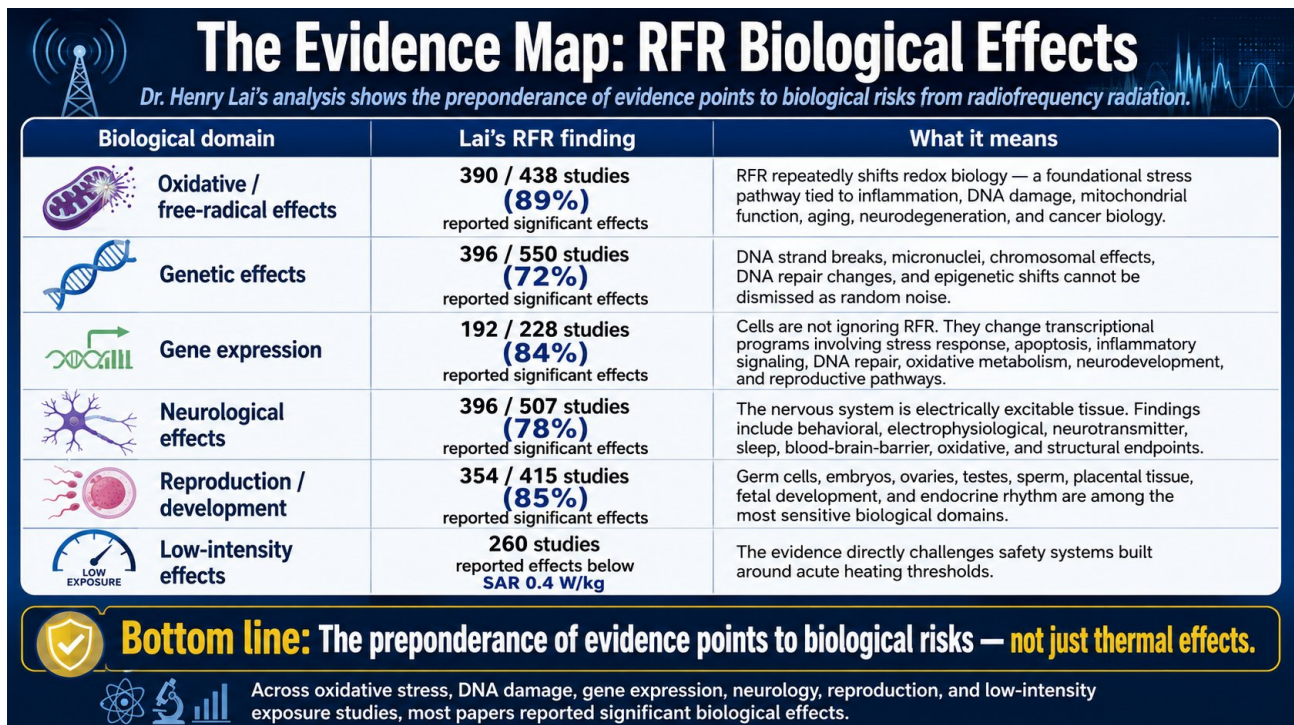


THE STANDARD MEASURES HEAT. BIOLOGY MEASURES TIMING.

Why America needs a Clean Ether Act, modern radiofrequency health standards, and Li-Fi-ready places for children

RF SAFE FLAGSHIP EVIDENCE + POLICY BRIEF • RESEARCH CURRENT THROUGH AUGUST 13, 2026

Invisible does not mean irrelevant. Non-ionizing does not mean non-biological. Legal exposure does not mean demonstrated safety.



Evidence-map preview. Full tallies, limitations, and source links appear in the brief.

How to read this brief

This is a forceful public-health advocacy document. It distinguishes controlled hazard evidence, quantitative risk estimates, mechanistic and susceptibility findings, human epidemiology, and registry surveillance. That hierarchy matters: no cancer-registry trend is presented as proof of radiofrequency causation, and no physiological response is automatically labeled harm.

HAZARD	Can a controlled exposure cause an adverse effect?
RISK	At what exposure and under what assumptions might harm occur?
MECHANISM	How can an imposed field change biological signaling or repair?
SURVEILLANCE	Which population patterns deserve investigation and follow-up?

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THE ACT Eight Clean Ether Act titles, Li-Fi compatibility, and child-priority low-RF spaces	THE RESPONSE Implementation roadmap, rebuttal matrix, research agenda, and source appendices

Executive judgment

The Federal Communications Commission's radiofrequency rules are compliance limits, not a modern demonstration of biological safety. They answer a narrow question: under specified test and averaging conditions, does radiofrequency energy stay below limits derived from an acute heating-and-behavior paradigm? They do not establish that years of pulsed and modulated, multi-source exposure are safe for developing children, pregnancy, fertility, sleep, the nervous system, genetically susceptible subgroups, or the environment.

That is no longer a theoretical criticism.

- The FCC's own 1996 rulemaking said both its previous 1982 framework and the revised limits shared a 4 W/kg specific absorption rate (SAR) basis. The revised rules were generally drawn from NCRP 1986 and ANSI/IEEE 1992 recommendations; the scientific and regulatory lineage therefore reaches into the early 1980s, even though the current framework was formally adopted in 1996. [FCC 1996 final rule](#)
- In 2021, the U.S. Court of Appeals for the D.C. Circuit held that the FCC failed to provide a reasoned explanation for why its retained limits adequately protect against non-cancer effects. The court specifically required the agency to address children, long-term exposure, ubiquitous wireless use, technological change, testing procedures, and environmental effects. The court did not decide the science or declare the limits unsafe; it found the agency's explanation legally inadequate. [Environmental Health Trust v. FCC](#)
- The U.S. National Toxicology Program (NTP) reported clear evidence of malignant heart schwannomas in male rats exposed to CDMA-modulated 900 MHz radiofrequency radiation, with malignant brain gliomas also related to exposure. NTP separately concluded that radiofrequency exposure caused DNA damage under the conditions it studied. [NTP TR-595](#) · [NTP program and FAQ](#)
- The Ramazzini Institute observed a statistically significant increase in malignant heart schwannomas in male rats after lifelong, far-field 1.8 GHz GSM exposure at much lower whole-body SARs than the NTP experiment. [Falcioni et al., 2018](#)
- A WHO-commissioned systematic review of animal cancer studies concluded with high certainty for increased malignant heart schwannomas; the final published record, including a 2026 corrigendum, reports high certainty for heart schwannomas and glial-cell tumors. [Mevisen et al., 2025](#) · [2026 corrigendum](#)
- A corrected WHO-commissioned review of experimental male-fertility evidence assigned high certainty to reduced pregnancy rate, with other sperm and reproductive findings contributing to the evidence base. [Cordelli et al., 2024](#) · [corrigendum](#)
- In 2026, Ronald Melnick and Joel Moskowitz applied benchmark-dose modeling and traditional toxicological uncertainty factors to animal cancer and fertility data. Their published estimates place the current public whole-body limit 15–114 times above a modeled cancer-protective level for one hour of daily exposure and 121–909 times above it for eight hours; their fertility estimate is 8–24 times lower than the current limit. These are peer-reviewed risk estimates based on stated assumptions—not determinations adopted by EPA, FDA, or FCC—but they demonstrate what happens when RF is evaluated with methods used for toxic and carcinogenic agents instead of only an acute heat threshold. [Melnick & Moskowitz, 2026](#)
- Dr. Henry Lai's continuously updated literature compilation reports that majorities of studies in oxidative stress, genetics, gene expression, neurology, reproduction and development found statistically significant effects. His June 2026 tallies include 390 of 438 oxidative/free-radical papers, 396 of 550 genetic-effects papers, 396 of 507 neurological papers, and 354 of 415 reproduction/development papers. These tallies are a broad evidence map, not a formal meta-analysis; they do not by themselves grade study quality, effect size,

independence, or publication bias. Their value is that they make the repeated signal impossible to dismiss as one laboratory or one endpoint. [Lai compilation](#)

- A double-blind, sham-controlled 2025 human experiment found that a common CACNA1C variant modified the effect of a standardized 3.6 GHz 5G exposure on non-REM sleep-spindle frequency. It is a physiological endpoint, not proof of injury, but it directly demonstrates that population averaging can conceal genotype-dependent response. [Sousouri et al., 2025](#)

The policy conclusion is straightforward: compliance with an acute energy-absorption limit cannot be treated as proof of protection against chronic, waveform-specific, developmental, reproductive, neurological, carcinogenic, or susceptible-subgroup effects.

Congress should enact a Clean Ether Act: a technology-positive public-health framework that modernizes exposure standards, restores independent research, establishes child-priority low-RF environments, requires transparent exposure information, reforms federal preemption, and makes wired and optical networking—including IEEE 802.11bb Li-Fi compatibility—the default pathway for high-bandwidth indoor connectivity.

This is not anti-technology. It is pro-biocompatible technology.

The single most important distinction: compliance is not a safety finding

The public hears “FCC compliant” and reasonably assumes “tested safe.” Those phrases are not equivalent.

An RF compliance test evaluates whether a device or facility meets prescribed limits under prescribed conditions. For portable devices in the United States, the best-known localized limit is 1.6 W/kg averaged over one gram of tissue. The public whole-body SAR limit is 0.08 W/kg. Current rules also use power-density limits and time averaging; FCC regulations permit general-public exposure to be averaged over periods up to 30 minutes for applicable SAR or power-density evaluations. [FCC RF safety](#) · [47 CFR update](#)

Those numbers are meaningful for the harm model they were designed around: excessive tissue heating. They are not measurements of oxidative stress, DNA repair, sleep architecture, neurodevelopment, fertility, calcium-wave timing, immune signaling, mitochondrial reserve, senescence, apoptosis, or recovery after repeated exposure.

The difference can be stated without jargon:

A compliance test can show	A compliance test does not establish
Energy absorption stayed below the applicable limit in the test configuration	No chronic biological effect occurs below that limit
A modeled or measured peak met a spatial and temporal averaging rule	Modulation, pulse structure, duty cycle, phase, and intermittency are biologically irrelevant
One device or site met an authorization requirement	Lifetime multi-source exposure is safe
The device did not exceed a heat-protection threshold	Children, pregnancy, fertility, illness, aging, or susceptible genotypes are adequately protected
The product is legal to market or operate	The exposure has been evaluated like a toxicant or carcinogen

The phrase “within the limit” describes regulatory compliance. It is not a clinical finding, a lifetime cancer-risk calculation, a developmental toxicology assessment, or a guarantee that every biologically relevant parameter was tested.

The historical record: the framework is older than “the 1996 guidelines”

Calling the limits “1996 guidelines” is incomplete. The FCC formally adopted its revised framework in 1996, but the underlying logic predates that action.

1. 1982: ANSI issued C95.1-1982, a radiofrequency exposure standard centered on an SAR threshold.
2. 1985: The FCC began using the ANSI standard for environmental RF evaluations.
3. 1986: NCRP Report 86 supplied recommendations later used in the FCC’s 1996 rulemaking.
4. 1992: ANSI/IEEE C95.1-1992 revised the standard but retained the acute-heating foundation.
5. 1996: The FCC adopted revised limits. Its final rule explicitly stated that the old and new approaches shared a 4 W/kg SAR basis and that the changes did not reflect a fundamental change in the RF safety criterion.

6. 2013–2019: The FCC opened and then closed an inquiry into the adequacy of the limits, retaining them while updating several compliance procedures.
7. 2021: The D.C. Circuit remanded the 2019 decision because the FCC had not adequately explained protection against non-cancer effects, including concerns involving children, long-term exposure, modern technology, testing procedures, and the environment.
8. 2026: The FCC’s public RF-safety materials still list the 1.6 W/kg localized public limit, while the peer-reviewed animal-cancer, reproductive, mechanistic, and risk-assessment literature has continued to grow.

The crucial historical sentence appears in the FCC’s own 1996 final rule: the more conservative revised limits did not arise from “a fundamental change in the RF safety criteria for SAR.” [FCC 1996 final rule](#)

That history matters because technology evolved from occasional voice calls to a dense ecosystem of smartphones, Wi-Fi, Bluetooth, wearables, smart appliances, tablets, laptops, small cells, connected classrooms, and continuous background signaling. A limit’s age is not by itself proof of failure. But a framework must be able to show that it still asks the right health questions. The 2021 court found that the FCC had not supplied that reasoned explanation.

The category error: “non-ionizing” is not the same as “non-biological”

Radiofrequency photons do not carry enough energy individually to ionize atoms the way X-rays and gamma rays do. That physical fact is important—and routinely misused.

It supports the statement that RF does not damage DNA by the same direct ionization mechanism as ionizing radiation. It does not support the universal claim that RF cannot affect biology by any indirect, collective, resonant, electrochemical, thermal-microgradient, membrane, redox, calcium-signaling, or gene-regulatory pathway.

Medicine supplies an unambiguous counterexample to the slogan “non-ionizing means biologically inert.” In 2024, FDA authorized the TheraBionic P1, a handheld radiofrequency electromagnetic-field generator that delivers specific amplitude-modulated frequencies for hepatocellular carcinoma. FDA’s description says the emitted fields may stop cancer cells from dividing. Therapeutic conditions are not equivalent to ambient Wi-Fi or phone exposure, and a treatment does not prove an environmental hazard. It does prove the narrower and decisive point: low-power, amplitude-modulated RF can be engineered to produce biological effects without being an ionizing radiation treatment. [FDA TheraBionic P1](#)

The proper question is therefore not “Can a non-ionizing signal ever affect biology?” The answer is yes. The public-health questions are:

- Which frequencies, modulations, pulse structures, intensities, durations, and tissue geometries produce effects?
- Which effects are adaptive, neutral, therapeutic, adverse, or context-dependent?
- What happens after repeated exposure without adequate recovery?
- Which life stages, genotypes, diseases, co-exposures, and tissues change susceptibility?
- Which dose metric best predicts each outcome?

SAR remains useful, but it cannot be assumed to be the complete biological dose metric before those questions are answered.

The evidence architecture: four layers, one policy conclusion

RF Safe's case does not depend on one study, one mechanism, or one cancer trend. It rests on convergence across four layers.

1. Controlled hazard evidence

Animal and cell experiments can control exposure, timing, modulation, tissue collection, and confounding. The NTP and Ramazzini bioassays, experimental fertility literature, sleep-EEG studies, and mechanistic experiments belong here. Controlled experiments are strong for establishing that an effect can occur under defined conditions. Translation to everyday human risk still requires dosimetry and exposure modeling.

2. Quantitative risk estimation

Benchmark-dose modeling and uncertainty factors translate controlled findings into proposed health-protective levels. The 2026 Melnick–Moskowitz analysis belongs here. Its assumptions must be visible, debated, and replicated. What cannot be justified is refusing to perform the calculation while continuing to call a heat-based limit health protective.

3. Mechanism and susceptibility

Oxidative stress, DNA damage, calcium dynamics, mitochondrial function, gene expression, sleep electrophysiology, and genotype-by-exposure interactions address biological plausibility and identify vulnerable receivers. No single mechanism must explain every finding. Environmental stressors commonly act through multiple, context-dependent pathways.

4. Human epidemiology and surveillance

Case-control studies, cohorts, and cancer registries ask whether effects are observable in people. They are indispensable and difficult: historical exposure reconstruction is weak, technology changes, latency can be long, self-reported use is error-prone, tumor classifications change, and people are rarely truly unexposed. Conflicting human findings should change the certainty and size of a risk estimate; they do not erase controlled animal hazards or prove that today's limit protects every endpoint.

When a ubiquitous exposure has controlled hazard signals, high-certainty animal conclusions for key endpoints, plausible pathways, subgroup response, low-cost exposure-reduction options, and unresolved long-latency human risk, the appropriate public-health response is prevention plus better science—not reassurance by assumption.

Dr. Henry Lai’s evidence map: a repeated signal across biological domains

Dr. Henry Lai’s literature compilation is one of the broadest domain-by-domain maps in bioelectromagnetics. As of the June 2026 update cited by RF Safe, it included more than 3,000 peer-reviewed abstracts across RF, ELF, and static fields.

Radiofrequency radiation

Biological domain	Papers reporting significant effects	Share
Oxidative/free-radical effects	390 of 438	89%
Genetic effects	396 of 550	72%
Gene expression	192 of 228	84%
Neurological effects	396 of 507	78%
Reproduction/development	354 of 415	85%
Low-intensity oxidative studies at SAR ≤ 0.40 W/kg	110 of 114	96%

Lai also catalogued 260 low-intensity RF studies reporting biological effects below 0.40 W/kg. In RF Safe’s source material, 79% of these effect studies were in vivo and 70% involved repeated or chronic exposure; the mean SAR was 0.072 W/kg and the median 0.028 W/kg.

ELF and static fields

Biological domain	Papers reporting significant effects	Share
Oxidative/free-radical effects	319 of 353	90%
Genetic effects	363 of 424	84%
Gene expression	232 of 252	92%
Neurological effects	364 of 397	92%
Reproduction/development	82 of 105	78%

These counts are not a substitute for systematic review. A statistically significant paper is not automatically a high-quality paper; multiple papers may come from related laboratories; endpoints and exposure systems differ; null findings matter; publication bias is possible; and a tally does not calculate a pooled effect size.

But dismissing the compilation because it is not a meta-analysis makes the opposite error. A signal replicated across oxidative biology, genetic endpoints, transcription, neurology, reproduction, species, tissues, frequencies, and laboratories demands an exposure framework capable of testing those domains. The current framework largely does not.

The honest conclusion is not “89% proves every wireless exposure is dangerous.” It is: a heat-only assurance is scientifically too narrow for an evidence base in which non-thermal biological responses are repeatedly reported across independent domains.

Oxidative stress: the most repeated mechanistic signal

Oxidative stress is a state in which reactive species and antioxidant defenses are out of balance. It can affect lipids, proteins, mitochondrial function, calcium handling, inflammatory signaling, DNA integrity, and cell fate. It can be a transient adaptive signal or a contributor to pathology; context, magnitude, timing, and recovery determine which.

The oxidative-stress literature illustrates both the strength of the signal and the methodological dispute.

- Lai’s 2026 compilation reports significant oxidative or free-radical effects in 390 of 438 RF papers and in 110 of 114 low-intensity studies at or below 0.40 W/kg.
- A 2016 review by Yakymenko and colleagues examined 100 studies and reported oxidative effects in 93. [Yakymenko et al., 2016](#)
- A WHO-commissioned 2024 systematic review by Meyer and colleagues identified 56 studies meeting its inclusion criteria and included 52 in meta-analyses. It found possible increases in some biomarkers and tissues but judged the overall evidence “very low certainty,” citing high risk of bias, inconsistent measures, and the limitations of single biomarkers. [Meyer et al., 2024](#)
- Melnick and colleagues responded that the WHO review considered a much larger pool potentially eligible but excluded many studies because of biomarker rules, including studies relying on TBARS or DCFDA. They argued that excessive subdivision and exclusions manufactured uncertainty. [Melnick et al., 2025](#)

This disagreement cannot be resolved by counting alone or by declaring “very low certainty” equivalent to “no effect.” It should be resolved experimentally.

A federal research program should preregister multicenter protocols that use:

- validated dosimetry and continuous thermal monitoring;
- blinded exposure and outcome assessment;
- multiple redox biomarkers in the same system;
- mitochondrial and cytosolic sensors measured in real time;
- positive controls and sham controls;
- several cell types, sexes, life stages, and exposure waveforms;
- exposure–recovery cycles, not only immediate post-exposure snapshots;
- raw-data publication and independent replication.

If the dominant stress signal disappears under rigorous, replicated conditions, policy should learn that. If it persists, exposure limits should be rebuilt around it. The present standard cannot settle the question because oxidative biology is not its protection endpoint.

DNA damage, genome maintenance, and gene expression

The claim “RF photons cannot directly break chemical bonds” does not answer whether RF exposure can indirectly alter DNA damage or repair through reactive species, calcium signaling, replication stress, chromatin organization, spindle function, or altered expression of repair genes.

Lai's compilation reports significant genetic effects in 396 of 550 RF papers and gene-expression changes in 192 of 228. A 2025 scoping review and evidence map of 517 DNA-damage studies found 59% of all studies reported effects; the balance among higher-quality experimental studies was 48% effect versus 52% no effect. Importantly, in vivo and epidemiological studies were more often positive than in vitro studies, while higher-quality in vitro evidence shifted toward null. This is a mixed evidence base—not a scientifically empty one. [Weller et al., 2025](#)

NTP's subchronic genotoxicity study used the comet assay in rats and mice. Its overall conclusion was that RF exposure may be associated with increased DNA damage, with results varying by species, sex, tissue, and modulation. NTP's current FAQ states directly that exposure led to DNA damage under the study conditions while the mechanism and repair consequences remained unknown. [Smith-Roe et al., 2020](#) · [NTP FAQ](#)

That pattern—effects that depend on tissue, sex, modulation, or biological state—is not a reason to average everything into “no effect.” It is a reason to identify the variables that control response.

The regulatory question is not whether every experiment is positive. It is whether a public limit can be declared protective when genome-maintenance endpoints are not part of the limit-setting architecture.

The receiver is part of the dose: genetic and molecular susceptibility

Public standards generally assume that a physical quantity averaged over mass and time adequately describes risk. Biology adds another term: the state of the receiver.

CACNA1C and human sleep physiology

CACNA1C encodes the principal pore-forming subunit of the Cav1.2 L-type voltage-gated calcium channel, a major regulator of neuronal calcium entry.

In a 2025 double-blind, sham-controlled study, 34 healthy participants were genotyped for CACNA1C variant rs7304986 and exposed for 30 minutes before sleep to standardized 700 MHz and 3.6 GHz 5G signals. A significant genotype-by-exposure interaction appeared: 3.6 GHz exposure shifted sleep-spindle center frequency in T/C carriers across central, parietal, and occipital regions; the matched T/T group did not show the same response. [Sousouri et al., 2025](#)

This study does not establish disease or prove that a spindle-frequency shift is harmful. It establishes something policy has largely ignored: a standardized RF exposure can produce different measurable human neurophysiological responses depending on a common genotype.

An earlier observational study of about 2,040 people linked a different CACNA1C variant, rs2302729, with both reduced subjective sleep quality and self-rated electromagnetic sensitivity. As an observational association, it cannot establish that RF caused the symptoms. Together with the controlled experiment, it identifies a serious research direction: genotype-stratified provocation studies with objective endpoints. [Eicher et al., 2024](#)

The same field, different cellular fate

A 2026 Breast Journal study exposed three breast-cancer cell subtypes and normal fibroblasts to the same 1 Hz, 100 mT ELF magnetic field for two hours per day over five days. HER2-enriched SKBR-3 cells showed about a five-

fold rise in reactive oxygen species and roughly 78% apoptosis, while other cancer subtypes upregulated antioxidant genes and resisted much of the cell death; fibroblasts showed little change. [Shayeghan et al., 2026](#)

This was a strong ELF field, not ambient telecom RF. It is not evidence that Wi-Fi produces the same outcome. Its relevance is conceptual and experimental: the identical external field did not produce an identical biological dose. Molecular context determined whether the signal was buffered or amplified.

Engineered EMF transduction

In 2026, a Cell paper described an electromagnetic-field-inducible gene switch and identified CYB5B as an essential mediator. Activation depended on rhythmic calcium oscillations rather than generic calcium influx. The system used engineered biology and low-frequency fields; it is not proof that everyday telecom signals activate the same pathway. It demonstrates that cells can be designed—and in that system were observed—to transduce field timing into calcium dynamics and gene expression. [Kim et al., 2026](#)

Taken together, these studies justify a new principle for exposure science:

Dose is not only what leaves the transmitter or is averaged in tissue. Biological dose also depends on waveform, tissue architecture, gene regulation, redox state, channel expression, developmental stage, health status, and recovery capacity.

Cancer evidence: two major bioassays, a WHO review, and an unfinished human question

National Toxicology Program

NTP's \$30 million program took roughly a decade, examined nearly 3,000 animals and about 40 tissues, and used purpose-built reverberation chambers. Pregnant rats and their offspring were exposed to 900 MHz GSM- or CDMA-modulated RF; mice were exposed at 1,900 MHz. The rat study included multiple dose groups and lifetime exposure.

For male rats exposed to CDMA-modulated 900 MHz RF, NTP reported clear evidence of carcinogenic activity based on malignant heart schwannomas; malignant brain gliomas were also related to exposure. The peer-review panel elevated several draft conclusions. NTP also reported preneoplastic and non-neoplastic cardiac changes.

NTP's results cannot be directly converted into a personal cellphone risk. The animals received whole-body exposure, for long daily periods, at SARs above typical whole-body human exposure. That is what a hazard bioassay does: it challenges a biological system under controlled conditions to identify target organs and outcomes. NTP states that direct extrapolation is not straightforward, but also that some tumors observed in animals occur in humans and may be relevant. [NTP program](#)

The most troubling policy development is not that every uncertainty has been resolved. It is that NTP reports no current plan for additional RF exposure studies after completing technically difficult follow-up work with a system limited to older 2G/3G signals. The need is to improve the platform for 4G, 5G, Wi-Fi, realistic modulation, and chronic low-intensity research—not end the program.

Ramazzini Institute

The Ramazzini Institute exposed Sprague-Dawley rats from prenatal life until natural death to 1.8 GHz GSM far-field radiation for 19 hours per day at whole-body SARs of approximately 0.001, 0.03, and 0.1 W/kg. The study reported a statistically significant increase in malignant heart schwannomas among male rats at the highest exposure, with increases in Schwann-cell hyperplasia. [Falcioni et al., 2018](#)

NTP and Ramazzini differed in exposure intensity, frequency, field geometry, and duration. Their concordance on a rare tumor of the same cell lineage is therefore biologically important even as each study's design and statistical interpretation remain debated.

WHO-commissioned animal-cancer review

Mevissen and colleagues systematically reviewed the animal cancer literature. The review's synthesis and certainty grading have been criticized by ARPANSA, ICNIRP-aligned scientists, and other commentators for emphasizing the NTP and Ramazzini findings, relying on trends, and not conducting a conventional meta-analysis. The authors and supporters counter that rare tumors, significant dose trends, controlled bioassays, and concordance warrant high certainty.

The final publication record matters: a WHO-commissioned review did not conclude that the animal evidence was reassuring. It concluded high certainty for the heart-schwannoma signal, with the 2026 corrigendum reporting high

certainty for glial-cell tumors as well. That finding should trigger formal quantitative risk assessment by U.S. health agencies.

IARC

IARC classified RF electromagnetic fields as possibly carcinogenic to humans (Group 2B) in 2011 based on limited human and animal evidence available at that time. Group 2B describes the strength of evidence that an agent can cause cancer; it does not quantify the size of risk. [IARC 2011](#) · [Monograph 102](#)

NTP and Ramazzini were published after the 2011 evaluation. In its 2025–2029 priority report, IARC’s advisory group again designated RF-EMF a high priority for re-evaluation and recommended timing in the latter half of the five-year period while additional bioassays mature. The current classification remains 2B until a future working group completes a new monograph. [IARC priorities report](#)

Why the 2026 Japan–Korea studies do not erase the NTP finding

Two 2026 papers from Japan and Korea exposed male Harlan Sprague-Dawley rats from gestation through two years to 900 MHz CDMA-modulated RF at 4 W/kg. Each study used 70 animals per group, a single RF dose, sham controls, and cage controls. Both reported no statistically significant carcinogenic or genotoxic effect and described their findings as evidence against reproducible carcinogenicity at 4 W/kg. [Japanese study](#) · [Korean study](#)

These studies add high-quality data for one exposure condition. They do not reproduce the NTP design closely enough to falsify the NTP result.

Design feature	NTP rat bioassay	Japan/Korea follow-ups	Why it matters
Animals per group	90	70 in each country	Rare tumors require high statistical power
RF doses	1.5, 3, 6 W/kg plus sham	4 W/kg only plus controls	One dose cannot test a trend or reproduce the positive 6 W/kg condition
Modulations	GSM and CDMA	CDMA only	Waveform may be a biological variable
Sex	Male and female	Male	Narrows generalizability
Main interpretive strength	Dose trend and highest-dose comparison	Carefully controlled result at 4 W/kg	Answers a narrower question

In a July 2026 commentary, Melnick, Moskowitz, and Héroux calculated that if the expected heart-schwannoma incidence at 4 W/kg were about 4.4% against a 0.5% control rate, approximately 258 animals per group would be required for 80% power and 323 for 90% power. They noted that even combining the two countries yields only 140 per group. The Korean study nevertheless observed an unadjusted 3% heart-schwannoma rate in exposed animals versus zero in both control groups—a result that was not statistically significant and cannot be interpreted as a replication by itself. [Melnick et al., 2026](#)

The correct conclusion is neither “the follow-ups prove safety” nor “the null findings do not matter.” It is:

The 2026 Japan–Korea studies found no statistically significant carcinogenicity under their single 4 W/kg CDMA condition. Because they omitted the NTP’s positive 6 W/kg condition, used fewer animals, and had no dose trend, they do not invalidate NTP’s multi-dose result.

Future replications should be powered in advance for rare tumors, include at least three dose levels plus control, reproduce the positive dose and modulation, and publish raw pathology and dosimetry data.

The quantitative challenge: 15–909 times for cancer, 8–24 times for fertility

Melnick and Moskowitz asked the question the existing limit never answered: if animal cancer and reproductive findings were handled using health-protective risk-assessment methods, what whole-body SAR values would result?

For cancer, they used EPA benchmark-dose software on heart schwannomas and combined heart-schwannoma/brain-glioma outcomes, then linearly extrapolated from the lower confidence bound of a 1% benchmark response to an extra cancer risk of one in 100,000. Linear low-dose extrapolation is a standard default when a carcinogenic mode of action is unknown and no safe threshold is established.

They estimated per-hour whole-body SAR values of roughly 0.8–5 mW/kg for that risk target. Against the current 80 mW/kg whole-body public limit, the modeled gap is:

- 15–114× for one hour per day;
- 60–444× for four hours per day;
- 121–909× for eight hours per day.

For male fertility, they applied uncertainty factors of 10 for animal-to-human extrapolation, 10 for human variability, and an additional factor of three when a no-observed-adverse-effect level was not identified. The resulting proposed range was 3.3–10 mW/kg, or 8–24 times below the current public whole-body limit.

These calculations should be independently reproduced, sensitivity-tested, and debated in an open agency docket. The assumptions are consequential:

- linear low-dose extrapolation;
- a one-in-100,000 extra cancer-risk target;
- choice of tumor datasets and survival adjustment;
- time normalization of SAR;
- uncertainty factors for reproductive effects;
- use of whole-body SAR as the dose metric.

But no criticism of an assumption justifies the status quo by default. The policy comparison is asymmetric: Melnick and Moskowitz show their assumptions and calculate risk; the existing FCC limit was not derived from a chronic cancer or reproductive risk assessment at all.

The most defensible policy response is an independent EPA/NIEHS/FDA/FCC assessment that publishes the models, alternative assumptions, uncertainty ranges, and proposed protective levels.

Reproduction and development: why waiting for perfect human data is unacceptable

Sperm, ova, placenta, embryos, and fetal tissues depend on tightly regulated redox balance, calcium signaling, mitochondrial function, DNA fidelity, epigenetic programming, and developmental timing. These systems are not simply smaller adult tissues.

Lai's compilation reports significant reproduction or development effects in 354 of 415 RF papers. The WHO-commissioned experimental male-fertility review and its corrigendum identify reduced pregnancy rate as a high-certainty outcome, while the broader literature includes changes in sperm count, motility, vitality, morphology, testicular endpoints, hormones, and oxidative damage with varying certainty. A separate experimental pregnancy/birth-outcomes review reported statistically significant effects in several outcomes, including fetal resorption or death, fetal weight and length, malformations, and motor functions, while emphasizing uncertainty and heterogeneity. [Pregnancy/birth review](#)

Human observational fertility reviews have generally been less conclusive because personal RF exposure is difficult to reconstruct and self-reported phone use is a weak dose measure. That discrepancy does not make the controlled animal signal irrelevant. It shows why the next human studies need device-derived transmit-power and duty-cycle logs, semen and hormone measures, prospective pregnancy outcomes, and co-exposure data.

Public policy should not expose an entire generation first and ask developmentally specific questions later.

Neurology, sleep, and calcium timing

The nervous system is excitable tissue. Its function depends on voltage gradients, ion channels, neurotransmission, oscillations, synchronization, glial regulation, and sleep-dependent recovery.

Lai's compilation reports significant neurological effects in 396 of 507 RF papers. The endpoints are heterogeneous—electrophysiology, neurotransmitters, behavior, sleep, oxidative markers, blood-brain-barrier measures, and structure—so a single percentage cannot define clinical risk. It does establish a large research domain that an acute-heating standard does not directly protect.

The 2025 CACNA1C sleep-spindle experiment is important because it combines:

- realistic standardized 5G signals;
- sham control and double blinding;
- an objective high-density EEG endpoint;
- genotype stratification;
- a frequency-specific result.

This is the study design the field needs: not a questionnaire asking whether participants felt a signal, but a controlled exposure linked to objective physiology and a biologically relevant susceptibility marker.

Future studies should measure calcium frequency, amplitude, phase, jitter, localization, propagation, termination, and recovery—not only average intracellular calcium. If signal timing is the variable, an average can hide the effect.

What U.S. cancer trends show—and what they cannot show

RF Safe asked for a direct examination of cancer-rate increases. The responsible answer is more powerful than a sweeping claim.

The United States is not experiencing a uniform increase in every cancer or every brain tumor. The 2025 Annual Report to the Nation found that overall male cancer incidence stabilized through 2021 after earlier declines, female incidence increased about 0.3% per year from 2003–2021, and cancer mortality continued to fall. In children, total cancer incidence declined during the short 2017–2021 interval and brain/other nervous system cancer declined in that interval. [SEER Annual Report to the Nation](#)

At the same time, specific anatomically relevant tumor categories and age groups show surveillance signals that deserve focused investigation. Using NCI SEER*Explorer with SEER 21 data from the November 2025 submission through 2023, RF Safe’s supplied queries report:

Tumor signal	SEER observation	Essential interpretation
Non-malignant meningioma	6.59 per 100,000 in 2004 to 12.18 in 2023; +1.27%/yr ages 15–39 since 2009 and +1.97%/yr ages 40–64 since 2008	Strong increase, but mandatory reporting began in 2004 and ascertainment/imaging changed
Thyroid cancer	7.65 per 100,000 in 2000 to 15.35 in 2023; increase, then decline, then +5.10%/yr in 2021–2023	Screening and overdiagnosis are major contributors; trend still requires etiologic surveillance
Salivary-gland cancer	+0.73%/yr from 2000–2023	Anatomically relevant to near-head use, but a trend is not RF attribution
Glioblastoma	3.02 per 100,000 in 2000 to 3.15 in 2023 overall; +1.12%/yr ages 15–39 through 2019 and +0.60%/yr ages 75+	Overall rate is nearly flat; age-specific divergence warrants analysis

Source: [NCI SEER*Explorer](#), SEER Incidence Data, November 2025 submission. Exact query links and definitions are provided in the technical appendix.

The Central Brain Tumor Registry of the United States reports that approximately 73.7% of primary brain and other CNS tumors diagnosed during 2018–2022 were non-malignant, and meningioma accounted for 42.6% of all tumors and 57.4% of non-malignant tumors. A public discussion limited to “brain cancer” omits most primary brain/CNS tumors. [CBTRUS 2025 report](#)

Three rules should govern interpretation:

9. A rising registry rate does not prove RF causation. Screening, imaging, reporting rules, histologic reclassification, obesity, hormones, medical radiation, chemicals, infections, and other factors can change incidence.
10. A flat broad category does not prove no RF effect. Opposing trends by tumor subtype, age, sex, location, latency, and exposure can cancel in an average.
11. The correct study links exposure to outcome. Registry signals should generate nested case-control and cohort studies using operator/device records, transmit-power histories, tumor location, molecular subtype, laterality, latency, and co-exposures.

The surveillance conclusion is therefore not “wireless caused the increase.” It is: population data do not provide a clean bill of safety, and the specific signals are important enough to demand exposure-linked investigation.

Why apparently negative human studies do not close the question

A 2024 WHO-commissioned systematic review of human observational cancer studies concluded with moderate certainty that near-field mobile-phone exposure likely does not increase glioma risk. The large prospective COSMOS cohort likewise reported no increase in glioma, meningioma, or acoustic neuroma across cumulative call-time groups during a median follow-up of more than seven years. [Karipidis et al., 2024](#) · [COSMOS](#)

Those findings belong in any honest evidence review. So do their limitations:

- Call time is not absorbed dose; transmit power varies by network, signal quality, device, use mode, and body position.
- Modern use shifts exposure away from voice calls toward data, streaming, Wi-Fi, Bluetooth, wearables, and devices carried against the body.
- Prospective cohorts reduce recall bias but can dilute contrast if almost everyone is exposed and exposure categories are weak.
- A median seven-year prospective follow-up may be short for some solid-tumor latencies, even when participants had years of prior use.
- Broad tumor categories can hide location, molecular subtype, laterality, or susceptible subgroups.

Conversely, case-control studies reporting increased risk among the heaviest and longest-duration users are vulnerable to recall and selection bias. A 2020 meta-analysis of 46 case-control studies reported increased tumor risk above 1,000 cumulative call hours, but combining heterogeneous designs cannot reconstruct true dose. [Choi et al., 2020](#)

The advocacy conclusion should not pretend this tension does not exist. It should state why it does not justify inaction: human epidemiology has not supplied a sufficiently precise exposure instrument to overrule controlled hazard evidence or certify the current limit.

Biological dissonance and low-fidelity biology: RF Safe's testable synthesis

RF Safe uses biological dissonance to describe an imposed, time-structured environmental signal that disrupts the timing, coherence, or recovery of a living system. Low-fidelity biology describes the proposed downstream state: less precise signaling, higher repair burden, reduced reserve, and greater vulnerability to other stressors.

These terms are an organizing hypothesis, not an established clinical diagnosis. Their scientific value depends on testable predictions.

The framework predicts that biological response will depend on more than average energy:

- modulation and pulse structure;
- signal envelopes and low-frequency components;

- duration, intermittency, and recovery time;
- sequence and phase of multiple signals;
- tissue and subcellular localization;
- calcium-channel and redox-system state;
- genotype and gene regulation;
- developmental window;
- sleep, infection, chemicals, metabolic disease, and other co-stressors.

It also predicts that the earliest and most sensitive endpoints may be fidelity measures rather than a named disease.

Measure fidelity, not only disease

The next generation of studies should quantify:

- calcium oscillation frequency, amplitude, phase, jitter, propagation, spatial localization, termination, and recovery;
- mitochondrial membrane potential, electron-transport efficiency, ATP reserve, oxygen consumption, and mitochondrial ROS;
- redox oscillations, glutathione state, lipid peroxidation, oxidized DNA bases, and antioxidant response;
- transcriptional timing, stress-response genes, inflammatory cytokines, DNA-repair activity, chromatin accessibility, and epigenetic state;
- membrane excitability, synaptic function, EEG oscillations, sleep architecture, and autonomic recovery;
- sperm function, ovarian and placental endpoints, embryo development, and multigenerational outcomes;
- immune signaling, senescence, apoptosis, cell-cycle checkpoints, and recovery after exposure ends.

The decisive experimental contrast is not merely exposed versus sham. It is waveform-preserved versus time-scrambled exposure at the same average SAR, with continuous thermal control. If biology responds differently to the two, timing contains biologically relevant dose information that SAR averaging misses.

The Clean Ether Act: a public-health and innovation program

The Clean Ether Act is RF Safe's proposed federal framework for making connectivity compatible with biological protection. It should be drafted as a technology-forcing transition law, not a prohibition on communication.

Its goal is simple:

Move high-bandwidth indoor connectivity onto wires and light wherever practical; reserve radiofrequency transmission for mobility, emergency, and uses where it is genuinely needed; and protect children and other vulnerable populations with the lowest reasonably achievable RF environment.

The Clean Air Act analogy must be used accurately. The Clean Air Act was not written only to remove lead from gasoline; it provided authority that enabled EPA's phased standards. EPA reports that on-road gasoline lead was eliminated and lead emissions fell by more than 99%. [EPA lead programs](#)

A Clean Ether Act should use the same public-health logic: define the hazard-control goal, set measurable standards, require monitoring and disclosure, create technology-neutral compliance pathways, fund transition, and tighten protections as evidence and alternatives improve.

Title I — Modern exposure standards

- Direct EPA, NIEHS, FDA, and FCC to conduct an independent quantitative RF risk assessment using animal cancer, reproductive, developmental, neurological, and mechanistic evidence.
- Replace a heat-only protection objective with endpoint-specific limits or action levels for chronic exposure.
- Require separate treatment of acute peaks, time-averaged exposure, cumulative duration, pulse structure, modulation, and recovery.
- Establish additional safety factors for children, pregnancy, illness, and susceptible subgroups.
- Require scheduled review at least every five years with public datasets and conflict-of-interest disclosure.

Title II — Biologically relevant premarket testing

- Test realistic modulation, duty cycle, beamforming, proximity, body contact, simultaneous radios, and worst-case adaptive power.
- Include chronic and developmental protocols for representative product categories.
- Require waveform-preserved and time-scrambled comparisons at matched SAR.
- Include oxidative, genotoxic, reproductive, neurophysiological, mitochondrial, immune, and recovery endpoints.
- Publish machine-readable exposure files and full negative as well as positive results.

Title III — Child-priority low-RF environments

Create a federal definition of child-priority spaces, including childcare, K–12 classrooms, pediatric and maternity care, libraries' children's areas, and sleeping areas in federally supported housing.

For these spaces:

- use fiber and Ethernet as the default network backbone;
- provide wired ports and power at desks, beds, teaching walls, and access points;
- use optical wireless for mobility where appropriate;
- keep RF radios off by default when a wired or optical connection is available;
- minimize transmit power and duty cycle rather than maximizing coverage everywhere;
- provide scheduled low-RF sleep and recovery periods;
- prohibit placing continuously transmitting infrastructure immediately adjacent to occupied child-rest areas unless no practical alternative exists and exposure is publicly documented.

Title IV — Li-Fi compatibility and optical-ready procurement

IEEE ratified the 802.11bb light-communications standard in 2023. It provides an interoperable framework for light-based networking that can work alongside the 802.11 ecosystem. Li-Fi is not a universal replacement for every radio link, but it is a technically credible indoor pathway that confines signals by room, reduces RF demand, and uses visible or infrared optical carriers. [IEEE 802.11bb](#) · [IEEE Spectrum overview](#)

The Act should:

- require that federally procured laptops, tablets, and classroom devices become 802.11bb-compatible or support a vendor-neutral optical interface on a staged schedule;
- require optical-ready cabling, power, ceiling/wall locations, and network control in new child-priority construction and major renovation;
- fund multi-vendor pilots in schools, hospitals, libraries, and housing;
- require optical safety, accessibility, cybersecurity, roaming, and performance certification;
- maintain RF fallback for emergencies, outdoor mobility, and legacy access during transition;
- prevent vendor lock-in by requiring open standards and interoperable management.

Title V — Right to know and exposure telemetry

- Require devices to display current transmit power, active radio, duty cycle, and proximity state in a standardized dashboard.
- Provide user-selectable low-exposure modes that reduce power, background transmissions, and unnecessary scanning.
- Require plain-language disclosure of the distance and configuration used for compliance testing.
- Create a personal, privacy-preserving cumulative exposure log that users can opt into for research and self-management.
- Require fixed facilities to publish machine-readable operating power, antenna patterns, and time-varying exposure estimates.

Title VI — Independent research and surveillance

- Rebuild an independent NTP/NIEHS RF program capable of testing 4G, 5G, Wi-Fi, Bluetooth, wearables, optical/RF hybrid systems, and future waveforms.
- Fund preregistered, multicenter replication with independent dosimetry and pathology review.
- Establish genotype-stratified human provocation studies with objective EEG, autonomic, endocrine, redox, and sleep endpoints.

- Link high-quality exposure records to cancer, fertility, pregnancy, neurodevelopment, and sleep outcomes under strict privacy controls.
- Fund alternatives research, including Li-Fi, passive connectivity, low-duty-cycle protocols, and adaptive minimum-power networking.
- Enforce the federal electronic-product radiation program's duty to research and minimize unnecessary exposure under 21 U.S.C. §360ii. [U.S. Code](#)

Title VII — Restore accountable local protection

Section 704 of the Telecommunications Act of 1996 limits state and local authority to regulate the placement, construction, and modification of personal wireless facilities on the basis of environmental effects when facilities comply with FCC rules. [47 U.S.C. §332\(c\)\(7\)\(B\)\(iv\)](#)

The Clean Ether Act should amend this provision so federal limits remain a floor, not a ceiling on health-protective siting. Local measures should be evidence-based, nondiscriminatory, technically feasible, and subject to prompt review, while allowing communities to create larger setbacks from child-priority spaces, require co-location, demand lower-power designs, and prefer fiber or optical alternatives.

Title VIII — Transition funding and accountability

- Create grants for wired and Li-Fi upgrades in schools, childcare, healthcare, and low-income housing.
- Prioritize communities with dense infrastructure and limited ability to reduce personal exposure.
- Establish public performance metrics: RF power avoided, percentage of traffic shifted to wire/light, uptime, cost, and user experience.
- Require annual public reports and an inspector-general audit of research independence, procurement, and enforcement.

A practical implementation roadmap

Years 0–2: measure, standardize, and pilot

- Establish the interagency RF health and technology office.
- Open the quantitative risk-assessment docket.
- Publish standard exposure telemetry and waveform file formats.
- Launch independent replication of NTP and priority reproductive studies.
- Fund at least 100 multi-vendor wired/Li-Fi pilots across schools, pediatric facilities, and housing.
- Freeze new federal procurement that cannot disclose transmit power and duty cycle after a reasonable compliance period.

Years 3–5: make low-RF design the default in child-priority spaces

- Require wired backbones and optical-ready infrastructure in new construction and major renovation.
- Require Li-Fi-compatible or vendor-neutral optical interfaces in federally procured education and healthcare devices.
- Establish child-specific exposure action levels and quiet-period requirements based on the interim risk assessment.

- Launch exposure-linked fertility, pregnancy, sleep, and cancer surveillance cohorts.

Years 6–10: scale and tighten

- Apply revised endpoint-based limits.
- Expand optical compatibility to mass-market devices.
- Shift routine indoor network traffic to wire or light wherever feasible.
- Ratchet down unnecessary RF power using performance-based targets.
- Review local-authority restoration, enforcement, health data, cost, and technology performance.

Immediate protections while policy catches up

Individuals should not be forced to solve a system-level exposure problem alone. Still, low-cost steps can reduce close-proximity exposure without abandoning connectivity:

- use speaker mode or a wired headset for calls;
- keep transmitting devices off the body when practical;
- prefer Ethernet for stationary computers, televisions, and workstations;
- place routers away from beds, desks, and child-rest areas;
- use airplane mode during sleep when connectivity is not needed;
- download content before travel or weak-signal use;
- avoid unnecessary wireless transmitters in infant sleep spaces;
- choose minimum-power settings and turn off unused radios.

Distance and reduced transmit time matter. NTP itself directs the public to FDA advice to reduce call time and use speaker mode or a headset. These measures are not a substitute for protective standards, and they should never be used to blame people who cannot control their workplace, school, housing, or neighborhood exposure.

The rebuttal matrix

“Non-ionizing radiation cannot cause biological effects.”

False as a universal claim. Non-ionizing RF is used deliberately in medicine, and controlled studies report physiological and molecular responses. The real question is which conditions produce adverse outcomes.

“The exposures are below FCC limits.”

That establishes compliance with the current framework. It does not show that chronic oxidative, genetic, reproductive, neurological, or susceptible-subgroup effects were tested or prevented.

“Brain cancer rates are stable.”

Overall glioblastoma is nearly flat in SEER through 2023, and recent national childhood brain-cancer trends declined. Other specific categories and age groups rose. Registry trends alone neither prove nor disprove RF causation because they lack individual exposure and are affected by diagnosis and reporting.

“The Japan and Korea studies failed to reproduce NTP.”

They found no statistically significant effect at one 4 W/kg CDMA condition. They used fewer animals, omitted the positive 6 W/kg NTP dose, included no dose trend, and did not test GSM. They narrow one question; they do not falsify the NTP design.

“WHO found very-low-certainty oxidative evidence.”

The review identified serious study-quality problems and judged the pooled evidence very uncertain. That is not evidence of absence. The contrast with Lai and Yakymenko reflects different inclusion, biomarker, and weighting rules. The solution is preregistered, multi-marker, multicenter replication.

“Animal exposures were higher than normal human exposure.”

Yes, especially in NTP. Hazard studies use controlled challenges to identify target organs. Ramazzini reported a concordant rare tumor at much lower whole-body SAR. Translating these findings requires formal risk assessment, which the current limit never performed.

“There is no single accepted non-thermal mechanism.”

Environmental health policy does not require one mechanism to explain every endpoint before acting. Oxidative stress, calcium signaling, mitochondrial function, DNA repair, and context-dependent gene regulation form testable pathways. Lack of a completed mechanism map is a research gap, not a safety certificate.

“Li-Fi cannot replace every wireless connection.”

Correct—and irrelevant to the proposal. The Clean Ether Act uses fiber/Ethernet and Li-Fi to replace avoidable indoor RF traffic while preserving RF for mobility, emergency, outdoor, and fallback use.

The research agenda that can end the argument

The debate persists partly because the field keeps repeating studies that measure the wrong thing, use incomparable waveforms, underpower rare outcomes, or average away susceptibility.

RF Safe calls for a transparent national protocol:

12. Publish the signal: full I/Q or equivalent waveform files, carrier, bandwidth, modulation, duty cycle, peak-to-average ratio, polarization, and spectral components.
13. Measure tissue dose: SAR and power density plus spatial microdosimetry, induced fields, thermal microgradients, and uncertainty.
14. Control heat continuously: temperature at relevant tissue and subcellular scales where feasible.
15. Test timing: matched-average-energy waveform-preserved, time-scrambled, continuous-wave, and sham conditions.
16. Test dose shape: at least three exposure levels plus control, including low intensity and a challenging high dose.
17. Build in recovery: repeated exposure–recovery cycles with longitudinal sampling.
18. Stratify the receiver: sex, age, pregnancy, genotype, disease state, mitochondrial reserve, and co-exposures.
19. Use objective endpoints: calcium dynamics, redox, mitochondria, DNA damage and repair, multi-omics, electrophysiology, fertility, development, pathology, and behavior.
20. Power rare outcomes: prespecified calculations and adequate group sizes.
21. Replicate independently: common protocol, independent laboratories, blinded analysis, shared raw data, and publication regardless of result.

If such a program finds no reproducible chronic effects below a revised threshold, it will strengthen public confidence. If it finds effects, it will identify the dose metrics and protections needed. Either outcome is better than regulating by inherited assumption.

The case for action now

Public-health action does not require a claim that every Wi-Fi router causes disease or that every cancer trend is wireless-driven. It requires a proportionate judgment about evidence, exposure, vulnerability, alternatives, and the cost of error.

The evidence supports five conclusions today:

22. RF can be biologically active without ionizing tissue.
23. The current U.S. limit is rooted in an acute heating paradigm and was not derived from chronic cancer, fertility, developmental, neurological, or susceptible-subgroup risk assessment.
24. Controlled animal evidence and the final WHO-commissioned review record establish a serious cancer hazard signal; reproductive evidence is also strong enough to quantify and act on.
25. Biological response varies by waveform, tissue, molecular state, genotype, sex, life stage, and recovery—variables poorly represented by a single averaged energy metric.
26. Wired and optical technologies make meaningful exposure reduction compatible with modern connectivity.

The remaining uncertainty is not a reason to preserve a standard that does not answer the relevant questions. It is a reason to build a standard that does.

The public message

Invisible does not mean irrelevant.

Non-ionizing does not mean non-biological.

Legal exposure does not mean demonstrated safety.

A heat-compliance standard cannot be assumed to protect biological timing, repair, development, fertility, or susceptible people.

The body is not a passive bag of salt water waiting to be heated. It is an electrical, rhythmic, redox-sensitive, developmentally timed network. Wireless signals are time-structured environmental inputs. Whether biology “decodes” their communications content is not the claim; whether waveform and timing change biological response is an empirical question—and the evidence says that question belongs in safety testing.

RF Safe calls the consequence of persistent mismatch biological dissonance and the resulting loss of precision low-fidelity biology. Those concepts now need the most rigorous possible tests.

America should not have to choose between connectivity and children's biology. The engineering pathway exists: fiber, Ethernet, minimum-power radio, optical wireless, exposure telemetry, better testing, and independent science.

The next era of connectivity should be judged not only by speed, coverage, and profit, but by biological compatibility.

Pass the Clean Ether Act. Make Li-Fi compatibility a national priority. Restore independent RF research. Protect child-priority spaces now.

Technical appendix A — exact SEER query links supplied for the tumor-trend analysis

- [Non-malignant meningioma, age groups, SEER 21, 2004–2023](#)
- [Thyroid cancer, age groups, SEER 21, 2000–2023](#)
- [Salivary-gland cancer, age groups, SEER 21, 2000–2023](#)
- [Glioblastoma, age groups, SEER 21, 2000–2023](#)

Non-malignant brain/CNS tumors became nationally reportable for diagnoses beginning January 1, 2004, which is an essential limitation for early trend interpretation. [SEER reporting rule](#)

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Editorial integrity statement

This paper is advocacy. Its purpose is to make the strongest evidence-based case for precautionary reform. It distinguishes controlled hazards, modeled risks, mechanistic findings, human associations, surveillance trends, and RF Safe’s hypotheses so that rhetorical force does not depend on overstating what any one study proves. “Biological dissonance” and “low-fidelity biology” are RF Safe’s proposed synthesis and research framework, not established medical diagnoses. Cancer-registry trends are surveillance signals, not proof of RF causation. The Clean Ether Act is a policy proposal, not existing law.