

Response to HHS Request for Information on Electromagnetic Fields (EMFs), Radiofrequency (RF) Radiation and Wireless Radiation Exposure

Docket No. HHS-OASH-2026-0397

Submitted to: U.S. Department of Human Health Services, Office of the Assistant Secretary for Health

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Response to Question 2: Respondent Perspective

This submission is made from the perspective of an independent researcher. It is not submitted on behalf of, or with funding from, any equipment manufacturer, telecommunications carrier, infrastructure provider, industry association, or government regulatory body. My research publication details can be found at <https://www.researchgate.net/profile/Steve-Weller>

Statement of Basis for Submission

This submission draws on several complementary sources of evidence including peer-reviewed research, my own research and reports from government and intergovernmental bodies. These include the U.S. National Toxicology Program (NTP), the Federal Communications Commission (FCC) and Australian Radiation Protection and Nuclear Safety Agency (ARPANSA), the International Agency for Research on Cancer (IARC) and the World Health Organization (WHO). It also draws on an independent scoping review and evidence map of the RF-EMF genotoxicity literature, an independent assessment of the evidence concerning RF-EMF and cancer using the Bradford Hill viewpoints, and a comparative analysis of approaches adopted by two international radiation protection authorities.

The submission further incorporates an analysis of public submissions to the 2019-2020 Australian Parliamentary Inquiry on 5G [1-3]. Symptoms reported by respondents to that inquiry are compared with symptoms reported in two smart-meter datasets: Lamech 2014, a peer-reviewed case series, and Halteman 2011, a survey report [4, 5].

Finally, the submission draws on my broader research into epistemic issues in evidence interpretation and the governance of scientific uncertainty. These analyses examine how differences in evidentiary frameworks, assumptions and approaches to uncertainty may contribute to divergent interpretations of the same body of evidence.

Full citations are provided in the consolidated, numbered reference list at the end of this submission.

Overview

The United States Human Health Services (HHS) has released an RFI that is effectively asking the following question: What does the evidence actually show about RF-EMF and human health, and are current RF-EMF guidelines/standards, surveillance systems and research priorities adequate? This submission answers this question from a position that will be unfamiliar to HHS in one respect: it does not treat the reassurance of ICNIRP, the WHO International EMF Project, or the FCC as a reliable proxy for “the science.” Those bodies are one set of institutional actors among several. The evidence set out below, includes animal carcinogenicity data, mechanistic and genotoxicity research, an analysis of a selection of WHO-commissioned systematic reviews (SRs) in 2024-25, the documented influence of funding sources on reported outcomes, and the history of how the WHO/IARC World Cancer Report has treated this evolving literature. Overall, it indicates that their settled position of safety reassurance is unjustified and that the evidence has not been treated as a neutral body of findings to be weighed impartially. Instead, the selection, characterization and relative weighing appear to have been influenced by the prevailing institutional interpretation of RF-EMF health risks in order to *maintain regulatory status quo*.

Importantly, this submission does not claim that RF-EMF is a proven, established human carcinogen in the sense that tobacco smoke and ionizing radiation are proven carcinogens. It is a claim that the totality of the evidence, considered across animal, mechanistic, and epidemiological domains, and without deferring automatically to the institutions that have minimized it, is substantially stronger than “no evidence of harm”. It is also a claim that the governance structures that are currently responsible for setting limits are not well suited to registering that strength. The sections below are organized around the RFI’s own numbered questions and integrates two bodies of my own independent research: an evidence assessment of the RF-EMF cancer and genotoxicity literature, and an analysis of the governance and institutional dynamics that have shaped how the evidence has been used, or not used, in setting federal policy. Where both analyses converge on the same underlying point, for example, that funding source and institutional affiliation are more predictive of a study’s reported findings than its methodological quality, the evidence and the governance analysis are presented together rather than repeated separately, because the pattern is the same pattern viewed from two directions.

Part I: Existing Scientific Evidence (Questions 12 – 15)

I.1 The Animal Carcinogenicity Baseline and the Case for Re-Classification

The starting point for any honest evaluation of RF-EMF carcinogenicity is the animal bioassay evidence, because it is the most rigorously conducted body of evidence available compared to Human epidemiological evidence. The U.S. National Toxicology Program (NTP) and the independent Ramazzini Institute in Italy, working under different

exposure protocols and without coordinating with one another, each reported statistically significant increases in cardiac schwannomas in male rats, and both observer elevated incidences of glioma in RF-EMF-exposed rats (with the glioma signal reaching statistical support in the NTP study) [6-8]. The concordance between these two independently conducted studies is notable because they differed substantially in both exposure intensity and study design.

The NTP study used controlled, whole-body exposure over approximately two years, whereas the Ramazzini study exposed animals from prenatal life until natural death under a significantly lower intensity, far-field exposures designed to represent environmental conditions (i.e., equivalent to a mobile phone base station exposure). This convergence across different exposure conditions provides independent experimental corroboration and strengthens the biological plausibility of an association between RF-EMF exposure and specific tumor development. It has also led to a number of researchers to argue that RF-EMF now satisfies criteria more consistent with an IARC 2A (probably carcinogenic) or even Group 1 (carcinogenic to humans) classification, rather than remaining as Group 2B (possibly carcinogenic) [9-11]. The IARC Advisory Group has itself identified RF-EMF as a high priority agent for re-evaluation in both its 2019 and 2024 priority-setting reports, a signal that the accumulated evidence since 2011 is regarded internationally as substantial enough to revisit [12, 13].

The most recent, and most rigorous animal evidence review, comes from the WHO-commissioned SR conducted by Mevissen et al. (2025), covering 52 experimental studies including 20 chronic bioassays, with no studies excluded on risk-of-bias grounds [8]. For brain tumors, across five chronic bioassays involving 1,902 mice and 6,011 rats. The review found increases in glial cell-derived neoplasms in two chronic bioassays in male rats and assessed the certainty of evidence for increased glioma risk as high. For cardiac tumors, across four chronic bioassays comprising six experiments (roughly 2,165 rats and 720 mice), statistically significant increases in malignant cardiac schwannomas were found in two rat bioassays, again with the certainty of evidence assessed as high. These are not equivocal, borderline findings buried in a mass of null results, they are specific, replicated, high-certainty findings in the tumor types that have also been seen in human epidemiological studies for the heaviest and longest mobile phone users (glioma and acoustic neuroma, which is a schwannoma of the vestibular nerve) [14, 15].

I.2 The 2024-2025 WHO-Commissioned Systematic Reviews: A Case Study in Contested Evidence

HHS should not read the recent WHO-Commissioned SRs on RF-EMF and cancer as settled, disinterested science. They are contested in the scientific literature itself, and the nature of that contest is instructive for how HHS should weigh “the state of the science” going forward.

The human evidence review (SR1a). Karipidis et al. (2024) authored the WHO-commissioned systematic review and meta-analysis of human observational evidence on RF-EMF and cancer risk [16]. Using GRADE-based certainty assessments, the review concluded that near-field exposure of mobile phone use is unlikely to increase the risk of glioma, meningioma, acoustic neuroma, pituitary and salivary gland tumors in

adults, or brain tumors in children, and that far-field exposure from fixed transmitters is not associated with increased childhood leukemia or pediatric brain tumors. It is worth noting that Karipidis is the Vice-Chair of ICNIRP and an employee of the Australian Radiation Protection and Nuclear Safety Agency (ARPANSA), the Australian commonwealth regulator responsible for Australia's RF-EMF exposure standards. These overlapping institutional roles, together with the alignment between SR1a's conclusions and the existing regulatory framework, constitute a material conflict of interest that HHS should weigh directly in assessing the review's independence and evidentiary conclusions, rather than treat as incidental biography.

Multiple independent research groups also challenged the SR1a review on substantive methodological grounds [17, 18], including: reliance on studies with significant design flaws; disproportionate weight given to non-significant results at low cumulative exposure rather than statistically significant elevated odds ratios reported in the highest-exposed groups; the use of crude "ever/never" phone use categories that do not reflect actual dose; omission of studies showing increased brain tumor incidence; reliance on studies with short latency in a disease known for long latency; exclusion of laterality analyses (whether the tumor occurred on the same side of the head as habitual phone use); non-standard pooling of primary studies; and risk-of-bias ratings critics considered inappropriately assigned. Karipidis et al. (2025) rejected these criticisms as false claims and defended the review as rigorous and independently peer reviewed [19]. Frank et al. (2025) issued a rebuttal identifying what they characterized as incorrect and misleading statements in Karipidis et al. response [20]. Hardell and Nilsson (2025) went further still, describing certain methodological decisions, specifically exclusion of studies with the highest cumulative exposure and the assignment of inappropriate risk-of-bias ratings, as tantamount to scientific misconduct [18]. These allegations remain contested, but the intensity of the exchange itself is evidence that HHS should not treat SR1a's conclusions as neutral or the final word on human epidemiological evidence.

A separate meta-analysis by Moon et al. (2024), published around the same timeframe as SR1a and drawing on much the same underlying evidence base, reached materially different conclusions. Pooled odds ratios rose with more concrete exposure sub-categories, and the authors declined to interpret statistically insignificant cohort-study findings as evidence of no association, emphasizing instead the well-known limitations of cohort designs for long-latency outcomes [21]. Moon et al. also critiqued the NTP/OHAT risk-of-bias tool, which was the same tool used by Karipidis et al., as poorly suited to observational epidemiology, since several of its domains are designed for randomized controlled trials. Moon et al. instead prioritized bias domains specifically relevant to mobile-phone epidemiology (selection bias, exposure misclassification, recall bias, ipsilateral versus contralateral exposure) [21]. Given that two systematic reviews, published within months of each other, drawing on largely the same overlapping evidence, and applying different but defensible methodological choices, reach substantively different conclusions is itself the central finding HHS should take from this literature. That is the "state of the science" on RF-EMF and cancer is not settled by citing one WHO-commissioned review while ignoring its contemporaneous rebuttal.

The animal evidence review (SR2) and its critique. Mevissen et al.’s animal systematic review, described previously, was itself attacked by Karipidis et al. (2025) (along with related commentary from the same regulatory-aligned network i.e., BfS) [22, 23]. The basis for complaint was that it had not adequately weighed the totality of evidence, had placed undue emphasis on a small number of positive findings, and should have included a quantitative meta-analysis (with Karipidis et al. citing a meta-analysis by Pinto et al. as a feasibility precedent). Mevissen et al.’s rebuttal was unusually direct, stating Karipidis et al. “do not appear to have experience in the analysis of animal toxicology” and that “basic knowledge in toxicology is necessary” to assess the studies correctly [24]. On the meta-analysis point, they argued that pooling data across different species, strains, sexes, exposure modalities, frequencies and endpoints “makes little biological or statistical sense”, and that qualitative narrative synthesis followed accepted practice for non-combinable data. It is noted that when the data are highly heterogeneous, performing a meta-analysis is inconsistent with Cochrane guidance, which advises against pooling studies that are too dissimilar to yield a meaningful combined estimate [25]. They further noted that “The authors criticizing the NTP study that was highlighted by Karipidis et al., including ARPANSA (2019); FDA (2020); the International Commission on Non-Ionizing Radiation (2020), have never criticized a negative finding in any study nor the conclusions drawn from negative findings; this suggests the potential for bias in these reviews.” [24].

The pattern across both exchanges is the same: the small number of authors associated with ICNIRP and affiliated regulatory bodies critique independent positive findings on methodological grounds; independent researchers respond that the critique itself reflects institutional bias, selective evidentiary standards, or an absence of relevant technical expertise. HHS should not resolve this by simply defaulting to whichever side happens to hold the WHO, FCC, FDA or ICNIRP institutional badge, instead it should recognize that the dispute is evidence of exactly the “asymmetric evidentiary standards” problem described in Part III below, and so should weigh both reviews and their respective critiques, accordingly.

I.3 Why the Absence of a Linear Dose-Response Trend Does Not Demonstrate Safety

A recurring argument used to dismiss positive epidemiological findings is that population-level brain tumor incidence has not been continually increasing despite decades of rising mobile phone use, and therefore is incompatible with a causal relationship. This line of reasoning is frequently paired with the related claim that the absence of a clear dose-response relationship in the aggregated observed data further undermines causality. For example, Karipidis et al. (2024) explicitly reported “no observable increase in mRRs for the most investigated neoplasms (glioma, meningioma, and acoustic neuroma) with increasing time since start (TSS) use of mobile phones, cumulative call time (CCT), or cumulative number of calls (CNC)”. They further concluded that formal dose-response meta-analyses likewise showed no evidence of rising risk with high cumulative exposure metrics [16]. At face value, these claims seem like a reasonable inference. However, it is not, for two fundamental and independent reasons that regulatory bodies and their affiliates consistently fail to engage with.

First, biological responses to RF-EMF at non-thermal levels are frequently non-monotonic, not linear. An independent scoping review and evidence map of RF-EMF genotoxicity, conducted with colleagues, identified U-shaped relationships between RF-EMF intensity and DNA damage, and more importantly between exposure duration and DNA damage, mirroring similar U-shaped patterns observed in free radical production and oxidative stress [26, 27]. This is consistent with hormesis, a well-studied phenomenon in which low or transient stress activates adaptive cellular defenses, such as the Nrf2 pathway, AMP-activated protein kinase (AMPK) signalling, mitochondrial biogenesis, heat shock proteins, DNA repair pathways, autophagy, and endogenous antioxidant systems, while more sustained or intense stress overwhelms those same defenses and produces damage [28-38]. Calabrese and Baldwin estimate that biphasic, hormetic dose response relationships occur in roughly 40% of toxicology experiments, more often than classical threshold models [38]. Cross-mode hormesis is another important and related area for consideration, where RF-EMF pre-exposure has been shown experimentally to reduce genotoxic damage from a subsequent exposure to a different genotoxic agent (e.g., ionizing radiation, UV or chemical mutagen challenge), and has been reported repeatedly in the literature [39-47]. None of this is fringe biology; it is standard toxicology theory that appears to be routinely ignored when the exposure in question is RF-EMF specifically.

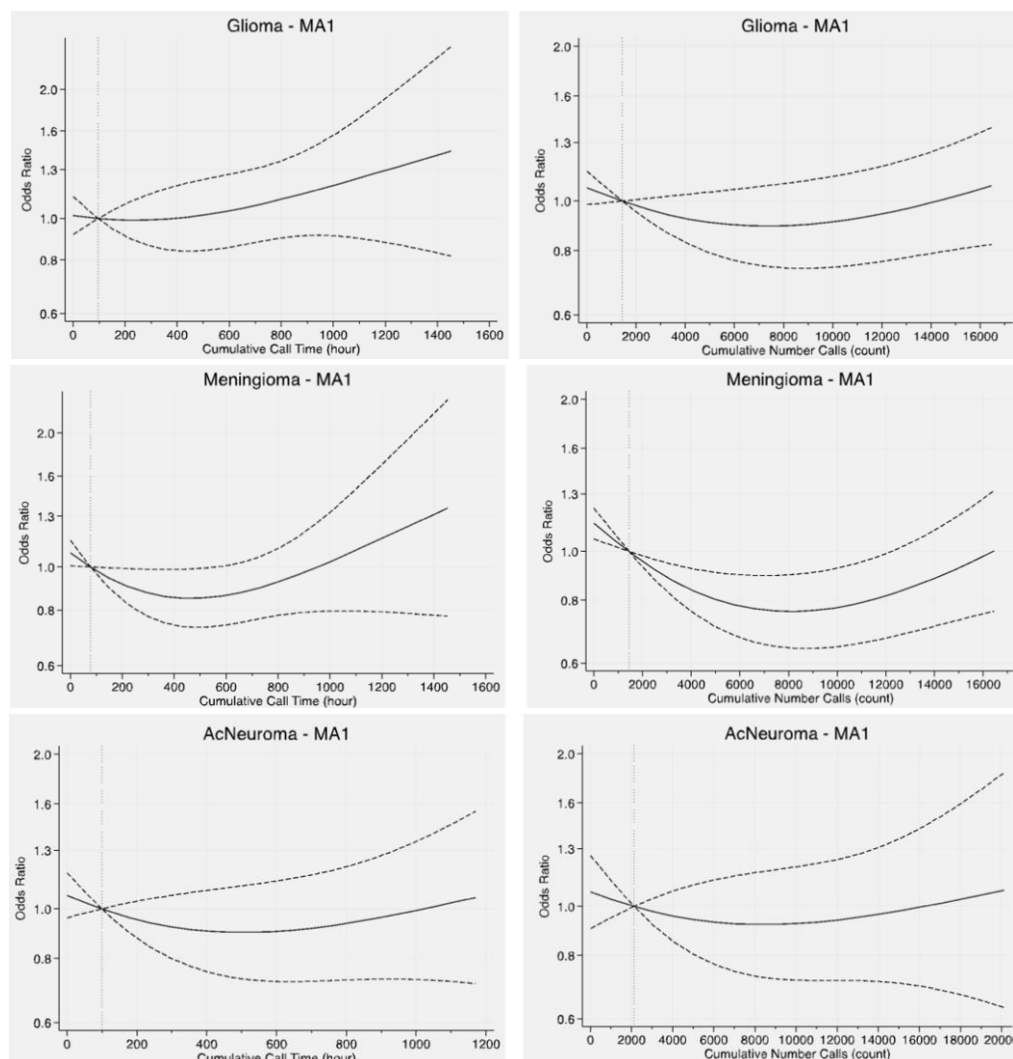


Figure 1: Various brain tumor risk profiles. Source: Karipidis et al. (2024)[16]

The practical consequences are significant. Several of the dose-response presentations in Karipidis et al. (2024) systematic review of observational studies on RF-EMF and cancer displayed U-shaped and J-shaped patterns, with risk estimates falling below 1.0 at intermediate exposure before rising again in the heaviest-exposed users (see Figure 1) [16]. Interphone showed a similar pattern, reduced odds ratios among “regular” users and an elevation only in the highest exposure category (see Figure 2) [14]. Both analyses attributed these curves primarily to selection bias, differential recall bias, and exposure misclassification [14, 16, 48]. This interpretation is coherent under conventional monotonic dose-response assumptions, yet is not compelled by the data alone.

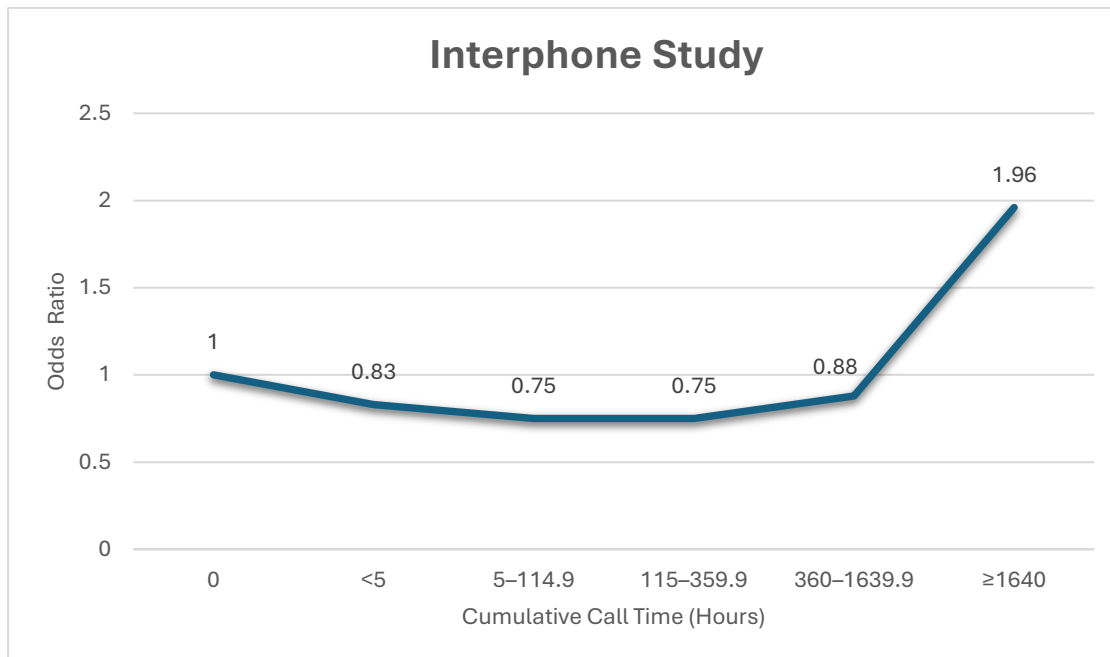


Figure 2: Interphone risk ratio for Glioma based on cumulative call time. Source: Interphone (2010) [14]

Contemporary toxicology recognizes hormesis and non-monotonic dose responses (NMDR) for a range of low-dose stressors. Adaptive or protective effects at intermediate exposures followed by adverse effects at higher doses are documented. Neither Karipidis et al. nor the primary Interphone analyses formally tested hormetic or biphasic models as competing explanations. The statistical models employed were designed to detect monotonic increases (or overall null associations), so intermediate risk estimates below 1.0 were treated as statistical noise and biases rather than potential signals of adaptive biology.

Validation studies of Interphone’s exposure recall found substantial random error, with larger variance and occasional over-estimation among cases. Purely random, nondifferential misclassification tends to attenuate associations toward the null; more complex combinations of selection bias and differential error can, under certain modelled conditions, generate a J-shape even under a true null. However, a plausible bias is not a demonstrated bias, and validation studies of the Interphone cohort found random exposure misclassification affecting both cases and controls, the kind of error that would be expected to *attenuate* risk estimated toward the null, not manufacture a spurious but significant signal for high cumulative exposure [49, 50].

Hardell and colleagues' successive case-control studies likewise showed odds ratios below 1.0 in lower exposure or shorter latency strata before elevations in risk appeared with longer latency and higher cumulative (especially ipsilateral) use. They have argued that residual recall bias is unlikely to fully account for the consistency of the latency and exposure-response patterns across datasets [51, 52].

Second, and more consequentially, the exposure represented by mobile phone use is neither homogeneous nor temporally stable. Both the technology and the way it is used have changed substantially across successive generations of mobile devices, making conventional epidemiological measures of exposure increasingly poor representations of the anatomical and temporal characteristics of RF-EMF exposure. This issue is particularly relevant to populations studied from around 2010 onwards, as smartphones progressively replaced earlier handsets and altered the patterns of use. Early first- and second-generation handsets carried external antennas positioned close to the head (brain) during a call. Modern smartphones use embed, multi-band antenna systems, with antennas distributed within the device and often positioned at the lower section of the device, changing the spatial distribution of absorbed RF energy to the lower head and neck region [53, 54]. At the same time, mobile phone use has shifted from predominantly intermittent voice calls toward a much broader range of activities, including data transmission, messaging, streaming and applications with cellular, Wi-Fi and Bluetooth components potentially contributing to exposure at different times and distances from the body. Transmission power has also changed, for example, a 3G/4G call will generally require less power than older 2G GSM calls under comparable conditions [55]. Consequently, two individuals reporting the same duration of "mobile phone use" may have experience significantly different exposures in terms of frequency, modulation, intensity, proximity and anatomical location.

This creates a particular problem for epidemiological studies, especially cohort studies in which exposure is often characterized using broad measures such as having a mobile phone subscription and years of phone ownership which provides an imperfect proxy for dose. Even case-control studies which capture more fine-grained usage details may still fail to capture important changes in the physical characteristics and the anatomical distribution of the exposure. The problem becomes amplified in more recent populations, for whom the category "mobile use" encompasses technologies and behaviors that differ considerably from those prevailing during the early mobile-phone era. Thus, an hour reported phone use in 1989 cannot necessarily be treated as an equivalent exposure of one hour use in 2026. The resulting exposure misclassification is unlikely to be random if technology, handset design, network generation, usage behaviors and anatomical proximity have all changed systematically over time. Therefore, an ecological study measuring aggregated brain-tumor incidence across this period is therefore not necessarily assessing a stable exposure. Instead, it is averaging across a moving and increasingly heterogeneous exposure landscape. If a health effect were dependent on particular combinations of frequency, modulation, intensity, duration, proximity or anatomical target, those effects could be diluted or obscured when exposure is collapsed into broad measures of mobile-phone use.

The implication of this reasoning is that RF-EMF should not be conceptualized solely as a candidate brain-specific carcinogen, evaluated against a single brain-tumor

incidence curve, but as a possible systemic carcinogen exposure capable of affecting whichever tissue(s) sit in closest proximity to a device that is now worn or carried against the body for most of the day [55, 56]. This warrants closer investigation of anatomical proximate cancers that are independently rising in incidence, including the thyroid, salivary gland, breast, prostate, rectal and others (see Figure 3), while also considering the broader, unexplained global rise in early-onset cancers in high-income countries [57, 58], rather than treating stabilized brain-tumor incidence as a closed question.

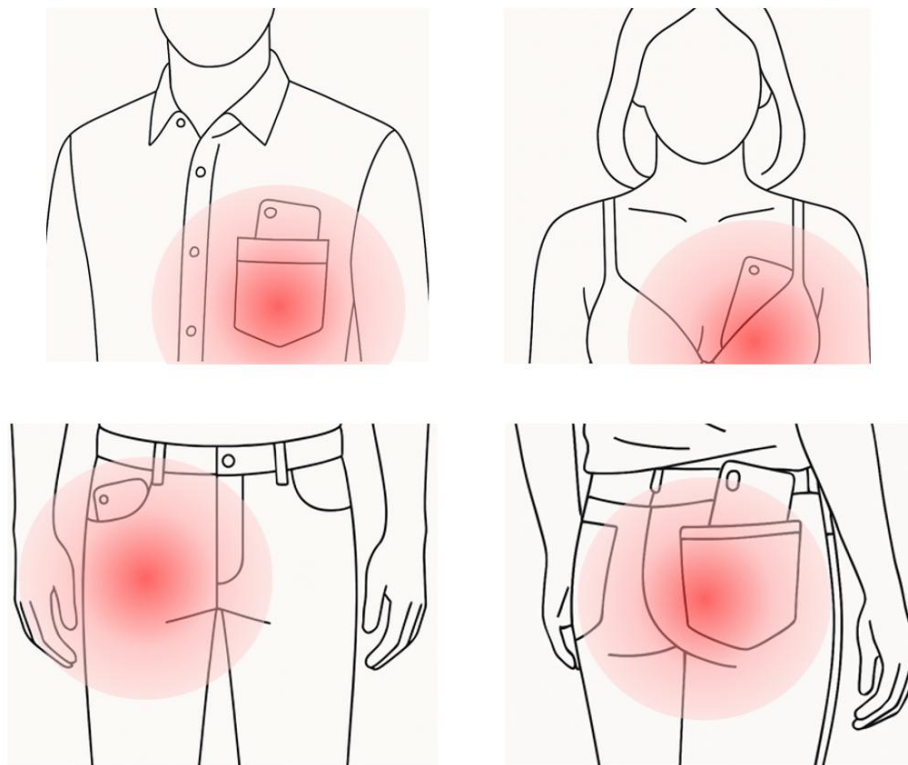


Figure 3: Mobile phone placement, RF-EMF absorption patterns and potential areas of cancer risk.
Source: Author's Thesis

It is also relevant, though not dispositive, that the telecommunications industry has long be confronted with evidence and scientific claims raising questions about the potential health effects associated with its products. Robert C. Kane, a Motorola engineer and research scientist with more than three decades of telecommunications experience, published *Cellular Telephone: Russian Roulette: A Historical and Scientific Perspective* in 2001. Writing from his personal experience within the industry, Kane described research concerning potential biological effects of RF-EMF exposure, including DNA damage, memory loss and tumors [59]. His account cannot by itself establish whether the telecommunications industry as a whole knew or accepted this position, it does however provide an insider account that the issue was recognized within parts of the industry well before the present regulatory debate.

More concrete evidence comes from Motorola's own internal documents. In December 1994, before the Lai-Singh RF-EMF DNA-break findings had been published, Motorola officials and its public-relations advisers discussed how to respond to the research. An internal strategy document referred explicitly to "war-gaming" the science and described plans to identify third-party experts who could challenge aspects of the

research and reassure the public about its implications [60, 61]. The document therefore establishes not merely that the findings were known to Motorola, but the company was actively developing a communications strategy to minimize the significance before publication. These are tactics that have previously been employed by the tobacco industry.

Subsequent litigation provides a further, though legally distinct, illustration of how questions of RF health effects have been mediated through evidentiary rules as well as through the underlying science. Michael Patrick Murray, a former Motorola handset technician diagnosed with a brain tumor, and twelve other consolidated plaintiffs initially sued Motorola and other telecommunication companies in 2001, alleging that long-term exposure to cell-phone radiation caused brain tumors. In *Motorola, Inc v. Murray* (2016), the *en banc* D.C. Court of Appeals abandoned the jurisdiction's *Dyas/Frye* "general acceptance" test for the admissibility of expert testimony and adopted the reliability framework of *Daubert* and Federal Rule of Evidence 702. Following remand, the trial court excluded the plaintiffs' proffered expert testimony under Rule 702 and granted summary judgement to the defendants. In July 2025, the D.C. Court of Appeals affirmed those rulings, holding that the plaintiffs had failed to provide admissible expert testimony establishing general causation. However, the outcome should be understood for what it was: a determination concerning the admissibility and sufficiency of expert evidence under the applicable legal standard, rather than a scientific determination that RF exposure cannot produce health relevant biological effects at or below permitted public exposure limits. The court's decision concerned whether the plaintiffs' proffered experts and methodologies satisfied Rule 702 and whether, without admissible general-causation evidence, the litigation could proceed. The distinction matters because scientific evidence and legal admissibility operate under different standards and serve different purposes.

The litigation history is also relevant because the change from *Frye* to *Daubert* was itself actively contested. Industry and business interests, including *amici* supporting Motorola, urged the D.C. Court of Appeals to abandon the *Frye* standard, and the court agreed, ultimately adopted the *Daubert*/Rule 702 reliability framework in 2016. The subsequent exclusion of the plaintiffs' experts illustrates the consequences of that change: after remand, the court applied the new reliability standard to the expert evidence, and the exclusion was ultimately upheld on appeal in 2025.

HHS should therefore treat this history as relevant context when considering what scientific and regulatory changes could improve the assessment of RF-EMF exposure. The point is not that litigation outcomes establish scientific causation, nor that the existence of industry advocacy proves misconduct. Rather the record demonstrates that questions surround RF-EMF biological effects have not only been shaped by the accumulation and interpretation of scientific evidence, but also by institutional responses to adverse findings, the strategies used to communicate and manage uncertainty, along with the legal rules governing which expert evidence is permitted to reach a fact-finder.

I.4 The Institutional Treatment of RF-EMF Cancer and Mechanistic Evidence: IARC's Word Cancer Reports, 2003 – 2020

The clearest documented example of how institutional evidence-handling, rather than the underlying data, has shaped the official RF-EMF narrative is the treatment of RF-EMF across four successive editions of the IARC World Cancer Report. The 2003 and 2008 editions used identical language, noting epidemiological signals from leukemia and brain tumors, but describing the evidence as “inconsistent and limited”, while also asserting that experimental evidence “suggests that radiofrequency fields cannot cause DNA mutations” [62, 63]. The verbatim repetition across a five-year gap suggests a cautious institutional position being maintained, emphasizing limitations in the evidence even amid emerging data.

The 2014 edition continued this cautious framing, prominently featuring the Danish cohort study (which reported no association despite well-documented significant methodological weaknesses) while omitting Hardell's positive-association studies and selectively noting limitations in positive association studies, without meaningfully engaging with alternative interpretations [64, 65]. Notably, the same 2014 report contained an insert from Dr. Robert A. Baan, the IARC officer who had overseen the production of the IARC Monograph volume 102 covering radiofrequencies, and offered a more balanced perspective that explicitly flagged the then-ongoing NTP carcinogenicity study as a study of interest that could help clarify RF-EMF's carcinogenic potential [6, 64]. The fact that two perspectives coexisted within the same report demonstrates perspective on RF-EMF risk were not monolithic within IARC and the WHO, and suggests that interpretive frameworks and institutional affiliation, not solely data, were shaping the narrative.

By the time of the most recent World Cancer Report (2020), the NTP study had been completed and had reported **“clear evidence” of carcinogenicity alongside evidence of genotoxicity in rodents**, and the **Ramazzini Institute had independently corroborated the same tumor types** under different, lower-intensity exposure conditions [6, 7, 66, 67]. However, the 2020 report made no reference to any of these studies. Instead, it reiterated that no consistently identified carcinogenic mechanism has been established, and again attributed positive finding to “various forms of bias such as recall bias” [67]. The contrast between Baan's 2014 insert, and the 2020 report is stark: in 2014, the pivotal animal studies were described as pending and important, by 2020, with the results published and positive, they were simply omitted. This is a documented, checkable pattern of selective evidence inclusion by the world's leading cancer research authority, and simply not a matter of interpretation. It is also relevant that the corresponding 2020 section was co-authored by an ICNIRP Commissioner (at the time) with documented links to an industry funded research foundation [67, 68]. This fact does not prove by itself intent, but it does reinforce why institutional affiliation and funding source must be treated as material information when HHS conducts and evaluates future evidence reviews, exactly as this RFI itself asks respondents to do (Instructions, “if you rely on existing exposure guidelines, regulatory frameworks or scientific assessments, please identify the specific framework(s)”).

This pattern, however, is not confined to IARC's flagship report. The claim that "no established mechanism exists" is repeated across WHO and ICNIRP assessments (and is implicit in the thermal-only foundations used by the FCC and ARPANSA) as though it was dispositive [67, 69]. It is not, the absence of an established mechanism is a statement about the current state of mechanistic consensus, not evidence that no mechanism exists, and Part I.5 below sets out why the oxidative-stress mechanism now has a substantially and rapidly growing evidentiary base of exactly the kind that regulatory bodies have chosen not to consider and weigh.

I.5 Oxidative Stress, the "Key Characteristics of Carcinogen" Framework, and the Upcoming IARC Re-Evaluation

The claim that no plausible carcinogenic mechanism exists for RF-EMF rests heavily on the fact that RF photons (in the order of 10^{-6} – 10^{-4} eV) are far too low in energy to directly ionize atoms or break chemical bonds (roughly 10 eV and above is required) [67, 70-74]. This is true, but it treats direct ionization as the only route to genotoxicity, which is not correct even within the accepted carcinogenesis literature. Since 2019, IARC has formally incorporated the "Key Characteristics of Carcinogens" (KCC) framework into its evaluation process for all agents, and the planned RF-EMF evaluation will apply this to RF-EMF research for the first time. KCC characteristic #8 is explicit "induces oxidative stress" and this is precisely where the experimental RF-EMF literature is strongest. Numerous *in vitro*, *in vivo* and several human studies report increased reactive oxygen species (ROS) production, altered antioxidant enzyme activity, oxidative damage to DNA, lipids and proteins at non-thermal exposure levels [34, 75-80].

The magnitude of this evidence base is itself instructive. Independent reviews report significant oxidative stress effects in 83 to 93% of hundreds of experimental studies across diverse models and endpoints [26, 76, 81, 82]. Against this, the most recent WHO-commissioned systematic review of oxidative stress evidence included fewer than 15% of relevant studies catalogued in the independent ORSAA database, it also had restricted its frequency range to 800 - 2450 MHz (far narrowed than the original protocol's intent of 100kHz - 300 GHz), excluded epidemiological data entirely, applied exposure thresholds that removed most low-level exposure studies, and discounted standard oxidative stress assays, including malondialdehyde on the grounds of "limited endpoint specificity" along with 8-oxoG, and Fpg-modified comet assays [83, 84]. The review also appeared to require a linear dose-response relationship to credit an effect, despite non-monotonic responses being well documented in this literature [26, 27, 85, 86].

This is not a minor methodological quibble. It is a demonstration of what independent researchers have termed asymmetric evidentiary standards. A biological process (oxidative stress and downstream evidence of genotoxicity) that is treated as centrally important to carcinogenesis for virtually every other agent that IARC evaluates is subjected to unusually restrictive inclusion criteria, a narrower frequency range, and a higher bar for what counts as a "real" effect, when the agent in question is RF-EMF specifically. Similar dynamics of elevated evidentiary bars, selective skepticism, and institutional "caution" appear with other high-stakes, high-economic-value agents

(certain chemicals, pharmaceuticals and other industrial products) and especially when large liability or regulatory costs are at stake. This is the single clearest evidentiary basis for this submission's position that HHS should not defer automatically to the existing WHO/ICNIRP evidence-synthesis and judgements when it convenes its own evaluation under this RFI.

I.6 Non-Ionizing Radiation is not Automatically Non-Carcinogenic: The UV Precedent

Ultraviolet radiation (UVA/UVB) is, like RF-EMF, non-ionizing, yet it is an established IARC Group 1 human carcinogen, causally linked to melanoma and non-melanoma skin cancers [87]. UV causes DNA damage both through direct photochemical mechanisms (cyclobutene pyrimidine dimers, 6-4 photoproducts) and through indirect, ROS-mediated oxidative pathways producing recognized biomarkers such as 8-oxoG and 8-OHdG, the very same oxidative lesions reported in RF-EMF experimental studies [26, 88, 89]. UV-induced oxidative damage is causally linked to mutations in genes such as BRAF and NRAS in melanoma [88], the same genes noted in the fastest growing cancer by incidence (in the last decade), thyroid cancer, particularly papillary thyroid cancer [90, 91].

The comparison is not offered to suggest that RF-EMF and UV are equally potent carcinogens, also noting UV's penetration is limited to superficial tissue, whereas RF-EMF penetrates more deeply and diffusely, but to make a narrower and more important point, that the entire electromagnetic spectrum, ionizing and non-ionizing alike is bioactive, capable of generating free radicals, either directly (ionizing) or via effects on endogenous cellular processes. The carcinogenic potential therefore clearly does not require direct-ionization mechanisms that regulators treat as a precondition for taking RF-EMF more seriously in this space [78, 92, 93]. The International Commission for Radiological Protection (ICRP)'s own linear no-threshold model for ionizing radiation exists precisely because a safe threshold for cancer induction could not convincingly be established, so ICRP manages this uncertainty prudently rather than waiting for certainty [94]. This illustrates that some institutions are willing to act under incomplete mechanistic certainty, unlike ICNIRP, the WHO International EMF Project, and national regulators such as the FCC and ARPANSA.

RF-EMF's biological effects, including oxidative stress, disrupted calcium signaling, altered gene expression, impaired DNA repair, DNA strand breaks and oxidative damage, have all been reported at exposure levels below current thermal limits [26, 34, 76, 77, 79, 80]. These observed effects are more subtle, indirect, and like low-level ionizing radiation, their effects are likely cumulative and stochastic. Importantly, subtlety is not the same as non-existence, and UV demonstrates that regulators are capable of recognizing non-thermal, non-ionizing carcinogenic mechanisms when the agent is not RF-EMF.

I.7 Applying the Bradford Hill Viewpoints to RF-EMF

An independent Bradford Hill causal assessment of the RF-EMF and cancer literature was conducted by me for this RFI response (see Table 1), balancing evidence from the NTP and Ramazzini lifetime bioassays, the Interphone study, the Hardell case-control

series, the WHO-commissioned systematic reviews and the ORSAA evidence-mapping database [6-8, 14, 51, 84, 95, 96]. It is worth noting that Hill's viewpoints were never intended as a checklist requiring unanimous satisfaction, but as a structured way of weighing the evidence where randomized trials are impossible, which is exactly the exercise this RFI has indirectly asked respondents to undertake.

Table 1 Bradford Hill's viewpoints applied to RF-EMF evidence

CRITERION	WHAT IT REQUIRES	ASSESSMENT FOR RF-EMF EVIDENCE
STRENGTH	Stronger associations (higher relative risk) are more likely causal	Moderate overall: moderate in animal studies with clearer evidence for specific rare tumors; moderate in better designed case-control studies with long latency and high cumulative exposure (Hardell group; CERENAT); weak or null in most cohort studies (Danish Cohort, Million Women Study etc.)
CONSISTENCY	Repeated observation across studies, population and time	Mixed but improving for animal evidence and for a subset of human studies that adequately assess long-term heavy use; heterogeneity partly explained by exposure assessment differences, latency, technology change, and usage pattern shifts
SPECIFICITY	Specific exposure → specific outcome (less emphasized in modern multifactorial disease)	Weak, as expected for an exposure acting via oxidative/genotoxic pathways with multiple downstream outcomes; supportive but not decisive
TEMPORALITY	Exposure must precede outcome	Satisfied in the animal lifetime studies and in human analyses with adequate lag) NTP, Ramazzini, Hardell, CERENAT
BIOLOGICAL GRADIENT	Dose-response relationship	Supported in key studies: INTERPHONE and Hardell found increased risk with higher cumulative use/duration; NTP showed dose trends; Ramazzini showed effects at lower levels (non-linear); non-monotonic (NMDR) responses are common in low dose toxicology and should not be treated as absence of a gradient
PLAUSIBILITY	A reasonable biological mechanism	Strengthened by convergent evidence for ROS generation, oxidative stress, DNA damage, altered gene expression and related cellular stress pathways
COHERENCE	Does not conflict with known disease biology	Coherent: patterns resemble other recognized carcinogens (low-dose ionizing radiation, UV-A/B); multisite effects and non-cancer outcomes (oxidative stress, neurological, fertility effects) fits known biology
EXPERIMENT	Evidence from intervention or removal of exposure	Moderately supportive: NTP and Ramazzini bioassays provide the principal experimental evidence; controlled human experiments are ethically constrained
ANALOGY	Similar known causes support the hypothesis	Supportive: reasonable analogy to UV and other low-level radiation effects acting through similar ROS/Oxidative/DNA damage pathways

The strongest support arises from temporality, experiment, and plausibility. Lifetime animal studies demonstrate that exposure precedes tumor development, and mechanistic research provide biological credible, non-RF-EMF-specific pathways (oxidative stress, ROS, DNA damage, altered signaling, gene expression change) [6, 7, 14, 66, 97-99]. Evidence for strength, consistency, and biological gradient is more mixed, concentrated in the NTP/Ramazzini studies and in case-control studies with long-term and high cumulative use, while many cohort studies show weak or null associations, findings that have themselves been subject to criticism for exposure misclassification, short follow-up and inadequate consideration for latency for slow developing tumors [100-105]. Specificity offers little support, but Hill developed this

criterion for infectious disease, and diseases like cancer are multifactorial, so are not expected to satisfy it strongly.

Overall, this assessment indicates that RF-EMF satisfies several Bradford Hill viewpoints to a meaningful degree, particularly for glioma and cardiac schwannoma. This is a materially stronger evidentiary position than the “no evidence of harm” formulation used by most regulatory communications, because it is supported by convergent animal, mechanistic, and selected epidemiological findings, even though it does not yet reach the certainty of established tobacco smoking and ionizing radiation exposure. As Frank (2025) has argued, precautionary approaches can be justified from a public health perspective once multiple Bradford Hill viewpoints are supported, without every viewpoint needing to reach the highest possible evidentiary standard [106]. HHS should adopt this framing directly. The correct policy question is not “has causation been proven” but “does the pattern of evidence across all nine viewpoints justify precautionary action”, and on that question the answer set out above is a resounding and definitive yes.

I.8 Response to Question 13: Cumulative Exposure Patterns Across Wireless Technologies

The evidence reviewed above bears directly on how HHS should think about cumulative exposure from 4G, 5G, 6G, Wi-Fi, satellite communications, IoT, wearables, smart homes, smart cities, autonomous vehicular radar, wireless medical devices and communication-enabled smart meters. The single most important point is a structural one: nearly all of these technologies add to, rather than replace, existing RF-EMF exposure. A modern smartphone alone now maintains near-continuous transmission across cellular, Wi-Fi, Bluetooth, and GPS bands simultaneously, a pattern of exposure that did not exist when the foundational epidemiology on mobile phones and brain tumors was designed [55]. Layering 5G/6G small- and micro-cell infrastructure, IoT sensors, devices that are worn on the body for extended periods and smart-home/city networks into this baseline compounds simultaneous multi-frequency exposures in ways that current RF standards/guidelines, built around single-source, single frequency thermal limits, are not specifically designed to evaluate [55]. I am not aware of any adequate, independently replicated epidemiological or exposure reconstruction research investigating the cumulative, simultaneous-multi-source exposure profile now typical of daily life on health, and this is identified as a priority research gap in Part VI below rather than a question this submission can answer definitively technology-by-technology. Today it is impossible to isolate a single technology and understand its impact on health given there is no reliable unexposed control pool (for all forms of anthropogenic RF-EMF) to compare against.

Current regulatory limits are defined and enforced primarily as time-averaged SAR values for near-field exposures (6 or 30 minutes under current ICNIRP and FCC rules depending on the exposure condition) [69, 107]. This averaging framework permits short duration instantaneous SAR levels that can substantially exceed the numerical limit, provided the rolling average remains compliant. Modern devices exploit this allowance through dynamic power control and time-averaged SAR (TAS) algorithms, producing high-intensity bursts while staying within the average limit.

Because the averaging windows themselves are derived from predominantly thermal time constants, they are poorly matched to non-thermal biological responses that may depend on frequency, rapid field variation and/or modulation characteristics rather than long-term average energy disposition. What the mechanistic evidence set out in Parts I.3, I.4 and I.5 does establish is that duration and the pattern of exposure, not necessarily intensity, are likely to be more important determinants of biological outcome. In particular, the submission author's RF-EMF genotoxicity scoping review found long-duration exposures at extremely low intensities (including levels < 0.001 W/kg) were associated with a higher proportion of positive effect outcomes. Surveillance and standard-setting frameworks built around time averaged SAR values from a single source will therefore systematically understate real-world cumulative dose and exposure patterns most relevant to those findings.

I.9 Response to Question 14: Potential Environmental Effects

Biological effects of low-intensity RF-EMF have been reported experimentally and in field studies across plants, insects, and animals at exposure levels well below current human public-exposure limits [108]. Reported effects include alterations in plant metabolism, reduced reproductive success in insects, behavioral or physiological disruption in birds and mammals [109-111]. Specific to pollinators, RF-EMF exposure has been linked to changes in navigation, colony behavior, reproduction, and survivability, including altered work-bee "piping" behavior, with RF-EMF being proposed as a contributing driver to broader insect decline [112, 113]. These findings have prompted independent scientists and concerned members of the public to question the adequacy of human health-only exposure thresholds as a basis for protecting ecosystems more broadly [114-117]. HHS's mandate is human health, but given the RFI explicitly asks about environmental effects, HHS should recognize that the existing evidence base, while smaller than the human health literature, is not negligible and the current exposure guidelines adopted by the FCC are not designed with ecological protection in mind at all.

I.10 Response to Question 15: Evidence Concerning Wireless Infrastructure

On proximity to homes, schools, childcare and healthcare facilities, and workplaces; cumulative neighborhood exposure; antenna density; and small/micro-cell deployments, the strongest available evidence in my own research is not epidemiological but institutional. The 2019-2020 Australian Parliamentary Inquiry [1] into 5G, in which public submissions repeatedly invoked the precautionary principle and called for independent, industry free investigation along with a deployment moratorium pending safety confirmation:

We recommend a moratorium on the roll-out of the fifth generation, 5G for telecommunication until potential hazards for human health and the environment have been fully investigated by scientists independent from the industry. (Submission 417)

... request a moratorium on the deployment of 5G until there is independent research that shows that this technology is safe and that there is a biologically based exposure limit implemented in Australia, which also includes the Precautionary Principal. ... There should be

an immediate independent investigation into developing stricter guidelines and regulations surrounding the public exposure to RF EMR. (Submission 393)

Government and industry-aligned submissions took the opposite position, treating the absence of established harm as sufficient grounds to continue deployment:

Health authorities around the world, including ARPANSA and the World Health Organization (WHO), have examined the scientific evidence for possible health effects from telecommunications sources. Current research indicates that there is no established evidence for health effects from radio waves used in mobile telecommunications. This includes the upcoming roll-out of the 5G network. ARPANSA's assessment is that 5G is safe.
(Submission 317 - ARPANSA)

These are not equivalent propositions, and HHS should not treat them as such when it evaluates comments on wireless infrastructure siting under this RFI. Demonstrating that a technology is entirely safe is generally not possible. However, the absence of “established” harm does not establish an absence of risk, particularly for exposures that are chronic, often non-consensual, and population-wide. The referenced inquiry illustrated that the underlying dispute is not about whether the same body of evidence supports a given conclusion, it is about which burden of proof, and which threshold for precautionary action should apply before infrastructure is sited near homes, schools and childcare facilities.

With regards to antenna density and small-cell deployment specifically, this burden-of-proof question becomes more urgent precisely because small-cell 5G/6G architecture by design multiplies the number of nearby emission sources per unit area compared to legacy macro-cell towers. This automatically increases cumulative neighborhood background exposure even when any single small-cell is operating well within existing public exposure limits, a cumulative exposure question that per-source thermal based limits are not designed to answer.

Health symptom surveys near base stations

Although none of the following papers were designed to study electromagnetic hypersensitivity (EHS) per se, all reported symptoms in people living near mobile phone base stations overlap with EHS non-specific symptoms, and bear directly on Question 15's concern with proximity to houses. In this section I compare two French survey papers reporting on the same 530 study population (Santini et al. 2003) [118, 119] with two recent studies from Aizawl, Mizoram, India: a symptom survey of 309 residents (Sailo et al, 2025) [120] and a blood-parameter study of 101 residents (Laldinpui et al. 2026) [121] of which I was a co-author for both Indian papers. Also included is a symptom survey of 278 adults in Hilla, Iraq (Hussain et al 2026) [122]. The Hussain study points the same way as the other studies being compared but is of a lower quality and so should be given limited weight. Table 2 summarizes the designs and findings.

Table 2 Comparison of the four base-station studies

Feature	Santini et al. 2003 (France) [118, 119]	Sailo et al. 2025 (Mizoram) [120]	Laldinpui et al. 2026 (Mizoram) [121]	Hussain et al. 2026 (Iraq) [122]
Design	Questionnaire survey; recruited through press, radio and websites; returned by mail	Home questionnaire, interviewer present but not assisting; 30 base stations (900 and 1800 MHz)	Blood study, 10 base stations (2022–23); door-to-door; nurse and laboratory blinded to exposure status	Face-to-face interview survey, Hilla, 2017–18; 4 base stations
Sample	530 (270 men, 260 women); about 21% in reference group	183 within 300m; 126 reference (≥ 400 m); matched on demographics	50 within 60 m; 51 reference (≥ 300 m); adults aged 18 – 45, matched on age and gender	278 responded (92.6%); Table 1 counts imply 180 within 300m and 92 beyond 500m; ≥ 5 years' residence; 59% male
Exposure metric	Self-estimated distance bands and position relative to antenna; no field measurements	Distance (laser tape) and in-home power density (103 homes); exposed mean 4.8 vs reference 0.7 mW/m ²	Distance (laser tape) and in-home power density; exposed about 4.6 – 4.8 vs reference 0.05 mW/m ²	Self-estimated distance (<300 m vs >500 m); no field measurements
Outcome	16 – 18 non-specific symptoms, rated 0 – 3	25 yes/no symptoms in four categories	Complete blood count with differentials; serum amylase and cortisol	Symptom checklist; Table 1 shows 7 items
Main finding	Complaints significantly more frequent than reference within 300m; symptoms drop out at different distances (nausea, appetite, vision to about 10m; irritability, depression, memory, concentration, dizziness to about 100m; sleep headache, discomfort to about 200m; fatigue to 300m)	All symptoms except weight problems varied significantly with distance and with power density; power density added 37 – 51% explained variance in each symptom category	Higher total leukocytes in exposed group; power density predicted total leukocytes, basophils and absolute monocytes; 24% (12/50) of exposed vs 10% (5/51) of reference had basophils above the reference limit	Insomnia, dizziness, headache and memory loss higher within 300 m; anxiety and depression lower.
Sex	Women reported 7 symptoms more often within 300m; no sex difference in reference group	Overall pattern not altered by gender	Sex moderated blood effects (TLC and basophils higher in exposed women; monocytes higher in exposed men)	No sex-specific analysis
Age	Older respondents more sensitive; no significant difference in those ≤ 20	Older respondents reported more ear, visual, joint, nerve and chest symptoms regardless of exposure; exposure-related inflammatory symptoms higher in those <40	Basophil excess mostly in those under 30; cortisol higher in exposed older group	Not analyzed
Duration	No significant change from <1 to >5 years (irritability excepted)	Some symptoms higher at 9 – 18 than 5 – 8 years; not significant in regression; no residents <5 years	No effect of 5 – 10 vs 11 – 20 years, or of distance within 60m	Not analyzed (all ≥ 5 years)
Mobile phone use	Little influence; sleep disturbance lower in phone users	Moderated some symptoms at >5 h/day; no main effect in ANOVA	Daily hours predicted lower basophil and lymphocyte counts; inverted-U for lymphocytes outside reference limits	Not measured

Feature	Santini et al. 2003 (France) [118, 119]	Sailo et al. 2025 (Mizoram) [120]	Laldinpuii et al. 2026 (Mizoram) [121]	Hussain et al. 2026 (Iraq) [122]
Acknowledged limits	Self-selected, media-recruited sample; distance self-estimated; no measurements	Cross-sectional; self-report, binary responses; interviewers knew distance (not power density); distance overlap between groups	Small sample; cross-sectional; single-point measurements; frequencies not separable; no pre-exposure baseline	Small sample; cross-sectional; mobile phone use and field density not measured

Where the papers agree. Across different countries, decades, recruitment methods and exposure assessments, the same symptom cluster recurs among people living closer to base stations than among a distant reference group: headache, sleep disturbance, fatigue, irritability, concentration and memory difficulties, dizziness, nausea and loss of appetite. This overlaps with the cluster of reported symptoms in the Australian Parliamentary Inquiry into 5G and smart meter datasets in Part III.8 [1, 4, 5]. The Iraqi survey points the same way for insomnia, dizziness, headache and memory loss. In the Mizoram studies the differences appeared at measured power densities of roughly 2 - 7 mW/m², well below the Indian public limit of 450 mW/m² at 900 MHz [120]. Both Mizoram studies also excluded homes near transformers, high-voltage lines and radio television broadcast transmitters.

Duration of residence added little in any of the papers, but this is a weak test: no Mizoram resident had lived near the tower for less than five years, so onset in the first months of exposure could not be examined.

Where they differ. The main difference is exposure assessment. Santini et al. and Hussain relied on distance estimated, while both Mizoram studies measure power density in the living room and also took distance measurements. Sex and age patterns are not consistent. Santini et al. reported greater sensitive in women and older people; Sailo et al. found no sex effect and, for exposure-related inflammatory symptoms, a stronger effect in those under 40; Laldinpuii et al. found sex-specific blood patterns. Mobile phone use mattered more for the blood measurements than for symptoms. These inconsistencies should be reported rather than smoothed over and may reflect differences in population, dietary habits, exposure and measurement, or chance in modest samples. Hussain et al. also found anxiety and depression somewhat less frequent near the towers than further away, and the relative frequencies of symptoms near vs further away (about 1.1 - 1.6 for symptoms in Table 2) were smaller than in the Mizoram survey [120, 122]. Santini et al. also examined position relative to the antenna and reported that facing the antenna was worst within 100m, consistent with antenna directionality [118, 119].

However, distance is a poor surrogate for exposure, and the Mizoram data shows why. Field strength near a base station depends on antenna height, tilt and direction, line of sight, and can be attenuated by buildings and vegetation, not on distance alone. In Aizawl's hilly terrain, Sailo et al. found that some homes more than 400m from a tower had line of sight and power densities as high as some homes within 300m, while other homes closer than 300m were partly or wholly shielded; distance and measure power density were strongly negatively correlated ($r_p = -0.9$) [120]. Santini et al. themselves caution that even measured fields near base stations fluctuate with call traffic and reflection and so may not represent true exposure [119]. Hussain et al. likewise

classified exposure by distance estimates, with a 200m gap between their near (<300m) and far (>500m) groups [122]. Misclassification and reduced exposure contrast can dilute real differences in reported symptoms.

The practical consequence is visible in Sailo et al.'s own data Grouped by distance, symptom frequency is not monotonic. For several symptoms (fatigue, sleep problems, memory problems and problem-solving difficulty) the 51 - 300m group reported symptoms less often than the group beyond 400m. When grouped by measuring power density (0 - 3, 3 - 5 and 5 - 8 mw/m²), the proportion reporting each symptom rose from lowest to the middle to the highest band for every one of the 25 symptoms listed in their Table 3, for example, concentration problems were 4.7%, 51% and 87.9%; memory problems were 16.3%, 59.2% and 92.3%; headache 44.1%, 81.6% and 95.6% [120]. Santini et al. showed a cruder version of the same gradient with the number of symptoms still significant falling stepwise as distance increases [119]. Neither is a precise exposure-response curve, but a graded decline in complaints with increasing distance or decreasing measured field strength is the pattern a dose-dependent effect would produce, and it corresponds to the biological-gradient viewpoint assessed in Table 1 shown in Part 1.7.

Linking the blood findings to the symptom findings. The two Mizoram papers come from the same city and research group, with the participants of the second study being a subset of the first (309 vs 101 residents). Table 3 sets out where the findings align and where they do not.

Table 3 Symptom findings [120] alongside blood findings [121]

Symptom finding (Sailo et al. 2025) [120]	Blood finding (Laldinpui et al. 2026) [121]	Interpretation and caveats
Allergy and increased infections more frequent near towers, with the excess concentrated in those under 40 (at 3.1 – 5.0 mW/m ² : allergies 54% vs 16%; infections 75% vs 20%)	Differential and absolute basophils higher with greater power density; 24% of exposed residents above the reference limit, most under 30	Basophils release histamine and mediate allergic inflammation. The two studies agree in direction and in the younger age group. This supports an immune-activation hypothesis; it does not show that the same individuals had both findings, or that exposure caused either.
Inflammatory symptom category (headache, chest pain, allergy, infections): power density explained 51% of variance	Absolute monocytes rose with power density; total leukocyte count higher in exposed group	Monocytes are a marker of inflammatory activity. The authors propose chronic low-grade inflammation and suggest testing inflammatory signaling and degranulation markers directly [121].
Mood-energy symptoms (anxiety, irritability, fatigue, sleep problems): power density explained 48% of variance	Cortisol and amylase: no exposure effect in regression; smoking and age dominated amylase; some categorical differences only	No concordance. Stress-hormone measures from a single afternoon sample did not track these symptoms, so the blood study does not support a simple stress-hormone explanation for them.
Mobile phone use above 5 hours per day moderated some symptoms but had no main effect	Daily phone hours strongly associated with basophil and lymphocyte changes (inverted-U for lymphocytes outside the reference range)	Base station and handset exposure appear to affect different cell types. The symptom survey's two-category phone-use measure (≤5 vs >5 hours) is probably too coarse to detect this, as its authors note.
Years of residence not a significant predictor	Years of residence had no effect (5–10 vs 11–20 years)	Consistent, but neither study includes residents of less than five years.

What this evidence does and does not show. Both Mizoram studies are cross-sectional, use modest samples and single-point measurements, and include many statistical tests. The symptom data are self-reported and some participants could see

the towers, so expectation effects cannot be excluded for the questionnaire results; in the symptom study the exposed group was also restricted to people at home during the day. The blood study's blinded sampling and laboratory analysis reduces concern for its endpoints, and because the participants in both studies were not recruited on the basis of an EHS label, the finding addresses part of the expectation question raised in Part III.8 without fully resolving it. Most blood differences are shifts still within or near normal ranges, although a minority were outside them. I do not claim these studies establish causation; however, they are consistent with, and add biological along with dose-response support to the symptom reports that regulators currently dismiss as not being "established".

Research gap and replication. I am aware of relatively few studies that combine in-home field measurement with symptom reporting near mobile phone base stations, fewer still that add blinded objective biomarkers, and I am not aware of a published attempt to directly replicate the Mizoram designs (due to recency of the study), with either a positive or a null result. The nearest is Hussain et al.'s 2026 survey in Hilla, Iraq, which tested only the distance-symptom association, without field measurements or biomarkers and found the same direction of difference for insomnia, dizziness, headache and memory loss [122]. Related work is mixed and should be acknowledged: a large German population-based survey reported slightly more health complaints among people living with 500m of a base station and noted that the higher prevalence of health complaints could not be fully explained by attributions or concerns [123]. A companion analysis using field measurements found no association between measured RF-EMF and the health outcomes studied, although bedroom exposures in that study were reported to be mostly very low, often near the dosimeter's detection limit and so may have suffered from a lack of contrast [124]. Because exposure contrast, measurement method and endpoints differ, such results cannot simply be pooled with the Mizoran findings. The absence of direct replication is itself the point: HHS should treat the question as under researched rather than closed.

Implications for HHS. Siting and surveillance studies should (i) measure power density at the residence, and report it alongside distance, rather than substituting distance for exposure (Part IV.1); (ii) pair symptom capture with blinded objective endpoints, such as differential leukocyte counts, hormone and neurotransmitter profiles, oxidative stress and DNA damage markers, and include personal dosimetry where feasible; (iii) include cases with short resident times and, where possible, before-and-after designs around new installations; and (iv) be independently replicated, including in the United States, by researchers with no industry, military or ICNIRP-affiliated ties (Part VI).

Part II – Public Experiences and Clinical Observations (Questions 3 – 4)

This submission is grounded primarily in published literature and institutional analysis rather than first-hand personal health experience and so therefore I have not completed the personal-experience fields (exposure type, onset timing, diagnosis, exposure-reduction outcomes) that Question 3 invites individuals to provide. Two points from my own research are nonetheless directly relevant to how HHS should weigh clinical and occupational observations submitted by others under this RFI.

First, documented clinical case evidence of short-latency cancers following occupational RF-EMF exposure exists and should not be dismissed on latency grounds alone. A published sentinel case series has reported cancer patients with occupational exposure to non-ionizing electromagnetic fields presenting with unusually short latency [56]. Case series of this kind are inherently limited in what they can establish about population-level risk but they are exactly the kind of clinical pattern-recognition signal that Question 4 asks healthcare professionals and researchers to report, and HHS's surveillance recommendation (Part IV below) should be designed to make it easier, not harder, for such signals to be systematically collected and aggregated rather than left as isolated case reports or worse, unreported.

Second, the historical record shows that industry actors and defense-related institutions were aware of a broad range of reported non-thermal biological effects, including neurological, cardiovascular, reproductive, genotoxic and potentially carcinogenic effects decades before current safety guidelines were finalized, even when causal interpretation of these early findings remained contested [125-127]. This institutional awareness, combined with the litigation history discussed in Part I.3 (the Kane disclosures and the *Murray v. Motorola* case) is directly relevant context for HHS in weighing why individual and clinical reports of adverse effect have historically received a more skeptical reception from regulatory bodies than the underlying signal may have warranted.

Part III – Exposure Standards, Risk Assessment, and Regulatory Governance (Questions 5, 6 and 11)

III.1 The Thermal-Only Paradigm: Origin and Limits

FCC RF-EMF exposure rules, like ICNIRP's guidelines recognize only acute tissue heating and nerve stimulation as established mechanisms of harm, and set their exposure reference levels accordingly. This is not simply a conservative starting point that has been supplemented as the evidence accumulated; instead, it is a paradigm whose origins are administrative and military as much as biological. Steneck's historical analysis of the development of U.S. microwave exposure standards found that early RF protection limits emerged from a mix of military utility, engineering feasibility, and administrative judgement reflecting Cold War institutional priorities, not solely from an objective determination of biological safety [128-130]. Critics argue this legacy continues to shape contemporary governance, making it structurally difficult for the evidence of chronic, non-thermal biological effects to gain regulatory traction [131-133].

The practical consequence is that findings of oxidative stress, genotoxicity, altered gene or protein expression, neurological effects, reproductive effects or electrophysiological changes at intensities below the current heating threshold are treated as insufficiently established for regulatory purposes almost by definition, regardless how consistently they are reported. This is due in part because the framework used for assessment privileges thermal effects and was never designed to

register non-thermal effects as relevant in the first place. This is a different problem from ordinary scientific disagreement about data quality, which is normal and healthy in biomedical science; it is a foundational assumption (only thermal effects matter) being protected from the evidence that would test it, with any challenging finding routing automatically into “methodologically flawed” or “not yet established” categories [69, 114, 132, 134]. FCC rules have remained largely unchanged for decades despite substantial growth in population-level exposure and a growing body of experimental and epidemiological evidence of non-thermal biological effects with potential health consequences. The current approach relies on what the late Dr Neil Cherry described as constructive dismissal [134], which provides regulatory stability and has the practical, if not necessarily intended, effect of insulating existing military, telecommunications, and industrial applications from disruption.

III.2 Two Models for Managing Uncertainty: ICNIRP/FCC Compared with ICRP

The most instructive comparison available to HHS is not between RF-EMF safety advocates and skeptics, but between two international expert bodies that both manage radiation exposure under scientific uncertainty and reach very different institutional answers about how to do it. ICNIRP (and FCC’s approach is largely similar) for non-ionizing radiation, and the International Commission on Radiological Protection (ICRP) for ionizing radiation [69, 94].

Both institutions rely on systematic evidence review, seek to identify exposure conditions associated with harm, and aim to provide internationally harmonized guidance. They differ fundamentally in how they treat incomplete causal knowledge. ICRP guidelines were developed specifically to manage uncertainty about low-dose ionizing radiation, and rather than requiring definitive proof of harm at low doses, ICRP adopted a prudent risk-management posture that assumes some risk may exist even when the magnitude is uncertain. This was operationalized through justification, optimization, and dose limits. ICRP’s optimization is expressed with the as low as reasonably achievable (ALARA) principle [94]. The purpose of ALARA is not to eliminate uncertainty but to manage it by reducing avoidable exposure wherever reasonably practicable, without waiting for causal certainty.

FCC rules by contrast, emphasize only health effects considered scientifically established, excludes the environment from protection scope, and does not implement an ICRP-style optimization mechanism for managing residual uncertainty. ICRP further situates its protection framework with an explicit ethical structure, identifying beneficence/non-maleficence, prudence, justice and dignity as core values, along with accountability, transparency, and inclusivity as procedural values intended to sustain public trust precisely when scientific certainty is incomplete [135]. No equivalent ethical framework is readily apparent in FCC’s rules or in the process by which its scientific assessments are developed. While FCC has conducted public consultation during rules review, there are legitimate concerns about whether public and independent-scientist input was substantively engaged with, rather than simply logging concerns as a procedural formality.

This comparison should directly inform HHS’s Question 5: An ALARA-equivalent optimization principle for RF-EMF, by reducing exposure where reasonably practicable, independent of whether causal harm has been conclusively established, is both technically and ethically coherent with how the U.S. federal government manages a structurally similar uncertainty problem in ionizing radiation low-level exposures. There is no principled reason available in the science itself for treating non-ionizing and ionizing radiation asymmetrically in this respect. Asymmetry is a governance choice, not a scientific finding.

III.3 Institutional Composition, Lack of Epistemic Diversity, and the “Echo Chamber” Problem

Concerns about ICNIRP’s and the WHO International EMF Project’s trustworthiness are inseparable from concerns about who sits on these bodies. RF-EMF protection committees are heavily weighted towards physics, engineering, dosimetry, and exposure assessment, with comparatively limited representation from clinical medicine, oncology, toxicology, environmental sciences and reproductive biology [132, 134]. This is not a claim that physical-science expertise is misplaced, dosimetry obviously matters, but that a narrow disciplinary composition shapes both which questions get asked and which forms of evidence are found persuasive. Leszczynski and others have argued this contributes to an “echo chamber effect” and groupthink dynamics where dissenting opinions or alternative viewpoints may be overlooked or dismissed [136, 137]. Analyses of the broader guideline development network found substantial personnel overlap among ICNIRP, the WHO International EMF Project, and national radiation-protection authorities [138, 139], creating conditions for epistemic reinforcement, with the same interconnected group of experts contributing to guideline development, conducting major evidence reviews, and interpreting the resulting literature for policymakers. Meanwhile, more than 260 independent scientific experts in this domain who endorsed the 2015 International EMF Scientist Appeal to the UN and WHO calling for greater precaution have not had any representation within these same governance or assessment structures [115].

Mercer’s analysis of the WHO International EMF Project describes this dynamic candidly: the project has sought not only to assess the evidence but to promote global harmonization of RF-EMF standards and this harmonization mandate has shaped how uncertainty and precaution are framed in international policy discussion [133]. As Mercer put it directly:

“The mainstream ‘regulatory science’ position has been that it is unlikely EMF’s constitute a significant health risk and therefore minimal regulatory intervention or significant precautionary considerations are required.” [133]

Viewed through this lens, the marginalization of non-thermal findings is not simply a function of insufficient evidence. It is a predictable output of an evidentiary framework that prioritizes harmonization, technical continuity, and mechanistically “established” effects over precautionary interpretation of genuine biological uncertainty. Mercer further argued the WHO EMF project has used appeals to technology determinism and

bespoke definitions of “sound science” to define the boundary between legitimate scientific assessment and policy advocacy in ways that exclude competing perspectives from the policy-making arena entirely [133].

This dynamic resembles Janis’ classic account of groupthink, where highly cohesive, prestigious expert groups operating under pressure for consensus can suppress dissent, filter out inconvenient information, and develop an illusion of invulnerability [140]. Long-standing radiation protection committees have presented a consistently unified public position on RF-EMF while resisting external criticism and maintaining limited epistemic diversity in both membership and evidence review. This approach produces a self-reinforcing cycle in which only evidence compatible with the thermal paradigm is deemed “sound”, while findings that challenge it are labelled “not established”, and the absence of establishment is then cited as proof that no revision is needed. Whether or not every element of this critique is accepted in full, it has direct implications for institutional legitimacy, and HHS’s own recommendations under Question 17 should explicitly address disciplinary and viewpoint diversity on any advisory panel convened as a result of this RFI, for exactly this reason.

III.4 Vested Interest and the Political Economy of RF-EMF Governance

The wireless industry is one of the largest markets in the world, generates more than a trillion dollars in revenue, with deep structural interconnections to military and surveillance infrastructure [141]. Wireless communications underpin telecommunications networks, radar, intelligence capability, smart city platforms, and emerging digital infrastructure more broadly. This creates a dense network of financial and political incentives that has plausibly contributed to selective research funding, controlled framing of RF-EMF risk communication and the marginalization of inconvenient findings.

From a military perspective, stricter RF-EMF regulation could constrain essential communication, navigation and defense systems; the U.S. Department of Defense has itself expressed concern that reduced spectrum access could compromise national-security and military-mission effectiveness, the same issues would presumably hold true if exposure limits were lowered [142]. From a commercial perspective, 5G/6G, IoT, smart city infrastructure and “Industry 4.0” automation are projected to generate trillions of dollars in economic value by 2030 [143, 144], creating strong incentives for operators, manufacturers, and governments to prioritize deployment speed over precaution, and making stricter exposure limits, which would require significant network design, costly to implement. Acknowledging potential health risks also carries the risk of consumer backlash and vendor liability, which can itself slow public-sector initiatives in surveillance, transportation, automation, and urban planning. This dynamic has been observed in documented reluctance of regulators including the FCC and ACMA (Australia) to tighten exposure limits, attributable in part to industry lobbying and the cost of retrofitting infrastructure [145-149].

HHS does not need to establish the motives of any individual scientist or regulator. Intent to shape the science is well documented for some industry actors (Part I.3 & III.5), while for expert bodies, the documented facts support potential conflicts of interest, institutional capture and asymmetric standards, whatever the intent is behind them. It

is important that HHS recognize that RF-EMF governance cannot be treated as a purely technical question of exposure measurements and biological mechanisms; it is also a question of the economic dependence, political incentive, and public trust operating on the same evidentiary terrain, and that conventional expert driven risk assessment struggles to resolve a disagreement of this kind because the disagreement extends beyond the interpretation of evidence into questions of values and acceptable risk.

III.5 Funding Source as a Predictor of Study Outcome: The Bibliometric Evidence

The clearest, most concrete evidence available to HHS on this point is not circumstantial, it is a direct bibliometric finding. A comprehensive bibliometric analysis undertaken as part of an independent scoping review of the RF-EMF genotoxicity literature, conducted by me and co-authors, found that independent research groups predominantly reported statistically significant evidence of DNA damage following RF-EMF exposure. However, research groups that predominantly reported null findings were interconnected through recurring co-authorship networks. Where funding sources were declared, research groups reporting predominantly null findings (>70%) were more closely networked through regulatory, military, or telecommunications-linked authorship clusters, while independent research groups reported a substantially higher proportion (>70%) of significant DNA damage findings [26].

This pattern is directly consistent with prior findings that vested interest-linked funding is associated with a greater likelihood of null or no-effect conclusions [61, 150-153]. It is also, notably, a variable that the recent WHO-commissioned systematic reviews on RF-EMF did not examine. None of them tested sponsorship or researcher-affiliation as an independent determinant of study outcome through sensitivity analysis, subgroup analysis, or meta-regression, despite this being a well-established methodological check available when quantitative synthesis is feasible [8, 16, 83, 154-159].

HHS should read this finding for what it is: when funding source or institutional affiliation predicts a study's conclusion more reliably than the quality of its methodology, the scientific process supporting that conclusion has been compromised, whether or not any individual researcher intended it. This creates conditions in which research can function as product defense rather than disinterested inquiry. Studies can be designed with insensitive exposure parameters to reliably generate null results that can then be used to dilute independent positive findings, producing a manufactured doubt and inconclusiveness that mirrors in mechanism, if not in intent, the historical tactics of the tobacco industry. This dynamic has contributed to a trust crisis that was clearly visible in the 2019/20 Australian Parliamentary Inquiry into 5G, described in Part I.10 above. When the public cannot distinguish genuine scientific consensus from industry/government regulator funded reassurance, "uncertainty" itself becomes strategically useful for preserving the regulatory status quo, at direct cost to precautionary public-health protection. This is precisely the dynamic Question 5 of this RFI is asking HHS to evaluate when it asks what regulatory changes would improve the assessment and disclosure of RF-EMF exposure. Mandatory sensitivity analysis for funding source in any federally

commissioned systematic review is a concrete, low-cost, immediately implementable answer.

Observable markers of asymmetric evidentiary treatment

Parts I.4, I.5, III.3 and III.8 each describe a different instance of the same problem: evidence of non-thermal biological effects of RF-EMF is held to a different standard from evidence for other agents and from the treatment of evidence of no effect. This selection draws those instances together as a set of observable markers that HHS can test for itself in any evidence review it commissions or relies on. In my submission I distinguish three tiers of claim throughout: *documented fact* (a record anyone can check), *reasonable inference* (what the documented facts support), and my own *judgement* (stated as such). The markers below are all in the first tier.

Table 4 Markers of asymmetric evidentiary treatment, with where each is documented in this submission

Marker	What a reviewer can check	Where documented
1. A higher bar for RF-EMF than for other agents	Whether inclusion criteria, assay acceptance, consistency and monotonic dose-response requirements applied to RF-EMF are the same as those applied by the same body to other agents. For example, the WHO-commissioned systematic review on RF-EMF and oxidative stress used a narrower frequency range than its own protocol, selected <15% of the available research, was looking for a monotonic dose-response, excluded epidemiology, gene expression studies related to oxidative stress, and discounted standard oxidative stress assays, even though oxidative stress is a recognized key characteristic of carcinogens.	I.5 [83, 84]
2. Funding source or affiliation predicts conclusions	Whether study outcome varies with sponsor or author affiliation, and whether the review tested for it. None of the WHO-commissioned reviews did.	III.5 [8, 16, 26, 83, 152, 154-159]
3. Uncertainty used strategically	Whether “further research is needed” is applied selectively, and whether evidentiary rules are changed in ways that restrict admissibility of expert evidence. Documented industry intent: A 1994 internal Motorola memo (below).	I.3; III.5; below [60, 160]
4. Psychological or nocebo attribution as the default	Whether a psychological explanation is acceptable on positive evidence, or by exclusion when objectives tests are negative. Precedents AHIs and ME/CFS.	III.8
5. Null findings treated as expected, positive findings as flawed	Whether the same methodological scrutiny is applied to both positive and null findings. Authors who critiqued the NTP and Ramazzini studies were reported not to have criticized any negative studies; the pivotal pair of positive studies were also omitted from the 2020 World Cancer Report despite being flagged as studies of interest in the 2014 World Cancer Report.	I.2 [8]; I.4 [64, 67]
6. Overlapping authorship and affiliation in the evidence chain	Whether the same small group writes the guidelines, conducts the reviews and critiques dissenting work. Declared links between reviewers, ICNIRP, industry, military, and the regulators they advise.	I.2; I.4 [67, 68] ; III.3 [138, 139]
7. Critique directed at the researcher rather than the data	Whether responses to unwelcome findings address the data and methods, or the competence, motives or standing of the authors.	I.2 [8, 20, 22, 101, 161]; III.3

Documented industry intent. At least for some industry actors, intent to shape the science is on the record rather than inferred. As discussed in I.3, in 1994 a Motorola spokesman wrote an internal memo to a public-relations firm discussing how the company had “war-gamed” the findings of Lai and Singh, who reported DNA strand breaks in rat brain cells after microwave exposure [162]. The memo, first reported by Microwave News and confirmed to exist by Motorola, described preparing responses to

press inquiries, lining up outside experts to point out weaknesses in the study, and coordination between the company, the industry trade association (CTIA) and an industry-funded research program [60]. The memo does not show that Lai and Singh's findings were wrong, and some of the points it proposed to make (for example that the study used a different frequency from cellular telephone) were scientifically legitimate at the time. Its significance is that it documents a coordinated communications strategy to manage genotoxic findings, in the research area on which Part I.5 and this submission's mechanistic argument depend.

What the markers do and do not show. Any one marker may have innocent explanations: such as sincere scientific disagreement, differences in training and resource constraints. My point is that they co-occur, that they consistently favor one conclusion, and that for several of them the direction of asymmetry is the same across bodies that share personnel. For regulators and expert bodies, the documented facts support conflicts of interest, groupthink and asymmetric evidence standards [100, 114, 136, 137, 149, 163]. Whether those patterns reflect conscious intent, motivated reasoning or structural incentives is a question that does not need HHS to resolve, intent is already documented in this submission and identifies industry, military and regulatory actors, The remedy (the need for independent research) is the same in each case.

A symmetry test, can be applied to this submission too. The same markers can and should be applied to studies that report effects. Several studies relied on this submission have limitations that I have has stated openly, including exposure misclassification, self-selected, unadjusted analyses and recall bias in case-control designs (Part I.10). A review that scrutinizes positive and null findings by the same pre-specified criteria is the standard I asks HHS to adopt.

What would change my position. I would consider revising the conclusion of this section if (i) funding and affiliation sensitivity analyses of the existing reviews showed no association with outcome; (ii) If a more inclusive systematic review investigating RF-EMF and oxidative stress that encompassed all the available and relevant studies, produced the same conclusions as the recent WHO-commissioned review; or (iii) independent replication of the NTP and Ramazzini bioassays, with comparable power and exposure, failed to reproduce the tumor findings. These are tests HHS/NIH is able to run.

Note: I do not consider the recent "NTP Lite" studies conducted in Japan and Korea to be an equivalent replication due to design and methodological deficiencies [164-166]. It is also instructive to consider the affiliation of the NTP Lite designers, authors and funding sources for these recent works.

III.6 RF-EMF as a "Wicked Problem" and the Case for Post Normal Science

RF-EMF exposure meets the definition of a wicked problem: a rapidly expanding, deeply embed technology intersecting with unresolved questions about long-term health and environment consequences, where technology cannot realistically be eliminated or substituted, and where technological, economic, infrastructural, and public-health interests are in genuine tension [3]. Establishing harm with the certainty regulators and

industry demand can take decades of epidemiological follow-up along with focused experimental studies, but ignoring early warning signs carries its own serious risk if those signals are eventually substantiated, a pattern the historical record on tobacco and asbestos demonstrates repeatedly (Part V below covers this in more detail).

From a Post Normal Science (PNS) perspective, this is a policy domain in which facts remain uncertain, values are contested, stakes are high, and decisions cannot be indefinitely postponed pending definitive evidence [167, 168]:

“The scientific system now faces a crisis of confidence, of legitimacy and ultimately of power. The ‘mainstream’ is reductionist in style, and increasingly linked to industry. By contrast, the ‘post-normal’ approach embodies the precautionary principle. It depends on public debate, and involves an essential role for the ‘extended peer community.’” Ravetz [167, page 347]

Applied to RF-EMF, this means exposure standards cannot be treated as purely technical products of dosimetry. This is because they encode judgements about what counts as harm, which evidence is weighted most heavily, what degrees of uncertainty are acceptable, and how risk is balanced against economic and social benefit. These judgements are not value-neutral even when a body such as ICNIRP frames its approach as purely rigorous, evidence-based science aimed at avoiding “speculative or unproven risks” [69, page 484]. PNS’s central prescription is about moving beyond closed expert models toward meaningful incorporation of an “extended peer community”, by including clinicians, toxicologists, affected communities, and independent researchers, into evidence evaluation and policy formulation. This is directly responsive to what HHS is doing by issuing this RFI in the first place, and this submission urges HHS to treat that open, extended participation model as the template for how structures the advisory process that follows this RFI, not merely as a one-time public comment exercise that reverts afterward to the same closed expert bodies critiqued above.

III.7 Response to Question 5(p): Classification Systems and Exposure-Related Characteristics

Question 5(p) asks how a classification system should account for operating frequency, power density, SAR, field strength, modulation, duty cycle, beamforming, simultaneous multi-frequency exposure, exposure duration distance from source, body proximity, and cumulative exposure over time. The evidence set out in Part I.3 above is directly responsive: any classification system built only around time-averaged SAR or instantaneous peak SAR from a single stationary source can systematically mischaracterize real-world exposure, because (i) duration and temporal pattern of exposure, not only intensity, are important determinants of biological outcomes; (ii) device design changes, antenna relocation, beamforming, and multi-band simultaneous transmission have altered the anatomical distribution temporal characteristics of exposure in ways that conventional epidemiological research does not adequately track; and (iii) cumulative, simultaneous multi-source exposure from concurrent 4G/5G/6G, Wi-Fi, Bluetooth, and IoT connections is poorly represented in both conventional exposure assessments and most experimental research.

Current ICNIRP and FCC approaches necessarily simplify this multidimensional exposure environment into a limited number of measurable quantities and defined exposure categories. This simplification has considerable regulatory value because it facilitates standardization, measurement, compliance testing, enforcement and consistent application across large numbers of devices and exposure scenarios. However, regulatory tractability should not be confused with biological completeness. The more complex the exposure environment becomes, the greater the potential for a simplified classification system to lose information relevant to biological response(s) and health.

This issue is particularly important because the available evidence does not consistently support an assumption that biological response is determined by exposure intensity alone or that the relationship between exposure intensity and biological response is necessarily monotonic. The RF-EMF genotoxicity scoping review and evidence map discussed in this response found evidence across experimental systems that reported DNA damage and oxidative stress may depend on exposure duration, temporal exposure pattern and signal modulation characteristics, and that these factors could be more important determinants of reported effects, especially for exposure levels below those associated with established thermal responses [26].

This is consistent with the broader evidence for non-linear and non-monotonic biological responses to environmental exposures. A classification system that assumes a simple monotonic relationship, that is higher SAR or power density equals greater biological effect, may therefore be appropriate for the specific thermal endpoints for which current limits were developed, but it is not necessarily sufficient for characterizing biological responses to chronic low-level persistent exposures.

The parameters identified in Question 5(p) are also unlikely to operate as independent variables. Frequency, modulation, waveform, duty cycle, peak and average power, beamforming, source distance, body orientation, anatomical location, simultaneous sources and exposure duration are likely to interact. Consequently, it would be unrealistic to assume that a single formula could assign biological significance to each parameter independently or combine them into a universal cumulative "dose". Nor should a new classification system assume in advance that biological response is proportional to total absorbed energy or exposure duration.

Moreover, RF-EMF biological effects that occur through mechanisms that are not adequately described by a simple intensity-dependent thermal model, the appropriate exposure-response relationship may be probabilistic rather than deterministic. This raises the possibility that stochastic or other non-monotonic processes may contribute to biological responses at lower exposure levels and therefore requires an approach analogous in principle to the treatment of stochastic effects in low-dose ionizing-radiation risk assessment by implementing justification and optimization through ALARA.

The appropriate objective should instead be to develop a more informative exposure-characterization system capable of retaining the multidimensional nature of real-world exposure while remaining sufficiently practical for regulatory use. Conventional quantities such as SAR, power density and field strength should remain important

measures of exposure magnitude and thermal compliance, but they should not be treated as complete descriptors of exposure or as universal surrogates for RF-EMF exposure induced biological effects.

Anatomical distribution is one example of information that can be lost through excessive simplification. Current approaches commonly distinguish exposure using broad categories such as whole-body, head/torso or limbs. Such categories may be useful for standardized compliance testing, but provide limited representation of many contemporary exposure scenarios. A mobile device carried directly against the chest, for example, produces a substantially different spatial exposure pattern from the same device held adjacent to the head. Within the torso itself, tissues may be separated from the transmitting antenna by only centimeters or less, and the distribution of absorbed energy can vary with device orientation, antenna location, operating mode and tissue type. A classification system that assigns such situations merely to a broad body or torso category can therefore conceal potentially important differences in anatomical exposure.

The same problem arises with dynamic network technologies. Beamforming, adaptive power control, multiple simultaneous connections and operation across multiple frequency bands mean that exposure is increasingly variable in time, frequency and spatial distribution. A measurement made at one location, frequency or instant may therefore provide only a partial representation of an individual's exposure history.

Accordingly, HHS should avoid simply replacing the current system with another fixed list of independent exposure variables or a single composite dose metric. Instead, the classification system should adopt a tiered, multidimensional exposure-characterization approach. Routine compliance assessment could continue to use established metrics such as SAR, power density and field strength. However, device-specific laboratory testing provides an opportunity for substantially more detailed characterization. For example, computational electromagnetic modelling using tools such as CST Studio Suite or equivalent platforms, potentially incorporating AI-assisted modelling and optimization, can be used to examine how antenna configuration, operating frequency, modulation, device orientation, proximity to the body and anatomical geometry influence the spatial distribution of absorbed energy under defined operating conditions. Such modelling could provide substantially richer exposure profiles for handsets and other RF-emitting devices than a single reported SAR value.

These device-level exposure profiles could then inform experimental research and epidemiological exposure assessment by providing more realistic exposure surrogates, without requiring every individual participant or real-world exposure environment to be characterized at the same level of detail. This would provide a practical means of improving exposure classification while recognizing the inherent limitations of attempting to measure the full multidimensional exposure environment in everyday life.

Most importantly, the classification system should distinguish between exposure magnitude and exposure pattern. Where the evidence indicates that duration, temporal pattern and signal characteristics can modify biological response, particularly for reported non-thermal effects, these characteristics should not be treated merely as

secondary adjustments to an intensity-based metric. They should be retained as explicit dimensions of exposure classification. This would allow the system to accommodate both monotonic exposure-response relationships, where they are supported, and non-monotonic or otherwise non-linear relationships where the evidence indicates that biological response cannot be adequately predicted from intensity alone.

III.8 Response to Question 11: Potentially Sensitive Populations

Short Answer. Yes. Exposure assessment and risk evaluation should differ for sensitive populations, and the case for this is broader than the categories listed in Question 11 alone would suggest. Current limits are derived for a general population on the premise that one threshold protects everyone within it. The evidence below indicates that susceptibility to RF-EMF varies between individuals, that part of the variation has an identifiable biological basis, and that a defined group of people report disabling symptoms at exposure levels the general-population model treats as safe. The same symptom cluster, and the way it has been explained away, also appears in a recent officially investigated setting (anomalous health incidents) discussed below. Although my doctoral research does include a population-stratified ecological study investigating mobile phone usage and thyroid cancer, it contains no dedicated cohort data on pregnant women or people with implanted medical devices (see the responses to Question 11(a) – (e) below. However, where my research does contribute, lies mainly in parts (f) and (g): Individuals with pre-existing biological susceptibility, and individuals who report electromagnetic hypersensitivity (EHS) or microwave sickness symptoms as defined by Merriam-Webster’s medical dictionary [169]. Some quotes from individuals who responded to the Australian Parliamentary Inquiry into 5G provide important context.

I have been medically diagnosed as being EHS (electrohypersensitive) ... My life is continually being impacted by wireless radiation from smart meters, Wi-Fi, mobile phones, etc. My accessibility to public places is limited, as I simply cannot tolerate being near these devices. I have had to give up work as a teacher because of my sensitivity to Wi-Fi in the classroom. My symptoms include severe head pressure, blurred vision, burning sensation on my skin, muscle aches and pains, insomnia, cognitive disturbances and tinnitus. (Submission 393)

and

During my service in the military I was exposed to high levels of EMR from military communications equipment (electronic, wireless and electrical) over long-term and prolonged periods of time ... The EMR exposure had a bearing on my health during my military service and since 1980 and over time it materialised into significant physical health problems suffering ME, CFS, FMS¹; and now chronically and severely debilitated with peripheral neuropathy and distal myopathy and neurasthenic symptoms ... Today, these illnesses are coined “electrohypersensitivity (EHS)”. (Submission 168)

¹ Myalgic encephalomyelitis (ME), chronic fatigue syndrome (CFS), and fibromyalgia (FMS) are interconnected, complex chronic illnesses characterized by disabling fatigue, widespread pain, and post-exertional malaise.

These accounts are consistent with a broader body of evidence suggesting that individual susceptibility and reported health effects warrant consideration in RF-EMF exposure assessment and risk evaluation.

Basis and framework relied upon. This response draws on peer-reviewed research including: Luo et al. 2020 [170], two smart-meter symptom datasets [4, 5], the public submissions to the 2019-2020 Australian Parliamentary Inquiry into 5G [1, 3], NIH studies and related official and press reporting on anomalous health incidents, and ICNIRP's own framework documents, particularly its 2002 general approach to protection against non-ionizing radiation, its 2020 principle differences statement, and its 2020 RF-EMF Guidelines [69, 171-173]. The ICNIRP framework, and the FCC rules that are similar in intent, is the framework this response tests, which currently assumes that a limit set for the general population protects everyone, irrespective of individual susceptibility.

ICNIRP has acknowledged in the past that a general population limit may not protect everyone. ICNIRP's 2002 general approach statement recognized that groups within a population differ in their tolerance of exposure and named children, the elderly and chronically ill people as groups who might not tolerate one or more forms of non-ionizing radiation less well, and accepted that separate guideline levels might be necessary or useful. It also said that some guidelines "may still not provide adequate protection for certain sensitive individuals" and that, where such situations are identified, specific advice should be developed [171]. ICNIRP's more recent position differs in emphasis, where the 2020 principles statement hold that people with medical conditions are now part of the general public and are protected by the guidelines, and its explanatory material describes the 2020 Guidelines as protecting against adverse health effects regardless of age or health status [172, 173].

Three features of this shift matter for HHS. First, the example ICNIRP gives of more sensitive groups is reduced thermoregulatory capacity in young children and the elderly [173], and the reduction factors it applies to derive restrictions accommodate biological variability [69]. However, these factors are applied to thresholds for the effects ICNIRP recognized, such as heating and nerve stimulation (Part III.1). Variability in susceptibility to non-thermal effects are outside what the framework was built to register. Second, I am unaware of any evidence published between 2002 and 2020 establishing that susceptibility to RF-EMF is uniform across individuals. Third, ICNIRP's own principles statement acknowledges that its guidelines are not intended to protect individuals with certain clinically conditions that increase susceptibility, exemplified by the heritable DNA-repair disorder xeroderma pigmentosum in the context of UV radiation. Although this example concerns UV rather than RF-EMF, it illustrates a broader limitation of population-level protection: individuals with particular biological susceptibilities may fall outside the protection the guidelines are designed to provide [173]. There are two questions for HHS: Firstly, whether such susceptibility is confined to rare, clinically diagnosed disorders or extends to common genetic variation? The evidence that follows suggest the latter. Second, what ethical obligations arise when population-level exposure guidelines are applied to a population whose members may differ in their biological susceptibility, particularly when exposure is widespread,

individual consent is limited, and the framework explicitly acknowledges that certain genetically susceptible individuals fall outside its intended protection?

Genetic Susceptibility: Luo et al. (2020). A population-based case-control study from the Yale School of Public Health/School of Medicine research group, the first of its type to examine a gene-environment interaction for cellphone use and thyroid cancer, provides direct evidence that susceptibility is not uniform [170]. The study conducted in Connecticut in 2010-2011, included 440 thyroid cancer cases and 465 population controls with genotyping for 823 single nucleotide polymorphisms (SNPs) in 176 genes, and tested each gene for interaction with cell phone use. Ten SNPs showed interactions at $P < 0.01$. Among people with the common genotype, cell phone use was not associated with thyroid cancer. Among the carriers of the variant genotype it was, for six SNPs, with odds ratios ranging from 1.59 to 2.64 (for example, MDM2 rs1695147: OR 2.52, 95% CI 1.30 – 4.90; PAK6 rs11070256: OR 2.36, 95% CI 1.30-4.30), and the authors reported that the association strengthened with increasing duration and frequency of use [170]. Although the identified polymorphisms occurred within different genes, they converge on a remarkably consistent set of biological pathways involved in cellular stress responses, including oxidative stress regulation, DNA damage signaling, apoptosis, epigenetic regulation and cellular proliferation.

Central to this response is the p53 tumor suppressor pathway, regulated in part by MDM2, which coordinates cellular decisions between DNA repair, cell-cycle arrest, and apoptosis following genotoxic stress [174, 175]. Genetic variations affecting this axis may therefore modify cellular sensitivity to DNA damage [170]. In parallel, epigenetic regulators such as HDAC4 influence transcriptional responses through chromatin remodeling and have been implicated in cellular adaptation to oxidative stress [176, 177]. The mechanistic literature reviewed in part I.5 identifies oxidative stress and oxidative DNA base damage as the leading candidate pathway for RF-EMF genotoxic effects [26, 34, 76, 77, 79, 80]; an individual capacity to repair damage or respond to cellular stress that differs between people is precisely the variable that would be expected to determine who is harmed by a given exposure.

The regulatory significance lies in the structure of the results rather than any single odds ratio. In a population that mixes carriers of the SNPs and those who don't, an average effect is diluted. With the Luo et al. study, the association sits primarily in the variant-carrying group. An exposure limit set on the average response of a general population, and defended by the absence of an average effect, would not protect the subgroup in which the effect occurs. This is one study that establishes what matters most for Question 11 and more are needed. Gene-environment interaction for RF-EMF can be tested in existing epidemiological designs, with the first test producing a positive signal, and therefore the general population model cannot be assumed adequate until more studies are done.

It is also worth considering the discussion of technology changes in I.3, because the authors of the Connecticut study noted that antenna's had only recently moved to the bottom of the smartphone (in the final year of the study), resulting in peak exposure more likely to fall on the neck where the thyroid is located rather than the brain [170]. Because this design change occurred so late in the study window, and given the multi-year latency typically associated with radiation-related thyroid carcinogenesis, any

contribution of the new antenna location to the observed associations would have had limited time to manifest (i.e., the bulk of exposure for most participants still reflected earlier device designs). This warrants further investigation by HHS, given my own research findings of significant increases in thyroid cancer incidence in Australia and internationally in the last decade, particularly in the young adult cohort [91].

People who report EHS: An analysis shows convergent symptom evidence. EHS is predominantly a self-reported condition in which people attribute their symptoms to EMF exposure. The WHO does not recognize it as a medical diagnosis but acknowledges that the symptoms are real and can be disabling [178], ARPANSA likewise acknowledges the symptoms and advises affected people to seek medical advice [179]. My analysis of submissions to the Australian Parliamentary Inquiry on 5G [1] found that 115 people referred to EHS in their public submissions, of which 68 described themselves or a family member as having EHS; a further 11 described symptoms commonly associated with EHS without using the label. Table 5 compares the symptoms most often reported in the Parliamentary Inquiry submissions with two smart-meter EHS symptom datasets, 1) a case series of 92 Victorian residents compiled from a public register of self-reports [4], and 2) a survey in which 318 respondents from the United States [5], who reported new or worsened symptoms after wireless “smart” utility meters were installed. I have normalized the symptom categories across the three datasets utilizing scientific judgement.

Table 5 Symptoms most frequently reported in three datasets

Symptom	5G Inquiry (2019/20) % of 115/531	Rank	Lamech (2014) % of 92	Rank	Halteman (2011) % of 318	Rank
Sleep Problems	84 (73%/15.8%)	1	47 (51%)	1	48%	1
Headaches	81 (70%/15.3%)	2	44 (48%)	2	40%	2
Concentration difficulty	62 (54%/11.7%)	3	29 (32%)	5	34%	4
Fatigue	61 (53%/11.5%)	4	31 (34%)	4	33%	5
Dizziness	44 (38%/8.3%)	5	20 (22%)	6	32%	6
Nervous system/ sensory effects	35 (30%/6.6%)	6	18 (20%)	7	17%	7
Nausea	34 (29.5%/6.4%)	=7	15 (16%)	8	16%	8
Tinnitus	34 (29.5%/6.4%)	=7	32 (35%)	3	37%	3
Dermal effects	25 (22%/4.7%)	9	7 (8%)	9	12%	9

Notes: Inquiry percentages as shown against 115 who noted symptoms and also all 531 submissions respectively; the tie between nausea and tinnitus is shown as =7. Lamech counts (n=92) have been converted to percentages; Halteman values were provided as percentages of 318 submissions.

The three datasets come from different countries and periods (2011, 2014 and 2019-2020), different exposure contexts and different method of ascertainment, yet they converge. Sleep problems and headaches rank first and second in all three, and dermal effects ranks last in all three. Rank agreement between the inquiry and each smart meter dataset is high (Spearman $\rho \approx 0.78 - 0.8$), and between the two smart meter datasets is very high (Spearman $\rho \approx 0.98$). The main discordance is Tinnitus, ranked third in both smart meter datasets and ranked eighth in the 5G inquiry. Excluding it, rank agreement across all three becomes $\rho \geq 0.98$. The reason for the tinnitus difference cannot be determined from these data. Difference in ascertainment (free-text submissions to the parliamentary inquiry, register entries and a survey questionnaire)

are one candidate. These correlations are descriptive only, there are nine categories from samples that are not part of any defined population sample and rank agreement says nothing about causation.

However, the same cluster identified in these surveys and submissions is not new. Merriam-Webster's medical dictionary defines microwave sickness, reported in early soviet medical literature, as headaches, anxiety, sleep disturbance, fatigue and difficulty concentrating along with cardiovascular and central nervous system changes, held to be cause by prolong low-intensity microwave exposure [169]. Government-sponsored literature from the 1970's and 1980's record overlapping subjective complaints among exposed workers, including headache, fatigue, dizziness, sleep disturbance and difficulty concentrating with a 1972 U.S. Navy bibliography [125], a 1976 US Defense Intelligence Agency review of Eurasian literature [127], and a 1981 NASA review [126] providing corroborating evidence. Table 20 of the NASA review, for exposure at 750kHz-200MHz, shows headache, sleep disturbance and fatigue reported more often by workers with 7 - 16 years of employment than by workers with 1 - 6 years (headache 32.9% vs 20.5%; sleep disturbance 23.3% verses 13.7%l fatigue 17.8% verse 12.3%; with 73 workers in each group), and neurological symptoms appearing only in the longer employed group (15.1% vs 0%) [126]. This is a duration pattern in an occupational population, of the kind I.3 argues should be examined, and it bears on Question 11(e).

A similar cluster has also recently been described in an officially investigated setting. Since 2016, U.S. government personnel and family members have reported anomalous health incidents (AHI), popularly called Havana syndrome" which has been characterized by abrupt perceived sound or head pressure, followed by headaches, dizziness, cognitive difficulty and other symptoms [180]. I do not claim that RF exposure has been confirmed to be the cause of AHIs, or that any foreign actor is responsible; both questions remained unresolved in the public forum and are outside the scope of this submission. However, the case is relevant to Question 11 for three reasons: its symptoms overlap with the EHS and microwave sickness literature (Table 6) the way in which a psychological explanation has been used as the default where no mechanism has been demonstrated, and what it suggests about how pulsed exposures are handled in current FCC rules and ICNIRP guidelines. The second and third are taken up after the discussion of provocation studies below.

Symptom overlap.

Table 6 compares the nine symptom categories in Table 5 with the microwave-sickness definition already cited [169] and with two published summaries of AHI symptoms [181, 182].

Table 6 Symptom categories of Table 5 compared with microwave sickness and AHI descriptions

Symptom	EHS-related datasets	Microwave sickness (Merriam-Webster) [169]	AHI: NIH release [181]	AHI: published symptom summary [182]
Sleep problems	Rank 1 in all three	Sleep disturbance	Not listed	Insomnia
Headaches	Rank 2 in all three	Headaches	Headache	Headaches
Concentration difficulty	Ranks 3–5	Difficulty concentrating	Cognitive dysfunction	Difficulty concentrating, memory loss, confusion
Fatigue	Ranks 4–5	Fatigue	Self-reported fatigue	Fatigue
Dizziness	Rank 5–6	Not listed	Dizziness	Dizziness, trouble walking
Nervous system / sensory effects	Rank 6–7	Central nervous system changes	Head pressure, perceived sound	Sound sensitivity, ear pain and pressure
Nausea	Rank 7–8	Not listed	Not listed	Nausea
Tinnitus	Rank 3 in both smart-meter datasets	Not listed	Perceived sound (not specified as tinnitus)	Tinnitus
Dermal effects	Rank 9 in all three	Not listed	Not listed	Not listed

Notes: The AHI columns are lay or news summaries (the second is a reprint of a general audience article that draws its list from journal papers) and the mapping involves judgement. Eight of the nine categories appear in at least one AHI description, although dermal effects do not. These symptoms are non-specific and common to concussion and other conditions; however, overlaps show that a similar symptom cluster is being described in three contexts.

What this evidence does and does not show. All three symptom datasets are self-reported and self-selected, none measure individual exposure, and none can exclude an expectation (nocebo) contribution; nor do they confirm that RF-EMF caused the reported symptoms. What they show is narrower and, for Question 11, sufficient: a definable group of people, in several countries and across six decades of literature, reports of a cluster of reproducible symptoms in association with RF-EMF emitting equipment; and a similar cluster has been reported in a recent official investigation whose cause remains unexplained; the cluster matches occupation descriptions that predate the current public debate about wireless harm; and current exposure protection frameworks contain no mechanism to evaluate it. The Lamech case-series adds that the vast majority of its cases did not describe themselves as EHS sufferers before wireless smart meter were installed, which the study author takes to point to the possibility that smart meter emissions have characteristics that lower the threshold for symptom development [4]. Sensitivity may therefore be acquired as well as inherited, which strengthens the case for treating it as a population category that HHS must assess rather than a fixed trait of a few individuals.

Why laboratory provocations studies do not resolve the question. ARPANSA, citing the WHO, told the Australian Parliamentary Inquiry on 5G that EHS has no clear diagnostic criteria, that science has not shown EMF exposure to be the cause, and that under controlled laboratory conditions people with EHS cannot detect RF sources more accurately than others (Submission 317). The WHO-commissioned systematic review of human experimental studies likewise reported no significant effect of RF-EMF on headaches, sleep disturbance or composite symptoms across 41 studies, and no reliable perception of exposure [155]; the companion review of observational studies rated the certainty of its evidence on tinnitus, migraine and non-specific symptoms as low [159]. My personal view is that these reviews answer a narrower question than the

one Question 11 asks. Failing to detect and RF source under short-term blinded conditions is not the same as being unable to develop symptoms, and people reporting EHS commonly describe symptoms that are delayed in onset and slow to resolve, which most brief provocation designs are not built to capture [183]. The absence of agreed diagnostic criteria [183] also makes it difficult to define who should be tested. As Part III.5 notes, none of the WHO-commissioned reviews tested whether sponsorship or affiliation predicts study outcome. Many provocation studies showing null findings received industry or workers compensation funding or both.

The reasoning applied to provocation studies, that negative subjective test results show the absence of an exposure-related illness has led some researchers to attribute symptom development to a placebo effect. A close parallel in the handling of anomalous health incidents (AHI), including those associated with Havana Syndrome, has been observed.

What the NIH studies found, and what they did not for AHI. NIH clinical researchers evaluated more than 80 affected personnel and adult family members against matched health controls, including controls with similar work assignments. An MRI was performed on average 80 days after symptom onset (and as early as 14 days). The team found no consistent set of imaging abnormalities and no differences in most clinical and biomarker measures. Affected participants did self-report more fatigue, post-traumatic stress and depression, and 41% met criteria of a functional neurological disorder (FND) or had significant somatic symptoms, most often persistent postural-perceptual dizziness (PPPD) [181, 184, 185]. The NIH investigators' own conclusions were more guarded than the headline. They stated that the symptoms were very real, that an absence of MRI-detectable differences does not exclude an adverse event affecting the brain at the time of the incident, and that markers of an external cause may no longer be detectable or may not be identifiable with current methods and sample size [181]. An earlier study of 40 affected personnel reported differences in brain volume and structure compared with controls, though its authors said their meaning was unclear [186]. The two studies differ in design and timing and are not concordant, and neither is conclusive.

How the findings are being used. One school of thought treats AHIs as a psychogenic or socially constructed condition ("shell shock" – type stress reactions, mass psychogenic illness) [182, 187]. Against this, a 2020 National Academies committee judged directed, pulsed RF energy the most plausible mechanism for a subset of cases with distinctive acute features [180], and a 2022 intelligence community expert panel likewise found pulsed electromagnetic energy a plausible explanation for some cases [188]. The author of this submission does not adjudicate between these. The point is structural. Where no "acceptable" mechanism or biomarker has been demonstrated, a psychological attribution has been adopted by exclusion rather than on positive evidence, and the NIH finding that 41% met FND or somatic symptom criteria has been cited in support. However, that inference does not follow. FND and PPPD are diagnosed as positive clinical features and do not identify what triggered them; the PPPD consensus criteria themselves list a physical precipitant (vestibular, neurological and other medical event) among the possible triggers alongside psychological distress, and that it is not a psychiatric condition [189]. Meeting criteria that is therefore compatible

with a physical initiating event. Expectation (nocebo) effects may contribute in some individuals, as they may in any illness. But a nocebo explanation is itself a causal hypothesis, and requires evidence that symptoms track belief rather than exposure. It should not be assumed because exposure has not been measured.

The ME/CFS precedent. A comparable pattern of denial is documented for myalgic encephalomyelitis/chronic fatigue syndrome (ME/CFS). For decades the condition was approached as predominantly psychological and behavioral, an approach reinforced by the PACE trial of cognitive behavioral therapy (CBT) and graded exercise [190]. Independent re-analyses of the trial data challenged the reported recovery rates [191]. In 2021, the UK's NICE guideline withdrew its recommendation of graded exercise and stated that cognitive behavior therapy should not be offered as a cure[192], and a 2015 Institute of Medicine report had already characterized ME/CFS as a serious, chronic, systemic disease [193]. Patients described as exhibiting fear-driven or expectation-driven were, on this record, describing a physically illness that the available tools had not characterized. The same question has been raised for Long COVID, where some researchers suggested a somatic symptom disorder that is compatible with a nocebo effect [194]; however, a population-based cohort analysis found no evidence that confirmation of infection status acted as a nocebo [195]. The lesson for EHS is not that every attribution to expectation is wrong. It is that a nocebo or psychogenic label has repeatedly been applied to symptom clusters before a mechanism was known and has been difficult to dislodge once applied. This misattribution has had real consequences for patient care and research. This is another area where the “asymmetric evidentiary standards” problem as described in Parts I.5 and III.5 exists.

The Norwegian self-experiment report. On 14 February 2026 the Washington Post reported, citing four people familiar with the matter, that in 2024 a Norwegian government scientist who doubted that pulsed-energy devices could harm the human brain built a pulsed microwave device based on what is believed to be classified information and tested it on himself, and developed neurological symptoms similar to those of AHIs [196]. I am not aware of whether the exposure parameters (peak and average power, pulse width and repetition rate, distance, duration) or the clinical findings have since been published. It is unlikely the scientist would have exposed themselves to a device operating at thermal levels; however, I acknowledge this consideration is speculative. Given the limited information available, there are three possible readings. If the exposure exceeded the limits, the report is consistent with the existing protection framework but raises the question of whether RF-EMF exposure can produce AHI/EHS like symptoms. If it complied with time-averaged limits but not the intent of the pulse provisions, it shows that duty cycle and modulation (Part III.7, and HHS Question 5(p)) are not adequately captured and handled. If it complied with all limits, it would directly challenge current FCC rules and ICNIRP guideline adequacy. HHS should be in a position to find out which, and the answer is material to its review.

Institutional handling of the AHI investigations. Reporting and commentary on the AHI investigations describe several features that parallel those documented in Parts I.4, II.3, and III.5. The 2022 intelligence-community assessment was issued before the expert panel had completed its own report, and the panel's chair has been reported as saying the agencies largely disregarded the panel's work [197]. A House Intelligence

Committee subcommittee's interim finding in December 2024 reportedly concluded that the initial assessment lacked analytical integrity and was highly irregular, and that the intelligence community's response had likely impeded the collection of evidence [197]. In the 2025 update, two agencies revised their earlier position on whether a foreign actor possesses a capability that could produce some of the symptoms, while five maintained that foreign involvement was very unlikely [196, 197]. A former Defense Intelligence Agency investigator has said that the bar for proof was set to high, an all too familiar story when it comes to the treatment of RF-EMF evidence and potential harm [197] (Part III.5). The relevant observations for HHS are that independent scientists lacked access to the evidence, that the conclusions preceded the expert review, that disagreement within and between agencies are publicly visible, and that an unusually high standard of proof was applied to a possible exposure-effect link.

What the AHI precedent does and does not establish. It does not establish that AHIs are caused by RF-EMF exposure, that anyone was targeted by a foreign agency, or that continuous low-level exposure from base stations, Wi-Fi or handsets are harmful. The reported Norwegian device was a microwave pulsed source, and nothing in this precedent should be read across directly to the exposures discussed in Part I.10 due to a lack of concrete information. What it does show is the following: First, official bodies have themselves judged RF energy as a plausible cause for neurological symptoms. Second, a psychological attribution has been used as a default, similar to EHS, although NIH investigators themselves said negative tests do not exclude an event. Third its symptoms overlap with microwave sickness and EHS (see Table 6). HHS should therefore not treat an absence of detectable biomarkers, or a negative blinded provocation test, as evidence that an exposure-related illness is absent. The ethical dimension should also be recognized. If pulsed RF-EMF exposure is capable of producing lasting neurological symptoms, then patients who were told their symptoms are psychogenic, and the care and research decisions that followed warrant review. Congress has already directed compensation to AHI-affected personnel, which implies an acceptance that the harm is real, whatever its cause.

Recognition and institutional handling of RF-EMF associated subjective symptoms. The AHI precedent has a regulatory counterpart in the handling of EHS. Although some of this evidence is drawn from an Australian inquiry, it is directly relevant to the HHS. In my own assessment, the FCC's recognition and handling of EHS is similar to ARPANSA's, with the difference being that the FCC has limited in-house radiation-protection science expertise. The Australian parliamentary inquiry record illustrates the consequence. ARPANSA acknowledges that the symptoms are real and advised affected people to seek advice from a medical practitioner [179], yet it has no mechanism to investigate individual reports, and medical practitioners in turn look to ARPANSA for guidance, thereby creating a gap and a circular problem. The 5G Inquiry Committee accepted that the reported symptoms are genuine but that EHS is not a recognized diagnosis [2]. What this means is affected people are validated in principle and offered nothing in practice. Approaches elsewhere differ: submissions to the 5G Inquiry noted that Sweden treats EHS as a functional impairment (Submission 393), and the EUROPAEM guideline [116] sets out an approach to the prevention, diagnosis and treatment of EMF-related health problems. Notably an Australian tribunal proceeding has also involved expert testimony from a physician who treats patients reporting EHS

[198]. HHS does not need to resolve the causation debate to act on this: people can be harmed by an unaddressed environmental sensitivity whether or not its mechanism is yet understood.

A working hypothesis. I would like to offer HHS the following as a hypothesis, not as an established finding. The genetic evidence and the EHS evidence above may describe the same underlying axis: individual capacity to buffer cellular and oxidative stress. That capacity varies with genetics, age, health status, diet, sleep and other environmental exposures, with RF-EMF reported to act as both a cellular and oxidative stressor at non-thermal exposure levels (Part I.5). On this hypothesis, Luo et al. (2020) describe a genetic component of susceptibility, and EHS describes a group in whom buffering capacity may be exceeded or is reduced. The hypothesis is testable: it predicts differences in oxidative stress and DNA-damage biomarkers, and in repair-gene variants, between people who do and do not report symptoms under matched exposure (as suggested under the biomonitoring discussion in Part IV.3)

Summary response to Question 11(a)-(g).

- **Children and pregnant women (a, b).** My own research contains no dedicated cohort evidence. The foundational NTP and Ramazzini animal studies used prenatal lifetime exposure protocols [6, 7], so the existing positive carcinogenicity evidence derives substantially from developmental exposure.

A recent European study showed “Children show 1.5 - 1.9 times higher whole-body SAR than adults at all sub-6 GHz frequencies.... The results identify frequency-phantom combinations where compliance margins narrow: 3500 MHz and 7 GHz under beamforming, and children near 2 GHz under environmental exposure. The organ-specific SAR data for brain, eyes, skin, and genitals support epidemiological exposure assessment and dose modeling across current and prospective wireless bands.” [199]

ICNIRP itself named children as a group who might tolerate exposure less well [173], and under the duration-dependent model described in Part I.3, the uncertainty surrounding cumulative dose is greatest for children, who have the longest remaining lifetime over which exposure can accumulate.

- **Older adults and people with pre-existing medical conditions (c, f).** ICNIRP’s 2002 statement named the elderly and some chronically ill people as potentially less tolerant [171]. Luo et al (2020) showed that inherited gene variation modifies risk [170], and the EHS evidence shows a group with pre-existing or acquired sensitivity reporting disabling symptoms in settings where exposure is presumed to comply with current limits. Assessment for these groups should not assume that the general population limit applies to them unchanged.
- **Individuals with implanted medical devices (d).** My research contains no dedicated evidence on implanted devices, and this submission does not address them. Device-specific interference and safety testing falls with FDA’s device regulation remit (Part VI).

- **Workers with elevated occupational exposure (e).** The occupational duration pattern in the NASA review [126], the sentinel case series of short latency cancers [56](Part II), and a military serviceman's submission to the Australian Parliamentary Inquiry on 5G attributing long-term illness to occupational exposure (Submission 168) all point to this group. The ICRP framework already applies justification and optimization (ALARA) to occupationally exposed workers for ionizing radiation [94](Part III.2); there is no principled reason to withhold equivalent standards from RF-EMF occupational exposure.
- **Other populations (g).** People who report EHS, as set out above, and people who cannot avoid exposure, such as residents subjected to a mandated smart-meter rollout, which the Lamech (2014) case series notes removed a whole population's ability to avoid exposure [4]. The same is applicable to other wireless sources (4G, 5G, 6G, IoT, Public Wi-Fi, radar etc.) deployed by government, military, and private enterprises, including the telecommunications industry.

What should differ

- **Stratify, do not average.** Any federal evidence review or limit-setting exercise should include a susceptibility analysis (gene-environment interaction, pre-existing conditions, acquired sensitivity) and should not treat a null average effect as evidence of safety for subgroups.
- **Fund the susceptibility research.** NIH should support replication of gene-environment analyses such as Luo et al. (2020) in existing genotyped cohorts and biobanks, along with prospective exposure-controlled studies of people reporting EHS that allow for delayed onset and recover while also including oxidative stress, DNA damage and SNP biomarkers amongst others.
- **Record the signal.** CDC should adopt a working case definition for EHS-type symptom clusters and record reports without requiring established causation (Part IV.3; Part VI, recommendation 5).
- **Require positive evidence for psychological attribution.** A nocebo or psychogenic explanation for an RF-associated symptom cluster should be accepted only on positive and verifiable evidence rather than as a default explanation when subjective tests are negative or the mechanism contested.
- **Assess early and objectively.** Studies of people reporting EHS or AHI-type symptoms should include early, repeated objective assessment, including looking for potential biomarkers (Part IV.3), rather than a single late MRI or a subjective group provocation test that is designed to fail.
- **Establish the relevant pulsed-RF exposure parameters.** HHS, with the office of the Director of National Intelligence and the Department of Defense, should determine and publish results, to the extent classification permits, the symptoms and exposure parameters of the Norwegian test, and any other relevant device(s) obtained by U.S. agencies, and test them against current exposure limits including pulse-related provisions (Part VI, recommendation 8).

- **Optimize exposure.** An ALARA-equivalent principle (Part III.2) along with justification is, by construction, more protective of sensitive people than a threshold model that requires harm to be proven before changes are considered. Low-cost accommodation, such as wired alternatives or opt-out for people reporting symptoms from wireless utility smart meters, is consistent with these principles.

HHS's own research agenda (Part VI) should prioritize closing this gap by establishing dedicated, population-stratified exposure and outcome research for the groups named in the answer to Question 11, together with research on susceptibility, rather than treating the absence of such research as license to assume these populations require no differentiated protection.

Part IV: Measuring Human Exposure and Public Health Surveillance (Questions 7-10)

IV.1 Response to Questions 7 – 8: Exposure Assessment Methods and Improvements Needed

The most consequential, currently unaddressed methodological gap identified in this submission is the mismatch between how exposure is measured in existing and proposed surveillance approaches (cumulative years of device ownership, or aggregate time-on-device) and how exposure actually varies anatomically and temporally in real use (Part I.3). Ecological and longitudinal studies routinely track a crude proxy, years of mobile-phone subscription, without adjusting for where on the body the device is held or worn, how its antenna geometry and transmit power have changed across hardware generations, or how much time is spent connected via Bluetooth accessories vs handset-to-ear usage. Validated exposure-measurement improvements should therefore prioritize: (i) anatomically resolved dosimetry that tracks which tissues receive peak absorption for a given device generation and usage pattern, rather than a single whole-body or whole-head metric; (ii) duration- and pattern-weighted exposure metrics, given the mechanistic evidence for a non-monotonic, temporally dependent biological response. (Part I.3, I.5); and (iii) cumulative, multi-source exposure reconstruction capable of capturing simultaneous 5G/6G, Wi-Fi, Bluetooth, and IoT transmission rather than a single-source measurement (Part I.8, III.7).

IV.2 Response to Question 9: Information That Should Be Disclosed

Two categories of disclosure are directly supported by the evidence in this submission. First, device-level disclosure of anatomical SAR distribution and antenna location changes across product generations, so that exposure-reconstruction research is not permanently dependent on manufacturers' own historical engineering record, which are not systematically available to independent researchers. Second, and more fundamentally, disclosure of funding source and research institutional affiliation for any study or systematic review used to inform federal guidance, to be made a standing requirement rather than a voluntary disclosure, which is directly responsive to the

bibliometric funding-source finding set out in Part III.5, and to this own RFI's own instruction that respondents identify the basis of their submissions and any conflicts inherent in the frameworks they rely on.

IV.3 Response to Question 10: Data Sources and Surveillance Systems

On the specific surveillance mechanisms this RFI lists, the research I have been involved with supports the following observations rather than a comprehensive evaluation of each:

- **Disease registries** are useful for detecting aggregate incidence trends, but as demonstrated in Part I.3, are vulnerable to exactly the kind of exposure misclassification that can dilute a genuine, anatomically localized signal into apparent stability; registries tracking only broad tumor categories (e.g. glioma) without anatomical sub-site resolution risk missing localized shifts caused by changing device design or usage.
- **Occupational Monitoring** is the surveillance category most directly supported by existing clinical signals (the sentinel case series discussed in Part II) [56], and should be prioritized for expansion given the ALARA-equivalent argument set out in the summary section of Part III.8.
- **Adverse event reporting and disease/illness cluster investigation** are the categories best suited to capturing the kind of short-latency clinical pattern reported in the sentinel case series, but are only useful if clinicians and researchers have a low-friction, standardized channel for reporting suspected RF-EMF-associated symptoms/clusters, which does not currently appear to exist in a form comparable to, for example, vaccine adverse-event reporting systems.
- **Biomonitoring** of oxidative-stress and genotoxicity biomarkers (e.g., 8-oxoG, 8-OHdG, malondialdehyde and comet assay DNA damage) could help investigate the biological pathways identified in Part I.5 as potentially relevant to RF-EMF exposure. However, these biomarkers are influenced by multiple environmental, lifestyle and endogenous factors, limiting their specificity for RF-EMF-related effects. Their value would therefore depend on carefully designed studies incorporating individual exposure assessment, relevant confounder adjustment and appropriate comparison groups. Such research could provide complementary evidence on potential biological effects.
- **Longitudinal cohort studies** remain valuable but should, as per Part I.3 and III.7, be redesigned around anatomically resolved, duration weighted, cumulative multi-source exposure metrics rather than the “ever/never” or years of ownership proxies that have limited the interpretability of existing cohorts.
- **Environmental monitoring** should be extended, consistent with Part I.9, beyond human-exposure compliance measurement to include ecological indicator species (pollinators in particular), given the existing evidence of RF-EMF effects on insect navigation, reproduction, and colony behavior occurring below current public exposure thresholds.

The unifying recommendation across all of these categories is procedural rather than technical: The EEA's “Late Lessons from Early Warnings” framework, discussed in Part

V below, specifically recommends using longer-term (decadal) monitoring and research, and integrated lay, local and specialist knowledge into surveillance design, both of which speak directly to how HHS should structure any new RF-EMF surveillance system arising from this RFI.

Part V: Learning from History - The Case for Precautionary Governance

Establishing harm to the standard regulators currently demand i.e., reliable long-term dose-response data, replicated and consistent or converging evidence across study types, can take decades. Ignoring early warning signs during that interval has serious historical precedent for going badly wrong. For example: the regulatory neglect of early warning about tobacco and asbestos delayed action for decades despite mounting evidence of harm (see Figure 4 below), and at the cost of wide-spread preventable harm.

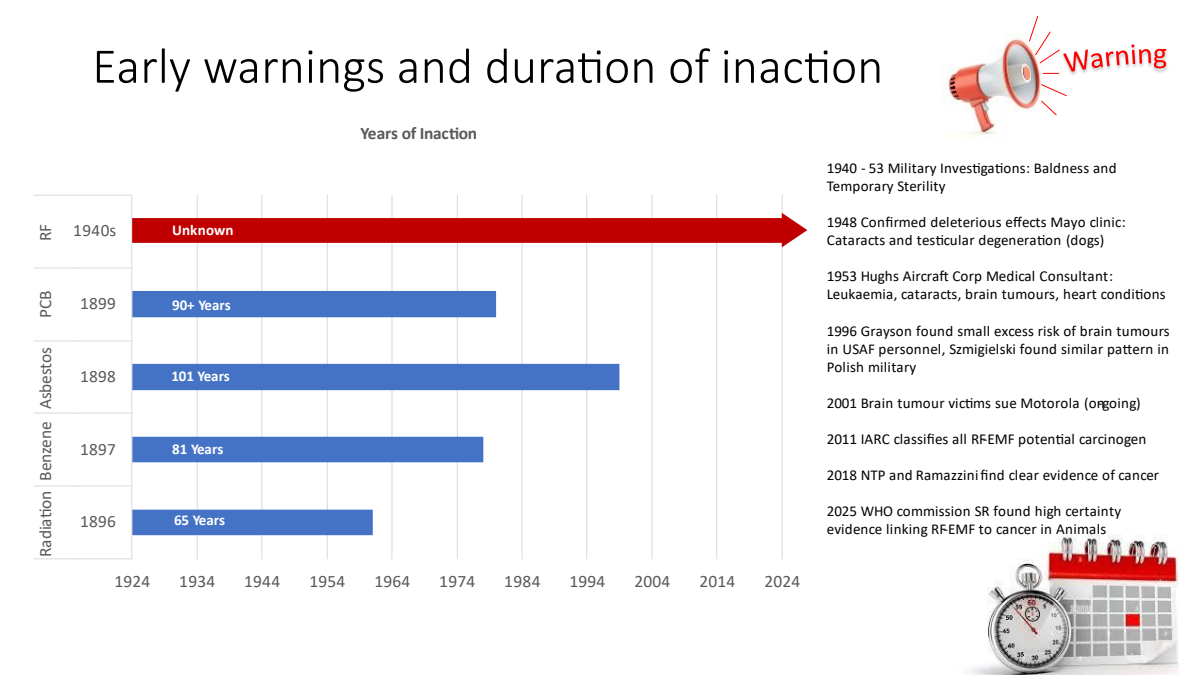


Figure 4 Early warnings and duration of inaction

The EEA’s “Late Lessons from Early Warnings” [200] distilled this and other historical regulatory failures into twelve principles for managing scientific uncertainty and institutional blind spots, which are reproduced below because they provide a precise blueprint to deal with the specific governance failures identified throughout this submission (see Table 7)

Table 7 The European Environment Agency (EEA) Twelve Late Lessons from Historical Case Studies

A. Identify/Clarify the Assumptions, Values and the Case for Action

1. Manage “uncertainty” and “ignorance” as well as “risk”.
2. Identify and reduce “blind spots” in sciences used.
3. Assess and account for all pros and cons of action/inaction.
4. Analyze and evaluate alternative options to the agent/activity under scrutiny.
5. Incorporate diverse stakeholder values.
6. Avoid “paralysis by analysis” by acting to reduce hazards via the precautionary principle.

B. Broaden Assessment Information

7. Identify and reduce interdisciplinary obstacles to learning.
8. Identify and reduce institutional obstacles to learning.
9. Integrate lay, local and specialist knowledge.
10. Identify and anticipate “real world” conditions.
11. Ensure regulatory and informational independence.
12. Use more long-term (i.e., decades) monitoring and relevant research.

These twelve principles appear to have been overlooked by ICNIRP, a private, non-transparent, non-governmental organization, and by the government agencies worldwide that defer to its assessments, despite the strengthening scientific evidence of RF-EMF harm and despite the availability of this exact guidance on how to act under uncertainty.

It is worth noting that the first documented early warning is now more than seventy years old, where a 1953 medical consultant’s report prepared for the Hughes Aircraft Corporation identified leukemia and brain tumors as possible risks associated with RF-EMF exposure (refer to Figure 5). Seventy-three years later, the association between RF-EMF and brain tumors continues to be argued vigorously in the peer-reviewed literature (Parts I.1 – I.7 above), without firm action from ICNIRP or national authorities, such as the FCC and FDA, in the interim.

As Lin, a former ICNIRP commissioner has observed, “the paradigms of ‘an ounce of prevention is far better than a pound of a cure’ appear to have vanished with little trace.” [201]



Figure 5: The growing evidence of harm from RFR 1940 – 2025: Important milestones

Answering the standard objection. The most common argument against revising exposure limits is economic, where the cost of re-engineering network infrastructure and devices is seen to be expensive, and institutions feel such measures might prove unnecessary if future research eventually confirms current public exposure limits are safe. This argument implicitly assumes that the cost of precaution exceeds the potential cost of harm, an assumption that the tobacco and asbestos precedents show to be a poor bet historically, and one that in public health is generally resolved by erring on the side of caution precisely because the downside of getting it wrong is asymmetric. That is, a wrongly precautionary standard costs money, while a wrongly permissive one costs population health at a scale that is very difficult to reverse once established. It is also worth noting, as a matter of record, that the telecommunications industry is not waiting for regulators to act. Manufacturers have already been relocating the primary antenna in the phone chassis away from the brain (an industry reaction to early evidence of brain tumors?), adopting beamforming and beam-steering capabilities, developing new antenna designs, and incorporating metamaterials specifically to direct RF-EMF signals away from the body. These engineering changes are being made in the absence of a regulatory mandate, which suggests that further exposure reduction is technically and economically achievable without the significant disruption the standard objection assumes.

The cost of continuing not to act. Withholding protective measures until conclusive proof of harm is available is a reactive rather than a proactive prevention posture, with concrete costs. It delays the precautionary updates to safety guidelines that chronic, mixed, real-world exposure scenarios actually require, and this delay itself becomes a credibility risk for HHS, FDA and the FCC as public awareness of the underlying evidence grows. It also functionally tilts regulatory priorities towards economic continuity over public health. Both industry and national-regulatory scientists have repeatedly argued the evidence is “not yet conclusive” and further research is needed before acting, while vested interests have simultaneously funded studies more likely to produce null findings (Part III.7), a combination that, whatever the intent, has the aggregate effect of allowing industry sectors to continue operating under fewer restrictions than the totality of the evidence would otherwise justify.

The precautionary paradox. Anthropogenic RF-EMF exposure represents a wicked problem [202] because wireless technologies are rapidly evolving, deeply embedded in modern society, and associated with unresolved questions regarding their long-term health and environmental consequences. Unlike many other potential hazards, these technologies cannot readily be eliminated or substituted without substantial social, economic, and infrastructural consequences. This creates a precautionary paradox. As societal dependence on wireless technology increases, so too do the practical, economic, and political costs of acknowledging potential risks and reconsidering established regulatory assumptions. Scientific uncertainty can consequently become a justification for maintaining the status quo rather than a reason for precautionary consideration.

The paradox is particularly concerning because the same technological expansion that increases the difficulty of regulatory change also increases the scale of population and environmental exposure. This concern is further compounded when evidence

suggesting harm is subjected to more stringent scrutiny, with methodological limitations, inconsistencies, and unresolved questions receiving disproportionate emphasis, while findings reporting no effect are treated as comparatively reassuring and expected. Such asymmetry risks marginalizing potentially important warning signals and allowing uncertainty to perpetuate existing policy assumptions rather than prompt their critical reassessment.

For HHS, the present moment offers an opportunity to resist this dynamic. With wireless technologies more deeply embedded in daily life than ever before, and a Health Secretary directed mandate to examine the relevant literature under the MAHA Strategy, the task is not to presume harm, but to ensure that uncertainty is not mistaken for evidence of safety, that competing interpretations receive rigorous and even-handed consideration, and that precaution remains a legitimate basis for public health deliberation.

Part VI: Federal Research Priorities and Coordination (Question 16 – 17)

Based on the evidence and epistemic analysis set out above, this submission recommends the following as the highest-priority research gaps and coordination reforms to FDA, NIH and CDC:

1. Mandatory funding-source and affiliation sensitivity analysis in any federally commissioned systematic review. No current WHO-commissioned RF-EMF systematic review has tested sponsorship or affiliation as an independent determinant of outcome (Part III.7). Any equivalent federal evidence synthesis conducted following this RFI should build this analysis in as a standing methodological requirement, not an optional addition.
2. Adoption of the Key Characteristics of Carcinogens (KCC) framework, and specifically KCC characteristic #8 (“induces oxidative stress”), as an explicit evaluation criterion in domestic federal RF-EMF risk assessment, in parallel with, and not contingent upon, IARC’s own upcoming re-evaluation (Part I.5). HHS does not need to wait for IARC to formally reclassify RF-EMF before applying a framework IARC itself has already adopted for all other agents.
3. Development and application of anatomically resolved, duration-weighted, cumulative multi-source exposure-reconstruction methodology, replacing “years of device ownership” or “ever/never use” as the default epidemiological exposure proxy (Parts I.3, I.8, III.7, IV.1). This is in my assessment, the single most consequential and tractable methodological gap identified in this submission, because it affects interpretability of essentially every past cohort study in this field. The proposed methodology should be applied in new epidemiological studies (case-control and prospective cohort designs, with registry linkage and operator traffic records for objective usage) that account for how technology and usage changes. These studies should examine cancer at the anatomical sites estimated

to have received the highest cumulative absorbed dose for each usage pattern, apply realistic latency lags, and analyze laterality where relevant (Part I.3).

4. A standing, low friction clinical and occupational adverse-event reporting channel specific to suspected RF/EMF-associated health effects, modelled on existing federal adverse-event surveillance infrastructure, to systematically aggregate the kind of sentinel case-series signal described in Part II rather than leaving such findings as isolated case reports or unreported (Part IV.3)
5. Diversity of advisory-panel composition for any federal RF-EMF evaluation task force convened as a result of this RFI, to explicitly include oncology, toxicology, reproductive biology, independent (non-industry, non-military, non-ICNIRP-affiliated) biomedical researchers, representatives from the affected community alongside physics, engineering and dosimetry expertise that has historically dominated these panels (Part III.3)
6. Explicit adoption of an ALARA-equivalent optimization principle for RF-EMF exposure, extending the logic HHS and other federal agencies already apply to ionizing radiation under the ICRP framework, rather than continuing to require conclusive proof of harm as a precondition for any exposure reduction guidance (Part III.2, III.8).
7. Establish the exposure parameters of the pulsed RF-EMF device implicated in anomalous health incident investigations, including the reported Norwegian self-experiment and any device obtained by U.S. agencies. A test can then be conducted against ICNIRP and FCC limits, including pulse-related provisions, with NIH/NTP and FDA leading the biological evaluation (Part III.8). This is the most direct test of whether current Guidelines register pulsed or non-thermal effects, and it can be done without resolving question of attribution.
8. A pre-specified, symmetric evidentiary standard for any federal RF-EMF evidence review. Inclusion criteria, assay acceptance, dose-response requirements and risk-of-bias assessment rules should be published for independent feedback before the review begins, and applied identically to studies reporting effects and null results. The review should report funding and affiliation sensitivity analysis (recommendation 2) and the markers in Table 4 as a standing checklist (Part III.5).
9. Consider conducting Single-case experimental design (SCED) studies of people who report EHS, with the inclusion of objective biological endpoints. Group provocation studies average over findings over individual and typically rely on short exposures and subjective reporting (Part III.8) and should be avoided. NIH should fund a series of single-case (N-of-1) experimental studies where each participant serves as their own controls, with randomized, blinded alternation of active and sham exposures, individually tailored designs that select sources and signals, washout and recovery periods that match the recipient's symptom onset and resolution patterns. Each study should combine symptom diaries with objective measures taken at baseline and repeatedly under exposure: oxidative-stress status (total oxidant status [TOS], total anti-oxidant status [TAS] and oxidative stress index [OSI]), DNA and oxidative damage markers, along with genomic, proteomic and

metabolomic profiles. Participants should also be genotyped to test whether given gene variants, including DNA-repair and stress response genes (Part III.8), differ between people who report EHS and matched controls. Individual results can be analyzed with single-case statistical methods and then pooled across participants. Recruitment can draw on the reporting channel in recommendation 5.

Part VII: Additional Information (Question 18)

I would welcome the opportunity to provide the full doctoral thesis research underlying this submission, including the genotoxicity scoping review and bibliometric analysis, thematic analysis of the Australian Parliamentary Inquiry into 5G, and comparative analysis of RF-EMF and ionizing radiation risk assessment and management, should HHS wish to examine the underlying research in greater detail. I would also welcome the opportunity to provide further detail on any specific recommendation in Part VI on request.

The central request this submission makes of HHS is procedural as much as substantive: that HHS conduct its own evaluation of this evidence directly, rather than deferring to the existing WHO/ICNIRP evidence synthesis apparatus whose institutional composition, funding-source blind spots, and document history of selective evidence treatment are set out in detail above. The evidence reviewed in this submission does not establish that RF-EMF is a proven human carcinogen at current exposure limits or causes EHS. It does establish that the confident public reassurance historically offered by ICNIRP, the WHO International EMF Project, and the FCC has been maintained in part by treating contested, asymmetrically scrutinized, and institutionally conflicted evidence syntheses as though they were settled, disinterested science. HHS's own Health Secretary has directed this Department to examine the literature because "our science must keep pace" with technologies embedded in daily life. Taking that mandate seriously requires HHS to weigh independent evidence on its merits, including where it conflicts with the institutions that have historically defined the "mainstream position", and to adopt the precautionary, ALARA equivalent governance model this submission has set out as the appropriate response to a genuinely unresolved and consequential public and environmental health question.

Yours Sincerely,

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