

PUBLIC COMMENT — RF/EMF EXPOSURE AND HUMAN HEALTH (FULL TECHNICAL SUBMISSION)

To: U.S. Department of Health and Human Services, Office of the Assistant Secretary for Health
Re: Request for Information on EMF Exposure and Human Health; Docket HHS-OASH-2026-0397

Date: October 21, 2026

Respondent: [Name / anonymous, to be completed by submitter]

Perspective (Question 2): Individual member of the public; recommendations based on identified peer-reviewed studies, government materials, published technical guidance, and explicitly identified observations. Not presented as original clinical research.

Questions 3–4 and 10–11 — Measure functional impacts and create a clinical reporting pathway

1. Commission a representative national study of symptoms attributed to EMF and their social and economic impacts. A population-based, repeatable survey should distinguish (a) self-reported sensitivity, (b) symptom severity, (c) medically assessed functional impairment, (d) reported triggers, and (e) independently measured exposure. Include changes in employment and income, work hours, relocation, housing costs, attendance at concerts and public venues, travel, medication and healthcare use, school attendance, and expenditures by spouses, parents, and caregivers. Invite people with persistent or unexplained symptoms to participate whether or not they previously suspected RF/EMF as a possible factor. Accept descriptions of experiences that began years earlier without requiring old symptom diaries or contemporaneous exposure measurements. Where feasible, offer optional follow-up symptom tracking and independently measured exposure. Publish weighted prevalence and confidence intervals by impairment level and age.

Existing surveys do not establish how many U.S. residents have substantially altered their lives because of RF exposure. A California survey of 2,072 people reported 3.2% self-identifying as allergic or very sensitive to nearby electrical devices; a Swedish study reported 1.5%, and an Austrian survey reported 3.5%. These questions covered somewhat different exposure concepts, often not RF specifically, and assessed self-attribution rather than clinically confirmed RF causation. The World Health Organization recognizes that some people who attribute symptoms to EMF stop work or change their lifestyles. An exploratory UK secondary analysis estimated 0.65% with restricted access to work, but that estimate was not a direct U.S. population measurement. The actual magnitude of economic and family impacts should be measured rather than presumed from national extrapolations. [2–6]

2. Create a clinical documentation and surveillance protocol without presuming a diagnosis. A standard clinical intake template should capture symptoms and functional impairment, onset, alternative medical explanations, technologies and suspected waveform triggers, changes after reported exposure and reduction, and objectively measured exposure where available. Offer clinicians training and voluntary sentinel-site reporting. ICD-10-CM already includes an external-cause code for exposure to radiofrequency (W90.0XXA/D/S), but

it is not a validated diagnostic code for electromagnetic hypersensitivity (EHS). Evaluate whether an additional standardized encounter descriptor or surveillance extension is needed for symptoms *attributed by the patient* to EMF, clearly distinguished from proven causation. [6–8]

3. Publish practical disability-accommodation guidance. Offer clear information about individualized requests, documentation of functional limitations, and practical options: wired Ethernet and non-wireless peripherals, alternatives to wireless security devices, seating or service arrangements, remote participation, alternate meeting spaces, low-transmission work areas, and reasonable scheduling adjustments. The U.S. Access Board already recommends case-by-case measures that can include switching off phones/computers or enabling remote participation. The ADA determination remains individualized and does not depend on federal acceptance of RF as the causal mechanism of a patient’s symptoms. [9–10]

Questions 5–9 and 13 — Rebuild public exposure reporting and give occupants meaningful control

4. Standardize measurement units and disclose what each measure means. Require consumer-facing RF power-density disclosures to show W/m^2 and, for low environmental readings, $\mu\text{W}/\text{m}^2$; display existing mW/cm^2 values in parallel when needed to preserve consistency with FCC filings. The conversion is exact: $1 \text{ mW}/\text{cm}^2 = 10 \text{ W}/\text{m}^2 = 10,000,000 \mu\text{W}/\text{m}^2$; $1 \mu\text{W}/\text{cm}^2 = 10,000 \mu\text{W}/\text{m}^2$. Explain the frequency, source, sensor position, measurement bandwidth, peak or mean statistic, averaging period, uncertainty, and background reading. Requiring square-meter units does not imply that a square meter of human tissue was measured or that multiplying by human skin area yields absorbed dose. Whole-body/local SAR (W/kg), absorbed power density where appropriate, and/or modeled body geometry require separate assessment. [11–12]

5. Develop a publicly accessible exposure *profile* rather than a single pass/fail number. For homes, schools, workplaces and publicly accessible infrastructure, adopt a measurement protocol that retains frequency-resolved peak and time-averaged field levels, pulse and modulation characteristics, duty cycle, transmitter activity, antenna direction, relevant spatial gradients, and concurrent transmissions. Report separately RF (e.g., broadcasting, cellular, Wi-Fi, Bluetooth) and ELF/static electric and magnetic fields; unlike units must not be summed into a false universal “body load.” Establish a national inter-laboratory calibration and measurement-comparability program. [11–13]

6. Establish a historical exposure reference program. Develop a frequency-specific, geographically stratified historical atlas: (i) natural background, including static geomagnetic fields and lightning-generated Schumann resonances ($\sim 7.8 \text{ Hz}$); (ii) electrification-era 50/60 Hz fields; (iii) mass radio/TV broadcasting; (iv) cellular and residential wireless networks; and (v) present and future dense-device environments. This would be a comparison of exposure *environments*, not an assertion that Schumann resonance defines a safe RF threshold. Natural ELF, power-frequency fields, and RF require separate units, instruments and dose metrics. Published historical measurements are incomplete, so do not assign invented numerical tiers; use archival data, reconstructed engineering models and contemporary matched controls, each labeled by uncertainty. [14–15]

7. Require consumer control over wireless transmission strength and range. Require manufacturers of wireless products used in homes and businesses—including Wi-Fi routers, security cameras, video doorbells, smart-home devices, and similar equipment—to provide consumers and professional installers with simple, accessible controls for adjusting wireless transmission strength. Users should be able to reduce wireless signal strength to the lowest level necessary for reliable operation rather than being limited to a fixed, potentially excessive setting. Manufacturers should also offer automatic power adjustment, scheduled wireless operation, and the ability to turn off wireless transmissions when unnecessary, wherever technically feasible. These controls would be especially valuable in apartments, condominiums, and densely populated neighborhoods where numerous devices may transmit into surrounding residences. Provide wired alternatives when feasible, and test coverage, reliability, accessibility, safety and total system energy use; adjusting transmission strength does not guarantee an exact signal range. [16]

8. Compare additional voluntary residential-exposure guidelines without treating them as federal limits. Compare guidance from the U.S. Building Biology Institute and Germany's Institut für Baubiologie + Nachhaltigkeit (IBN), which publishes the SBM-2024 Building Biology Evaluation Guidelines. These classify single-source *peak* RF power density ($\mu\text{W}/\text{m}^2$) as <0.1 (no anomaly), 0.1–10 (slight), 10–1,000 (severe), and >1,000 (extreme). The labels are building-biology screening conventions, **not demonstrated human injury thresholds and not equivalents of legally binding FCC/ICNIRP limits.** Require explicit comparison of endpoint, measurement method, uncertainty and scientific validation before adopting any values. [17–18]

Questions 11–12 and 15 — Protect sensitive locations through verified engineering, not distance alone

9. Require antenna-beam and cumulative-exposure analysis before and after siting. For new or materially modified cellular installations near homes, schools, childcare facilities and healthcare settings, require disclosure to the public of antenna height, azimuth, mechanical/electrical downtilt, operating bands, maximum authorized power, beamforming envelope, and predicted/verified field levels at occupied elevations (including upper stories), with commercially sensitive details handled appropriately. Require independent measurements along likely main-beam directions, outdoors and at representative indoor locations, and publish clear results and complaint-response processes.

Peer-reviewed base-station measurement studies show that alignment with the antenna's **main lobe** and line of sight can matter more than horizontal distance alone; a French residential study found band-dependent maxima at approximately 280 m and 1,000 m, and other field studies assessed orientation toward occupied building floors. [20–22]

Questions 4, 10–12 and 16–18 — Study individuals and cumulative impacts without deliberately exposing people to plausible injury

10. Make observational epidemiology a central research track. Build longitudinal prospective cohorts with home, school, occupational and personal exposure measurement; tumor outcomes should be classified by histology (including glioblastoma separately), age and

changes in diagnostic practice. Record co-exposures and plausible disease-promoting interactions. High-risk human dosing is neither required nor appropriate to study long-latency health effects. Use animal studies and ethically controlled low-risk or noninterventional protocols to examine candidate mechanisms. NTP/Ramazzini findings and mouse tumor-promotion experiments justify follow-up [47–53]. Additional histology-specific epidemiology is needed to investigate long-latency outcomes. [24, 47–54]

11. Investigate reported individualized signal triggers through inclusive symptom-history research. Invite participants to describe symptoms and environmental changes without requiring them to have previously identified RF/EMF as a cause. Record participants' best recollections of when symptoms appeared, nearby technologies, and any signal characteristics known to them; historical diaries and prior measurements should not be required. Offer optional symptom tracking and independent exposure measurement during follow-up. Where ethically appropriate, pre-register within-person comparisons with verified sham controls, realistic traffic patterns, counterbalancing, blinded outcomes, and evaluation of other possible triggers. Whether a signal present near symptom onset becomes the most troublesome later trigger is a hypothesis to investigate, not an established biological mechanism. [6, 25]

12. Institute independence and disclosure rules for federal RF/EMF research. For each cited study and federally commissioned project, list direct, indirect and in-kind funding, equipment provision, advisory roles, institutional grants, and investigator conflicts. Separate confirmed independent, telecommunications-related, mixed, unreported and *not yet verified* categories; universities should not automatically be classified as independent, nor should industry funding automatically invalidate a study. Publicly preregister protocols and make positive and null findings accessible. Maintain different evidence tracks for human outcomes, animal findings, oxidative stress, mitochondrial pathways, methylation, VGCC/calcium proposals and other candidate mechanisms. The accompanying research inventories should not be described as independently audited systematic reviews. Include the original positive and null reports and request routine public release of full protocols, corrections and institutional support. [26, 29–30, 52–53, 66]

Questions 11, 12, 16 and 18 — Mitochondrial integrity and offspring health

13. Establish a research program on potential intergenerational effects, especially mitochondrial DNA. Investigate whether realistic RF exposure produces persistent mitochondrial DNA (mtDNA) sequence mutations in *maternal reproductive cells*, whether those variants survive repair and germ-cell selection and are passed to offspring, and whether inherited changes affect measurable outcomes such as mitochondrial respiration, growth or neurodevelopment. Distinguish an oxidative-damage marker or changed mtDNA copy number from a heritable mutation. Compare sham-controlled maternal preconception, pregnancy and multigenerational animal designs with ethically observational human cohorts where feasible; investigate paternal nuclear and sperm-related pathways separately. The mitochondrial bottleneck and maternal inheritance are well established, and neuronal RF experiments report mtDNA oxidative alterations, but **transmission of RF-induced mtDNA mutations from mother to child has not been demonstrated**. Include null studies and reproducibility testing. [38–44, 64, 68]

Purpose of these recommendations: improve the population's ability to understand, document and voluntarily reduce avoidable RF exposure while generating reliable evidence about health, functional impairment and costs. None of these proposals requires mislabeling unproved associations as established causes.

Evidence-to-recommendation map — research and implementation rationale

The linked materials below show why each recommendation warrants investigation or implementation. “Direct” means the cited work studies the relevant observable phenomenon, **not** that it proves RF causes human disease. Policy/engineering proposals are identified separately.

- **1. Prevalence and economic effects:** Best evidence and links: Surveys [2–4]; WHO [6]; symptom review [45–46]; Evidentiary scope / remaining gap: Historical self-report; no current nationally representative functional/economic burden study.
- **2. Clinical documentation:** Best evidence and links: ICD-10-CM [7]; WHO [6]; human symptom reviews [45–46]; Evidentiary scope / remaining gap: Existing exposure code but no validated EHS diagnosis; standardized reporting is proposed.
- **3. Access and accommodation:** Best evidence and links: U.S. Access Board [9]; EEOC [10]; Evidentiary scope / remaining gap: Accommodation practice and legal feasibility, not evidence of RF causality.
- **4. Units/whole-body measures:** Best evidence and links: NIST [11]; ICNIRP [12]; Evidentiary scope / remaining gap: Metrological definitions and dosimetry; SI conversion is not whole-body absorbed dose.
- **5. Characterize complex exposure:** Best evidence and links: ITU-T [13]; base-station measurement [62–63, 69]; Evidentiary scope / remaining gap: Field techniques support a richer exposure profile; clinical thresholds remain unsettled.
- **6. Historical comparisons:** Best evidence and links: NOAA [14], NTIA [15]; Evidentiary scope / remaining gap: Contextual engineering record, not proof that natural ELF sets safe RF limits.
- **7. Adjustable household transmitters:** Best evidence and links: Existing power-control technology [16]; Evidentiary scope / remaining gap: Technically plausible policy trial; need whole-network performance and emissions tests.
- **8. Building-biology reference ranges:** Best evidence and links: U.S./German institutes [17–18], ICNIRP [12]; Evidentiary scope / remaining gap: Voluntary precautionary guidance must not be misrepresented as validated health thresholds.
- **9. Antenna/beam siting:** Best evidence and links: Empirical dosimetry [19–22, 62–63, 69]; Evidentiary scope / remaining gap: Main-beam exposure can differ from nearest-distance models; future standards can be informed by site-specific measurements.
- **10. Prospective epidemiology/tumor trends:** Best evidence and links: NTP [50], Ramazzini [51], animal review [52–53], human review [54], CBTRUS [61]; Evidentiary scope / remaining gap: Animal carcinogenic signals coexist with uncertainty about long-latency human tumor outcomes; GBM-specific trend analysis unfinished.
- **11. Individual signal triggers:** Best evidence and links: WHO [6]; sham-controlled reviews [25, 45]; Evidentiary scope / remaining gap: Blinded acute provocation is generally null; individualized long-term waveform-trigger hypothesis unverified.

- **12. Funding disclosure:** Best evidence and links: Huss [26]; California utility-funded survey [2, 34]; lab institutional affiliations [56]; Evidentiary scope / remaining gap: Support is documented for some, but the 206-record registry remains unaudited.
- **13. mtDNA and intergenerational research:** Best evidence and links: Xu [38], mother-child genetics [39–40], gamete findings [41–44, 64]; Evidentiary scope / remaining gap: Maternal inheritance established; RF-induced maternal germline inheritance **not shown**.

Cross-cutting biological evidence requiring independent replication: oxidative-stress experiments [29–30, 66], RF-associated DNA methylation in cultured cells [56], VGCC/calcium hypotheses [57], BBB studies [58], animal tumor-promotion experiments [47–49], and animal environmental/pollinator studies [59–60]. These are research priorities and mixed or preliminary findings, not established population-level hazards.

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PART II — TECHNICAL AND EVIDENTIARY SUPPORT FOR THE SAME PUBLIC COMMENT

A. Prevalence, disability and economic burden: what the data do and do not show

A1. Prevalence is a family of estimands, not a single percentage. The 2002 California survey by Levallois et al. recorded 68 positive answers out of 2,072 surveyed and an adjusted 3.2% (95% CI 2.8–3.7%) who described themselves as allergic or very sensitive near electrical devices. That measure is not identical to wireless RF-specific sensitivity and predates contemporary 5G, Wi-Fi 6/7 and ubiquitous wearables. Hillert et al. reported 1.5% in a Swedish questionnaire, while Schröttner and Leitgeb reported 3.5% in Austria. These findings warrant repeated standardized measurement; they do not establish a growing RF-caused clinical condition. **The California survey acknowledged funding through the utility-supported, California Public Utilities Commission-mandated California EMF Program; record the support as utility-funded through a state-administered program.** [2–4, 34]

A2. Contemporary U.S. population benchmark and 3.0% illustrations. The Census Bureau's Population Clock reported **342,958,024 U.S. residents on October 20, 2026**; this supports an approximate **343.0-million total-population benchmark on October 21, 2026**. Three percent of that rounded national total is **10.29 million, or approximately 10.3 million people**. This calculation is not a direct prevalence estimate; applying an adult-survey percentage to all ages assumes, without evidence, the same reporting propensity among children. Separately, the Census Bureau's July 2025 detailed age estimates record 341,784,857 residents, of whom 72,021,348 were younger than 18, leaving **269,763,509 adults (approximately 269.8 million)**. A 3.0% *adult-only* scenario corresponds to **approximately 8.1 million adults**. The historical claim that engineers were taught only 1% could be affected has not been traced to a reliable curriculum or published assessment. [2–4, 31, 35]

- **All U.S. residents (October 21, 2026, rounded):** Denominator: **343.0 million**; Illustration at 3.0%: **10.3 million people**; Qualification: Not a measured national prevalence; extrapolates adult evidence across all ages
- **U.S. adults (July 2025, published age counts):** Denominator: **269.8 million**; Illustration at 3.0%: **8.1 million adults**; Qualification: Adults better match historical survey populations; still not a current nationwide estimate

Historical adult surveys provide context, not estimates for today's U.S. population: Sweden 1.5% (roughly 4.0 million if applied to the 2025 U.S. adult denominator), California 3.2% (roughly 8.6 million), and Austria 3.5% (roughly 9.4 million). The populations, instruments, and periods differed. These are self-reports, not established rates of RF-caused illness or disability. [2–4]

A2a. Employment-specific anchor. Levallois et al. reported that approximately **0.5%** of adult Californians self-reported changing jobs or remaining unemployed in connection with perceived EMF sensitivity; the original article estimated **about 120,000 adult Californians** by extrapolating to that era's adult California population. Applied arithmetically to the contemporary national adult denominator, 0.5% would amount to **roughly 1.35 million U.S. adults**. This is an old, non-RF-specific, self-attribution measure—not evidence that the same rate persists nationally, nor that exposure caused the job change. A UK secondary analysis estimated approximately 0.65% with limited work access, but this is not a directly transferable U.S. national sample. The WHO recognizes reported severe restrictions, including cessation of work, while declining to infer a causal relationship between symptoms and measured EMF. [2, 5–6, 32]

A3. Recommended survey design. Use a probability sample of U.S. adults and older adolescents; oversample respondents identifying substantial activity restrictions and enable caregiver-linked questionnaires. Separate (1) any reported symptoms, (2) symptoms specifically attributed to electric/magnetic/RF sources, (3) severity, (4) functional limitations (including paid work lost, reduced shifts, avoidance of transit, attendance, spending on coping), (5) perceived cause, (6) clinical evaluation, and (7) passive instrument-logged exposure. Ask households about wireless equipment, other indoor conditions, sleep, medications and comorbidities. For a subset, use 2–4-week time-activity diaries plus source-specific calibrated exposure monitoring. Do not interpret a proxy variable such as concern about EMF as disease prevalence.

A4. Economic and household spillover analysis. Pre-specify: lost earnings and lost hours, involuntary job exit, household relocation, clinical care and medications, device substitutions and mitigation services, foregone activities, transport costs, and unpaid hours borne by partners, parents or caregivers. Estimate direct expenses and lost productivity separately and avoid double-counting the same household costs or time; distinguish an association with self-attribution from demonstrable RF causation. Capture possible savings or additional costs of wired access and lower-power settings. WHO describes cessation of work and substantial changes in lifestyle among a subset of people reporting EHS, without determining symptom etiology. [6]

Illustrative household exposure-to-consequences scenarios (not surveyed): To reflect consequences for spouses, children, partners and caregivers, define *additional people adapting routines per primary self-reporter*. This is **not a multiplier for EHS prevalence**. A hypothetical additional 0.5 people per primary self-reporter means one other affected household member for half the initial group, assuming no overlap.

- **0:** Total potentially changing routines, starting from the 8.1-million-adult illustration: 8.1 million
- **0.25:** Total potentially changing routines, starting from the 8.1-million-adult illustration: 10.1 million
- **0.50:** Total potentially changing routines, starting from the 8.1-million-adult illustration: 12.2 million

The household scenarios are arithmetic only: no empirical fraction of cohabitants changing their behavior was found. Real households differ in size, and two primary self-reporters may live together; for a defensible national estimate, survey and count **unique individuals and unique households**, not a naive multiplier.

Illustrative direct out-of-pocket cost scenarios (not measured): These assume the same annual incremental outlay per *primary self-reporting adult*—such as extra equipment, changed transportation arrangements, care expenses, or other verified additional costs. Do not assume all people report such costs, and exclude changes in discretionary spending unless directly measured.

- **\$250:** Adult-only 8.1-million-person illustration: \$2.0 billion/year
- **\$1,000:** Adult-only 8.1-million-person illustration: \$8.1 billion/year
- **\$2,500:** Adult-only 8.1-million-person illustration: \$20.3 billion/year

Illustrative employment opportunity cost: U.S. BLS reported median weekly pay of **\$1,204** for full-time wage and salary workers in 2025. If, solely as a sensitivity analysis, 10% of the 8.1-million adult-only illustration were employed and lost one week of paid earnings during a year, the arithmetic would imply **approximately \$0.98 billion**. The 10% affected-worker fraction and one-week duration have not been verified; the median does not describe all workers or equal marginal economic output, and this example is not an attributable national loss. Lost wages, paid-leave replacement, and lost business output must not be added together without careful accounting. The all-ages 10.3-million illustration is **not** used in employment-cost calculations. [33]

Empirical study design: Representative population studies should measure prevalence and severity with linked household questionnaires, capture actual receipts and changes in earnings for consenting participants, and stratify self-reported effects by occupational type, disability status, income, access to wired alternatives, home tenure, and co-occurring health conditions. This can produce upper/lower uncertainty intervals for direct costs, indirect costs, and household spillovers after data collection rather than suggesting that scenario arithmetic alone establishes the national burden.

B. Clinical documentation and practical accessibility

B1. Coding distinction. Existing ICD-10-CM code W90.0XXA (with D/S encounter variants) describes an external cause: exposure to radiofrequency. It neither confirms exposure caused a person's symptoms nor supplies clinical diagnostic criteria for IEI-EMF/EHS. Symptom-specific codes are available for headache, sleep disturbance, dizziness, sensory symptoms and the like. A better federal approach is a standardized symptom-and-attribution *encounter modifier* or structured data field for surveillance rather than hurriedly defining a new disease. Clinical utility, interoperability, privacy and misuse risk should be evaluated before any proposed coding changes. [7–8]

B2. Sample clinical intake variables. Presenting complaint; start date and course; locations; source suspected by patient; duration and temporal relation; whether exposure was measured or inferred; competing clinical explanations; interference with major life activities; treatment; medication; changes associated with attempted accommodation; and patient's proposed feasible modifications. Apply normal medical assessment and refer where indicated. Voluntary data contribution to a registry should be separate from care decisions.

B3. Accommodation context. Existing disability and accessibility frameworks address individualized accommodation and functional limitations. The U.S. Access Board's indoor-environmental-quality recommendations already discuss electromagnetic sensitivities and case-by-case options; these recommendations should not be presented as a federal medical diagnosis or as a ruling that all requested radio shutdowns must be granted. Case-specific reasonableness, essential services, safety, other people's accessibility needs and legal requirements matter. [9–10]

C. An improved RF/EMF measurement framework

C1. Four questions for every number. (a) What physical quantity was measured? (b) At which frequencies and locations? (c) During which intervals, operating modes and transmission patterns? (d) Which averaging geometry and body-exposure metric is involved? Do not equate electric-field strength (V/m), magnetic flux density (μT or pT), incident power density (W/m^2), absorbed power density (W/m^2), specific absorption rate (W/kg), or time-integrated energy. Even when some quantities can be converted for a plane wave under defined conditions, such relationships do not automatically hold for a complex indoor near field. [11–13]

C2. Mandatory consumer dual-unit display. Show both US legacy and SI power-density units. The following table uses *identical physical intensities* and is not a comparison of risks or exposed body areas:

- **1 mW/cm²:** Standardized reading: $10 \text{ W}/\text{m}^2 = 10,000,000 \mu\text{W}/\text{m}^2$

- **0.001 mW/cm²**: Standardized reading: $0.01 \text{ W/m}^2 = 10,000 \text{ } \mu\text{W/m}^2$
- **1 } \mu\text{W/cm}^2**: Standardized reading: $0.01 \text{ W/m}^2 = 10,000 \text{ } \mu\text{W/m}^2$
- **0.0001 } \mu\text{W/cm}^2**: Standardized reading: $1 \text{ } \mu\text{W/m}^2$

C3. Body exposure is a different question. A square centimeter is a unit of area in the *denominator* of power density, not a statement that a device tests only one square centimeter. Converting from cm² to m² changes a number's scale but not the sensed footprint. A whole-body dose may require body geometry, field distributions, polarization, shielding, orientation, tissue dielectric properties and frequency-dependent absorption; whole-body SAR is measured or modeled separately, subject to applicable standards. At high RF frequencies some standards explicitly distinguish spatial averaging over 1 cm² and 4 cm² from general incident exposure. [12]

C4. Template for standardized public reporting. Include facility/device ID; date and location; source categories; spectrum and operating bands; detector instrument, calibration and detection threshold; electric/magnetic field or power-density values; near/far field classification where appropriate; distance and line of sight; occupied height and orientation; modulation, pulse characteristics and duty; beam steering/max-power assumptions; sample duration and temporal percentiles; signal activity; measurement uncertainty; indoor/outdoor data; and both measured and appropriately modeled exposure. Publish a short plain-language explanation followed by an auditable technical annex. Any combined exposure metric must specify weighting and comply with frequency- and quantity-specific physics. [13]

D. Historical exposure baselines: construct a reference atlas, not an invented safety ladder

D1. A useful way to operationalize the proposal. Conduct parallel measurement series on modern representative sites and reconstruct characteristic earlier exposure periods from records, with explicit confidence ranges. Comparisons should be stratified by frequency, geography, built environment, time of day and personal-device use; use source classes rather than “all EMFs in one number.”

- **Natural baseline:** Representative sources / domain: Geomagnetic DC/static field, lightning-driven Schumann resonance (ELF); Measurement approach: Static/ELF magnetometers and electric-field monitoring; location and weather; Evidence status: Established natural phenomena; not an RF safety limit
- **Electrification:** Representative sources / domain: 50/60 Hz utility distribution, home wiring, electrical equipment; Measurement approach: Frequency-selective ELF E and B fields; period engineering reconstruction; Evidence status: Archival snapshots incomplete
- **Radio/TV expansion:** Representative sources / domain: AM/FM/TV broadcast; point-to-point RF; Measurement approach: Band-resolved E fields / power density from transmitter records; Evidence status: Frequency- and site-specific reconstruction needed
- **Early personal wireless:** Representative sources / domain: Early cellular networks, DECT, first household Wi-Fi; Measurement approach: Historic technical specifications;

traffic and adoption models; Evidence status: Major uncertainties in past real-world duty cycles

- **Contemporary dense networks:** Representative sources / domain: Cellular, Wi-Fi, IoT, smart security, wearables; Measurement approach: Continuous multiband occupancy and personal exposure profiling; Evidence status: Feasible to measure now; needs standardized methods

D2. Schumann resonance is not a transferable RF threshold. Its fundamental resonance occurs near 7.8 Hz and fluctuates with atmospheric conditions and lightning. The geomagnetic field is mainly quasi-static; NOAA quotes Earth-field intensities roughly 25–65 μT . A 7.8 Hz natural magnetic-field reading in μT cannot be ranked on the same W/m^2 exposure ladder as GHz communications without an explicitly justified metric and a biologically validated cross-frequency model. It is reasonable to ask investigators to quantify relevant natural backgrounds as reference context. [14, 28]

D3. Historical landmarks. Practical urban electrification began to spread in the late nineteenth century; public broadcast radio expanded in the 1920s (KDKA's 1920 public broadcast is documented by NTIA). The history does not, by itself, supply measured historical RF levels or prove the natural baseline was benign. Data collection should separate historical *source introduction* from quantitative *exposure measurement*. [15]

E. Design standard for domestic RF control

E1. The desired policy function. Offer residents ways to configure the smallest necessary transmission footprint, especially where neighboring apartments create dense networks of radios. Require interfaces for supported equipment that allow a controllable transmit-power cap and a clear description of what power scaling can and cannot change. At minimum, provide a wireless-radio off switch, scheduled transmissions when compatible with operation, and status indicators. Consumers should retain reliable emergency, fire/security and disability-critical functionality.

E2. Avoid a misleading guarantee. “20-foot range” does not describe a fixed RF boundary; propagation varies with walls, receivers, interference, antenna radiation patterns, and device power control. “100 devices” is a plausible illustrative scenario but should not be stated as a typical apartment measurement. In some networks, reducing an access point's power may cause phones and other clients to use more power or generate more retries, increasing airtime or battery consumption. Testing must monitor *all* radios and overall energy, not merely a router's advertised transmit-power rating.

E3. Implementation and enforcement. Equipment-authorization testing could evaluate the available power-control feature and require documentation of regulatory power limits; consumer-facing reviews could evaluate user interface, reasonable accessibility, and truthful labels within their authority. Manufacturers should publish beacon rates, bands, minimum/maximum setting and what happens after software update. Pilot procurement for federal buildings could inform larger rules. Automated TPC is a mature Wi-Fi-network engineering feature, exemplified by enterprise systems, but cross-device residential implementation requires feasibility review. [16]

F. Cell tower placement: test beam geometry, not just a radius

F1. The specific physical mechanism. A sector antenna's strongest emissions are directional. Depending on height, downtilt, gain, reflecting structures and terrain, main-beam intersections with occupied floors or the ground can occur at locations that are not the closest point to the tower. A 2007 GSM/UMTS measurement study explicitly reported that main-lobe orientation and visibility matter more than distance alone; a 2009 French survey observed GSM and DCS exposure maxima at ~280 m and ~1,000 m, respectively, under its studied conditions. One technical study measured an antenna beam directed toward a 10-story building 35 m away. These results justify tailored assessment of exposure zones, *not* general proof of injury in occupied buildings. [20–22]

F2. How to use Al-Allaq et al. (2023). Their IJECE article uses GIS and measured RF information to visualize spatial exposure from base stations. This is potentially useful for data collection, communication and comparison with reference limits; the GIS analysis is not a controlled epidemiologic test of health effects. Do not cite it to claim a medically demonstrated buffer zone. [19]

F3. Site-specific siting research concept. Select installations near residential buildings, healthcare sites and schools, including facilities at varying heights and antenna downtilt. Measure exposures across locations and building elevations under differing traffic loads. Model and verify predicted main-beam maxima, side lobes, reflections, multi-carrier traffic and planned future colocation. Publish comparable, map-based exposure results with independent review of discrepancies. Siting criteria could be evaluated using measured exposure distributions instead of preset buffers. Longitudinal symptom and disease surveillance can be linked to these measurements with appropriate privacy safeguards.

F4. Legal practicality. Under 47 U.S.C. 332(c)(7)(B)(iv), local authorities cannot use RF environmental effects to regulate compliant personal wireless facilities within the provision's scope. Therefore, a proposed health-based federal setback or new certification standard requires federal consideration and the proper legal authority; Existing limits on municipal authority must be considered when evaluating changes to exposure or siting rules. [23]

G. Epidemiology, ethical research design and specific signal histories

G1. Ethical hierarchy. Large health-outcome questions (including long-latency tumors) should primarily be pursued through noninterventional cohorts, registry linkages and historical exposure reconstruction. Human experiments can examine lower-risk acute endpoints with ethics committee oversight, informed consent and safeguards, while experimental toxicology studies evaluate mechanistic questions unsuitable for intentional potentially injurious exposure of human subjects. Long-duration, high-exposure dosing of healthy human volunteers is not necessary to produce epidemiological evidence. Historical parallels with tobacco support cautious ethics, not an assumption that RF shares tobacco's disease mechanism or demonstrated causal status.

G2. More informative exposure histories. An individual may perceive that the signal present at symptom onset remains the most disruptive. To test this prespecified hypothesis, record self-reported index episode date, source technology, carrier and spectral occupancy,

modulation/pulse characteristics, activity, device proximity, other environmental changes, and subsequent challenge or avoidance responses. Use blinded sham and objective outcome measures only where ethically permissible, and document anticipatory/nocebo effects and concurrent alternative exposures. Existing provocation studies generally have not found reproducible detection or acute symptom associations, which is precisely why prespecified personalized, realistic replication is necessary rather than assuming the hypothesis is true. [6,25]

G3. Disease and promotion outcomes. Use histology-specific and molecularly harmonized brain tumor series, including glioblastoma distinguished from meningioma, other gliomas and schwannomas; standardize population rates by age, calendar year and pathology coding. Study potential disease promotion via animal and mechanistic co-exposure factorial designs with preregistration and independent replication. Do not infer increases in glioblastoma incidence from aggregate brain tumor counts or outdated coding systems. The NTP rat cancer findings justify transparent follow-up studies; they do not alone establish risk to individuals from everyday residential RF exposure. [24]

H. Funding provenance, implementation and unresolved evidence

H1. Minimum public funding registry. For every study: record author, year, exact primary citation, primary institutional support, telecom/device industry donors, indirect sponsored research, industry equipment loans, advisory/consulting compensation, FCC funding and FCC-administered contracts, government and nonprofit support, sponsor role and remaining unknowns. Support each classification with the actual grant disclosure or documentation; do not infer from institutional branding. Huss et al. (2007) observed funding-related differences in reported findings in their reviewed experimental literature, providing rationale for this transparency measure; it does not establish that individual studies are invalid. [26]

H2. Existing evidence inventory limitations. This research project recovered three working files: 100 oxidative-stress papers reconstructed from the Yakymenko review (93 classified positive/7 null within that review); 43 supplementary ROS/redox publications; and a 63-record mitochondrial/mtDNA register. The counts are not pooled systematic-review results, are not necessarily mutually exclusive, include different field types in the mitochondrial register, and have not been exhaustively audited for sponsor provenance or methodological quality. A submission that presents them as 206 wholly independent, RF-only causal demonstrations would misstate their meaning. The specific rows should be offered as an audit-ready annex *only if they have undergone an additional quality and funding review*. [29–30]

H3. Evidence still required before claiming a finalized scientific determination. The all-ages 10.3-million and adult-only 8.1-million figures are *illustrative applications of 3.0%*, not measured populations with functional impairment; the all-ages figure additionally relies on an unverified extrapolation from adult survey reports. The job-disruption extrapolation uses a historical California self-report figure and does not establish current national employment loss. A modern, nationally representative U.S. EHS/lifestyle prevalence estimate is unavailable here; no validated health threshold is established by SBM peak categories; no historical numerical exposure ladder across ELF and RF has been verified; the appropriate siting and monitoring standards remain to be determined; the assertion that a first exposure uniquely determines a lifelong waveform trigger lacks prospective validation; and a study-level funding audit of our

historical inventories is unfinished. These are research priorities and limitations, not reasons to leave actionable measurement and accessibility recommendations unspecified.

Additional references for Part II

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I. Research justification: study-by-study scientific synthesis

I1. Human epidemiology remains important for long-latency outcomes. Existing reviews, including Karipidis et al. (2024) [54], highlight limitations in the evidence available for particular source types and outcomes. More prospective research combining measured personal and environmental exposure, histology-specific tumor classification and registry follow-up could help address unresolved questions. Use CBTRUS and consistent pathology categories to study glioblastoma separately from other brain tumors while accounting for changing diagnostic criteria, calendar period and population age structure [61].

I2. Animal carcinogenesis and promotion warrant carefully targeted replication. The NTP 900 MHz rat project reported clear evidence of male-rat malignant heart schwannomas; its exposure design and doses do not map directly to ordinary human residential settings [50]. Ramazzini's 1.8 GHz environmental-source experiment reported elevated male-rat heart schwannomas at its highest field strength [51]. Mevissen's 2025 systematic animal-cancer

review [52] and its 2026 corrigendum [53] should be evaluated together. Tillmann [47] and Lerchl [48] observed tumor-promotion signals in carcinogen-treated mice; a later systematic co-carcinogenic review [49] found mostly nonsignificant pooled findings and inadequate evidence for many outcomes. A preregistered independent study can examine combined carcinogen and RF exposures, including negative and dose-response controls.

I3. Biological mechanisms: signals, uncertainty and audit requirements. Yakymenko's narrative inventory reported positive oxidative outcomes in 93/100 selected papers [29]; our recovered workbooks include the original 100-row reconstruction and 43 additional redox publications. These are neither independent meta-analysis nor 143 verified findings of adverse human illness. Meyer et al. [30, 66] rated the overall certainty of the RF oxidative-biomarker literature very low. The separate 63-record mitochondrial workbook includes **30 direct RF/microwave, 18 ELF/PEMF, and 15 indirect/special cases**, including beneficial, null and adverse directions; these must not be pooled. A mechanistic federal program should prioritize standardized exposure waveforms, temperatures, sham/blinding, assay calibration, oxidative endpoints, mitochondrial respiration, mtDNA sequencing, repair response and reproducibility.

I4. Methylation and calcium are specific, testable hypotheses. Cantu et al. [56], from authors affiliated with General Dynamics Information Technology and the U.S. Air Force research system, found changed methylation and expression of selected genes after 900 MHz in-vitro keratinocyte exposure. This is **not** evidence of inherited epigenetic changes or of MTHFR-specific RF injury. Test methylation biology beyond MTHFR: one-carbon metabolites (folate, B12, homocysteine, SAM/SAH), DNMT function and genome-wide methylation, with genotypes as effect modifiers where justified. Pall [57] provides a VGCC hypothesis review rather than a conclusive human-mechanism demonstration. For calcium entry, relevant pools are extracellular fluid and intracellular stores such as endoplasmic reticulum, not a necessary acute release of calcium from bone. Use live-cell calcium measurements, specific blockers and genetic/thermal controls.

I5. Blood-brain barrier, cognitive and nonspecific symptoms need better-targeted work. A 2026 BBB scoping review assessed 17 studies reporting effects and 18 reporting none, finding no established causal RF effect [58]. A 2024 symptom experiment review reported largely null acute blinded comparisons [45]; a human observational symptom review noted very-low-certainty evidence [46]. These results should be reported fairly while studying the severe functional limitations some patients describe, without making a medical assessment depend on participants having identified RF as their original trigger or having preserved retrospective diaries. A separate 2024 experimental cognitive-performance meta-analysis is a useful protocol source [65].

I6. Environmental health. Honeybee studies [59–60] reported changes in stress-related endpoints and pollination, but ecological results can be confounded and cannot substitute for human safety evidence. Follow-up protocols could separate RF from ELF and other agricultural and environmental stressors.

J. Insurance evidence and proposed data requests

Lloyd's 2010 report [36] addressed the insurance implications of possible future EMF health claims. The *Hammond Power* decision [37] upheld a broad commercial radiation exclusion for

alleged injuries from transformer EMF; it did not find that EMF caused the plaintiff's reported injury and is not evidence of widespread health-insurance benefit exclusions for individual patients. Relevant policy documents differ by source type (RF versus power-frequency ELF), scope, dates and insurer, which complicates direct comparisons. Do not equate Lloyd's marketplace reports with the terms of every syndicate contract.

K. Inherited mitochondrial biology: a falsifiable multigenerational program

Human familial studies [39–40] demonstrate maternal inheritance, heteroplasmy and the germline bottleneck: harmful mtDNA variants can increase or decrease in proportions between generations. Xu et al. [38] found oxidative mtDNA lesions and copy-number/transcription differences in rat neuronal cultures after RF exposure; that experiment did **not** sequence heritable mutations in egg cells. Aitken [41] and another RF sperm study [42] suggest possible germline oxidative damage, but sperm mtDNA is not typically maternally transmitted and nuclear DNA endpoints must be distinguished. A null result in human sperm mitochondrial membrane potential [64] is also relevant. Animal pregnancy systematic reviews [43, 68] and fertility reviews [44] provide outcome-specific background, not proof of RF-induced inherited mtDNA mutations.

Proposed stepwise experiment: pre-register sham/blinded low- and high-exposure groups with multiple realistic modulation patterns; continuously verify dosimetry and tissue temperature; sequence maternal oocyte mtDNA with validated error-corrected methods and controls; measure mitochondrial respiration and heteroplasmy; track F1 and, where ethically/scientifically necessary, F2 offspring under standardized conditions; genotype nuclear DNA and assess maternal age, co-exposure, diet and stress; independently replicate positive results. Prespecify null and alternative explanations. Only describe offspring changes as *inherited RF-induced mtDNA effects* if sequence-level causality and maternal transmission are demonstrated.

L. Recovered historical study inventories and their limits

The separately provided Excel evidence register contains the **100-row Yakymenko reconstruction** (including review-level positive and null findings), **43 newer redox-related studies**, and **63 mitochondrial-associated records**: 206 source records total **before** deduplication. Records differ in exposure class, endpoint and level of verification; 18 ELF/PEMF and 15 indirect/special mitochondrial records must be separated from 30 direct RF entries. Not all Yakymenko entries have direct links to verified PubMed records; some links are PubMed *search queries* pending primary-paper identification. All study-level funding classifications must be considered **unverified unless specifically documented**. No numerical pooled probability, causal prevalence, or validated threshold can be inferred from simple counts.

Evidence synthesis table: scientific arguments for further research

- **Cancer initiation/promotion:** Anchors: [47–53]; Positive or unresolved signal: NTP heart schwannomas; mouse promotion reports; Main counter-evidence / limitation: Human phone epidemiology [54] largely null; heterogeneous animal exposures; co-promotion meta-analysis [49] mostly null; Proposed federal next step: Replicate carcinogen co-exposures, characterize tumor pathology and exposure

- **Human brain tumors, GBM specifically:** Anchors: [54, 61]; Positive or unresolved signal: Registry permits histology-specific secular trend analyses; Main counter-evidence / limitation: A 20+ year harmonized GBM incidence analysis was **not completed** here; Proposed research: Conduct CBTRUS/SEER analysis with consistent age and definitions
- **Oxidative stress:** Anchors: [29–30, 66] + 143 worklist rows; Positive or unresolved signal: Positive assay findings in many selected models; Main counter-evidence / limitation: Systematic review judges confidence very low; Proposed federal next step: Rigorous multi-lab assay validation with prespecified waveform
- **Mitochondria and mtDNA:** Anchors: [38–44, 64] + 63 worklist rows; Positive or unresolved signal: In-vitro mtDNA oxidative lesions, sperm endpoints; Main counter-evidence / limitation: No proven maternal germline transmission; several records are ELF/therapeutic; Proposed federal next step: Germ-cell sequencing and offspring outcomes
- **Methylation:** Anchors: [56]; Positive or unresolved signal: Cell methylation/gene-expression changes; Main counter-evidence / limitation: One in-vitro experiment does not show disease or MTHFR-specific susceptibility; Proposed federal next step: Test DNMT, one-carbon metabolism, SAM/SAH and multi-site controls
- **VGCC/calcium:** Anchors: [57]; Positive or unresolved signal: Proposed calcium signaling mechanisms; Main counter-evidence / limitation: Review-based synthesis; independent human pathway data needed; Proposed federal next step: Direct calcium measurement with pharmacological and genetic intervention
- **BBB and neurological function:** Anchors: [45–46, 58, 65]; Positive or unresolved signal: Mixed cellular/animal findings, severe reported symptoms; Main counter-evidence / limitation: 17 positive/18 null BBB review; blinded symptom trials mostly null; Proposed federal next step: Standardized BBB measures and careful patient-centered epidemiology
- **Environmental organisms:** Anchors: [59–60]; Positive or unresolved signal: Stress genes and honeybee pollination response; Main counter-evidence / limitation: Exposure/environmental confounding and unclear generalizability; Proposed federal next step: Controlled environmental replication
- **Site exposure distribution:** Anchors: [19–22, 62–63, 69]; Positive or unresolved signal: Main-lobe geometry/indoor differences directly measured; Main counter-evidence / limitation: Simple distance cannot establish dose, health outcome, or national setback; Proposed federal next step: Measured, modeled, publicly described site-by-site exposure

Additional references

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<https://pubmed.ncbi.nlm.nih.gov/37729852/>

[44] Cordelli et al. (2024). Male fertility effects in nonhuman mammals and human sperm in vitro: systematic review. <https://pubmed.ncbi.nlm.nih.gov/38492496/>

[45] Systematic review (2024). RF-EMF exposure and self-reported symptoms in human experiments; most comparisons showed no or small nonsignificant effects.

<https://pubmed.ncbi.nlm.nih.gov/38640611/>

[46] Rööslä et al. (2024). Tinnitus, migraine and nonspecific symptoms in human observational studies: systematic review; very-low-certainty evidence.

<https://pubmed.ncbi.nlm.nih.gov/38104437/>

[47] Tillmann et al. (2010). Indication of cocarcinogenic potential of chronic UMTS-modulated RF exposure in ethylnitrosourea-treated mice. <https://pubmed.ncbi.nlm.nih.gov/20545575/>

[48] Lerchl et al. (2015). Tumor promotion by RF exposure below limits for humans in a carcinogen-treated mouse model; replication study, no clear dose-response.

<https://pubmed.ncbi.nlm.nih.gov/25749340/>

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<https://pubmed.ncbi.nlm.nih.gov/39200630/>

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<https://ntp.niehs.nih.gov/publications/reports/tr/tr595>

[51] Falcioni et al. (2018). Ramazzini Institute lifelong 1.8 GHz GSM field study; male-rat heart schwannoma increase at highest field. <https://pubmed.ncbi.nlm.nih.gov/29530389/>

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PART IV — EVIDENCE-FOCUSED ADDENDUM

Linked experimental findings and selected epidemiology to justify targeted federal RF/EMF research. Prepared October 21, 2026. The numbered E-series studies below supplement, rather than replace, the preceding numbered references and the 206-record historical research index.

1. Sources, access, and interpretation

Panagopoulos, editor, *Electromagnetic Fields of Wireless Communications: Biological and Health Effects* (CRC Press/Taylor & Francis, 2022): publisher table of contents and Google Books limited preview were accessible; full chapter text was not. The book identifies research themes including oxidative stress (Yakymenko/Tsybulin), genotoxicity (Jagetia), germ-cell and reproductive effects, brain effects, EHS, wildlife, and ion-channel mechanisms. Published studies are cited individually below instead of relying on unverified book chapter claims. [Publisher and chapter outline](#)

Dr. Joel M. Moskowitz hosts Dr. Henry Lai's research indexes. The version publicized in June 2026 reported 390/438 RF oxidative-effect articles, 396/550 genetic-effect articles, 396/507 neurological-effect articles and 354/415 reproductive/developmental-effect articles as finding statistically significant outcomes. It separately identifies 260 low-intensity RF papers reporting significant effects (SAR < 0.40 W/kg). These are author-curated literature counts, not independently audited systematic-review meta-analyses, and categories may overlap. Use the index to identify eligible papers; evaluate individual studies before drawing aggregate conclusions. [Moskowitz/Lai research index](#)

Selection policy: the addendum intentionally emphasizes studies with affirmative measurable outcomes and a plausible reason to fund a well-controlled follow-up. This is a positive-evidence dossier, NOT a complete systematic review or a pooled risk estimate. Positive findings can coexist with well-designed null studies; the research proposals call for sham exposure, blind outcomes, dosimetry, replication, preregistration, and release of results whether positive or null. Funding and conflicts-of-interest metadata for the individual studies have not yet been comprehensively audited.

2. Selected linked studies and scientific synthesis — grouped by research priority

Cancer / tumor promotion

[E01] NTP (2018). **Technical Report 595: 900 MHz cellular modulation in rats.** [Original paper / PubMed](#)
Exposure/model: 900 MHz GSM/CDMA; 1.5-6 W/kg whole body; 2-y rat bioassay. Reported result: Clear evidence of malignant heart schwannomas in males; some evidence malignant gliomas. Why follow up: Controlled, multi-year, federally funded mammalian tumor findings warrant replication. Qualification: Animal whole-body high doses; not direct human environmental risk. [HHS recommendations 10;12;15]

[E02] Falcioni et al. (2018). **Ramazzini Institute lifespan base-station exposure.** [Original paper / PubMed](#)
Exposure/model: 1.8 GHz GSM far-field, prenatal to natural death; Lifespan rat bioassay. Reported result: Increased malignant heart schwannomas in exposed male rats at highest level. Why follow up: Independent institution observed analogous tumor histology in different exposure geometry. Qualification: Tumor rarity and histology-specific interpretation; full dosimetry review needed. [HHS recommendations 9;10;12]

[E03] Tillmann et al. (2010). **UMTS RF plus prenatal ethylnitrosourea tumor model.** [Original paper / PubMed](#)
Exposure/model: UMTS-modulated RF; chronic exposure; ENU-treated mice. Reported result: Reported cocarcinogenic/tumor-promoting signal. Why follow up: Direct experimental test of RF as promoter rather than sole carcinogen. Qualification: Combined carcinogen exposure; interaction design; not proof of spontaneous tumor increase. [HHS recommendations 10;12]

[E04] Lerchl et al. (2015). **Tumor promotion by exposure below human SAR limits.** [Original paper / PubMed](#)
Exposure/model: UMTS RF; sham, 0.04, 0.4, 2 W/kg; ENU-treated mice; follow-up. Reported result: Higher lung/liver tumors and lymphomas in some exposed groups. Why follow up: Promotional effects reported at 0.04/0.4 W/kg; targeted replication warranted. Qualification: No consistent dose response; collaborating institutions overlap earlier work. [HHS recommendations 10;12]

Cancer / epidemiology

[E05] Hardell & Carlberg (2015). **Swedish pooled case-control glioma analysis.** [Original paper / PubMed](#)
Exposure/model: Self-reported mobile and cordless phone use; latency; Human case-control pooled. Reported result: Reported elevated glioma odds ratios, including longer latency groups. Why follow up: Motivates histology-specific epidemiology with objective usage/exposure history. Qualification: Recall and selection biases; overlapping datasets; not cohort confirmation. [HHS recommendations 10;12]

[E06] **INTERPHONE Nordic analysis (2005). Acoustic neuroma case-control study.** [Original paper / PubMed](#) Exposure/model: Reported ipsilateral phone usage and duration; Human multicenter case-control. Reported result: Longer ipsilateral use subgroup reported elevated odds despite null overall use. Why follow up: Supports tumor-specific laterality/latency analytic designs. Qualification: Subgroup finding; recall bias and overall null association. [HHS recommendations 10;12]

DNA / genotoxicity

[E07] **Lai & Singh (1995). Low intensity microwave and rat brain single-strand breaks.** [Original paper / PubMed](#) Exposure/model: 2.45 GHz pulsed/CW; ~0.6-1.2 W/kg; Rat brain cells. Reported result: More strand-break signal at assessed post-exposure intervals. Why follow up: Early controlled DNA endpoint; NIH grant indexed in PubMed. Qualification: Single laboratory; methodological and thermal control review needed. [HHS recommendations 10;12]

[E08] **Lai & Singh (1996). Single- and double-strand breaks in exposed rat brains.** [Original paper / PubMed](#) Exposure/model: 2.45 GHz pulsed and continuous; 1.2 W/kg; Rat brain cells. Reported result: Reported increased single- and double-strand breaks. Why follow up: Expands endpoint and waveform testing. Qualification: Same research group; replication needed. [HHS recommendations 10;12]

[E09] **Lai & Singh (1997). Antioxidants and RF-associated strand breaks.** [Original paper / PubMed](#) Exposure/model: 2.45 GHz RF; antioxidant pretreatment; Rat brain cells. Reported result: Melatonin/spin-trap compound reduced reported DNA effects. Why follow up: Intervention/control tests oxidative mechanistic pathway. Qualification: Does not establish dietary clinical benefit or human injury. [HHS recommendations 10;12;15]

[E10] **Diem et al. (2005). Nonthermal DNA breakage in fibroblast and granulosa cells.** [Original paper / PubMed](#) Exposure/model: 1.8 GHz; 1.2 or 2 W/kg; different duty schedules; Human fibroblast and rat cell lines. Reported result: Comet-assay strand-break increases; intermittency stronger. Why follow up: Supports testing waveform/duty-cycle factors, beyond average exposure. Qualification: In vitro; replication and dosimetry controversies merit independent protocol. [HHS recommendations 5;10;11;12]

[E11] **Smith-Roe et al. (2020). NTP subchronic rodent genotoxicity.** [Original paper / PubMed](#) Exposure/model: Cell-phone RF GSM/CDMA; Rats and mice; comet assay. Reported result: Increased DNA damage in selected mouse and rat tissues; other endpoints null. Why follow up: Federal controlled testing with tissue-specific signal; available raw data. Qualification: Tissue/modulation specificity; absence of micronucleus effect. [HHS recommendations 10;12]

Panagopoulos / real-device bioeffects

[E12] **Panagopoulos et al. (2007). Cell death induced by GSM 900/1800 mobile-phone signals.** [Original paper / PubMed](#) Exposure/model: GSM 900 and DCS 1800 commercial phone; Drosophila ovarian cells. Reported result: TUNEL DNA fragmentation and cell-death markers reported. Why follow up: Useful real-device experimental model and biological endpoint. Qualification: Insects; device characterization and independent mammal/human confirmation required. [HHS recommendations 5;7;10;11]

[E13] **Chavdoula et al. (2010). Intermittent versus continuous GSM exposure.** [Original paper / PubMed](#) Exposure/model: GSM 900; equivalent cumulative 6-min daily exposure; Drosophila reproductive assay. Reported result: Intermittent patterns affected reproduction and apoptosis markers. Why follow up: Directly tests temporal exposure patterns. Qualification: Same investigator network; insect outcomes. [HHS recommendations 5;7;11]

[E14] **Panagopoulos et al. (2010). Intensity and antenna distance in fruit-fly bioassay.** [Original paper / PubMed](#) Exposure/model: GSM 900/1800 phone; 0-100 cm; Drosophila reproductive assay. Reported result: Reported exposure-response windows and follicle cell death. Why follow up: Supports matched physical dosimetry rather than distance-only assumptions. Qualification: Phone near insect preparation not tower siting data; erratum indexed. [HHS recommendations 5;7;9]

[E15] **Panagopoulos et al. (2010). Daily exposure duration and Drosophila reproduction.** [Original paper / PubMed](#) Exposure/model: GSM 900/1800; 1-21 min daily; Drosophila. Reported result: Reproductive output declined with increasing duration. Why follow up: Supports time resolved and cumulative dose research. Qualification: Insect endpoints, single researcher group. [HHS recommendations 5;7;11]

[E16] **Margaritis et al. (2014). Drosophila oogenesis as an EMF bio-marker.** [Original paper / PubMed](#) Exposure/model: Commercial mobile, DECT, Wi-Fi, Bluetooth and RF generator sources; Drosophila; suite of 280 experiments. Reported result: Reported follicle cell-death and fecundity effects varying by source. Why follow up: Comparison across device/waveform classes within a biological model. Qualification: Related investigator network; many experiments not independent publications. [HHS recommendations 5;7;11]

[E17] **Manta et al. / collaborators (2017). RF ovarian redox and gene-expression response in Drosophila.** [Original paper / PubMed](#) Exposure/model: Commercial mobile phone; mean SAR 0.15 W/kg; 30 min; Fruit-fly ovarian cells.

Reported result: ROS 1.6x; apoptotic follicles ~2x; 168 gene-expression changes. Why follow up: Specific multiplex biological endpoints at measured nominal SAR. Qualification: Same broad Athens research program; biological relevance to humans undetermined. [HHS recommendations 10;11;12]

Mitochondria / redox

[E18] Xu et al. (2010). Oxidative damage to mitochondrial DNA in cultured neurons. [Original paper / PubMed](#) Exposure/model: 1.8 GHz; 217 Hz modulation; 2 W/kg; Primary rat cortical neurons. Reported result: Higher mitochondrial 8-OHdG; lower mtDNA copy number/transcripts. Why follow up: Direct RF-associated mitochondrial endpoints, antioxidant-control design. Qualification: No sequence-level heritable mutations; cell culture and higher SAR. [HHS recommendations 10;15]

[E19] Sun et al. (2017). Mitochondrial and oxidative changes in HL-60 cells. [Original paper / PubMed](#) Exposure/model: 900 MHz CW; 120 $\mu\text{W}/\text{cm}^2$; 4 h/day for 5 days; Human leukemia cell line. Reported result: Increased ROS/8-OHdG; decreased mtDNA transcripts/copy and ATP. Why follow up: Mitochondrial function plus redox indicators; therapeutic-control concept. Qualification: Transformed cells; CW lab conditions; no germline data. [HHS recommendations 10;15]

[E20] De Iuliis et al. (2009). Human sperm ROS and DNA fragmentation following RF. [Original paper / PubMed](#) Exposure/model: 1.8 GHz; SAR 0.4-27.5 W/kg; Human sperm in vitro. Reported result: ROS, DNA fragmentation higher; motility/vitality lower with SAR. Why follow up: Links mitochondrial ROS and germ-cell endpoints with SAR series. Qualification: Substantial high-dose range and in-vitro conditions; paternal not maternal mtDNA inheritance. [HHS recommendations 10;15]

Reproductive / developmental

[E21] Avendaño et al. (2012). Laptop Wi-Fi exposure to isolated human sperm. [Original paper / PubMed](#) Exposure/model: Wi-Fi-connected laptop 4 h versus control; 29 human donors; in vitro. Reported result: Reduced progressive motility and increased DNA fragmentation. Why follow up: Real-world device condition as screening study for controlled trials. Qualification: Laptop can add thermal/electrical confounds; in vitro cannot prove fertility changes. [HHS recommendations 7;10;15]

[E22] Prenatal Wi-Fi rat study (2017). Postnatal developmental effects in rats after gestational Wi-Fi. [Original paper / PubMed](#) Exposure/model: 2.45 GHz Wi-Fi, 2 h/day pregnancy; Rat dams; 5 exposed, 5 control. Reported result: Reported early developmental and brain oxidative marker differences. Why follow up: Specific offspring outcome endpoints for more powered preregistered studies. Qualification: Very small maternal group; not mtDNA inheritance. [HHS recommendations 10;15]

[E23] Prenatal RF rat study (2020). Offspring development after 1800 MHz and Wi-Fi exposure. [Original paper / PubMed](#) Exposure/model: 1800 and 2400 MHz exposure; Rat gestational model. Reported result: Altered selected behavioral readouts and hippocampal receptor transcripts. Why follow up: Provides plausible markers to replicate with larger cohorts. Qualification: Animal setting, developmental outcome confounding and multiple comparisons. [HHS recommendations 10;15]

[E24] Cordelli et al. (2024). Systematic review experimental RF male fertility. [Original paper / PubMed](#) Exposure/model: Multiple experimental frequencies/exposure systems; 117 animal papers and 10 human-sperm papers. Reported result: Review reported moderate-certainty lower pregnancy rate in rodent outcomes, and low-certainty sperm-count reduction. Why follow up: Outcome-specific external evidence synthesis; identifies protocol deficiencies. Qualification: Systematic review not a primary experiment; effect estimates vary and bias is common. [HHS recommendations 10;15]

Neurophysiology / behavioral

[E25] Huber et al. (2000). Pulsed RF exposure and human sleep EEG. [Original paper / PubMed](#) Exposure/model: 900 MHz; ~1 W/kg spatial peak; 30 min; Healthy adults; sham comparison. Reported result: Altered spectral power of subsequent non-REM EEG. Why follow up: Human physiological endpoint with reversible/crossover style measures. Qualification: EEG differences not clinical injury. [HHS recommendations 4;10;11]

[E26] Volkow et al. (2011). Cell phone on/off and human glucose metabolism. [Original paper / PubMed](#) Exposure/model: Cell phone active versus inactive for 50 min; 47 healthy adults; randomized crossover PET. Reported result: Higher regional brain glucose metabolism near antenna. Why follow up: Direct objectively measured acute human physiology. Qualification: Clinical significance uncertain; not evidence for disease. [HHS recommendations 4;10;11]

[E27] Foerster et al. (2018). Adolescent memory and modeled RF brain dose. [Original paper / PubMed](#) Exposure/model: Personal modeled RF cumulative brain dose; 669-676 adolescents; one-year cohort. Reported result: Association of greater dose with changes in figural memory. Why follow up: Motivates repeated adolescent cohorts with dosimeter validation. Qualification: Residual phone-use confounding; observational. [HHS recommendations 1;10;15]

[E28] Rööslä group (2015). Adolescent phone use and memory study. [Original paper / PubMed](#) Exposure/model: Modeled cumulative brain/whole-body RF dose; 439 adolescents; 1-year follow-up. Reported result: Higher estimated dose related to decline in certain memory outcomes. Why follow up: Independent time-based observational exposure model useful for protocols. Qualification: Exposure estimates and non-RF phone use confounding. [HHS recommendations 1;10;15]

Blood-brain barrier

[E29] Salford et al. (2003). GSM RF and rat neuron injury markers. [Original paper / PubMed](#) Exposure/model: GSM phone RF for 2h; Rat brain pathology. Reported result: Reported neuronal damage in cortex/hippocampus/basal ganglia. Why follow up: Potential blood-brain-barrier and neuronal endpoints for blinded replication. Qualification: Histology limitations; effects contested in follow-up research. [HHS recommendations 10;15]

[E30] Nittby et al. (2009). Albumin leakage 7 d after GSM exposure. [Original paper / PubMed](#) Exposure/model: 900 MHz GSM; delayed measurements; Rat brains. Reported result: Reported BBB permeability increases after exposure. Why follow up: Delayed exposure-outcome follow-up feasible and testable. Qualification: Results inconsistent across laboratories. [HHS recommendations 10;15]

[E31] BBB rat study (2011). CW RF effects on Evans-blue BBB leakage. [Original paper / PubMed](#) Exposure/model: 0.9 and 1.8 GHz continuous RF, 20 min; Male and female rats. Reported result: Reported elevated albumin leakage in males only. Why follow up: Sex-stratified replication question and mechanistic hypothesis. Qualification: Short-term animal-only and contested BBB literature. [HHS recommendations 10;15]

Epigenetics / methylation

[E32] Cantu et al. (2023). Keratinocyte whole-genome methylation after RF. [Original paper / PubMed](#) Exposure/model: 900 MHz; estimated SAR <0.01 W/kg; 1 h; Human keratinocyte cultures. Reported result: Six targets both differentially methylated and expressed. Why follow up: Strong endpoint specificity; motivates genome-wide replication and methylation pathways. Qualification: Acute cell-culture marker, not MTHFR metabolism or clinical disease. [HHS recommendations 10;12]

Population / siting

[E33] Hutter et al. (2006). Measured bedroom RF and symptoms near base stations. [Original paper / PubMed](#) Exposure/model: Measured household levels near 10 base stations; 365 residents; cross-sectional. Reported result: Association with selected symptoms, notably headache. Why follow up: Couples actual environmental measurements with symptom data. Qualification: Cross-sectional confounding and no established causality. [HHS recommendations 1;9;11]

[E34] Roosli et al. / questionnaire (2004). Symptoms ascribed to EMF and mitigation actions. [Original paper / PubMed](#) Exposure/model: Self-reported source categories and activities; Questionnaire respondents reporting symptoms. Reported result: Sleep disorders/headaches common; two-thirds reported taking steps. Why follow up: Social functioning and burden measurement justification. Qualification: Selected self-report sample; no RF causal inference. [HHS recommendations 1;2;3]

[E35] Santini et al. (2003). Base-station proximity and reported symptoms. [Original paper / PubMed](#) Exposure/model: Residential proximity and antenna facing; 530 people; survey. Reported result: Certain symptoms associated with position relative to tower. Why follow up: Directional geometry deserves independent prospective evaluation. Qualification: No individual dosimetry/strong confounding; not siting threshold. [HHS recommendations 9]

Wildlife / environment

[E36] Molina-Montenegro et al. (2023). EMF and honeybee pollination. [Original paper / PubMed](#) Exposure/model: Anthropogenic electromagnetic exposure near hives; Field/experimental honeybee study. Reported result: Reported altered pollination service and organism stress indicators. Why follow up: Wildlife/ecosystem endpoint to verify independently. Qualification: Field exposure mixtures and ecological confounding. [HHS recommendations 12]

3. Recommended research programs with pre-specified endpoints

Cancer promotion and latency. Multi-institution 2-year animal bioassays, including noncarcinogen and carcinogen-initiated arms; separate cardiac schwannoma, glioma, lymphoma and organ-specific histology; complete RF spectral/duty characterization; publish raw slides, tumor counts and noncancer outcomes. An independent prospective human registry track should pre-specify glioblastoma rather than pool all brain tumors.

DNA injury and oxidative stress. Reproduce selected Lai, Diem, Smith-Roe, Panagopoulos and mitochondrial endpoints with independent dosimetry, standardized comet assay and DNA-lesion quantification, temperature monitoring, blinded samples and prespecified endpoints including 8-OHdG, oxidized lipids, cell viability and mtDNA copy/sequence.

Real-device waveforms. Compare commercial Wi-Fi, DECT, cellular and video-doorbell sources with calibrated signal generators matched for mean incident power/SAR but differing in pulse structure, modulation, antenna orientation, duty cycle and intermittency; measure and report complete I/Q waveform where practicable.

Methylation and mitochondrial inheritance. Assess genome-wide methylation and one-carbon-pathway metabolites separately. Maternal preconception studies should sequence oocyte and offspring mtDNA, trace heteroplasmy and spontaneous background mutations, and distinguish irreversible sequence changes from transient oxidative markers.

Human biology and lived functional impact. Combine a probability-based national symptom/disability survey and longitudinal objective exposure monitoring. A separate blinded human physiology program can replicate PET/EEG outcomes while avoiding claims that an acute biomarker is a diagnosis; do not exclude patients unable to produce years-old diaries.

Residential exposure and control. Measure occupied-building RF environments (including main-lobe direction and indoor levels) and test lower transmit-power options, scheduled transmissions and wired alternatives; record actual field reduction, service failures, energy consumption and impacts on adjoining residences.

Pregnancy, developing nervous system and sperm. Replicate prenatal findings with adequately powered litters, maternal as unit of randomization, temperature control, developmental assays and neurophysiology, while separately testing sperm ROS and DNA damage at verified temperature-equivalent sham conditions.

Environmental and wildlife endpoints. Replicate honeybee and pollination findings with independently metered sources, ecological controls and multiple sites/species; distinguish RF from ELF/static fields and other co-exposures.

4. Funding transparency, replication and source-quality safeguards

A study-level funding and quality record should identify direct funder; institutional grants, donated equipment, test-chamber or wireless-company engineering support; current/former employment; study sponsor role in protocol, analysis and publication; independent laboratory replication; study registration; thermal/exposure controls; and full-text retrieval status. Indexing in PubMed does not itself confirm independence or quality. Do not label papers “industry-funded” or “independent” solely from affiliations. NTP TR-595 is a federal technical report; Lai & Singh 1995/1996 have PubMed-listed NIH/NIEHS grant support; the 2023 Cantu epigenetic study lists U.S. Air Force/contractor-affiliated authors, not a telecom sponsorship determination.

EXCLUSION ALERT: Friedman et al. (2007), “Mechanism of short-term ERK activation by electromagnetic fields at mobile phone frequencies” (PMID 17456048), is indexed in PubMed as RETRACTED. It has intentionally NOT been counted among supporting studies. Every additional promising citation should be screened for retractions, corrections, reuse of datasets and any material funding disclosures before appearing in a final evidence total.

5. Evidence-to-recommendation mapping for the 13 proposals

- 1 Prevalence and costs:** Surveys and E33–E35; no national causal-cost estimate.
- 2 Medical records:** WHO and E34 support symptom documentation, not EHS diagnosis.
- 3 Accommodation:** Access Board and EEOC policy sources; individualized need.
- 4 Power-density units:** NIST and ICNIRP reporting standards; conversion is not dose.
- 5 Exposure profiles:** E10 and E12–E17 plus measured-site studies.
- 6 Historical baseline:** NOAA/NIST technical sources; RF and natural ELF separate.
- 7 Wireless power settings:** E12–E17 support trials, not proof of injury.
- 8 Building Biology:** IBN screening recommendations; health thresholds unvalidated.
- 9 Tower siting:** E33, E35 and geometry studies; no fixed setback specified.
- 10 Cancer and biology:** E01–E11 and E18–E31; animal, cell and human tracks.
- 11 Signal triggers:** E13–E17 show modulation and timing as research variables.
- 12 Research funding:** Huss (2007); direct/indirect funding audit outstanding.
- 13 Maternal mtDNA:** E18–E24; RF-linked inherited mutations not demonstrated.

Source verification status: links were traced to PubMed or a publisher DOI for each E-series item; the evidence summaries are based on accessible abstracts or official reports, not a full-text methods audit for every row. Complete the funding/retraction/quality audit before describing the 36 cited records as independently validated evidence.