, the PUC’s default policy is one of deference to municipalities in matters concerning the design and location of wireless facilities. In a 1996 opinion adopting the general order governing wireless facility construction, the PUC states the general order “recognize[s] that primary authority regarding cell siting issues should continue to be deferred to local authorities. ... The [PUC’s] role continues to be that of the agency of last resort, intervening only when a utility contends that local actions impede statewide goals ....” (*Re Siting and Environmental Review of Cellular Mobile Radiotelephone Utility Facilities* (1996) 66 Cal.P.U.C.2d 257, 260; see also *Re Competition for Local Exchange Service* (1998) 82 Cal.P.U.C.2d 510, 544.)<sup>12</sup> The order itself “acknowledges that local citizens and local government are often in a better position than the [PUC] to measure local impact and to identify alternative sites. Accordingly, the [PUC] will generally defer to local governments to regulate the location and design \*\*249 of cell sites ....” (PUC, General order No. 159A (1996) p. 3 (General Order 159A), available at <<http://docs.cpuc.ca.gov/PUBLISHED/Graphics/611.PDF>> [as of Apr. 3, 2019].)

The exception to this default policy is telling: The PUC reserves the right to preempt local decisions about specific sites “when there is a clear conflict with the [PUC’s] goals and/or statewide interests.” (General Order 159A, *supra*, at p. 3.) In other words, generally the PUC will not object to municipalities dictating alternate \*\*\*424 locations based on local impacts,<sup>13</sup> but it will step in if statewide goals such as “high quality, reliable and widespread cellular services to state residents” are threatened. (General Order 159A, at \*1125 p. 3.) Contrary to plaintiffs’ view of the respective spheres of state and local authority, the PUC’s approach does not restrict municipalities to judging only whether a requested permit would impede traffic. Instead, the PUC accords local governments the full scope of their ordinary police powers unless the exercise of those powers would undermine state policies.

Plaintiffs argue our construction of [section 7901](#), and a decision upholding the City’s authority to enforce the Ordinance, will “hinder the roll-out of advanced services needed to upgrade networks [and] promote universal broadband” and will “stymie the deployment of 5G networks, leaving California unable to meet the growing need for wireless capacity created by the proliferation of ... connected devices.” This argument is premised on a hypothetical future harm that is not cognizable in a facial challenge. (*Pacific Legal Foundation v. Brown* (1981) 29 Cal.3d 168, 180, 172 Cal.Rptr. 487, 624 P.2d 1215; see also *Arcadia Unified School Dist. v. State Dept. of Education* (1992) 2 Cal.4th 251, 267, 5 Cal.Rptr.2d 545, 825 P.2d 438.)

In sum, neither the plain language of [section 7901](#) nor the manner in which it has been interpreted by courts and the PUC supports plaintiffs’ argument that the Legislature intended to preempt local regulation based on aesthetic considerations. The statute and the ordinance can operate in harmony. [Section 7901](#) ensures that telephone companies are not required to obtain a local franchise, while the Ordinance ensures that lines and equipment will not unreasonably incommode public road use.<sup>14</sup>

#### *B. The Ordinance Does Not Violate [Section 7901.1](#)*

Plaintiffs next contend that, even if not preempted, the Ordinance violates [section 7901.1](#) by singling out wireless telephone corporations for regulation. [Section 7901.1](#) provides in relevant part that, consistent with [section 7901](#), municipalities may “exercise reasonable control as to the time, place, and manner” in which roads are “accessed,”

and that the control must “*be applied to all entities in an equivalent manner.*” (§ 7901, subds. (a), (b), italics added.)

\*1126 Before trial, the parties stipulated to the following facts. First, that the City requires all utility and telephone corporations, both wireless and nonwireless, to \*\*\*425 obtain temporary occupancy permits to “access” public rights-of-way during the *initial* construction and installation of equipment facilities. These permits are not subject to aesthetic review. Second, that the City requires only wireless \*\*250 telephone corporations to obtain site-specific permits, conditioned on aesthetic approval, for the *ongoing* occupation and maintenance of equipment facilities in public rights-of-way. The trial court and the Court of Appeal held that [section 7901.1](#) only applies to *temporary* access to public rights-of-way, during initial construction and installation. Because the parties had stipulated that the City treats all companies equally in that respect, the lower courts found no violation of [section 7901.1](#).

Plaintiffs argue the plain language of [section 7901.1](#) does not limit its application to temporary access to public rights-of-way. Rather, the introductory phrase, “consistent with [section 7901](#),” demonstrates that [section 7901.1](#) applies to both short-and long-term access. Plaintiffs also suggest that the legislative history of [section 7901.1](#) supports their position, and that the lower courts’ interpretation of [section 7901.1](#) “results in an incoherent approach to municipal authority.”

Plaintiffs’ arguments are unpersuasive. [Section 7901.1](#) allows cities to control the time, place, and manner in which roads are “accessed.” (§ 7901.1, subd. (a).) As the competing arguments demonstrate, the “plain meaning of the word ‘accessed’ is ambiguous.” (*T-Mobile West, supra*, 3 Cal.App.5th at p. 358, 208 Cal.Rptr.3d 248.) It could refer only to short-term access, during the initial installation and construction of a telephone equipment facility. But it could also refer to the longer term occupation of public rights-of-way with telephone equipment. (*Ibid.*) Though it would be odd for a statute authorizing local control over *permanent* occupations to specifically allow for control over the “time” of such occupations, the statute’s plain language does not render plaintiffs’ construction totally implausible.

However, the legislative history shows that [section 7901.1](#) only deals with temporary access to public rights-of-way. “This bill is intended to bolster the cities[’] abilities with regard to *construction management* ....” (Sen. Rules Com., Off. of Sen. Floor Analyses, 3d reading analysis of Sen.

Bill No. 621 (1995–1996 Reg. Sess.) as amended May 3, 1995, p. 3, italics added.) Before [section 7901.1](#)'s enactment, telephone companies had been taking the “extreme” position, based on their statewide franchises, that “cities [had] absolutely no right to control construction.” (Assem. Com. on Utilities & Commerce, Rep. on Sen. Bill No. 621 (1995–1996 Reg. Sess.) as amended July 7, 1995, p. 2.) [Section 7901.1](#) was enacted to “send a message to telephone corporations that cities have authority to manage their construction, without \*1127 jeopardizing the telephone [corporations’] statewide franchise.” (Sen. Rules Com., Off. of Sen. Floor Analyses, 3d reading analysis of Sen. Bill No. 621 (1995–1996 Reg. Sess.) as amended May 3, 1995, p. 3.) Under [section 7901.1](#), cities would be able to “plan maintenance programs, protect public safety, minimize public inconvenience, and ensure adherence to sound construction practices.” (Assem. Com. on Utilities and Commerce, Rep. on Sen. Bill No. 621 (1995–1996 Reg. Sess.) as amended July 7, 1995, p. 2.)

[22] To accept plaintiffs’ construction of [section 7901.1](#), we would have to ignore this legislative history. (*T-Mobile West*, *supra*, 3 Cal.App.5th at p. 358, 208 Cal.Rptr.3d 248.) Contrary to plaintiffs’ argument, construing [section 7901.1](#) in this manner does not render the scheme incoherent. It is

eminently reasonable that a local government may: (1) control the time, \*\*\*426 place, and manner of temporary access to public roads during construction of equipment facilities; and (2) regulate other, longer term impacts that might incommode public road use under [section 7901](#). Thus, we hold that [section 7901.1](#) only applies to temporary access during construction and installation of telephone lines and equipment. Because the City treats all entities similarly in that regard, there is no [section 7901.1](#) violation.

### III. DISPOSITION

The judgment of the Court of Appeal is affirmed.

[Cantil-Sakaue](#), C. J., [Chin](#), J., [Liu](#), J., [Cuéllar](#), J., [Kruger](#), J., and [Groban](#), J., concurred.

### All Citations

6 Cal.5th 1107, 438 P.3d 239, 245 Cal.Rptr.3d 412, 19 Cal. Daily Op. Serv. 3067, 2019 Daily Journal D.A.R. 2886

### Footnotes

- 1 The plaintiffs named in the operative complaint were T-Mobile West Corporation, NextG Networks of California, Inc., and ExteNet Systems (California) LLC. T-Mobile West Corporation has also appeared in this litigation as T-Mobile West LLC. NextG Networks of California, Inc. has also appeared as Crown Castle NG West LLC and Crown Castle NG West Inc. (*T-Mobile West LLC v. City and County of San Francisco* (2016) 3 Cal.App.5th 334, 340, fn. 3, 208 Cal.Rptr.3d 248 (*T-Mobile West*)). Not all plaintiffs install and operate the same equipment, but there is no dispute that they are all “telephone corporation[s],” as that term is defined by [Public Utilities Code section 234](#), nor that all of the equipment in question fits within the definition of “telephone line” in [Public Utilities Code section 233](#). All unspecified statutory references are to the Public Utilities Code.
- 2 The Ordinance was codified as article 25 of the San Francisco Public Works Code.
- 3 The Court of Appeal discussed other provisions of a previous enactment of the Ordinance that are not in issue here. (*T-Mobile West*, *supra*, 3 Cal.App.5th at pp. 340-341, 208 Cal.Rptr.3d 248.) We review the current version of the Ordinance. (*Kash Enterprises, Inc. v. City of Los Angeles* (1977) 19 Cal.3d 294, 306, fn. 6, 138 Cal.Rptr. 53, 562 P.2d 1302.)
- 4 Plaintiffs’ first, second, fourth, and fifth causes of action are not before us. The first cause of action was resolved in plaintiffs’ favor by summary adjudication. The second was dismissed by plaintiffs before trial. The fourth was resolved in City’s favor by summary adjudication. And the fifth was resolved in plaintiffs’ favor after trial.
- 5 This case does not involve the construction or installation of lines or equipment across state waters. Thus, we limit our discussion to lines installed along public roads and highways, which we refer to collectively as public roads.
- 6 There is some uncertainty regarding the standard for facial constitutional challenges to statutes and local ordinances. (*Today’s Fresh Start, Inc. v. Los Angeles County Office of Education* (2013) 57 Cal.4th 197, 218, 159 Cal.Rptr.3d 358, 303 P.3d 1140.) Some cases have held that legislation is invalid if it conflicts in the generality or great majority of cases. (*Guardianship of Ann S.* (2009) 45 Cal.4th 1110, 1126, 90 Cal.Rptr.3d 701, 202 P.3d 1089.) Others have articulated a stricter standard, holding that legislation is invalid only if it presents a total and fatal conflict with applicable constitutional prohibitions. (*Ibid.*; see also *Tobe v. City of Santa Ana*, *supra*, 9 Cal.4th at p. 1084, 40 Cal.Rptr.2d 402, 892 P.2d 1145.)

We need not settle on a precise formulation of the applicable standard because, as explained below, we find no inherent conflict between the Ordinance and section 7901. Thus, plaintiffs' claim fails under any articulated standard.

7 In this context, a franchise is a "government-conferred right or privilege to engage in specific business or to exercise corporate powers." (Black's Law Dict. (10th ed. 2014) p. 772, col. 2.)

8 All Internet citations in this opinion are archived by year, docket number, and case name at <<http://www.courts.ca.gov/38324.htm>>.

9 The predecessor of section 7901, [Civil Code section 536](#), was first enacted in 1872 as part of the original Civil Code. (*Anderson v. Time Warner Telecom of California* (2005) 129 Cal.App.4th 411, 419, 28 Cal.Rptr.3d 289, citing *Sunset Tel. and Tel. Co. v. Pasadena* (1911) 161 Cal. 265, 273, 118 P. 796.) [Civil Code section 536](#) contained the "incommodious" language, as did its predecessor, which was adopted as part of the Statutes of California in 1850. (Stats. 1850, ch. 128, § 150, p. 369.)

10 *Visalia* interpreted a predecessor statute, [Civil Code section 536](#), which was repealed in 1951 and reenacted as section 7901. (Stats. 1951, ch. 764, pp. 2025, 2194, 2258 [reenacting Civ. Code, former § 536 as [Pub. Util. Code, § 7901](#)].)

11 The Ninth Circuit has addressed this issue twice, coming to a different conclusion each time. In *Sprint PCS Assets v. City of Palos Verdes Estates* (9th Cir. 2009) 583 F.3d 716, the Ninth Circuit found no conflict between [section 7901](#) and a local ordinance conditioning permit approval on aesthetic considerations. (*Palos Verdes Estates*, at pp. 721-723.) In an unpublished decision issued three years earlier, the Ninth Circuit had reached the opposite conclusion. (*Sprint PCS v. La Cañada Flintridge* (9th Cir. 2006) 182 Fed.Appx. 688, 689.) Due to its unpublished status, the *La Cañada Flintridge* decision carries no precedential value. (*T-Mobile West*, *supra*, 3 Cal.App.5th at p. 355, 208 Cal.Rptr.3d 248, citing *Bowen v. Ziasun Technologies, Inc.* (2004) 116 Cal.App.4th 777, 787, fn. 6, 11 Cal.Rptr.3d 522.)

12 In its 1996 opinion adopting general order No. 159-A, the PUC left implicit the portions of the statutory scheme it was applying. In its 1998 opinion, the PUC clarified the respective regulatory spheres in response to arguments based on [sections 2902, 7901, 7901.1](#) and the constitutional provisions allocating authority to cities and the PUC. (See *Re Competition for Local Exchange Service*, *supra*, 82 Cal.P.U.C.2d at pp. 543-544.)

13 Among the PUC's express priorities regarding wireless facility construction is that "the public health, safety, welfare, and zoning concerns of local government are addressed." (General Order 159A, *supra*, at p. 3.)

14 We dispose here only of plaintiffs' facial challenge and express no opinion as to the Ordinance's application. We note, however, that plaintiffs seeking to challenge specific applications have both state and federal remedies. Under state law, a utility could seek an order from the PUC preempting a city's decision. (General Order 159A, *supra*, at p. 6.) Thus, cities are prohibited from using their powers to frustrate the larger intent of [section 7901](#). (*Pacific Telephone II*, *supra*, 197 Cal.App.2d at p. 146, 17 Cal.Rptr. 687.) Under federal law, Congress generally has left in place local authority over "the placement, construction, and modification of personal wireless service facilities" (47 U.S.C. § 332(c)(7)(A) ), but it has carved out several exceptions. Among these, a city may not unduly delay decisions (47 U.S.C. § 332(c)(7)(B)(ii) ) and may not adopt regulations so onerous as to "prohibit or have the effect of prohibiting the provision of wireless services" (47 U.S.C. § 332(c)(7)(B)(i)(II) ). If a city does so, a wireless company may sue. (*Sprint PCS Assets v. City of Palos Verdes Estates*, *supra*, 583 F.3d at p. 725.)

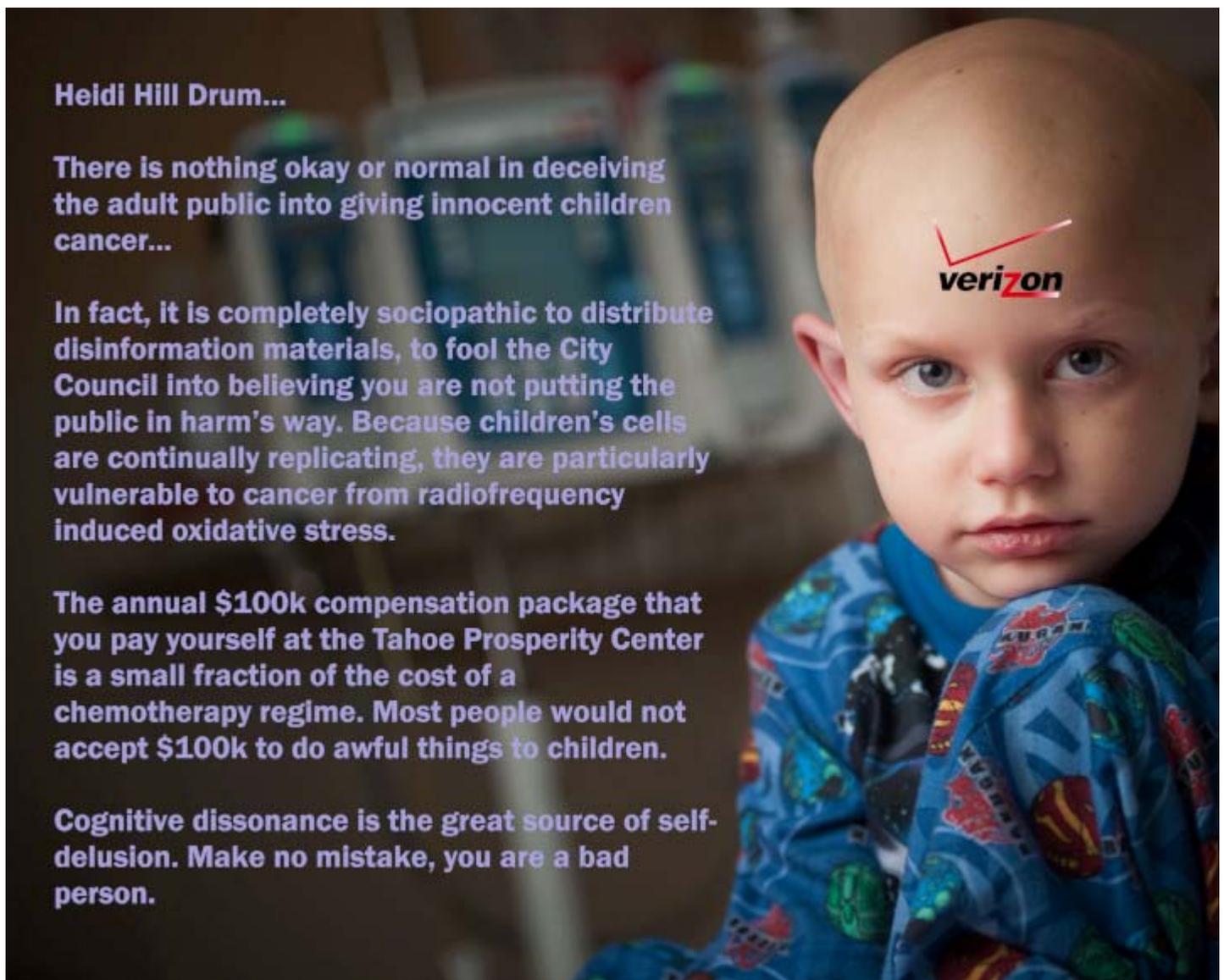
## **Bridget Cornell**

---

**From:** Polly Higgins <polly.higgins@legalprivilege.ch>  
**Sent:** Thursday, April 7, 2022 1:13 PM  
**To:** Joanne Marchetta; John Marshall; Marja Ambler; Katherine Hangeland  
**Subject:** TRPA--H.O.: Hearings Officer Meeting Agenda Item No V.B Verizon/Tahoe Seasons New Telecommunications Facility  
**Attachments:** Heidi Hill-Drum\_cancer.pdf

Dear TRPA Hearings Officer,

I just have one tiny thing to say...



Thanks,

Polly Higgins

**Heidi Hill Drum...**

**There is nothing okay or normal in deceiving the adult public into giving innocent children cancer...**

**In fact, it is completely sociopathic to distribute disinformation materials, to fool the City Council into believing you are not putting the public in harm's way. Because children's cells are continually replicating, they are particularly vulnerable to cancer from radiofrequency induced oxidative stress.**

**The annual \$100k compensation package that you pay yourself at the Tahoe Prosperity Center is a small fraction of the cost of a chemotherapy regime. Most people would not accept \$100k to do awful things to children.**

**Cognitive dissonance is the great source of self-delusion. Make no mistake, you are a bad person.**

