

Kendall Sawyer

Attachments: Problems evaluating RF health impacts Ishai Davis Taylor Birnbaum 2023 (2).pdf

From: lendri purcell
Sent: Monday, March 20, 2023 10:00 AM
To: -- City Council
Subject: Allow Opt-out for "Smart" water meters

---Warning: Use caution before clicking any attachments. THIS EMAIL IS FROM OUTSIDE OUR EMAIL SYSTEM.---
Dear City Council,

Please ensure Petalumans' privacy and safety with regard to "smart water meters."

Please insist this program:

- Include an "Opt-out" in the wireless EMR (electromagnetic radiation) smart meters
- Ensure their **related EMR telecommunications facilities and antennas do not violate Petaluma's Wireless Ordinance**, which does not allow 5G antennas and telecom facilities in residential neighborhoods and schools
- Disallow the distribution, selling, etc., of user data to any parties other than City of Petaluma

This project is not a Cell Tower Developer project, and therefore the City has 100% power/control to use full, free discretion.

Please ensure full precaution to protect resident privacy and safety. Research on cumulative impact of wireless device and antenna proliferation has not been proven safe, especially for children (whose bodies are still developing and vulnerable to EMR) and medically vulnerable and disabled individuals.

As the attached paper concludes:

"There is a plethora of both experimental and epidemiological evidence establishing a causal relationship between EMF and cancer and other adverse health effects including adverse effects on fetal development and the endocrine system. Increases in biochemical alterations such as DNA damage, increased production of free radicals and other signals found to be predictive of cancer and other degenerative diseases have been clearly demonstrated."

Further, residents should have a right to privacy. This should be something we can opt out of, with any/all usage data protected and not shared outside of City Public Works.

Thank you. Sincerely, Lendri Purcell



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Problems in evaluating the health impacts of radio frequency radiation

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ABSTRACT

In an effort to clarify the nature of causal evidence regarding the potential impacts of RFR on biological systems, this paper relies on a well-established framework for considering causation expanded from that of Bradford Hill, that combines experimental and epidemiological evidence on carcinogenesis of RFR. The Precautionary Principle, while not perfect, has been the effective lodestone for establishing public policy to guard the safety of the general public from potentially harmful materials, practices or technologies. Yet, when considering the exposure of the public to anthropogenic electromagnetic fields, especially those arising from mobile communications and their infrastructure, it seems to be ignored. The current exposure standards recommended by the Federal Communications Commission (FCC) and International Commission on Non-Ionizing Radiation Protection (ICNIRP) consider only thermal effects (tissue heating) as potentially harmful. However, there is mounting evidence of non-thermal effects of exposure to electromagnetic radiation in biological systems and human populations. We review the latest literature on *in vitro* and *in vivo* studies, on clinical studies on electromagnetic hypersensitivity, as well as the epidemiological evidence for cancer due to the action of mobile based radiation exposure. We question whether the current regulatory atmosphere truly serves the public good when considered in terms of the Precautionary Principle and the principles for deducing causation established by Bradford Hill. We conclude that there is substantial scientific evidence that RFR causes cancer, endocrinological, neurological and other adverse health effects. In light of this evidence the primary mission of public bodies, such as the FCC to protect public health has not been fulfilled. Rather, we find that industry convenience is being prioritized and thereby subjecting the public to avoidable risks.

1. Introduction

The perennial question of the biological impacts of Radio Frequency Radiation (RFR) constitutes an especially challenging matter that has come to the fore recently, in part driven by public concerns over the introduction of 5G mobile communications. 5G Small Cell base stations are permitted to be sited as close as 3 m from the ground in proximity to homes, schools and offices in many locales in the US. In the U.S. alone, the industry estimates that up to one million new antennas will be required. 5G ranges broadly from 800 MHz to 100 GHz (Document). As Lin (2022a) has noted, for the higher mm-wave bands, wider spectrum is only accessible over short distances and will depend on the construction of numerous new cells in the dense urban environment. Despite Industry

claims (5G, [EMF Exposure and Safety, 2020](#)), an increase in the number of transmitters is expected to lead to much higher levels of exposure for the general public (Blackman and Forge, 2019). This has provoked public concerns regarding the potential health impacts of RFR.

For nearly a century, well-established controlled bioassay protocols have traditionally formed the foundation for predicting and setting limits for public health exposures to pharmaceuticals, pesticides, radiation, and other agents. Yet as regarding the potential impacts of RFR, positive adverse experimental findings on RFR-induced carcinogenicity that have historically provided guidance for preventive policies, have been subjected to extraordinary and unprecedented attacks. The same can be said for studies of individuals exposed to RFR that solely confirm whether or not past harm has taken place. Research and training in this

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multidisciplinary field of bioelectromagnetics are currently not a funding priority. Further, human studies are especially problematic in light of the widespread use of these technologies, the lack of suitable control groups, the general failure to fund relevant studies, and the recent publication of a few limited or fundamentally flawed, yet widely publicized, reports purporting to show no health risks (Castaño-Vinyals et al., 2022; Grimes, 2021; Karipidis et al., 2021; Schüz et al., 2022; Wu et al., 2015), some of which will be reviewed below.

In 2011, the International Agency for Research on Cancer (IARC) concluded, largely based on epidemiological evidence, that mobile phone radiation—a form of RFR—constituted a possible human carcinogen. In 2018, the U.S. National Toxicology Program (NTP) reported the largest animal study ever conducted on this topic (National Toxicology Program (NTP), 2018); the data provided clear evidence that mobile phone radiation caused cancer in male rats, along with cardiac and other systemic damage, as well as DNA damage in multiple organs in both rats and mice. Despite this evidence, a number of recent widely publicized reports have rejected and discredited the NTP findings as well as a substantial toxicological literature demonstrating a range of health impacts. (Brzozek et al., 2021; Castaño-Vinyals et al., 2022; Grimes, 2021; Karipidis et al., 2021). These critics contend that the weight of evidence does not support a finding of significant negative impacts of RFR. (Momoli et al., 2017; Peres, 2010; Schüz et al., 2022), even though the results of the NTP report and parallel findings (Falcioni et al., 2018; Vornoli et al., 2019) from the Ramazzini Institute study of lower levels of exposure have not been disproven. For instance, Grimes (2021) dismisses the NTP finding by insisting that the exposure SAR level was too high and that this would cause internal heating of the animals. As we note below, a preliminary study was conducted to ensure that the exposure level used would not result in internal heating (M. E. Wyde et al., 2018). Karipidis criticized that whole body exposures were used and that the animals were free to roam (Karipidis et al., 2021). We point out that there is an inherent difficulty in persuading animals to make mobile phone calls and so whole-body exposures are employed. These exposures are more akin to the real world situation that people experience every day.

It is important to note that such dismissive studies presume that the sole biological impact of RFR is a consequence of heating. This presumption ignores a substantial body of independent studies finding that RFR induces numerous adverse biochemical changes affecting the formation of free radicals, the rates of cell growth and death, and cellular membrane transport. These changes are widely reported in organisms as diverse as plants, animals, and humans. Furthermore, the Directorate-General for Parliamentary Research Services (Belpoggi, 2021) of the European Union, and an independent Swiss government scientific advisory group (BERENIS) reaches similar conclusions, adding that “EMF (Electromagnetic Fields) are probably carcinogenic for humans, in particular related to gliomas and acoustic neuromas.” In addition, they add that “... 450–6000 MHz: these frequencies clearly affect male fertility and possibly female fertility too. They may have adverse effects on the development of embryos, fetuses and newborns”. This indicates that EMF/RF functions like a classic endocrine disruptor impairing both male and female reproductive functions.

In an effort to clarify the nature of causal evidence regarding the potential impacts of RFR on biological systems, this paper relies on a well-established framework for considering causation with respect to experimental and epidemiological evidence on carcinogenesis of RFR. Originally developed for the evaluation of infectious agents, Koch's postulates constitute the classic means of assessing causal evidence in medicine (Grimes, 2006). They rest on the requirement that ill animals or persons display evidence of exposure to a particular infectious agent prior to developing illness, such as the tuberculosis bacterium, and those that are not ill do not. Further support for inferring causation comes from studies finding that exposure to the specific agent in the healthy can induce illness. It is generally recognized that such postulates must be modified for the study of cancer, especially considering that infectious

agents such as Epstein-Barr and Human Papilloma viruses can, but do not necessarily, induce the disease (Garcion et al., 2009; Moore and Chang, 2014).

For the study of causation for multiply caused, multi-stage chronic illnesses such as cancer, Sir Austin Bradford (Hill, 1965) adapted the Koch paradigm to include principles for inferring the existence of a causal relationship: strength, consistency, specificity, temporality, and biological gradient for evidence of increased risk. One recent assessment (Carlberg and Hardell, 2017) applied these considerations to epidemiological and experimental evidence on the potential carcinogenicity of RFR, and concluded that as of 2017, the record established compelling indications of causation, a position also expressed by the Israel Institute for Advanced Study international expert forum the same year (“Wireless Radiation and Health,” 2017). Recent experimental and epidemiological studies have added considerably to the record and have led Miller et al., 2018 to conclude that on the basis of evidence amassed as of 2018 RFR constitutes a class one proven human carcinogen. Another more recent report concurred (Hardell and Carlberg, 2020), as do the recent publications by Lin (2022b) and the (International Commission on the Biological Effects of Electromagnetic Fields (ICBE-EMF), 2022). This paper builds further on those records and provides an update on the science applying the principles for deducing a causal relationship between RFR and cancer.

These conclusions regarding the carcinogenic and other potential adverse effects of RFR are not shared equally, with strong dissent provided by a vocal number of industry-affiliated scientists (Foster et al., 2000, 2022; Grimes, 2021; Repacholi, 2010). While some that have questioned the causal nature of the relationship may be well-meaning, a disproportionate number of those who discount the data are in the direct or indirect employ of the affected telecom industries. As a result, the ability to carry out independent analysis of the matter remains hampered, fueled in no small part by the genuine complexity of the topic and by a well-organized effort to ‘manufacture doubt’ (Alster, 2015; Weller et al., 2022).

Given the unprecedented and exponentially rising growth in worldwide exposures to this technology, the lack of a vibrant well-funded program of training and research constitutes a major problem. Since the 1990s, panels of government and other experts have repeatedly examined the scientific evidence, found it wanting and called for more research to be conducted. Although the call for further research constitutes the one matter on which all are agreed, funding for this work remains quite limited. Thus, the principal output of such inquiries is to recommend research but has not resulted in major ongoing funding for such research. By the end of the 1990s, Motorola had closed its world-class bioelectromagnetics laboratory. The U.S. government programs on the subject were defunded by Congress at the same time. Thus, on this matter the absence of evidence is not proof of safety. Rather it is an indication of the intense struggle that has led to a lack of funding with respect to critical research questions, the failure to monitor human and environmental health impacts, and the ongoing manufacturing of doubt that has been documented by a number of experts (Davis, 2010).

Both the IARC (IARC, 2013) and the formatting (M. Wyde et al., 2018) carried out intensive evaluations of the health impacts of RFR in the past decade. Since these publications, a growing experimental literature has noted both negative and positive biological impacts of RFR in systems as far-ranging as plants, *C. elegans*, vertebrates and human public health (Levitt et al., 2021a, 2021b, 2022). Disputing these and other similar findings, Grimes (2021) claimed that there is no evidence supporting a causal link between RFR and carcinogenesis and that weak nonthermal levels of millimeter waves of 5G cannot possibly have any biological effect. He and others often ignore the full spectrum coverage of the 5 generation of mobile communications. The term 5G covers 5G NR (660 MHz–3500 MHz), 5G C band (3500 MHz–5000 MHz) and 5G High band (24 GHz–40 GHz), occupying frequency bands previously held by 3G and 4G mobile communications (5G Frequency Bands & Spectrum Allocations). Grimes asserted that the NTP study was so

deeply flawed that it did not constitute a valid finding. More recently, several senior advisors to the World Health Organization have reviewed studies published since the [NTP, 2018](#) determinations and have concluded that if RFR were evaluated based on more current studies, it would be upgraded to a probable if not confirmed human carcinogen ([Hardell and Carlberg, 2020](#); [Miller et al., 2018](#), [Lin, 2022a](#)).

To clarify the matter, this four-part review evaluates the epistemological foundations for concluding that RFR is carcinogenic in animals and humans. First, we explore possible mechanisms of action underlying biological impacts of non-ionizing RFR. Then we assess recent key experimental findings including detailed reports from the genetic toxicology component of the National Toxicology Program (NTP) study ([M. Wyde et al., 2018](#)). We also evaluate evidence from evaluations of exposed human populations obtained through case-control and population-based studies. Finally, we consider the weight of evidence that RFR constitutes a carcinogen and also promotes other negative health effects.

2. A discussion of relevant mechanisms

Historically agencies involved in evaluating RFR assumed that, apart from direct EM heating, there were only two possible mechanisms of RFR/tissue interaction that can lead to DNA damage, both of which involve ionization caused by an impinging photon ([Grimes, 2021](#)). The first is by a direct ionization of DNA by an RFR photon. The second, indirect route, is by the ionization of cellular water, leading to an excess of reactive oxygen species (ROS), such as OH^- and O_2^- , that can induce DNA damage ([Görlach et al., 2015](#)). However, the wavelength of RFR photons is at least three orders of magnitude too long to contain energy sufficient to produce ionization. Despite this, there are a number of alternative routes by which RFR can indirectly lead to cellular damage. An extensive literature has identified a number of other potentially relevant mechanisms of action that could underlie electromagnetic radiation impacts. For instance, a number of publications indicate that RFR disturbs the balance of cellular ROS, indirectly damaging DNA ([Luo et al., 2020](#); [Smith-Roe et al., 2020](#)), and interferes with cellular membrane integrity ([Desai et al., 2009](#); [Gautam et al., 2019](#); [Zhao et al., 2007](#)). Through these and other mechanisms, RFR can reasonably be expected to increase the risk that cancer and other degenerative diseases could develop ([Prasad et al., 2017](#)).

The production of ROS in the interior of a cell is a natural byproduct of cellular metabolic processes ([Forrester et al., 2018](#); [Ray et al., 2012](#)) and its regulation is critical for cell homeostasis ([He et al., 2017](#)). An imbalance of ROS leads to Oxidative Stress (OS) and has been linked to inflammatory diseases in general and cancer in particular ([Panieri and Santoro, 2016](#); [Yang and Lian, 2020](#)). RFR has also been shown to cause the perturbation of Voltage Gated Calcium Channels (VGCC) ([Brieger et al., 2012](#); [Pall, 2013, 2022](#); [Panagopoulos, 2019](#); [Panagopoulos et al., 2021](#); [Ullrich and Apell, 2021](#)) and to promote the activation of mitogen activated protein kinase (MAPK) activity ([Friedman et al., 2007](#)). VGCC are a class of membrane protein structures responsible for the transport of Ca^{2+} ions across the cellular membranes for electrical signaling and the initiation of many different cellular events ([Catterall, 2011](#)). Integral to these processes are ROS. The interplay between intercellular calcium and ROS for signaling and regulation is well established ([Görlach et al., 2015](#); [Mazars et al., 2010](#)).

To paraphrase the title of one research paper – ROS and Ca^{2+} – partners in sickness and in health ([Ambudkar and Muallem, 2016](#)), a recent review of the literature ([Panagopoulos et al., 2021](#)) pointed out that there is a plethora of studies demonstrating experimentally the disruption of VGCC by RFR at low intensities. A large number of these studies note that the pulsed nature of many RFR transmissions plays a vital role in affecting this interaction. A well-developed physical mechanism, known as the forced ion oscillation model, may account for why pulsed RFR is so bioactive ([Panagopoulos et al., 2000, 2021](#)). Simply put, the forced oscillation of free ions in or near the VGCC

protein complex triggers its activation when the time period (the reciprocal of the oscillation frequency) of the oscillation is close to the characteristic time for the opening and closing of the gate. Under normal circumstances the gate would be activated by the membrane potential caused by differing concentrations of ions on each side of the membrane. External pulsed electric fields, even though they are of low intensity, such as those used in telecommunications, can be a driving force acting on these gate ions precisely because they mimic the membrane potential ([Pall, 2013](#); [Panagopoulos et al., 2000, 2021](#)). It is unfortunate that their modulation frequency (not the carrier frequency) is in the widely employed region of 100 s Hz – 1 kHz (3G/4G MT, DECT, 5G (“[Chapter 2: Radio Transmission — 5G Mobile Networks: A Systems Approach Version 1.1-dev documentation](#),” n.d.)), close to the characteristic time of the VGCC ([Panagopoulos et al., 2021](#)). The same is true for other membrane ion gates ([Panagopoulos, 2019](#)).

[Fig. 1](#), reproduced and adapted from ref. ([Panagopoulos et al., 2021](#)), graphically represents the limits of the bioactivity herein described. Note that all mobile phone transmissions are bioactive. The effect of this disruption is minor but cumulative, leading to long term oxidative stress (OS) in the cell interior. Recent detailed biochemical studies of serum reported significantly heightened levels of OS in people living in the immediate vicinity (less than 80 m) of a cell phone base station, compared with those living far away (greater than 300 m) ([Zothansiam et al., 2017](#)). Furthermore, the expert advisory group, BERENIS, to the Swiss Federal Office for the Environment (FOEN) ([FOEN, n.d.](#)) recently reviewed evidence from the last 10 years for OS originating from RFR ([Mevisen and Schürmann, 2021](#)) concluding that “the majority of the animal and more than half of the cell studies provided evidence of increased oxidative stress caused by RF-EMF (*Radio Frequency Electromagnetic Fields*) or ELF-MF (*Microwave Fields*).” This, coupled with ample *in-vitro* studies ([Belpomme et al., 2018](#); [Belyaev, 2019](#); [Chowdhury et al. n.d.](#); [Durdik et al., 2019](#); [Kostoff et al., 2020](#); [Schuermann and Mevisen, 2021](#); [Yakymenko et al., 2014](#)) lends credence to the negative effects on human populations from exposure to RFR ([Kivrak et al., 2017](#)). Effects have been shown to vary depending on polarity, frequency, power-density, and a number of other parameters of RFR that are not well-studied.

However, not everyone has been convinced by this evidence. Consales et al. ([Consales et al., 2012](#)) in 2012 concluded that there was no incontrovertible evidence for a linkage between RFR and OS. This study did note that the generation of extracellular ROS responses varied with different cell lines, stimulating cell membrane nicotinamide adenine dinucleotide (NADH) oxidase in Rat1 and HeLa cells. The fact that RFR can have synergistic effects as well as both negative and positive impacts on ROS in various organs and species also adds to confusion regarding efforts to estimate its overall effect on carcinogenicity. While acknowledging that specific relevant biological endpoints can be altered by RFR, the study questioned the generality of these responses, noting that, “Differences in cell lines and experimental methods, used for both *in vitro* and *in vivo* exposure, might explain, in part, these still conflicting findings”. We concur that differences in measures of RFR as well as cell lines studies constitute important parameters that should be evaluated more carefully under any circumstance.

More recently, [Karipidis et al. \(2021\)](#) published a review stating that there is no confirmed evidence that RFR is hazardous to health, especially for the emerging 5G technologies. They rejected most published experimental studies for failures to provide detailed information on exposures, while also not providing that same information for their definition of “low-level” conditions. They also reported positive findings of increased cancer mortality in Belgium studies of military radar workers (exposed to higher frequencies occupationally), but not from other country-based studies. They noted that this could reflect the “healthy worker” effect, as military personnel are in general healthier than the general public. When the general population is employed as the comparison group in occupational studies of mortality, the risk ratio for death will tend to be underestimated because that group includes the

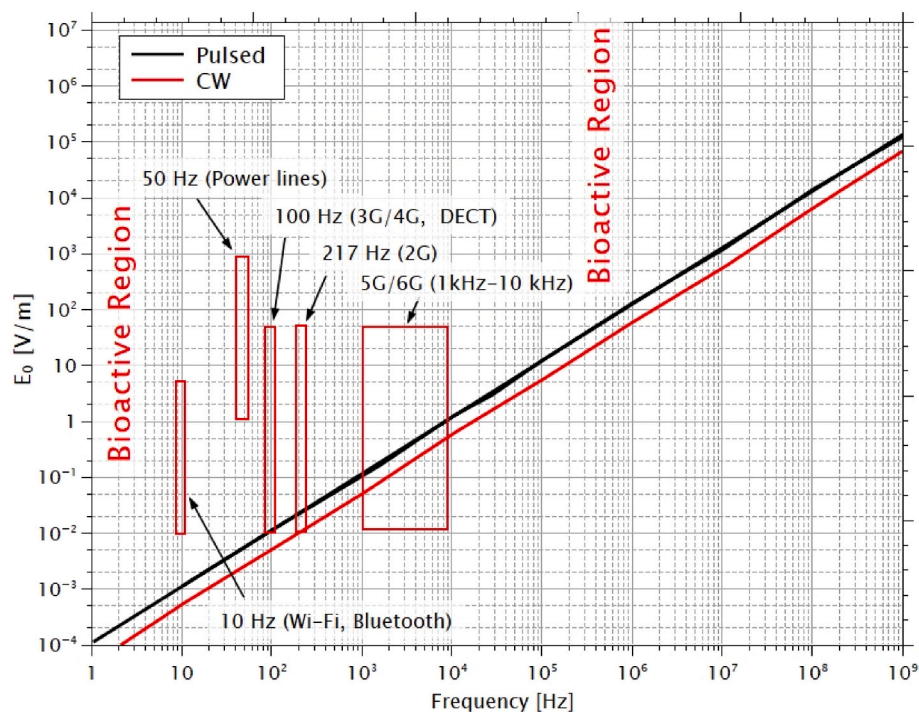


Fig. 1. Depicting the combinations between Electro-magnetic Field strength and modulation frequency of pulses carried on the base transmission frequency of the signal that can disrupt VGCCs in the membrane. The scales of the axes are logarithmic. The Y axis is the E-field strength and is related to power density (PD) of the signal by $PD = \frac{E^2}{377}$, where 377Ω is the impedance of free space. The red areas represent the combination of field strengths and frequencies of some common telecommunication protocols. The maximum power density for the base transmission frequency allowed by the FCC and ICNIRP is $PD = 10 \text{ W/m}^2$ (Wu et al., 2015). The black line represents the limit above which an oscillating field of frequency and strength represented by the line will disrupt a membrane ion channel. The red line is the same, but for continuous fields. The frequency limits of bioactivity are marked on the graph. (Reproduced and adapted with permission from (Panagopoulos et al., 2021)). (For interpretation of the references to colour in this figure legend, the reader is referred to the Web version of this article.)

unemployable who may have a higher rate of disease than the unexposed workforce.

In fact, both studies used overly stringent criteria that would exclude most critical studies. Effectively, they select which studies are to be accorded valid concerns and reject those that do not comport with their views and they have been criticized for such (Panagopoulos et al., 2021). Even so, both Karipidis and Consales recommend further intense study to resolve the uncertainty.

In a similar vein, Grimes (2021) published a review of cancer and RFR that discounted any possible link between the two and ignored more recent papers countering his conclusion. This “review” also applied criteria for inclusion that effectively eliminated a number of widely regarded experimental studies including the multi-year, thirty million dollar NTP evaluations in rats and mice and the Ramazzini Institute report of even lower levels of exposures such as those typically emitted by base stations. This rejection of these state-of-the-art studies rests on the presumption that the exposure conditions do not reflect human experience. In fact, the exposure chambers in these studies were constructed and reviewed at several stages of these projects in consultation with the renowned Swiss National Institute for Electrical Engineering. Consequently, we conclude that experimental evidence corroborates the link between ROS, OS, and cancer is well established and the link between them and excessive RFR exposure is clearly illustrated in the literature.

3. Epidemiology

The recent and widely read publication by Grimes in JAMA Oncology (Grimes, 2021) reviewed several epidemiological studies that he claimed found no association between RFR and cancer. These are the case-control studies of INTERPHONE study (The INTERPHONE Study Group, 2010), the Danish Cohort study (Frei et al., 2011), the CERENAT case-control study (Coureau et al., 2014), and the population-based Million Women study (Benson et al., 2013). All were completed between 2010 and 2013. A recent update of the Million Woman Study also reported a null result (Schüz et al., 2022). In one of these studies, the CERENAT study (Coureau et al., 2014) did indeed find an association of heavy mobile phone use with significantly increased risk of glioma. A

more recent study attempting to find a link between cellphone radiation and gliomas in children also produced a null result (Castaño-Vinyals et al., 2022). However, all of these studies have been shown to suffer from problematic definitions or incomplete cohorts and inaccurate exposure information, effectively biasing them in favor of the null result (Choi et al., 2020). For instance, Grimes cites the Million Women study as demonstrating no impact of cell phone radiation on cancer risk. That study relied on a single question about cell phone use put to fewer than 750,000 women in 2001 and 2011, asking if they “never”, “ever” or “daily” used a cell phone. Undertaken to evaluate the risk of breast cancer for post-menopausal women taking hormone replacement therapy, this was not the age group that were first adopters of cell phone use in the 1990s, when that study was begun. Only 18% of all these women reported any use of phones. As the study obtained no detailed information on type of cellphone use, nor use of cordless phones, that can emit substantial microwave radiation, the absence of a positive finding may reflect exposure misclassification as well as the fact that this age group reported relatively little use of phones. Women who used the phone once a day were lumped together with women who used the phone for hours a day. Most of the women reported cell phone use under 30 min or more a week. When this group of cellphone users was compared to those who reported never using a phone, no statistically significant associations were detected between phone use and brain cancer. In contrast, the CERENAT study did find increased brain cancer in several of their subgroups. It is the Grimes evaluation that ignored it.

In an analysis of nine epidemiological studies of brain cancers and mobile phones, Miller et al. (2018) noted increased risks. Vienne-Jumeau et al. (2019) while not observing heightened instances of brain tumor, did find robust epidemiological evidence of acoustic neuroma. Mialon and Nesson (2020) found mobile subscription rates significantly and positively associated with death rates from brain cancer 15–20 years later. Pareja-Peña et al. (2020) similarly found clear evidence that epidemiological studies detect a causal association between the exposure to RFR and the incidence of brain neoplasms. Boileau et al. (2020) found that use of a mobile phone for more than 30 min per day by expectant mothers impaired fetal growth. Choi et al. (2020) carried out a systematic review and meta analysis of case control studies and found evidence that cell phone use increased the risk of tumors. Although

challenged by de Vocht and Rösli (de Vocht and Rösli, 2021) and by Brzozek et al. (2021), the replies of Choi et al. (Moskowitz et al., 2021; Myung et al., 2021) effectively establish the validity of their findings. De Vocht's contention was that Choi et al. had unfairly combined different cancer types in their meta-analysis and that they had relied too heavily on the work by Hardell et al. (Hardell et al., 2013; Hardell and Carlberg, 2009). The reply, drafted by Myung (Myung et al., 2021), pointed out that such an approach was fair if the intention was to show the possibility of cancer formation, rather than the instance of a particular type of cancer. They also pointed out that this approach was perfectly acceptable when discussing other cancer risks. Furthermore, they addressed the high quality of the work done by the Hardell group as a reason to rely on their results. Hardell and Carlberg have also recently published studies demonstrating a causal link (Hardell and Carlberg, 2020, 2021). This list of studies represents only a portion of the published work produced during just the last two years.

4. Population studies

An interesting study recently published by Sato (Sato et al., 2019) explained that because of the long latency of brain tumors (up to 4 decades) the effect of cellphone radiation induced changes should only become evident now in population studies.

A case-control study from Connecticut between 2010 and 2011 by Luo et al. (2020) included 440 thyroid cancer cases and 465 population-based controls with genotyping information for 823 single nucleotide polymorphisms (SNPs) in 176 DNA genes. Using multivariate unconditional logistic regression models, they determined genotype-environment interaction between each SNP and cell phone use and estimated the association with cell phone use in populations according to SNP variants. While in the most common homozygote groups, no association was observed with cell phone use, ten SNPs showed highly significant interactions in all thyroid cancers and cell phone use, $P < 0.01$. In small tumors, increased risk was observed for 5 SNPs (rs1063639, rs1695147, rs11070256, rs12204529 and rs3800537). In large tumors, increased risk was observed for 3 SNPs (rs11070256, rs1695147, and rs396746). The authors conclude that their results indicate that genetic susceptibilities modify the associations between cell phone use and risk of thyroid cancer and provide further evidentiary support for determining that RFR is carcinogenic to humans.

4.1. Possible implications of pregnancy

Experimental studies finding prenatal effects of microwave radiation on behavior, brain, and other organ development of progeny are corroborated by some human studies as well.

Divan et al. (2008) evaluated mothers of 13,159 children that self-reported cell phone use during pregnancy and afterwards. They reported significantly increased odds ratios for behavioral problems for children who had possible prenatal exposures to cell phones. After adjustment for potential confounders, the odds ratio for a higher overall behavioral problems score was 1.80 (95% confidence interval = 1.45–2.23) in children with both prenatal and postnatal exposure to cell phones. Exposure to cell phones prenatally-and, to a lesser degree, postnatally-was associated with behavioral difficulties such as emotional and hyperactivity problems.

Birks et al. (2017) found that, “38.8% of mothers from the Danish cohort, reported no cell phone use during pregnancy, and these mothers were less likely to have a child with overall behavioral, hyperactivity/inattention or emotional problems” (Birks et al., 2017). Mothers self-reporting medium and high frequency cell phone use had OR (odds ratio) of 1.28 (95% CI 1.12, 1.48) for having a child with increased risks of hyperactivity/inattention. In addition, the association of cell phone usage with behavioral problems appeared consistent between and across cohorts.

As early as 2012 it was noted that EMF originating from cellphones

operating at 800–1600 MHz could lead to developmental deficiencies in mouse models (Aldad et al., 2012). Mice exposed *in utero* were hyperactive and suffered from impaired memory. A recent study studied EMF exposure on the placenta of pregnant rats (Kim et al., 2021). While it did find significant increases of cortisol in maternal circulating blood, these did not seem to be carried over to placental blood. However, one notes that the radiation source used in the reverberation chamber was an RFID source, rather than a cellphone. In the aforementioned studies the animals were exposed to SAR values of 1.6 and 4 W/kg respectively.

Finally, a recent review of the literature pertaining to possible EMF exposure effects on human pregnancy did find significant impacts (El Jarrah and Rababa, 2022). These included, heightened risks of miscarriage, changes to fetal temperature, variations in fetal heart rate variability, and changes in infant anthropometric measurements. However, the same study cautioned that there was wide variability in assessing exposure in cohort studies, in the type and duration of exposures involved. The authors, while concluding that there is a negative impact of EMF exposure on fetal development, called for more studies to be carried out.

4.1.1. *In vivo* experiments

In addition to the NTP and Ramazzini studies, a large body of *in vivo* animal studies has been published that demonstrate adverse effects from RFR. Frequently these studies are dismissed, principally because whole body exposures are used, rather than localized exposures. In fact, there is no way to have rodents simulate local exposures that take place during phone calls. The Federal Food and Drug Administration (FDA) review of literature 2008–2018 (FDA U.S., 2020) provided an especially restrictive overview of the topic (Balmori et al., 2020; Leach, 2020). Out of a possible 55 animal studies that could have been considered, the FDA report only used 37, of which 23 concluded that there was genotoxicity from RF exposure (Leach, 2020). The FDA report concluded that “Overall, based on certain limitations, these studies have not produced any clear evidence that RFR exposure has any tumorigenic effect.” In rejecting many animal studies, the main concern of the FDA was that most of these reports did not measure the animal's internal temperature.

The principal assumption of the FDA is that thermal damage is the reason for any carcinogenic effect and this only arises when the animal whole body temperature rises more than a single degree Centigrade—a rise that may occur under current limits. In fact, hyperthermia is currently an accepted treatment for some forms of cancer, as heat is recognized as being anti-carcinogenic. This reasoning is shared by such august bodies as the WHO EMF Project and ICNIRP in the establishment of their exposure limits (Grigoriev, 2010). However, recent research demonstrated that at levels of exposure to RFR considered to be non-thermal, there is indeed an elevation of body temperature of mice and that this is a physiological reaction to stress and not heating (Mai et al., 2020). This work used implanted Anipill temperature loggers (BodyCap (“e-Celsius Medical -,” n.d.), Paris, France) to record core temperature of mice during exposure to 900 MHz CW signal for 1 h periods. The results show a consistent temperature elevation of $\Delta T \sim 0.1$ to 0.3°C due to exposure, well within physiological limits. As the authors pointed out, the traditional method of gauging core temperature, as favored by the Federal Communications Commission (FCC), leads to elevated core temperatures due to physical discomfort and stress (Mai et al., 2020) of the animal.

As part of the NTP study, to ensure that internal temperatures were not disrupted by exposures to cell phone radiation comparable to those that humans can receive, Wyde et al. (M. E. Wyde et al., 2018) also used implanted probes to gauge the effect of RFR exposure on animals due to mobile transmissions. They also found little variation of core temperature in rats exposed to GSM- and CDMA-modulated RFR signals even up to SAR values of 6 W/kg. In short, the criticism of FDA that in most *in vivo* experiments that core temperatures were not measured and so are not valid, is unfounded.

4.2. The NTP study is inaccurately depicted

In evaluating the carcinogenicity of current levels of RFR from phones and other devices, the NTP study employed validated state-of-the-art methods that have been honed over more than four decades with the concurrence of the FDA, EPA, and other federal agencies. To date, the NTP has produced several hundred carcinogenicity assessments employing standard protocols. Despite this robust history, some pundits have dismissed many of the findings of the National Toxicology Program (NTP) Cell Phone Radio Frequency Radiation study (National Toxicology Program (NTP), 2018; [Smith-Roe et al., 2020](#); [M. Wyde et al., 2018](#); [M. E. Wyde et al., 2018](#)). Despite being approved at every stage of planning and operation by the FDA, some reject its findings claiming that they rely on “low power and questionable methods” ([Grimes, 2021](#)). The NTP study was a toxicology study of rats and mice to clarify the risks of disease from exposure to RFR ([Gong et al., 2017](#)). A similar study ([Falcioni et al., 2018](#); [Vornoli et al., 2019](#)) was carried out by the Italian Ramazzini Institute and concluded that “there is now clear evidence that RFR causes cancer in experimental animals.” RFR re-evaluation has also been listed as a priority by IARC ([Vornoli et al., 2019](#)).

Among the principal critiques of the NTP study are those from the anonymous FDA report ([FDA U.S., 2020](#)) and the International Commission on Non Ionizing Radiation Protection (ICNIRP) ([Protection \(ICNIRP\)1, 2020](#)), both of which were disputed in considerable detail ([Melnick, 2019, 2020](#)). It must be noted that the NTP study was commissioned by the FDA at the behest of the FCC (Federal Communications Commission), reviewed, and approved by them ([Melnick, 2019](#)). The study was funded to the tune of \$30 million and lasted 5 years. It was specifically designed to test the null hypothesis that cell phone radiation at non-thermal exposure intensities could not cause adverse health effects. By the definition of the FCC and ICNIRP “non-thermal” exposure intensities means that there is no more than a 1° rise in core body temperature resulting from acute exposures ([Melnick, 2020](#); “OET - Bulletin No. 65 (August 1997),” 2011). The NTP carried out initial studies to find the exposure limit for rats and mice in terms of SAR to maintain this limit and found that the maximum whole body exposure for such would be a SAR value of 6 W/kg ([M. E. Wyde et al., 2018](#)). One notes that the FCC limit for local exposure for the human head is 1.6 W/kg averaged over 6 min ([Proposed FCC changes to measuring and evaluating human exposure to radiofrequency electromagnetic fields and wireless power transfer devices are flawed: need for biologically-based standards, 2020](#)) with the phone 25 mm from the skin surface ([Gandhi, 2019](#)). In real life scenarios the cell phone is usually in contact with the skin during a conversation. Using the Standard Anthropometric Model (SAM), the National Agency ANFR of France routinely measured SAR values of 5 W/kg and above for over 450 mobile phone models ([Gandhi, 2019](#)) held in contact with the skin surface. In other words, claims, including those made by [Grimes \(2021\)](#), that the exposure limit fixed for the NTP study were too high are not valid. The criticism of the findings of the NTP study were adequately answered by [Melnick \(2019\)](#) and by [Leach \(2020\)](#). In particular the claim that whole body SAR was used instead of local SAR is invalid. Extensive care was taken in this study to assess the dosimetry for the animal assays ([Gong et al., 2017](#)). Further, there are no acceptable means of strapping a cellphone to the head of a rodent. Furthermore, Whole-body SAR provides little information on organ specific exposure levels ([IARC, 2013](#)) and the correct comparison is indeed the localized human head exposure level as defined above. The results of the NTP study were unequivocal. The null hypothesis that non-thermal RFR is incapable of causing harm was disproved! The study found cancers like glioma in the brain increased by 3% for GSM type signals, at all powers, and by 3.3% for CDMA type signals at 6 W/kg (National Toxicology Program (NTP), 2018; [M. Wyde et al., 2018](#)). Glial cell hyperplasia increased by 2–3% for both types of signal and for all signal strengths. Surprisingly, these changes occurred almost exclusively in male rats. For schwannomas of the heart, there was an increase of

2–5% for GSM type signals and an increase of 2–6% for CDMA type signals ([M. Wyde et al., 2018](#)). Genotoxicity was found in the frontal cortex of male mice (both modulations), leukocytes of female mice (CDMA only), and hippocampus of male rats (CDMA only) ([Smith-Roe et al., 2020](#)) and other effects. In short, criticism of the NTP study was unfounded.

4.2.1. Other avenues of negative health impacts

While the carcinogenicity of RFR has garnered considerable attention in the perennial debate over its health impacts, it is quite possible that other forms of damage dominate. Electromagnetic Hypersensitivity (EHS), a syndrome in which the sufferer experiences a number of debilitating conditions, ranging from tinnitus to headaches and even to a complete inability to function as a result of exposure to anthropogenic EMF, is thought to affect up to 10% of the population in some countries ([Dieudonné, 2020](#); [Genuis and Lipp, 2012](#)). However, due to the amorphous nature of what can be considered as an EHS symptom, some researchers feel that the number is actually closer to 1.5% of the population ([Tatoń et al., 2021](#)). A comprehensive review of its symptoms and its grounding as a physiological response to exposure to EMF was recently published (International Commission on the Biological Effects of Electromagnetic Fields (ICBE-EMF), 2022) by the International Commission on the Biological Effects of Electromagnetic Fields (ICBE), an independent NGO. They note that there are now guidelines for the diagnosis and management of EHS and that this syndrome can have measurable biomarkers, such as elevated oxidative stress, inflammatory markers and changes in cerebral blood flow.

The nature of EHS has long been disputed, with many researchers feeling that it is a nocebo effect rather than a physiological malady ([Baliatsas et al., 2015](#); [Bräscher et al., 2020](#); [McCarty et al., 2011](#)). However, EHS has been recognized in Sweden as a disability since 2001 ([Johansson, 2010](#)) and there is increasing evidence that the nocebo explanation cannot stand as the main cause, even if one accepts that there are psychosomatic aspects to the phenomena ([Dieudonné, 2020](#); [Johansson, 2006, 2010](#); [Singh and Kapoor, 2014](#)). Recent research has placed its physiological origins on a firmer footing. Belpomme et al. have identified specific neurological pathological disorders that can be linked to EHS ([Belpomme and Irigaray, 2020](#)). Johansson et al. has also detected subtle changes in the neuronal systems of the skin that can be associated with EHS ([Johansson, 2010](#)). However, in a recent and exhaustive review of the literature regarding EHS research, [Leszczynski \(2021\)](#) points out that there are inherent problems with the greater majority of studies either demonstrating or denying a correlation between the exposure to RFR and the existence of symptoms associated with EHS. He reports a number of problems including; widely disparate definitions of the symptoms (up to 71 in one study), in the reliance of a subjective measure, either from the subjects of the study or the research team involved, a lack of objective exposure measurements and the small statistical weight of most studies. As he points out, one cannot deny EHS based on these studies, as some do, but that it may well be better to consider it as a syndrome, encompassing a variety of effects induced by exposure and mitigated by the existence of cofactors in the subject's environment. Consequently, what may induce EHS in one person may have no effect on another, even if they too claim to suffer from EHS. The complexity of the situation requires individual studies of sufferers that include biochemical and physiological markers, in an effort to identify distinct physio/bio markers that can then be used in large scale studies ([Leszczynski, 2021](#)). Not everyone agrees with this assessment of the state of research (International Commission on the Biological Effects of Electromagnetic Fields (ICBE-EMF), 2022), with strong contrary comments from [Dieudonné \(2022\)](#) and from [Ofstedal et al. \(2021\)](#).

Despite these disagreements, the existence of EHS as a real debilitating syndrome affecting millions worldwide is not in dispute. As the level exposure to RFR is expected to drastically increase as 5G systems begin to dominate our urban environments, one may assume that the instances of EHS will only rise, along with its economic and personal

impacts.

5. Discussion

Industry and regulatory authorities should have the safety of the public as their paramount concern. However, the boundaries separating the regulator from the regulated are frequently blurred. In 2020 the FCC (“FCC Maintains Current RF Exposure Safety Standards,” 2019; Proposed FCC changes to Measuring and Evaluating Human Exposure to Radiofrequency Electromagnetic Fields and Wireless Power Transfer Devices are Flawed: need for biologically-based standards, 2020) extended the current allowed levels of exposure to the frequency region 3 GHz–300 GHz, effectively preparing the legal framework necessary for the whole-scale deployment of 5G C band and 5G High Frequency mobile communications infrastructure. Concurrently, ICNIRP published its recommended levels of exposure, confirming a standard that is already 27 years old (Protection (ICNIRP)1, 2020).

We concur with Hardell (Hardell and Carlberg, 2020) and Miller (Miller et al., 2018) that the grounds for inferring causation established by Sir Bradford Hill (1965) have generally been fulfilled regarding the capacity of RFR to induce cancer and other adverse health effects. Experimental and epidemiological evidence both indicate that with greater exposures there are greater responses. Further the biological foundation for concluding that such exposures are causal have been plausibly elucidated by several authorities to emanate from the capacity of RFR to induce OS along with other properties. The lack of consistency of studies is noteworthy, nonetheless. Regarding these inconsistencies, it should be added that several commenters have documented substantial conflicts of interest in institutions such as ICNIRP that have reached contrary views. Even so, the situation has been exacerbated by a failure to standardize nomenclature, by a lack of consistency in signal types, frequencies, polarity and other EM properties. To compound this some *in vitro* studies have not used standardized cell cultures. Evidence has mounted that pulsed lower-power signals can be highly disruptive, especially for immature stem cells (Durdik et al., 2019; Romeo et al., 2022).

The Precautionary Principle is defined by the United Nations Rio Earth Summit in 1992, in article 15 of the Rio Declaration as follows: “In order to protect the environment, the precautionary approach shall be widely applied by States according to their capabilities. Where there are threats of serious or irreversible damage, lack of full scientific certainty shall not be used as a reason for postponing cost-effective measures to prevent environmental degradation.” (Robbins, 2007). While it was initially invoked for environmental concerns, the Precautionary Principle has become the yardstick with which to gauge the impact of industrial and technological advancement on human and societal health. Since its inception it has become part of the regulatory scene (Hanson, 2018), adopted by the European Union in 2000, Australia in 1991 and a number of U.S. states. It has consequently drawn the ire of those who feel that it is overly restrictive and stifles advancement (Hansson, 2020; Peterson, 2007; Turner and Hartzell, 2004). Notwithstanding, the notion of preventing harm rather than proving damage has already occurred is a cardinal principle of public health that forms the foundation of policies to employ bike helmets, seat belts, and air bags. In those instances, indications of the clear benefits of safety led to their global implementation. The grounds for taking action to prevent harm rests on scientific foundations that specific actions can reduce or mitigate those harms. In the matter of RFR no such approach is evident, although we believe that one is sorely needed.

As pointed out by Prof. Butler in his working paper (Butler, n.d.), the ICNIRP Guidelines reject non-thermal health effects, despite a wealth of evidence including epidemiological, *in vitro*, and animal studies to the contrary, and that this is prevailing institutional logic amongst the world regulatory bodies who take their cue from ICNIRP (Butler, n.d.; Hardell and Carlberg, 2020). Given the weight of scientific evidence, only some of which is outlined above, it is clear that the Precautionary principle is

being ignored. A recent commentary by Lin concurs with this statement (Lin, 2022b).

Oversight by the FCC is concerning. It has frequently been pointed out that the routine employment of officials by a regulatory body from the industry it must regulate, constitutes an inherent conflict of interest. This is evident in the relationship between the FCC and the Mobile Phone Industry. As pointed out by Alster in his book “Captured Agency” (Alster, 2015), “But with the overwhelming application of money and influence, information and communications technologies have almost totally escaped political scrutiny, regulatory control, and legal discipline.”

6. Conclusion

There is a plethora of both experimental and epidemiological evidence establishing a causal relationship between EMF and cancer and other adverse health effects including adverse effects on fetal development and the endocrine system. Increases in biochemical alterations such as DNA damage, increased production of free radicals and other signals found to be predictive of cancer and other degenerative diseases have been clearly demonstrated. While the evidence is not consistent, the reasons for that inconsistency merit independent review and assessment. A number of industry-affiliated scientists have offered criticisms that are subject to bias, as we have outlined here. If progress is to be made in improving the public understanding of this complicated issue, it is imperative to insist on a complete picture of the evidence that relies on independent science.

While we may disagree strongly with the conclusions some critics have provided, we concur wholeheartedly that there is a need for a serious concerted program of research. No such program exists with support from National governments in the United States and Canada. The job of the government is to ensure the protection of Public Health. We earnestly hope that as the situation evolves, those in positions to create the training and funding for major interdisciplinary research programs in engineering, medicine, toxicology, and bioelectromagnetics will do so. In the meantime, we add our voices to those of more than four hundred experts in the field calling for discussion of a moratorium on 5G. Without such a program we are effectively conducting an uncontrolled experiment on ourselves, our families, and our children.

Indeed, the subject of RFR and carcinogenicity remains truly complex. Studies have to simulate intricate exposures that are taking place every day to billions of people around the world. Given the ubiquity of the technology, as we move ahead it will not be possible to find an unexposed control group in the modern world. The inarguable intricacy of the technology can easily become a way of confusing rather than clarifying the matter. The subject of the impact of RFR on human health is one of the most important topics of our age. It is one in which the general public seeks clear answers to a collective, but poorly defined angst. It is the job of experts to present the state of knowledge in clear and concise language that the layman can understand. The numerous omissions and distortions in recent articles originating from the industry perspective do not meet this criteria. The medical and public health communities deserve the whole story, no matter how complicated or unpalatable it may be. There is an abundance of evidence pointed towards deleterious effects of RFR exposure on human health. Further, the growing applications of low levels of RF in medicine through electroceuticals constitutes evidence per se of biological impacts (Mishra, 2017). Any agent that can be beneficial, whether aspirin or oncology drugs, can also have negative impacts. Consequently, it is imperative to insist on a complete picture of the evidence and not the whitewashed or distorted version currently promoted. The need to take into account the complete weight of the evidence in devising regulatory policies is widely ignored to our detriment. It is time that the Precautionary Principle be applied to RFR.

Declaration of competing interest

The authors declare the following financial interests/personal relationships which may be considered as potential competing interests: Guest Editor for Environmental Research P.B.I. Provided scientific opinion for the Environmental Health Trust P.B.I.

Data availability

No data was used for the research described in the article.

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