

Scientific Response to the HHS Request for Information on RF/EMF Exposure and Human Health

Question-by-question scientific response | HHS-OASH-2026-0397

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Basis of response. This submission provides independent scientific input based primarily on peer-reviewed research, governmental scientific reports, and methodological literature. It distinguishes biological responses, adverse health effects, hazard identification, and quantitative risk, and it does not imply institutional endorsement by the authors' institutions.

Executive Summary

This response addresses Questions 2 through 18 of the HHS Request for Information. Its central scientific recommendation is that RF/EMF exposure should not be treated as a single scalar quantity when evidence is evaluated. Frequency, modulation and waveform, duty cycle and temporal pattern, peak versus time-averaged intensity, exposure duration and recovery intervals, source geometry, near-field versus far-field conditions, polarization, simultaneous RF/ELF exposures, tissue-specific absorbed dose, and developmental timing can differ substantially across studies and technologies. Where biological equivalence has not been demonstrated, these exposure conditions should be analyzed separately rather than pooled simply because average SAR or power density is similar (Valberg, 1995; Belyaev, 2010; Belyaev et al., 2009; Markova et al., 2010; IARC, 2013; Belyaev and Dasdag, 2026).

A second central recommendation is that RF exposure should be characterized as part of the complete electromagnetic environment. Static magnetic fields (SMF) and extremely low-frequency (ELF) magnetic fields can be present in laboratories, exposure chambers, buildings, and the devices themselves. Experimental studies summarized by IARC have shown that weak ELF magnetic noise can attenuate or block several RF-induced responses, including ornithine-decarboxylase activation, spatial-learning deficits, and DNA damage, while the magnitude of the local static geomagnetic field can alter microwave resonance-type responses (Litovitz et al., 1997; Lai, 2004; Lai and Singh, 2005; Yao et al., 2008; Ushakov et al., 2006; IARC, 2013). Accordingly, background and co-exposure SMF/ELF conditions should be measured and reported in RF experiments and should be considered when apparent replication or non-replication is interpreted.

This point is supported by concrete experimental observations. In human lymphocytes, exposures at 905 and 915 MHz GSM and at 1947 MHz UMTS produced different responses in 53BP1/ γ -H2AX DNA-repair foci despite low, non-thermal SARs; the strongest response was observed with UMTS, whereas 905 MHz produced little or no effect in most conditions (Belyaev et al., 2009; IARC, 2013). In human stem cells and differentiated fibroblasts, the same general RF exposure paradigm produced different biological responses by cell type and exposure schedule (Markova et al., 2010). In a recent prenatal 3.5 GHz rat study, exposure throughout gestation produced more pronounced renal histological and DNA-damage findings than exposure limited to the last two weeks, illustrating the potential importance of exposure duration and developmental window (Gelenli Dolanbay et al., 2026).

The human evidence is also sensitive to exposure characterization. The WHO-commissioned cancer review concluded with moderate certainty that mobile-phone RF exposure likely does not increase risks of the most studied brain and head tumors, while evidence for fixed-site environmental exposure was more limited (Karipidis et al., 2024). However, most mobile-phone epidemiology relies on surrogate variables such as cumulative call time, years of use, or subscription history rather than direct tissue dose. Measurement error can materially alter risk estimates, and regression-calibration work in COSMOS illustrates that this uncertainty can be quantified rather than ignored (Reedijk et al., 2024).

The interpretation of negative human cancer evidence also requires care. Karipidis et al. (2024) concluded, using an adapted GRADE framework, that there is moderate-certainty evidence that near-field mobile-phone exposure likely does not increase the risk of several of the most studied brain and head tumors. That conclusion should be reported accurately, but it should not be treated as equivalent to the IARC evidence descriptor 'evidence suggesting lack of carcinogenicity.' Under the IARC criteria used in Monograph 102, negative evidence requires several adequate studies covering the relevant exposure range, mutually consistent null findings, narrow confidence intervals near unity, reasonable exclusion of bias, confounding and exposure misclassification, and adequate follow-up. Given the reliance of current mobile-phone epidemiology on surrogate exposure variables, incomplete characterization of cumulative dose and downlink exposure, technological heterogeneity, and limited follow-up for some outcomes, the evidence more strongly supports the statement that a convincing increase in risk has not been demonstrated under the exposure constructs studied than the stronger statement that carcinogenic risk has been excluded (IARC, 2013; Karipidis et al., 2024; Reedijk et al., 2024).

Population-based cancer-registry trends are valuable external consistency checks, but they should not be used as an independent refutation of analytical epidemiology unless registry completeness, latency, exposure prevalence, tumor classification, and expected effect size are all sufficiently characterized. Several registry-quality studies have documented incomplete ascertainment or differential capture of central nervous system tumors, and stable overall incidence can coexist with changes in specific anatomical or histological subtypes (Barlow et al., 2009; Larjavaara et al., 2008; Parkin and Bray, 2009; Tettamanti et al., 2019). Incidence-trend analyses can constrain very large population-level risks, but they do not exclude modest effects, effects confined to highly exposed groups, or site-specific risks that are diluted in aggregate incidence (Little et al., 2012; Deltour et al., 2012; de Vocht, 2016).

This distinction is methodological rather than semantic: absence of detected evidence is not automatically evidence of absence. A null or non-significant result becomes informative evidence against an effect only when the study was capable of detecting the effect of interest, including adequate statistical power, valid exposure classification, sufficient latency and follow-up, coverage of the relevant exposure range, and confidence intervals sufficiently narrow to exclude effects of practical importance (IARC, 2013).

For chronic and epidemiological exposure assessment, HHS should also distinguish exposure rate from cumulative dose. SAR is expressed in W/kg and represents the rate at which RF energy is absorbed per unit mass; it is therefore analogous to an absorbed-dose rate. Where dosimetry permits, cumulative absorbed RF energy should be estimated as the time integral of SAR ($\int \text{SAR} \cdot dt$, J/kg), while cumulative incident far-field exposure can be represented by the time integral of power density ($\int S \cdot dt$, J/m²). A simple intensity $\times$ duration metric is therefore more informative for long-term exposure than an instantaneous SAR value alone, although cumulative dose should not replace information on frequency, modulation, duty cycle, intermittency, peaks, and recovery periods because equal integrated doses may still represent biologically different exposures (ICNIRP, 2020; Belyaev and Dasdag, 2026).

We therefore recommend that HHS: (1) make exposure validity a formal evidence-quality domain; (2) avoid assuming biological equivalence across frequencies, modulation types, technologies, temporal patterns, or exposure geometries without supporting data; (3) support independent replication of influential positive and negative findings under harmonized exposure conditions; (4) develop prospective cohorts and environmental studies with repeated instrumental exposure assessment; and (5) support publicly funded, competitively reviewed RF/EMF research with preregistration, transparent sponsor-role disclosure, and open data where feasible. Public funding should not be interpreted as a reason to exclude industry-funded studies; rather, it provides an important independent replication pathway because associations between funding source and reported outcomes have been described in the RF experimental literature (Huss et al., 2007; van Nierop et al., 2010).

Central methodological point

An RF/EMF exposure is defined by more than intensity. Two exposures with the same average SAR or power density may differ in carrier frequency, modulation, pulse structure, duty cycle, spatial distribution, exposure/recovery schedule, and tissue dose. Risk assessment should preserve these distinctions unless biological equivalence has been demonstrated.

Responses to the HHS Questions

Question 2 — Perspective represented by this response

This response is submitted from a research perspective. The lead author, Igor Belyaev, is a radiobiologist with research experience in cellular, animal and human studies of ionizing and non-ionizing radiation, including cytogenetics, DNA-damage and repair responses, individual radiosensitivity, and biological effects of RF, ELF and static magnetic fields. He served on the IARC Working Group that evaluated the carcinogenicity of radiofrequency electromagnetic fields for Monograph 102 and on the WHO Task Group that assessed health risks from static electric and magnetic fields for Environmental Health Criteria 232. On 14 February 2012, he gave an invited presentation at the U.S. Federal Communications Commission titled “Exposure to Microwaves from Mobile Communication, DNA Repair, and Cancer Risk,” and he has also given an invited lecture at the U.S. Air Force Research Laboratory at Brooks Air Force Base, San Antonio, Texas. He and his coauthors received the Bioelectromagnetics Society’s 2010 Most Influential Bioelectromagnetics Journal Paper by Citation Award, presented in 2011, for their 2006 rat-brain gene-expression study. These credentials are provided only to explain the scientific perspective represented here and do not imply endorsement by IARC, WHO, FCC, the U.S. Air Force, or the authors’ institutions. The purpose of this response is not to advocate a predetermined regulatory outcome, but to identify methodological conditions needed for reliable health-risk assessment (WHO, 2006; IARC, 2013; Belyaev et al., 2006).

Question 3 — Individual experiences of potential adverse health effects

No individual or clinical case histories are submitted in this response. Self-reported experiences attributed to RF/EMF exposure may nevertheless be useful for hypothesis generation if they are documented prospectively together with objective exposure measurements, symptom timing, relevant medical history, potential confounders, and any exposure-reduction intervention. Such reports should not be treated as proof of causation.

A small number of well-documented individual cases may be informative if exposure measurements and a reproducible temporal pattern are available, but they should remain clearly labeled as observational. Blinded or sham-controlled follow-up is needed before causal interpretation.

Question 4 — Research observations that may help identify patterns

A recurring pattern in RF/EMF research is that biological response can depend on the detailed exposure condition rather than on intensity alone. In Belyaev et al. (2009), human lymphocytes exposed for 1 h to GSM 905 MHz, GSM 915 MHz, or UMTS 1947 MHz at low SARs showed markedly different effects on chromatin and 53BP1/ γ -H2AX DNA-repair foci: effects were observed at 915 MHz and were stronger with UMTS, while 905 MHz produced little or no effect in most conditions. IARC’s Monograph 102

summarized these experiments and explicitly noted the frequency- and signal-dependent pattern (Belyaev et al., 2009; IARC, 2013). The broader electromagnetic environment is another experimentally supported modifier. IARC Monograph 102 reviewed four studies in which superposed ELF fields up to approximately 5 μT significantly reduced RF-induced biological responses, including ODC activity, chick-embryo hypoxia sensitization, spatial-learning impairment in rats, and DNA strand breaks in rat brain cells (Litovitz et al., 1997; Di Carlo et al., 2002; Lai, 2004; Lai and Singh, 2005; IARC, 2013). Yao et al. (2008) likewise reported that superposed electromagnetic noise inhibited RF-associated DNA damage and ROS increases in human lens epithelial cells. For static fields, resonance-type microwave effects in *E. coli* varied with the net static magnetic field, including changes in effect magnitude and effective frequency under different SMF conditions (Belyaev et al., 1994; Ushakov et al., 2006; IARC, 2013). These data do not establish that every RF endpoint is sensitive to SMF/ELF background, but they show that such fields can be effect modifiers and should not be assumed irrelevant.

The same principle extends beyond carrier frequency. Markova et al. (2010) reported stronger inhibition of 53BP1 focus formation in human mesenchymal stem cells than in differentiated fibroblasts under defined GSM/UMTS exposures, showing that biological model and differentiation state can modify response. More recently, prenatal 3.5 GHz exposure of rats at approximately 24–28 V/m for 2 h/day was associated with renal histological alterations, increased comet-assay DNA damage, and altered Beclin-1/LC3 expression at 6 months; effects were generally more pronounced after exposure throughout gestation than after exposure during only the last two weeks (Gelenli Dolanbay et al., 2026). These studies do not establish human disease risk, but they directly support the need to preserve frequency, waveform, duration, developmental timing, and biological model in evidence synthesis.

Human biomonitoring also shows why measured exposure matters. Zothansiam et al. (2017) compared 40 residents living within 80 m of mobile-phone base stations with 40 controls living at least 300 m away and reported higher bedroom RF power density together with higher micronucleus frequency and oxidative-stress-related changes. Gulati et al. (2024) studied 24 adults living for at least five years in two neighboring German residential areas with measured GSM/LTE downlink exposure. Total chromosomal aberrations were higher in the higher-exposure group ($7.1 \pm 1.3\%$ versus $3.9 \pm 1.1\%$, $p < 0.001$), whereas micronuclei and several other endpoints were not significantly different. These small observational studies cannot establish causality, but they illustrate the value of combining objective exposure measurements with multiple biological endpoints (Zothansiam et al., 2017; Gulati et al., 2024; Belyaev et al., 2026).

Professional observations from practitioners who measure RF/ELF fields and implement exposure-reduction interventions may also be hypothesis-generating if reported with a denominator, pre/post measurements, the intervention used, timing of any reported change, co-interventions, and the frequency of no response or worsening. HHS could encourage a simple prospective registry for self-reported IEI-EMF in which standardized symptom diaries are paired with objective RF/ELF measurements; the purpose would be to identify reproducible patterns for later blinded study, not to infer causation from the registry itself.

For HHS surveillance purposes, research observations are most interpretable when each record identifies the endpoint measured, timing of the observation relative to exposure, population or biological model, exposure pattern and measurement method, and whether the finding was transient, persistent, replicated, or linked to a later clinical outcome. This structure would allow hypothesis generation without conflating biomarker changes with established adverse health effects.

Question 5 — Exposure standards, guidelines, regulatory frameworks, and needed improvements

Current frameworks serve different purposes. FCC rules and IEEE C95.1-2019 provide compliance and exposure-limit frameworks; ICNIRP 2020 is a health-protection guideline based on established adverse effects; IARC classified RF electromagnetic fields as Group 2B, possibly carcinogenic to humans, in 2011 and documented the evaluation in Monograph 102; and WHO/NTP/OHAT/GRADE approaches are evidence-synthesis frameworks. HHS should avoid treating conclusions from these frameworks as if they answer the same scientific question: compliance, hazard identification, and quantitative risk are not interchangeable endpoints (IARC, 2013; ICNIRP, 2020; Guyatt et al., 2008; OHAT, 2019).

The main technical improvement we recommend is explicit separation of exposure dimensions in both primary studies and risk assessment. Average SAR or power density is necessary but not sufficient to characterize an RF exposure. Frequency, modulation, waveform, bandwidth, duty cycle, peak-to-average ratio, intermittency, duration, recovery periods, spatial distribution, source geometry, and simultaneous RF/ELF fields should be reported and retained in evidence tables. Pooling across technologies should require a stated biological justification rather than an assumption of equivalence (Valberg, 1995; Belyaev, 2010; Belyaev et al., 2009; Misek et al., 2023; Belyaev and Dasdag, 2026).

In addition, HHS should require a cumulative-dose descriptor for chronic exposure whenever technically feasible. For tissue dosimetry this can be expressed as specific absorbed energy, i.e., the time integral of SAR (J/kg); for environmental far-field exposure, an incident-energy metric based on time-integrated power density (J/m²) can be reported alongside modeled tissue

dose. This does not mean that dose should be treated as the only relevant variable. Rather, dose should complement—and in long-term epidemiology often be more informative than—a single instantaneous or short-term SAR value, while the underlying temporal and spectral characteristics remain available for stratified analysis (Belyaev, 2010).

Exposure parameter	Why it should remain explicit	Illustrative evidence
Carrier frequency / band	Biological response may not be identical at nearby or widely separated frequencies.	Different 905, 915 and 1947 MHz lymphocyte responses (Belyaev et al., 2009; IARC, 2013).
Modulation / waveform	Signals with similar average intensity can differ in temporal structure.	GSM versus UMTS differences in DNA-repair foci (Belyaev et al., 2009; Markova et al., 2010).
Duration / developmental window	Cumulative time and developmental timing may affect persistence or phenotype.	One-year follow-up after prenatal-only 3.5 GHz exposure; related 2.45 GHz thyroid follow-up studies (Gelenli Dolanbay et al., 2025; Ozyilmaz et al., 2024; Özyilmaz et al., 2025).
Intermittency / recovery	Biological response can depend on exposure/recovery sequence, not only total energy.	Exposure–response synthesis emphasizes temporal patterns (Belyaev and Dasdag, 2026).
Geometry / tissue dose	External field strength does not uniquely determine local absorbed dose.	Near-field handset, far-field base station and whole-body rat studies are not interchangeable (IARC, 2013; NTP, 2018; Falcioni et al., 2018).
Biological model / life stage	Cell type, differentiation state and developmental stage may modify response.	Stem-cell versus fibroblast responses (Markova et al., 2010); prenatal rat findings (Gelenli Dolanbay et al., 2026).
Background / co-exposure SMF and ELF fields	Static and ELF magnetic fields can modify RF bioeffects in some experimental systems.	ELF noise attenuated several RF-induced endpoints; static-field magnitude altered microwave responses (Litovitz et al., 1997; Lai and Singh, 2005; Yao et al., 2008; Ushakov et al., 2006; IARC, 2013).

A practical classification framework should therefore preserve, at minimum, carrier frequency/band, electric and magnetic field strength or power density, local and whole-body SAR or absorbed-power density where applicable, modulation/waveform, duty cycle and temporal variability, beamforming or spatial focusing, simultaneous sources, source-body distance and geometry, exposure duration, and a cumulative exposure descriptor. These dimensions should remain available for stratified analysis rather than being collapsed prematurely into a single technology label.

Accordingly, HHS should ask systematic reviews to test heterogeneity by exposure parameters whenever data allow. A null pooled estimate across biologically dissimilar exposures should not automatically be interpreted as evidence that every component exposure is null; similarly, a positive result under one narrowly defined condition should not be generalized to all RF/EMF.

The same principle should apply to environmental electromagnetic co-exposures. RF studies should report the net static magnetic field (magnitude and, where relevant, orientation/gradient), ELF magnetic-field spectrum and amplitude, 50/60-Hz components, chamber or incubator fields, and any intentional or incidental magnetic-noise exposure. This recommendation follows long-standing exposure-characterization guidance in bioelectromagnetics and magnetobiology and is particularly relevant to replication studies (Misakian et al., 1993; Valberg, 1995; Makinistian et al., 2018).

As a proportionate risk-management measure, HHS could support practical, measurable methods for reducing avoidable exposure where this can be done without compromising essential connectivity or medical benefit. Such measures should be presented as exposure-reduction options, not as evidence that exposure below current limits is known to cause disease. Any recommendation should distinguish whole-body from localized exposure, the applicable averaging mass and time, frequency-dependent metrics, and the actual exposure condition rather than treat numerical limits or experimental exposure levels as interchangeable (FCC, 47 CFR §§ 1.1310 and 2.1093; ICNIRP, 2020).

Question 6 — Evidence concerning adverse health effects under current exposure limits

The available evidence does not support a simple statement that all RF/EMF exposures below current limits either are harmful or are harmless. Current U.S. and international exposure limits are designed to protect against established adverse effects within their specified frequency, averaging and dosimetric conditions. Compliance with those limits should not be interpreted as proof that no biological response can occur below them, while a biological response observed below a limit should not automatically be interpreted as an adverse health effect.

Human carcinogenicity: what a negative conclusion requires

Karipidis et al. (2024) reported moderate-certainty evidence that mobile-phone RF exposure likely does not increase the risk of glioma, meningioma, acoustic neuroma, pituitary tumors, salivary-gland tumors, or pediatric brain tumors. This is an important synthesis of the available epidemiology, but GRADE certainty and IARC evidence descriptors are not interchangeable. In the IARC framework applied to RF-EMF in Monograph 102, 'evidence suggesting lack of carcinogenicity' requires several adequate studies

that cover the full relevant range of exposure, are mutually consistent in showing no positive association at any observed exposure level, have narrow confidence intervals with upper limits close to unity, reasonably exclude bias/confounding/misclassification, and have adequate follow-up (IARC, 2013).

Current mobile-phone epidemiology does not clearly satisfy those stricter negative-evidence conditions. Exposure is generally represented by years of use, cumulative call time, number of calls, or subscription history rather than validated tissue dose; historical studies pool different mobile technologies and often do not quantify infrastructure-related downlink exposure; and the highest cumulative exposures and longest latencies are represented by relatively small subgroups. The appropriate interpretation is therefore that the available human evidence has not demonstrated a convincing increase in risk for the most studied tumors under the exposure metrics and follow-up available. That is scientifically different from demonstrating that RF exposure lacks carcinogenic potential (IARC, 2013; Karipidis et al., 2024; Reedijk et al., 2024).

For cancer epidemiology, cumulative exposure should be developed explicitly rather than inferred only from years of use. Li et al. (2012) provides a concrete example: in a population-based childhood-neoplasm study, the investigators combined mobile-phone base-station power with duration of operation to construct a watt-year exposure index and averaged it over the five years preceding diagnosis. This township-level metric was an ecological exposure proxy rather than absorbed tissue dose and was subject to exposure misclassification, but it illustrates why long-term cancer studies should preserve time-integrated exposure information rather than rely only on instantaneous field strength, distance from a source, or short-term SAR.

This issue is especially important because nondifferential exposure misclassification generally attenuates exposure-response associations. Thus, the statement 'no dose-response relationship was demonstrated using available surrogate exposure metrics' is more defensible than 'no dose-response relationship exists.' Incidence-trend analyses can provide useful external coherence but do not remove uncertainty about exposure validity, latency, tumor subtype, or highly exposed subgroups.

Cancer-registry incidence trends: useful consistency checks, not stand-alone refutations

Cancer registries are indispensable for population surveillance, but their ability to test an RF-carcinogenicity hypothesis depends on data quality and on the scale of the hypothesized effect. Completeness varies across registries and tumor types.

Methodological work on cancer registration has emphasized that completeness cannot be assumed and must be evaluated empirically (Bray and Parkin, 2009; Parkin and Bray, 2009). For central nervous system tumors, under-ascertainment may occur when tumors are diagnosed radiologically without histological confirmation, when reporting pathways differ between inpatient and outpatient care, or when classification and coding practices change over time (Larjavaara et al., 2008; Tettamanti et al., 2019). Independent validation studies illustrate this problem. Barlow et al. (2009) identified incomplete registration in the Swedish Cancer Register, while Tettamanti et al. (2019) found discrepancies between central nervous system tumors recorded in the Swedish Cancer Register and the National Inpatient Register. These findings do not imply that national registries are unusable; they show that registry incidence is an estimate with ascertainment error rather than a perfect census of all relevant tumors.

For mobile-phone epidemiology, the correct interpretation is therefore conditional. A stable overall brain-tumor incidence can argue against a very large, rapidly emerging population-wide risk if the registry is sufficiently complete and the assumed latency and exposure prevalence are realistic. It cannot, by itself, exclude a smaller excess risk, a risk concentrated among the highest-exposed users, a long-latency effect, or an increase limited to a specific tumor subtype or anatomical region.

This is borne out by published consistency analyses. Little et al. (2012) compared U.S. glioma incidence trends with risk estimates from case-control studies and found that observed incidence was broadly compatible with the more moderate risk estimates from INTERPHONE, although less compatible with substantially higher risk estimates from some Swedish analyses. Deltour et al. (2012) similarly concluded that Nordic incidence trends were inconsistent with some large risk scenarios but did not exclude all smaller effects. In England, de Vocht (2016) reported that overall brain-cancer incidence did not show the same pattern as some anatomical subtypes, including an increase in temporal-lobe tumors. That finding does not establish RF causality, but it demonstrates why aggregate incidence can obscure site-specific trends.

More recent Nordic analyses have again argued that observed glioma trends are not compatible with large increases in risk attributable to mobile-phone use (Deltour et al., 2022). These analyses should be included in the totality of evidence. Their strongest use is to place an upper bound on plausible population-level effects under specified latency and exposure assumptions, not to convert a population-level null trend into proof that no carcinogenic effect exists.

Registry evidence can support	Registry evidence cannot establish by itself	Conditions
Whether a large population increase is visible over time	That RF exposure has no carcinogenic effect	Adequate completeness; stable case ascertainment
Whether trends fit specified risk/latency scenarios	Exclusion of modest effects among highly exposed subgroups	Valid exposure-prevalence and latency assumptions

Trends in anatomic or histologic tumor subtypes	Attribution of a subtype trend to RF	Consistent classification and sufficient subtype-specific numbers
External consistency check for individual-level studies	Replacement of individual-level exposure analysis	Recognition of ecological and exposure-misclassification limits

Two large animal programs are especially relevant to hazard evaluation, although they do not directly quantify human risk under FCC-compliant everyday exposure. The NTP two-year rat study used whole-body 900 MHz GSM and CDMA exposures at 1.5, 3, and 6 W/kg and concluded there was clear evidence of carcinogenic activity in male rats based on malignant cardiac schwannomas; malignant gliomas in male rats were classified as some evidence of carcinogenic activity (NTP, 2018). The Ramazzini Institute study used far-field 1.8 GHz GSM exposure from prenatal life until natural death at 5, 25, or 50 V/m and reported an increased incidence of heart schwannomas in male rats at the highest exposure (Falcioni et al., 2018). Differences in dose, geometry, modulation, and whole-body versus far-field exposure mean that these studies should be compared mechanistically, not collapsed into a single generic 'RF' category.

The WHO-commissioned systematic review of laboratory-animal cancer studies by Mevissen et al. (2025), with its 2026 corrigendum, evaluated evidence for increased malignant heart schwannomas and glial-cell-derived brain tumors in male rats as high certainty. This assessment is material to hazard identification but remains contested. In a quantitative re-analysis, Belenki et al. (2026) argued that the certainty was overstated because of pooling decisions and statistical imprecision; their alternative synthesis assessed the certainty for malignant heart tumors as moderate and for malignant brain tumors as low. The diverging assessments emphasize the need to disclose study-selection rules, exposure and biological heterogeneity, uncertainty intervals, and the assumptions required to combine experiments. Neither assessment directly quantifies human risk under everyday exposure conditions (Mevissen et al., 2025, 2026; Belenki et al., 2026).

Japan–Korea follow-up studies do not constitute strict refutations of the NTP findings

The 2026 Japanese and Korean studies were developed under an international project intended to verify or clarify the reproducibility of the NTP findings (Ahn et al., 2022; Imaida et al., 2026; Kim et al., 2026). They are scientifically valuable independent chronic studies, but they provide less information than NTP for reproducing the specific rare-tumor dose-response pattern because they used male rats only, CDMA only, and a single 4 W/kg exposure level. NTP used 90 animals per sex per group, GSM and CDMA modulations, and whole-body SAR levels of 1.5, 3.0 and 6.0 W/kg plus sham controls. Exposure in NTP began on gestation day 5 and continued after birth through the two-year study. The international follow-up protocol likewise began prenatal exposure on gestation day 5; 70 male rats per group were assigned, with 68 exposed males available for final evaluation in the Japanese study (NTP, 2018; Ahn et al., 2022; Imaida et al., 2026; Kim et al., 2026).

These differences matter for interpretation. The follow-up studies omitted the NTP 6 W/kg group, did not reproduce the NTP multi-dose design, could not perform an exposure-trend analysis across several SAR levels, did not test the GSM arm, and had fewer animals available for evaluation. Imaida et al. acknowledged the limitations of a single 4 W/kg exposure and group size for detecting rare tumors. Melnick, Moskowitz and Héroux (2026) argued that substantially larger groups would be needed, under their assumptions, to achieve high statistical power for rare heart schwannomas. Their exact sample-size estimates are model-dependent, but the general point is sound: failure to reach statistical significance in a smaller, single-dose study cannot by itself invalidate a positive multi-dose study of a rare endpoint. The Japanese and Korean studies are therefore best interpreted as important independent follow-up studies under related, but not identical, conditions rather than as strict one-to-one replications capable by themselves of falsifying the NTP result.

Static and ELF magnetic fields are an example of a possible, but unproven, effect-modifying variable. Experimental literature summarized by IARC and later methodological work indicates that RF responses can depend on co-existing low-frequency or static magnetic-field conditions in some systems (IARC, 2013; Makinistian et al., 2018). This does not establish that SMF/ELF differences explain the divergent tumor findings in NTP and the Japan/Korea studies. It supports a narrower methodological recommendation: future replication studies should measure and report background static and ELF fields so that electromagnetic comparability can be evaluated rather than assumed.

Feature	NTP rat study	Japan/Korea 2026	Interpretive consequence
Animals/group	90 per sex/group	70 male rats/group assigned; 68 exposed males evaluable in the Japanese study	Lower information/power for rare tumors than the larger NTP groups
Sexes	Males and females	Males only	Female NTP findings not tested
Modulations	GSM and CDMA	CDMA only	GSM arm not replicated
SAR design	0, 1.5, 3, 6 W/kg	0, 4 W/kg	No multi-dose trend; 6 W/kg missing

Exposure timing	Exposure began on gestation day 5 and continued postnatally through the two-year study	Prenatal exposure also began on gestation day 5 under the follow-up protocol; chronic postnatal exposure followed	Broad life-course timing similar, but study designs and dose structures were not identical
Statistical interpretation	Pairwise and trend analyses across multiple doses	Single exposed dose	Less information for dose-response and rare-endpoint trend assessment
Electromagnetic equivalence	Validated NTP reverberation system	Independent reverberation systems	Nominal CDMA similarity does not prove complete field equivalence

The interpretation of the Japanese and Korean follow-up studies should distinguish a valid independent negative study from a fully powered, exposure-matched replication of an earlier positive study. Concerns about the single-dose design, lower power for rare tumors and omission of the NTP 6 W/kg arm have also been raised by Lin (2026). These critiques should be weighed together with the primary study reports and should not be taken as proof of a specific mechanism for the divergent outcomes.

Accordingly, the Japanese and Korean studies should be included in the totality of evidence, but their null results should not be described as having 'disproved' or 'invalidated' the NTP findings. A strict replication designed to test the NTP dose-response pattern directly would require comparable or greater power, several dose groups including the highest informative NTP exposure, matched modulation arms, harmonized dosimetry and exposure-system characterization, and pre-specified pairwise and trend analyses (Ahn et al., 2022; Melnick et al., 2026; Lin, 2026).

Low-intensity cellular and human biomarker studies add evidence of biological activity under specific conditions, but they do not by themselves establish adverse disease outcomes (Belyaev et al., 2009; Zothansiam et al., 2017; Gulati et al., 2024). HHS should therefore separate three questions: whether a biological change occurs; whether it is adverse and persistent; and whether it translates into a clinically meaningful increase in human risk.

Question 7 — Methods available for characterizing individual and population exposure

Exposure characterization should combine methods rather than rely on a single surrogate. Useful tools include frequency-selective environmental measurements, personal exposimetry, device/network transmit-power information, time-activity diaries, geospatial propagation models, and computational dosimetry for tissue-specific absorbed dose. These methods answer different questions and should be cross-validated where possible (Birks et al., 2018; Jalilian et al., 2019; Reedijk et al., 2024).

For handset epidemiology, cumulative call time or years of use are at best proxies for absorbed RF dose because output power changes with network generation, signal quality, adaptive power control, handset position, and user behavior. Regression calibration in COSMOS demonstrates that error in self-reported phone use can be estimated and incorporated into risk models (Reedijk et al., 2024). For fixed-site sources, distance alone is insufficient because antenna height, downtilt, sector orientation, propagation, building attenuation, traffic, and reflections determine actual field strength.

For epidemiological studies of brain and head tumors, exposure assessment should preserve laterality whenever feasible: the habitual side of handset use, the side of the head receiving the highest local exposure, and the anatomical side and location of the tumor. Ipsilateral and contralateral associations should be analyzed separately because a localized near-field exposure hypothesis predicts spatially specific effects. At the same time, retrospective reports of preferred phone side can be affected by differential recall after diagnosis; prospective collection or objective device/operator-derived information is therefore preferable (INTERPHONE Study Group, 2010).

Handset exposure should also not be reduced to RF alone. Misek et al. (2023) measured ELF magnetic fields from 15 mobile phones in different operating modes and showed model-, mode- and generation-dependent ELF components generated by the signal envelope and device hardware, with 2G phones generally producing higher ELF magnetic fields than 3G phones under the tested conditions. This does not establish a health effect from handset ELF exposure, but it demonstrates that some mobile-phone exposures are simultaneous RF+ELF exposures and supports documenting both components when biological or health outcomes are investigated.

A further limitation is that handset use captures only one component of total RF exposure. Contemporary real-world measurements show that downlink exposure from mobile-network infrastructure can contribute substantially to brain RF exposure. Lee et al. (2026) simultaneously measured downlink electric fields and uplink phone transmit power in commercial 3G/4G/5G networks and found that modeled whole-brain SAR from downlink exposure was comparable to or exceeded uplink SAR in most tested scenarios. Because downlink/base-station exposure can occur for much longer periods than active handset transmission, epidemiological studies should estimate cumulative dose from both uplink and downlink sources rather than treating mobile-phone use as a proxy for total RF exposure (Lee et al., 2026; Joseph et al., 2010; Bolte and Eikelboom, 2012; Birks et al., 2018; Sagar et al., 2018).

Question 8 — Improvements needed for real-world exposure assessment

HHS should promote repeated, source-resolved measurements instead of single spot measurements or distance-only classifications. At minimum, epidemiological and biomonitoring studies should preserve: frequency/band, technology generation, measured field or power density, temporal variability, duration, body/source geometry, simultaneous sources, and instrument uncertainty. Where tissue-specific effects are hypothesized, external field measurements should be complemented by dosimetry rather than treated as tissue dose.

The most important conceptual improvement is to avoid reducing multidimensional exposure to one number too early. For example, two subjects with similar time-averaged power density may experience different frequency mixtures, peak structure, duty cycles, and body positioning. These variables may be irrelevant for some outcomes, but that should be tested empirically rather than assumed. The same principle should guide experimental replication: an attempted replication is strongest when frequency, waveform, exposure schedule, dosimetry, temperature, biological model, and endpoint timing match the original study (Valberg, 1995; Belyaev and Dasdag, 2026).

For experimental reproducibility, 'exposure' should therefore mean the complete electromagnetic environment, not only the nominal RF carrier and SAR. Measurements should include background SMF and ELF conditions because these may differ across laboratories or exposure systems even when the RF protocol appears identical. Ferromagnetic chamber components, power supplies, incubators, current-carrying conductors, and permanent magnets in devices can alter the local DC/ELF environment. Mobile phones themselves can also emit ELF magnetic components, while smartphones can produce localized static-field hotspots; therefore, combined-field characterization may be relevant to both laboratory and real-world handset exposure (Belyaev, 2010; Misek et al., 2023; Zastko et al., 2026). At the same time, long-term studies need a physically meaningful cumulative quantity. We therefore recommend reporting both exposure rate and cumulative dose: e.g., SAR together with $\int \text{SAR} \cdot dt$ for tissue exposure, or power density together with $\int S \cdot dt$ for far-field environmental exposure. This dual reporting separates 'how intense' an exposure is from 'how much exposure' accumulates over time. It is particularly important for comparing short, relatively high handset exposures with low-level but persistent base-station exposure (Li et al., 2012).

Question 9 — Technical and exposure information that should be disclosed

For personal wireless devices, public technical information should include supported bands, applicable SAR or absorbed-power-density compliance values, test positions/separation distances, simultaneous-transmitter assumptions, and the conditions under which the device was tested. For fixed infrastructure, useful information includes operating bands, antenna height and orientation, sectorization, maximum authorized and typical operating power, beamforming characteristics where applicable, and the method used to establish public-access compliance.

For research use, machine-readable technical metadata would be particularly valuable because it would permit reconstruction of historical exposure during epidemiological follow-up and facilitate separation of frequency bands and technologies.

The information presented should also be audience-specific. Consumers and patients need clear device-use and test-condition information; residents, schools and local governments need source-specific environmental measurements and understandable uncertainty ranges; workers need task- and time-resolved occupational exposure data; healthcare providers need concise information on medical-device compatibility and the limits of symptom attribution; and researchers need machine-readable technical metadata sufficient for exposure reconstruction and replication.

Question 10 — Data sources and surveillance systems

No single surveillance system can answer RF/EMF health questions. The highest value would come from interoperable linkage of disease registries, longitudinal cohorts, environmental monitoring, occupational measurement, device/network metadata, and biomonitoring. Each source should retain enough technical detail to reconstruct exposure over time rather than only classify participants as 'exposed' or 'unexposed'. Cancer registries are essential for incidence trends but usually have little individual exposure information and should not be assumed to be perfectly complete. For CNS tumors, HHS should encourage periodic completeness audits and linkage with inpatient, mortality, pathology, imaging, and other relevant data sources so that non-histologically confirmed cases and classification changes are captured as consistently as possible (Barlow et al., 2009; Parkin and Bray, 2009; Tettamanti et al., 2019). Environmental monitoring networks can define geographic and temporal distributions but do not measure personal dose. Longitudinal cohorts can establish temporality but require repeated exposure validation as wireless technologies change. HHS should prioritize linkage and validation rather than treat any single registry trend as a definitive test of RF carcinogenicity.

Biomonitoring programs can add repeated biological endpoints when exposure is measured objectively, but they require pre-specified endpoints, appropriate controls and clear interpretation of clinical relevance. Adverse-event or symptom-reporting

systems can detect signals and unusual temporal patterns, but are vulnerable to reporting bias and cannot establish causality by themselves. Disease-cluster investigations can be useful when case definitions, geographic boundaries, latency assumptions and exposure reconstruction are specified in advance; otherwise they can generate misleading spatial associations. HHS should use these systems as complementary signal-detection tools rather than substitutes for analytical epidemiology.

Question 11 — Sensitive populations

Children, pregnant individuals, older adults, workers with elevated exposure, persons with implanted or wearable medical devices, and people with relevant pre-existing conditions should be considered explicitly in exposure assessment. Inclusion of these groups does not presume greater susceptibility; it recognizes that anatomy, physiology, developmental timing, device interaction, behavior and source proximity can differ from assumptions based on healthy average adults.

Developmental timing and long post-exposure follow-up deserve particular study. Gelenli Dolanbay et al. (2025) reported reproductive and cellular differences in male rat offspring examined one year after prenatal-only 3.5 GHz exposure, with more pronounced findings after exposure throughout gestation than after later gestational exposure. Related thyroid studies using a prenatal 2.45 GHz exposure model reported no significant differences in TUNEL-positive cells or H2A.X at six months (Ozyilmaz et al., 2024), while a one-year follow-up study reported histological differences together with increased apoptosis and H2A.X (Özyilmaz et al., 2025). These studies assessed long-term postnatal outcomes in offspring following prenatal exposure, rather than delayed somatic effects in the exposed dams. They do not by themselves establish a delayed causal effect or human risk; rather, they illustrate the importance of long-term follow-up of offspring, validated dosimetry, and pre-specified endpoints for persistence, recovery, and delayed effects.

Question 12 — Evidence at exposure levels below current federal limits

Biological findings have been reported at exposure levels that are numerically low or below some guideline reference values, but published experiments should not be labelled simply as 'below current U.S. federal limits' without checking the applicable frequency range, spatial and temporal averaging, averaging mass, whole-body versus local exposure, source geometry and simultaneous transmitters. Examples relevant to biological activity include low-SAR lymphocyte and stem-cell studies of DNA-repair-foci responses (Belyaev et al., 2009; Markova et al., 2010), measured environmental base-station biomonitoring studies (Zothansiana et al., 2017; Gulati et al., 2024), and developmental animal studies at 3.5 GHz (Gelenli Dolanbay et al., 2025, 2026). None of these findings, individually, demonstrates a population-level adverse health effect or regulatory non-compliance.

The appropriate HHS response is therefore not to ignore sub-limit findings, but to rank them by reproducibility, exposure quality, endpoint validity, and relevance to adverse outcome. Parameter-specific replication is critical. A negative experiment at a different frequency, waveform, exposure schedule, or biological model is informative, but it is not necessarily a direct replication of a positive experiment under another condition.

Question 13 — How newer and connected technologies affect cumulative exposure patterns

Newer and connected technologies should be evaluated by their actual exposure characteristics rather than by the technology label alone. The HHS categories include 5G, 6G, Wi-Fi, satellite communications, Internet-of-Things devices, wearables, smart homes and appliances, smart-city systems, autonomous vehicles, wireless medical devices, and smart meters. Across these categories, relevant variables include operating frequency, bandwidth, modulation, beamforming or spatial focusing, duty cycle, proximity to the body, coexistence with legacy networks and other sources, and duration of use.

5G and future systems can change exposure patterns through new frequency bands, dynamic resource allocation, beamforming, denser infrastructure, changing uplink/downlink balance and coexistence with legacy networks. Experimental programs should use standardized signals with documented bandwidth, modulation, duty cycle, traffic assumptions, field uniformity, SAR or absorbed-power-density metrics, and thermal monitoring. At higher frequencies, where absorption becomes increasingly superficial, skin and ocular dosimetry should be characterized explicitly; superficial energy deposition is a dosimetric fact and should not itself be interpreted as evidence of greater harm.

Question 14 — Potential adverse environmental effects

This response does not attempt a substantive ecological health-effects review. That is a scope limitation, not evidence that environmental effects are absent. Wildlife, livestock and plant evidence requires species-specific review with appropriate dosimetry and ecological context. HHS could support a separate systematic evidence map coordinated with environmental and agricultural agencies, emphasizing measured exposure, species-appropriate dosimetry, sham controls, replication and explicit signal characterization.

Question 15 — Evidence for evaluating wireless infrastructure

Infrastructure studies should not use distance from a base station as the sole exposure metric. Distance can be a useful screening variable, but actual exposure also depends on antenna height, downtilt, azimuth, traffic, propagation, building attenuation, other nearby sources, antenna density and deployment of small cells. Small cells may operate at lower power than macro-sites but can be closer to people and more spatially numerous; the net change in exposure therefore should be measured rather than inferred from site count alone. Studies near homes, schools, workplaces, hospitals and long-term-care facilities should characterize time-resolved, source-specific exposure rather than classify a location only by proximity.

A useful future design would combine repeated indoor and outdoor RF measurements, source-resolved downlink characterization, handset uplink information where feasible, time-activity data, and longitudinal biological or clinical outcomes. Uplink and downlink should be analyzed separately because they arise from different source geometries and temporal patterns, while a cumulative total-exposure estimate can be derived when a common dosimetric basis is available. This is directly relevant to mobile-phone epidemiology because base-station/downlink exposure may contribute materially to total RF exposure over long periods. Livestock should be addressed through dedicated, species-specific agricultural studies rather than extrapolated from human biomonitoring or wildlife observations; this submission does not claim a completed livestock health-effects review.

Question 16 — Highest-priority federal research gaps

1. Parameter-resolved exposure biology. Conduct controlled studies that vary frequency, modulation/waveform, duty cycle, exposure duration, and recovery interval systematically rather than treating RF as a single exposure. The goal should be to identify which parameters are biologically relevant and which are not, while examining potential differences in biological susceptibility across cell types, tissues, animal models, and human populations (Belyaev, 2010; Belyaev et al., 2009; Markova et al., 2010; Belyaev and Dasdag, 2026; IARC, 2013).
2. Complete electromagnetic-environment characterization. In RF experiments and replication studies, measure and report static magnetic fields, ELF magnetic-field spectra and amplitudes, 50/60-Hz components, chamber/background fields, and intentional or incidental magnetic noise. Where feasible, use matched SMF/ELF conditions across laboratories or deliberately vary them as pre-specified experimental factors to test whether they modify RF outcomes (Makinistian et al., 2018; Misek et al., 2023; IARC, 2013).
3. Independent replication. Replicate influential positive and negative findings using matched dosimetry, biological model, temperature control, exposure schedule, endpoint timing, and blinded analysis.
4. Long-term carcinogenicity replication. For direct tests of NTP-type findings, future studies should be designed to match or exceed the sensitivity of the original experiment: adequate group size for rare tumors; several dose levels rather than a single dose; inclusion of the highest informative exposure level; pre-specified trend tests; matched signal/modulation conditions; harmonized dosimetry and electromagnetic background; and complete pathology review. The Japan/Korea experience shows why a related follow-up study and a strict replication should not be treated as equivalent concepts (NTP, 2018; Ahn et al., 2022; Imaida et al., 2026; Kim et al., 2026; Melnick et al., 2026).
5. Modern 5G exposure systems. Use standardized, fully documented 5G NR downlink and uplink signals at bands of high population relevance, including approximately 700 MHz and 3.5 GHz, with measured and simulated dosimetry.
6. Developmental and life-course studies. Examine prenatal and childhood windows with long-term follow-up, informed by recent 3.5 GHz and 2.45 GHz animal studies but without presuming human susceptibility. Designs should distinguish exposure during development from postnatal exposure and pre-specify persistence, recovery and delayed outcomes (Gelenli Dolanbay et al., 2025; Ozyilmaz et al., 2024; Özyilmaz et al., 2025).
7. Cumulative RF dose. Develop and validate cumulative exposure metrics that integrate intensity over time. For tissue exposure, report time-integrated SAR (specific absorbed energy, J/kg) where feasible; for far-field environmental exposure, report time-integrated power density and, where possible, convert external measurements to organ-specific absorbed dose using computational dosimetry. Dose metrics should preserve source identity and signal characteristics so that equal cumulative energy is not assumed to imply biological equivalence (Belyaev, 2010; Li et al., 2012).
8. Prospective human cohorts. Use repeated personal or source-resolved instrumental measurements and calibration substudies rather than relying only on recall or subscription history (Reedijk et al., 2024).
9. Longitudinal base-station research. Combine measured residential exposure with repeated cytogenetic, molecular, or clinical endpoints and adequate sample size; current biomarker studies are small and cross-sectional (Zothansiam et al., 2017; Gulati et al., 2024).

10. Mechanistic integration. Link cytogenetics, DNA-repair signaling, oxidative stress, transcriptomics, methylomics, proteomics, and single-cell approaches to pre-specified mechanistic hypotheses rather than exploratory endpoint accumulation.
11. Tumor promotion/progression. Study whether RF exposure can influence growth or progression of already initiated cell populations in addition to initiation, because epidemiological patterns may differ under those mechanisms (Kundi and Hutter, 2026).
12. Open science. Require exposure files, calibration records, protocols, raw or appropriately de-identified data, image archives, and analysis code whenever feasible.
13. Publicly funded independent research. Establish competitive federal funding streams for key RF/EMF questions and independent replication. Funding source alone should not be used to accept or reject a study, but sponsor-related differences in reported outcomes have been observed in systematic analyses of RF experimental research (Huss et al., 2007; van Nierop et al., 2010). Public funding should be paired with preregistration, independent scientific oversight, transparent conflict-of-interest disclosure, and publication/data-access commitments.
14. Recovery and persistence. Determine whether transient or reversible RF-induced biological responses remain reversible under repeated or chronic exposure schedules, show adaptation, or become persistent after incomplete recovery. The study question should be pre-specified and should distinguish persistence of a biomarker from adverse tissue injury or disease.
15. Higher-frequency superficial-tissue dosimetry. For millimeter-wave and other higher-frequency exposures, improve dosimetry and thermal characterization of skin, cornea and other superficial tissues, including spatial peak structure and realistic intermittent exposure. This priority reflects different energy-deposition geometry, not an assumption of greater hazard.

Question 17 — Improving federal coordination

Federal coordination should assign complementary roles. HHS/OASH can coordinate a common research agenda and evidence standards; NIH/NIEHS/NTP can support independent mechanistic, epidemiological, developmental and chronic-animal research and replication; FDA can contribute health-evidence review, medical-device safety and interaction expertise, and coordination of device-related health questions; CDC can contribute public-health surveillance, epidemiologic methods, risk communication and structured approaches to symptom or disease-cluster investigation; FCC can provide device and infrastructure technical and compliance data needed for exposure reconstruction; and NIST can provide measurement traceability, reference methods and inter-laboratory dosimetry. Shared exposure metadata and interoperable data standards would make these roles more effective.

Federal grants should require prospective registration of primary hypotheses and endpoints, complete exposure documentation, disclosure of all funding and sponsor roles, and data-sharing plans. These safeguards address selective reporting and reproducibility regardless of funding source.

Question 18 — Additional evidence and recommendations

First, totality-of-evidence assessment should not be reduced to counting positive and negative studies. A study contributes according to exposure validity, risk of bias, statistical precision, biological relevance, replication, and consistency with other evidence streams. Bradford Hill considerations are useful for causal reasoning but should not be applied as a mechanical checklist (Hill, 1965; OHAT, 2019).

Second, the validity of the exposure construct should be separated from the reliability of its measurement. A self-reported variable such as cumulative call time may be measured reproducibly but still be only indirectly related to tissue RF dose. The WHO-commissioned review by Karipidis et al. (2024) concluded with moderate certainty that mobile-phone RF exposure likely does not increase risks of glioma, meningioma, acoustic neuroma and several other tumors. That conclusion should be represented accurately. At the same time, uncertainty about whether behavioral surrogates capture biologically relevant exposure can affect certainty even when the direction of the epidemiological estimate is unchanged (Karipidis et al., 2024; Reedijk et al., 2024).

Third, evidence synthesis should preserve exposure heterogeneity. If studies of 905 MHz GSM, 915 MHz GSM, 1947 MHz UMTS, 1.8 GHz far-field GSM, 900 MHz whole-body GSM/CDMA, 3.5 GHz developmental exposure, and 2.45 GHz developmental exposure are combined under the single label 'RF', biologically meaningful differences may be hidden. The correct approach is not to assume that every parameter difference is important, but to test whether it modifies outcome and to avoid pooling until equivalence is reasonably supported (Belyaev, 2010; Belyaev et al., 2009; NTP, 2018; Falcioni et al., 2018; Gelenli Dolanbay et al., 2025; Ozyilmaz et al., 2024; Özyılmaz et al., 2025).

Exposure heterogeneity also includes co-existing non-RF electromagnetic fields. A study conducted at a given RF frequency and SAR is not necessarily electromagnetically equivalent to another study if the static and ELF magnetic-field environments differ materially. This is not a speculative concern only: IARC reviewed experimental evidence in which weak ELF magnetic noise

reduced RF-induced responses, and static-field magnitude altered resonance-type microwave effects. Therefore, systematic reviews and replication assessments should record whether SMF/ELF background was measured and whether major differences between studies are known (IARC, 2013; Litovitz et al., 1997; Lai and Singh, 2005; Yao et al., 2008; Ushakov et al., 2006; Misek et al., 2023).

Fourth, HHS should explicitly distinguish absence of evidence from evidence of absence. Statistical non-significance is not evidence that an effect is zero. Evidence of absence requires a study or body of studies capable of excluding effects of relevant magnitude: adequate power, validated and sufficiently precise exposure assessment, coverage of the relevant exposure range, adequate latency and follow-up, low risk of bias/confounding, and confidence intervals narrow enough to exclude an important increase in risk. This principle is consistent with the IARC framework, in which evidence suggesting lack of carcinogenicity requires several adequate and mutually consistent studies with narrow confidence intervals and reasonable exclusion of bias, confounding, and exposure misclassification (IARC, 2013).

Cancer-registry trends provide a useful example of this principle. Where registry ascertainment is sufficiently complete and stable, stable population incidence is stronger evidence against a large, common, short-latency effect than against a modest, exposure-specific, subtype-specific, or long-latency effect. Registry data should therefore be used to test explicit quantitative scenarios, with sensitivity analyses for completeness, latency, diagnostic change, and exposure prevalence, rather than as a categorical argument that positive epidemiological findings are impossible (Deltour et al., 2012; Little et al., 2012; de Vocht, 2016; Deltour et al., 2022).

For RF epidemiology, this distinction has direct practical consequences. If exposure is represented mainly by call time or years of use, while actual tissue dose varies with network generation, adaptive power control, handset position, downlink exposure, modulation, and other parameters, a null association may reflect attenuation by exposure misclassification. For brain and head tumors, failure to preserve laterality can add further misclassification because the local handset exposure is asymmetric; ipsilateral and contralateral associations should therefore be analyzed separately while accounting for recall bias. Likewise, a null rare-tumor result in an underpowered single-dose animal study is not equivalent to evidence that the effect is absent. The scientifically appropriate wording is therefore often 'an effect was not demonstrated under the conditions and sensitivity of the study' rather than 'there is no effect.' Fifth, chronic-exposure epidemiology should distinguish exposure rate from cumulative dose. SAR (W/kg) is a rate quantity; for long-term exposure, the time integral of SAR provides a cumulative absorbed-energy metric (J/kg). Similarly, time-integrated power density can represent cumulative incident far-field exposure. A population-based study by Li et al. (2012) illustrates the epidemiological principle by integrating base-station power and duration into a watt-year exposure index, although that ecological metric was not a tissue dose. This distinction is particularly important when comparing brief handset uplink exposure with persistent base-station/downlink exposure. Recent real-world 3G/4G/5G dosimetry found downlink whole-brain SAR comparable to or exceeding uplink SAR in most tested scenarios, reinforcing the need to include infrastructure-related exposure in mobile-phone epidemiology (Belyaev, 2010; Li et al., 2012; Lee et al., 2026). However, cumulative dose should be analyzed together with frequency, modulation, waveform, intermittency, and exposure schedule rather than used as a universal scalar of biological equivalence.

Finally, HHS should consider the credibility value of independent public funding. The RF literature includes published analyses reporting associations between sponsor type and whether experimental studies reported effects (Huss et al., 2007; van Nierop et al., 2010). These analyses do not prove that any individual industry-funded or publicly funded study is biased. They do support a practical recommendation: pivotal findings should be replicated under public or otherwise independent funding, with preregistration, transparent protocols, and open data.

Concluding Statement

The classical toxicological principle commonly summarized as “the dose makes the poison” is also relevant to RF/EMF research. However, exposure dose should be interpreted together with frequency, modulation, waveform, geometry, intermittency, and exposure schedule rather than treated as a universal scalar.

The central scientific problem is not simply whether 'RF exposure' is harmful or harmless. RF/EMF is a family of exposures whose biological relevance may depend on frequency, modulation, waveform, intensity, temporal pattern, geometry, duration, tissue dose, biological context, and the surrounding electromagnetic environment. For chronic exposure, HHS should distinguish exposure rate from cumulative dose and encourage time-integrated metrics rather than relying on instantaneous SAR or power density alone (Belyaev, 2010). At the same time, cumulative dose must not erase signal-specific information or co-exposure conditions. Static and ELF magnetic fields can modify RF responses in some experimental systems, and mobile phones themselves can emit ELF magnetic components; therefore, replication and evidence synthesis should characterize the complete electromagnetic environment rather than only the RF carrier and nominal SAR (Misek et al., 2023). HHS can improve the evidence base by requiring complete exposure characterization, testing parameter dependence directly, funding independent replication, strengthening prospective human exposure assessment, and integrating epidemiological, animal, and mechanistic

findings with transparent uncertainty. This approach is neutral with respect to the eventual direction of the evidence; its purpose is to make that evidence more reproducible, interpretable, and useful for health risk assessment.

Given the expansion of the human, animal and mechanistic evidence base since IARC Monograph 102, HHS could support an updated, independent hazard evaluation of RF-EMF. Any future IARC re-evaluation should follow the current IARC Preamble, integrate human epidemiology, experimental-animal findings and mechanistic evidence, and should not predetermine whether the resulting classification remains Group 2B or changes (IARC, 2019; Mevissen et al., 2025, 2026; Belenki et al., 2026).

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