

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] Rösli 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.