

Protect Biological Fidelity

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A 5,000-word position statement, references, and six pathway illustrations. The combined submission packet also includes John Coates's complete comment as Attachment B.

Protect biological fidelity now

HHS must act now to reduce unnecessary wireless radiation exposure and protect the biological coordination on which healthy development, reproduction, and lifelong resilience depend. I submit this position statement as Katie Webb of QuantaX Technology. I support John Coates's accompanying comment and ask HHS to consider its complete evidence record, S4-Mito-Spin framework, and proposed developmental, cancer, metabolic, and immune pathways together with this statement. The central public-health obligation is to protect the capacity of living systems to develop correctly, regulate themselves, repair damage, and pass a sound biological foundation to the next generation. [1]

I believe low-fidelity biology is the organizing problem HHS needs to investigate and prevent. By biological fidelity, I mean the precision with which cells receive signals, coordinate energy and information, execute the appropriate response, and recover. By low-fidelity biology, I mean deterioration in that performance: mistimed signaling, unreliable coordination, excessive compensation, and reduced reserve. These terms identify a measurable research program. They direct attention upstream of diagnosis, where developmental decisions are made and the capacity to withstand later stress is established.

The evidence already demands action. Lifetime animal studies identified malignant tumors. Controlled prenatal experiments produced behavioral and brain-function changes. Human cortical-organoid research identified disrupted developmental differentiation. Molecular experiments identified field-responsive calcium signaling and gene regulation. Quantitative risk assessment using EPA methods produced protection levels far below existing public limits. HHS must bring these findings into a prevention program with funding, responsible officials, and deadlines. [2-7,9,13]

Future generations cannot participate in this docket. Their interests must be represented now. A decision to postpone protection preserves today's exposure conditions through pregnancies, childhoods, and reproductive lifetimes that cannot be repeated. My position is that HHS must protect developmental fidelity today, investigate transmission across generations directly, and make lower-exposure connectivity readily available. Waiting for a complete count of affected children would turn preventable exposure into the condition under which the evidence is collected.

Apply the cancer and reproductive evidence to public protection

The National Toxicology Program reported clear evidence of carcinogenic activity involving malignant heart schwannomas in male rats exposed to GSM- or CDMA-modulated radiofrequency radiation. It also reported some evidence involving malignant gliomas. The Ramazzini Institute independently reported increased heart schwannomas in male rats in a prenatal-to-lifetime experiment using substantially lower whole-body exposure levels. The convergence on a rare tumor deserves direct regulatory attention. HHS should use these studies to determine protective limits and to investigate the biological processes preceding tumor formation. [2,3]

The WHO-commissioned animal-cancer systematic review rated the evidence for increased glioma and heart schwannoma in male rats as high certainty. That finding must carry weight in HHS's response. A standard designed around acute heating does not answer the chronic-cancer question simply because a transmitter complies with it. HHS must publish how animal pathology, exposure duration, tissue susceptibility, and uncertainty affect its assessment of public protection. [4]

Melnick and Moskowitz's 2026 analysis makes the regulatory gap quantitative. Applying EPA benchmark-dose tools and low-dose extrapolation to animal tumor data, the authors calculated that the public whole-body limit of 0.08 W/kg is 15–900 times above their modeled exposure levels for one additional cancer per 100,000 people, depending on daily exposure duration. Their male-reproductive assessment yielded protection levels of 3.3–10 mW/kg, making the existing 80 mW/kg limit 8–24 times higher. These comparisons belong at the center of HHS's response. [5]

The authors applied established risk-assessment procedures: model the observed response, identify a point of departure, extrapolate toward lower exposure, and account for uncertainty and differences in susceptibility. EPA's guidance explicitly considers animal evidence, sensitive life stages, cumulative burdens, and the timing of exposure. HHS and EPA should apply that discipline to wireless radiation and publish a federal quantitative assessment. The comparison above concerns whole-body SAR; the localized one-gram device limit is a different measurement. Keeping the measures correct makes the regulatory demand stronger. [5,6]

I ask HHS to commission this assessment immediately, with public access to the models, assumptions, alternative calculations, and resulting recommendations. Require reviewers to explain how the proposed limits protect pregnancy, reproduction, childhood, and repeated daily exposure. Identify which endpoints determine the recommended level and which remain insufficiently tested. Pair the assessment with practical exposure reduction from the outset. A research timetable must include a protection timetable.

Cancer and fertility are also connected to the fidelity question. Tumors develop through failures of growth control, repair, and tissue regulation. Reproductive success depends on the integrity of gametes and the developmental information they carry. These are reasons to examine regulatory precision before pathology becomes visible. HHS should fund early measurements within long-term studies so that the same research identifies the warning signals, the later outcomes, and the interventions that interrupt the sequence.

An exposure assessment must preserve the periods that matter biologically. Record the pattern during conception, embryonic development, sleep, and recovery instead of collapsing an entire day into one number. Identify simultaneous sources and distinguish external field measurements from absorbed dose in the relevant tissue. When comparing studies, retain their actual frequency, duration, geometry, and biological preparation. This gives HHS a defensible basis for deciding which findings apply to which conditions and where additional measurements are required. The result should be a protection framework that can respond to changes in technology without discarding the biological endpoints that people need protected.

Put the Yale developmental findings at the center

The Yale study by Aldad, Gan, Gao, and Taylor is a direct experimental warning about developmental exposure. Pregnant mice were exposed to an active cellular telephone, and their offspring showed hyperactivity, impaired memory, and altered glutamatergic synaptic transmission in prefrontal-cortex neurons. The study connected an exposure during development to later behavior and measurable brain function. These findings belong prominently in the federal record on developmental protection. [7]

Senior author Hugh Taylor stated: “We have shown that behavioral problems in mice that resemble ADHD are caused by cell phone exposure in the womb.” That is the researchers' account of their experiment. I ask HHS to respond to the finding with a prenatal-protection program, independent replication, and modern exposure characterization. The question for public health is what action protects developing children while the remaining exposure and mechanism questions are resolved. [8]

The important sequence is exposure during a formative window followed by altered function later. A developmental system must produce the right cells, connections, and signaling relationships in the right order. A disturbance during construction can matter long after the original input has ended. HHS must examine the developmental sequence itself and the persistence of its consequences. An adult exposure study cannot stand in for an experiment during neural development.

Cakir and colleagues' human cortical-organoid study adds a different experimental level. RF exposure maintained radial-glia stem-cell identity and delayed differentiation; the investigators also reported rescue of developmental defects through BET-protein inhibition. This connects RF exposure with a manipulable regulatory pathway in a human-derived developmental model. HHS should replicate that result and measure the upstream calcium, mitochondrial, and chromatin events in the same preparation. [9]

Taken together, these experiments justify a coordinated program spanning prenatal animal exposure, human developmental models, and prospective pregnancy cohorts. The program must measure developmental stage, actual absorbed exposure, signaling dynamics, tissue organization, and later function. It must investigate the conditions under which a developmental disturbance begins and the conditions that prevent it. Those are actionable questions with consequences for maternal care, household technology, workplace practice, and public infrastructure.

A developing child gets one chance at each developmental window. That fact should govern the urgency of HHS's response. Families deserve practical guidance and affordable ways to reduce avoidable exposure before conception and throughout pregnancy. Schools and childcare facilities should receive the infrastructure and technical support to provide reliable connectivity with fewer unnecessary transmissions. The burden of resolving the evidence belongs with adequately funded public institutions, while protection must reach the people who cannot choose their surroundings.

Investigate the complete S4-Mito-Spin pathway

The S4-Mito-Spin framework gives HHS a connected set of mechanisms to test. Its proposed sequence runs from a structured electromagnetic input through field-responsive molecular machinery, altered ion-channel or calcium timing, mitochondrial and redox mismatch, changed gene regulation, and persistent loss of functional precision. Each step has a physical variable, a biological measurement, and an intervention capable of testing causal direction. John Coates's full comment supplies the extended architecture and its supporting literature. [1,31]

The S4 branch identifies the voltage-sensing machinery of voltage-gated ion channels as an initiating target. The research task is to determine whether a characterized field changes channel operation and whether altering the relevant sensor changes the response. Record membrane voltage, channel current, calcium events, and downstream function together. An intervention that selectively removes field sensitivity while preserving ordinary channel function would identify the receiving interface far more precisely than an isolated downstream biomarker.

Calcium timing already has an established role in cellular information processing. Dolmetsch and colleagues showed that calcium oscillations regulate the efficiency and specificity of gene expression. De Koninck and Schulman demonstrated frequency-sensitive decoding by CaMKII. These experiments establish why the timing

of a signal matters to the biological decision it produces. HHS should require measurements of pulse frequency, duration, spatial organization, and phase relationships alongside average calcium concentration. [10,11]

Rao and colleagues provide a direct RF anchor. In stem-cell-derived neuronal cells, an 800 MHz exposure at 0.5 W/kg increased spontaneous calcium spiking; their experiments implicated calcium-channel and intracellular signaling pathways. This supplies a reproducible experimental question about the relationship between an external waveform and an internal signaling pattern. HHS should independently reproduce the preparation and then test contemporary waveforms under matched absorption and temperature conditions. [12]

Kim and colleagues' 2026 Cell study identified CYB5B through a CRISPR screen as an essential mediator of an engineered electromagnetic-field-responsive gene switch. Activation depended on rhythmic calcium dynamics. This is a molecular demonstration that a defined electromagnetic input can engage a calcium-pattern-dependent route to gene expression. Its low-frequency operating conditions provide a starting point for transducer research and comparison with separately characterized telecommunications exposures. [13]

The mitochondrial branch asks how an initiating disturbance recruits energy metabolism, redox signaling, and compensation. Xie and colleagues reported a mitochondrial unfolded-protein response after 900 MHz exposure in mouse bone-marrow stromal cells and used JNK2 interference to test pathway involvement. HHS should extend this kind of intervention study with simultaneous calcium measurements, respiratory reserve, compartment-specific reactive species, and repeated physiological challenges. Follow recovery and retained burden rather than ending observation at the first changed signal. [14]

The spin branch concerns field-sensitive reaction chemistry. Usselman and colleagues observed changes in superoxide and hydrogen-peroxide signals under a 7 MHz magnetic exposure combined with a controlled static field. Sherrard and colleagues reported cryptochrome-dependent responses under pulsed magnetic stimulation. These studies identify experimental systems for testing how field parameters change reaction products and downstream cellular activity. Their frequencies, field strengths, illumination, and molecular conditions must travel with their results into replication. [15,16]

Density gating asks why different receivers respond differently. Measure channel abundance, mitochondrial organization, reactive chemistry, energy demand, and repair capacity before exposure, then test whether those measurements predict response. Add the persistence gate: turnover, clearance, recovery time, and developmental deadlines. Genotype belongs beside those measurements. Sousouri and colleagues' controlled human sleep study found a CACNA1C-genotype-dependent spindle-frequency response after 3.6 GHz exposure. HHS must investigate susceptibility that an overall average can conceal. [17]

The framework also requires a clean physical distinction between a modulated RF carrier and a separately applied ELF field. HHS should characterize both and test their coupling, with matched thermal controls and recorded waveforms. The historical Litovitz and Farrell experiments, in which added temporally incoherent magnetic fields suppressed reported embryo or enzyme responses, make temporal structure an experimental variable. The investigation should explain both response and suppression through measured biology. [18]

Measure loss of reserve before disease emerges

Low-fidelity biology describes the upstream vulnerability that these experiments should resolve. A cell can maintain an apparently normal output by spending more energy, activating additional repair, or changing its response threshold. A resting measurement can therefore miss the cost of maintaining that output. The relevant question is how reliably the system responds to a challenge, how much compensation it requires, and whether it returns to its previous operating state. The next challenge reveals how much reserve remains.

John's meta-disease construct organizes that question across tissues. In the proposed developmental pathway, mistimed signals change differentiation and tissue organization. In the cancer pathway, impaired correction allows errors to persist. In the metabolic pathway, energy supply and physiological demand become less closely coordinated. In the immune pathway, activation becomes poorly proportioned or fails to resolve. These are separate functional outcomes linked by a proposed loss of regulatory precision. The accompanying images show the connections HHS should investigate. [1]

Belyaev's and Markova's experiments on DNA-repair foci give the repair branch specific cellular endpoints. Volkow and colleagues' human PET experiment found a regional glucose-metabolism response after mobile-phone exposure, making spatially resolved metabolism another relevant endpoint. HHS should connect these measurements to repair completion, energy demand, tissue function, and recovery. A regional response deserves regional investigation even when a whole-organ average changes little. [19,20]

The ceLLM framework broadens the receiver to the organized living cell. Its membranes, channels, RNA, chromatin, organelles, and prior activity determine how an input becomes a response. The research program should distinguish immediate electrical and chemical state from slower changes in expression, structure, and cell identity. It should also test the proposed photonic-redox feedback branch with controlled optical access, calibrated photon measurements, and replay experiments. A model becomes useful when its measured components predict the next response. [1]

I support fidelity measurements that distinguish accurate reception, appropriate execution, reliable feedback, and beneficial adaptation. Test these functions during development and across repeated exposure and recovery cycles. The proposed fidelity debt is the cost of compensation carried forward into the next challenge. HHS should determine when that burden resolves, when it persists, and which interventions preserve function. This puts prevention before the loss of tissue performance becomes a diagnosis.

The aging literature strengthens the importance of the underlying question. Yang and colleagues experimentally investigated epigenetic information loss in mammalian aging, and Debès and colleagues linked transcriptional changes with longevity outcomes. These studies make maintenance of regulatory information a concrete biological subject. HHS should investigate whether environmental perturbations advance failures of coordination in susceptible systems and whether protecting recovery preserves reserve. The objective is to protect healthy function throughout life. [26]

Protect the biological information carried into the next generation

The generational case begins with development itself. Exposure during pregnancy reaches a developing organism while tissues and regulatory systems are being established. Effects arising in that window can shape later health, learning, reproductive capacity, and resilience. The Yale findings make that concern experimentally concrete. HHS must follow exposed offspring through development and adulthood instead of treating a normal birth or an early resting measurement as the end of the investigation. [7]

Paternal exposure also deserves direct investigation. Yan and colleagues exposed male mice to 2.0 GHz RF, with estimated whole-body SAR of 0.125–0.5 W/kg, and reported increased testicular germ-cell apoptosis and sex-specific differences in offspring growth and glucose metabolism. Standard measures of sperm quality and pregnancy rate did not capture those offspring findings. This is a direct reason to include offspring physiology in reproductive assessment and to extend the design across lower doses, modern signals, and additional generations. [21]

The experiment HHS funds must track the actual route of transmission. Following an exposure during pregnancy, the mother is F0, the fetus is F1, and the fetus's developing germ cells contribute to F2. F3 is the

first generation beyond those directly exposed compartments. After paternal preconception exposure, F2 is the corresponding first generation beyond the exposed sperm and resulting offspring. The research must follow these lineages to test transgenerational inheritance through the germline. [22]

Environmental information can reach offspring through molecular routes beyond a change in DNA sequence. Chen and colleagues transferred sperm small-RNA fractions from high-fat-diet male mice into normal zygotes and produced metabolic disturbances in offspring. Tomar and colleagues demonstrated sperm-to-oocyte transfer of mitochondrial RNAs and linked paternal dietary and mitochondrial state with offspring metabolism. These diet experiments identify molecular transmission routes that the RF research program should examine directly. [23,24]

The question is specific: does characterized RF exposure alter gamete RNA, chromatin, DNA integrity, or mitochondrial state in ways that change embryonic regulation and later function? Measure those events in sequence. Separate maternal and paternal lineages, test molecular mediation, and follow unexposed descendants. Determine whether preserving calcium-redox coordination prevents the gamete change and whether manipulating the identified carrier changes the offspring outcome. That is a causal research program directed toward prevention.

Generational studies should preserve a clear family tree for every animal, specimen, and result. Record which ancestors were exposed, when exposure occurred, which descendants remained unexposed, and how breeding partners were selected. Keep lineage identifiers linked to molecular and functional measurements. Follow apparently unaffected descendants as well as affected ones, because the research question includes delayed expression and differences in susceptibility. Establish funding that survives ordinary grant cycles, with specimen custody and data access maintained across institutions. This investment creates the capacity to identify a generational consequence before separate laboratories lose the information needed to recognize it.

The long horizon is biologically consequential. Korolenko and colleagues' 2026 rat study followed lineages after ancestral exposure to the fungicide vinclozolin across twenty generations, reporting persistent epigenetic changes and later disease and reproductive abnormalities. That chemical-exposure experiment establishes why the duration of generational follow-up matters. It gives HHS a reason to design RF studies that can detect inherited consequences beyond the initially exposed generation and to preserve the specimens needed for later analysis. [25]

The public-health case for protecting future generations also includes repeated exposure. A population can encounter the same environmental stressor during successive pregnancies without a germline inheritance mechanism. Policy that leaves avoidable exposure in place reproduces that opportunity for developmental disruption in every generation. HHS must address both routes: continuing direct exposure across generations and biological transmission after exposure ends. Each has different measurements, and both require attention now.

I reject a policy timetable that makes children wait for their grandchildren to demonstrate whether a preventable burden was inherited. Establish the multigenerational experiments now, preserve long-term funding, and reduce unnecessary exposure while they proceed. A protective action can be adjusted as evidence improves. A developmental opportunity that has passed cannot be scheduled again. Public institutions should carry the cost of investigation and prevention rather than leave future families to carry the consequences of delay.

Fund experiments that connect the pathway to the outcome

HHS should establish a coordinated biological-fidelity program with common protocols and independent laboratories. Begin with exposure systems that record carrier frequency, modulation, pulse structure, duty cycle, peaks, spatial absorption, temperature, and recovery intervals. Publish the actual waveforms. Compare sham, carrier-only, and pulsed conditions at matched average SAR, and characterize any separately applied low-frequency fields. Match the exposed biological preparation to the question being asked.

In developmental models, measure calcium timing, membrane potential, mitochondrial respiration, reactive-species location, transcription, chromatin regulation, cell-cycle progression, differentiation, and tissue organization on the same time axis. Follow the early response into later function. Use models that perform the developmental task under investigation: neural-tube closure requires a closure-capable preparation; cortical differentiation requires an appropriate cortical model. HHS should fund the relevant systems together and connect them through shared endpoints.

Use targeted interventions to locate the causal steps. Test CYB5B dependence and rescue, channel dependence, mitochondrial coupling, and downstream chromatin readers. Record an exposure-associated intracellular pattern, block its initiating interface, and replay the pattern with an independently controlled actuator. Compare faithful replay with a scrambled pattern matched for total activity. Determine whether correcting the timing restores the affected function. This design tests the proposed mechanism directly and creates a route to prevention.

The generational program should include maternal gestational exposure, paternal preconception exposure, both sexes, and adequately powered lineage comparisons. Follow maternal lines through F3 and paternal preconception lines through F2, then continue successful studies further. Maintain unexposed descendant groups and separate groups with repeated exposure in every generation. Measure gamete integrity, sperm and oocyte RNA, methylation, chromatin state, embryo development, fertility, behavior, metabolism, and tissue pathology. Retain samples and exposure records for independent analysis.

Control litter effects, parental physiology, handling, cage conditions, and background fields. Use cross-fostering and assisted-reproduction or molecular-transfer experiments where scientifically appropriate to distinguish germline transmission from gestational, parental-care, and shared-environment effects. Prespecify primary outcomes and statistical comparisons. Blind the outcome assessors. Require external replication before turning an experimental marker into a clinical test. These procedures make the program capable of identifying a mechanism that public protection can address.

Human research should begin before conception and follow pregnancy, infancy, childhood, and later reproductive health. Use measured exposure histories rather than screen time alone. Record device placement, active transmissions, occupational exposure, sleep, medication, nutrition, relevant co-exposures, and developmental timing. Obtain informed consent for genotype and biological sampling. Protect privacy while preserving enough exposure detail to distinguish a real biological pattern from a poorly measured average.

Restore a sustained NTP RF program and connect its long-term outcomes with these early fidelity measurements. Require a shared public repository for protocols, dosimetry, raw observations, analysis code, corrections, and prespecified results. Fund independent teams to reproduce the most consequential findings, including Yale prenatal behavior, cortical-organoid differentiation, CYB5B-calcium signaling, and genotype-conditioned sleep responses. Assign an accountable HHS lead and publish annual milestones. A program built around identifiable deliverables can produce protection rather than another unconnected list of studies.

Reduce avoidable exposure during critical windows

HHS should issue clear exposure-reduction guidance for prospective parents, pregnancy, childcare, schools, sleeping environments, and relevant workplaces. Explain how to reduce unnecessary transmissions and avoid carrying an actively transmitting device against the body when the task permits another arrangement. Make wired connections, radio-off controls, and appropriate optical alternatives practical choices. Guidance must be usable by families with ordinary budgets and by institutions without specialized engineering staff.

Public procurement should fund the infrastructure required to make those choices real. A school cannot implement a wired-first policy with tablets that lack suitable adapters or classrooms without adequate cabling. A hospital needs validated reliability, infection-control compatibility, accessibility, and emergency communications. HHS should support complete installations, staff training, maintenance, and measured verification. The public benefit comes from actual reductions in unnecessary exposure while essential services continue to work.

Require body-contact testing where contact is a foreseeable use of a wireless device. Publish test distance, averaging mass and time, simultaneous-transmitter conditions, operating modes, and the waveform characteristics needed to interpret exposure. Commission independent testing of retail devices. John Coates's accompanying comment includes the FCC source records behind this request. Families should be able to understand what was tested and whether that condition represents how a device is used. [1]

Protect people who have limited control over their environment. Pregnancy, childhood, disability, medical conditions, and workplace dependence affect a person's ability to change exposure. HHS should provide institutions with implementation standards and resources, including options for people reporting exposure-related symptoms. Establish respectful clinical assessment and exposure documentation. The success of a public-health program should be measured partly by whether meaningful choices reach those who cannot redesign their surroundings themselves.

Lower-exposure pilots should report occupied-room measurements, transmissions displaced, service reliability, costs, energy use, and accessibility. Collect baseline data before installation and publish follow-up results under realistic traffic conditions. Prefer solutions that reduce exposure at the source and can be maintained over time. A credible pilot should show the physical change it achieved and the resources needed to reproduce it elsewhere.

The responsibility must be institutional. Parents should receive understandable information and useful options, without being made responsible for infrastructure decisions they did not control. Agencies, manufacturers, network operators, schools, employers, and healthcare systems should each publish what they will do. HHS can coordinate this work immediately while the longer research program proceeds. Prevention should be visible in purchasing decisions, facility design, testing practices, and public guidance.

Make health agencies accountable for health protection

Public Law 90-602 supplies an existing federal foundation for action. Its electronic-product radiation-control provisions, now codified in the Federal Food, Drug, and Cosmetic Act, direct the Secretary to conduct a program protecting public health, support research to minimize unnecessary radiation exposure, evaluate exposure conditions, and develop and test exposure-reduction techniques. HHS should publish a wireless-radiation program that carries out those duties with a budget and named responsible offices. [27]

I ask HHS to make an explicit performance-standards determination under 21 USC 360kk, supported by the current cancer, developmental, reproductive, and mechanistic record. The agency should identify the hazards it is addressing, the evidence used, and the protection its chosen measures provide. EPA should contribute its

risk-assessment expertise. NIH and NIEHS should fund the experimental program, and CDC should develop surveillance and clinical communication. Coordination must produce decisions with identifiable ownership.

Congress should assign primary authority for RF health-protection standards to HHS and EPA through a clear statutory framework. The FCC should retain its communications and spectrum-management functions. Health standards require accountable medical, developmental, toxicological, and environmental expertise. The record should show who evaluated each major endpoint, how conflicts were managed, and why the final protection level was selected. Institutional familiarity with an older standard cannot replace a current health assessment.

I also support repeal of the Section 704 restriction codified at 47 USC 332(c)(7)(B)(iv), which limits state and local decisions based on RF environmental effects when facilities comply with FCC regulations. Communities need meaningful participation informed by current health evidence. Federal protection must be strong enough to protect vulnerable residents, and local consideration must be able to address the conditions people actually experience. [28]

Within ninety days, HHS should publish its work plan, lead office, initial exposure-reduction measures, and independent-review process. Within one year, it should publish the quantitative risk assessment, realistic device-testing recommendations, and funded developmental and generational protocols. Report progress annually against public milestones. Explain any missed deadline and provide a revised date. A request for information should lead to a public record of what the agency decided and what changed because people participated.

Public accountability should include a response matrix connecting the major evidence submitted to the agency's decisions. Identify the studies selected for replication, the protection measures adopted, the unanswered questions assigned for investigation, and the official responsible for each next step. Make the supporting calculations and protocols accessible to independent scientists. Invite affected communities to assess whether guidance is usable and whether institutional changes reach them. A technically detailed report has public value when its conclusions produce understandable instructions, funded implementation, and measurable protection. HHS should make that connection visible in every annual progress report and in its communications with Congress.

Build lower exposure connectivity through the Clean Ether Act

QuantaX Technology's perspective includes the engineering choices available to society. Useful connectivity can be delivered through fiber, Ethernet, and optical networks. HHS should evaluate those architectures by the unnecessary RF transmissions they displace, their accessibility, reliability, and operating costs. The design goal is to make lower-exposure service convenient enough to become the ordinary choice in settings where it meets the task.

I support the Clean Ether Act proposed in John Coates's accompanying comment. It calls for a staged transition of suitable indoor connectivity toward wired and optical systems, device interoperability, public-building and school pilots, and investment in domestic photonics. IEEE 802.11bb supplies an existing light-communications standard around which compatibility work can proceed. HHS should help turn that direction into funded demonstrations and measurable procurement requirements. [1,29]

John Coates is the named inventor on U.S. Patent 11,700,058 B2 involving optical wireless communications using germicidal light. The work demonstrates the multifunctional potential of photons within a designed network. Public research should evaluate communications, protective functions, and suitable operating conditions for each application. Fund independent performance testing and accessible implementations that can serve schools, healthcare, homes, workplaces, and public facilities. [30]

RF Safe has dedicated nearly thirty years to open-source design, with product designs developed for the public domain. That public-benefit direction should inform the transition. Support published interfaces, interoperable equipment, affordable adapters, and procurement that allows multiple suppliers to compete. The value of an exposure-reduction architecture should be demonstrated through measurements that independent engineers and communities can examine. Public investment should make effective options broadly available.

Start with facilities where children spend long periods and where a managed network is practical. Establish a baseline, install the wired or optical alternative, disable the unnecessary radios it replaces, and verify the resulting exposure during actual use. Provide training and maintenance funding so the installation continues to deliver its intended benefit. Preserve emergency communications and essential accessibility functions through tested operational plans.

The transition should also protect choice outside institutions. Require clear radio controls, dependable wired operation, and optical compatibility as the supporting ecosystem develops. Publish repair and support requirements that keep lower-exposure equipment usable. HHS should work with standards organizations, public purchasers, and Congress to remove practical barriers. Families should not need unusual technical knowledge or expensive custom installations to choose a network architecture that reduces avoidable exposure.

Protect fidelity before the developmental window closes

The evidence and the engineering options give HHS a concrete decision. It can apply modern risk assessment, fund the mechanisms and generational experiments, and begin reducing avoidable exposure now. Those actions reinforce one another. Better measurements improve protection; practical exposure reduction creates opportunities to evaluate implementation; long-term studies establish which early biological changes predict persistent harm. The program should be designed so that each result informs the next decision.

I ask HHS to accept the accompanying John Coates comment and the illustrated pathways as part of this submission's supporting record. His comment answers Questions 2 through 18 and supplies the expanded mechanisms, sources, FCC records, and legislative program. My statement emphasizes the responsibility running through those materials: protect the fidelity of development, reproduction, cellular regulation, and recovery before the resulting burden is carried into another lifetime. [1]

The duty to future generations requires decisions during the period when prevention remains possible. Once a developmental program has run under an avoidable stressor, later administrative review cannot rerun that pregnancy or childhood. HHS must account for that asymmetry when choosing its timetable. Fund the research that resolves the pathways, provide the infrastructure that reduces exposure, and make the institutions responsible report what they have accomplished.

Every year of delay includes pregnancies and childhoods that a later policy cannot protect retroactively.

My position is clear. Act on the cancer and reproductive evidence. Put the Yale developmental findings and human-organoid results into the protection program. Test biological fidelity and transmission across generations. Reinvigorate Public Law 90-602. Support repeal of Section 704's health-based siting restriction. Place health standards under health agencies and advance the Clean Ether Act. Protect the biological information on which the next generation will depend.

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References

Numbered references support the statement and pathway captions. Study corrections are linked with the original publications.

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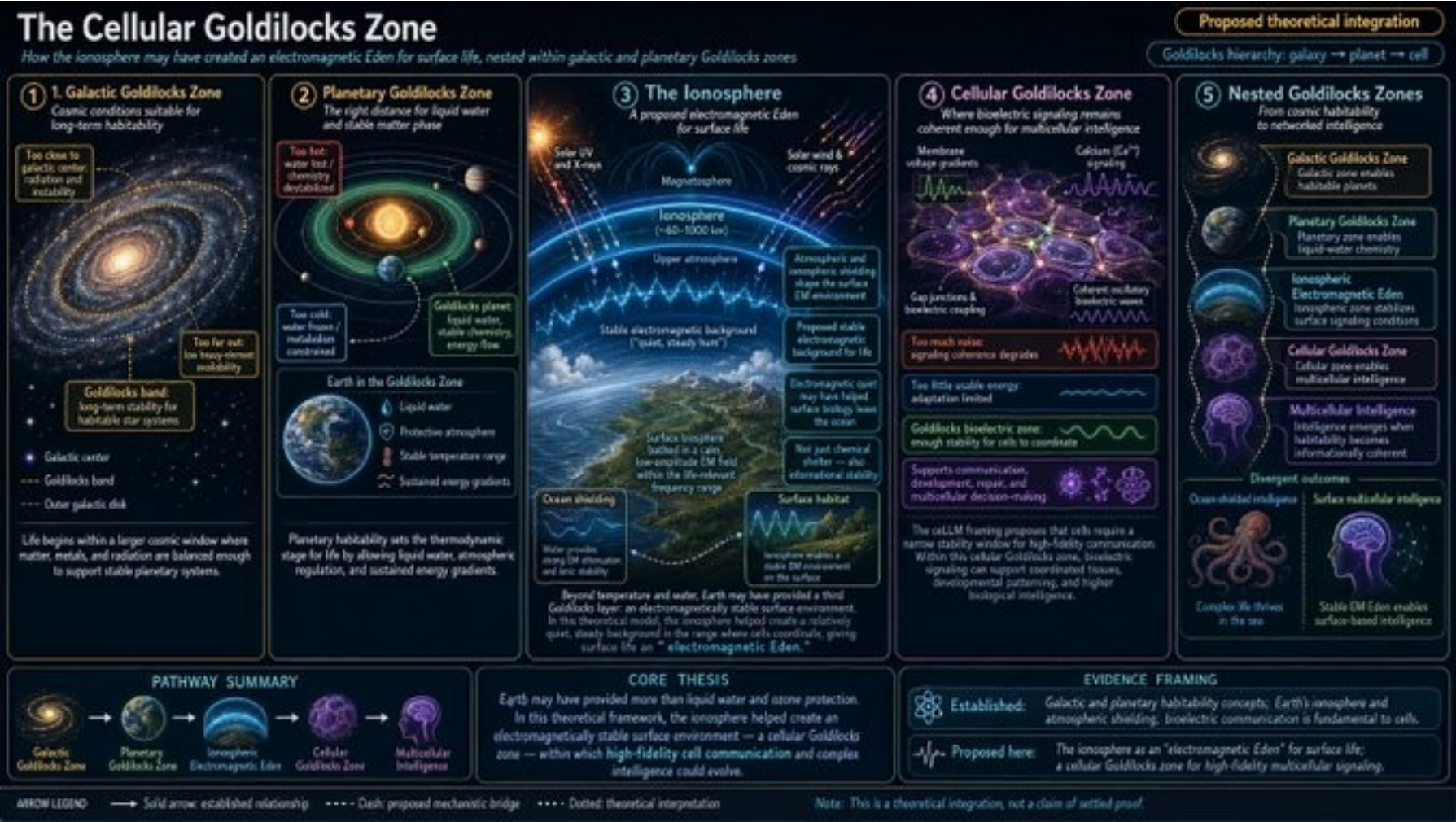
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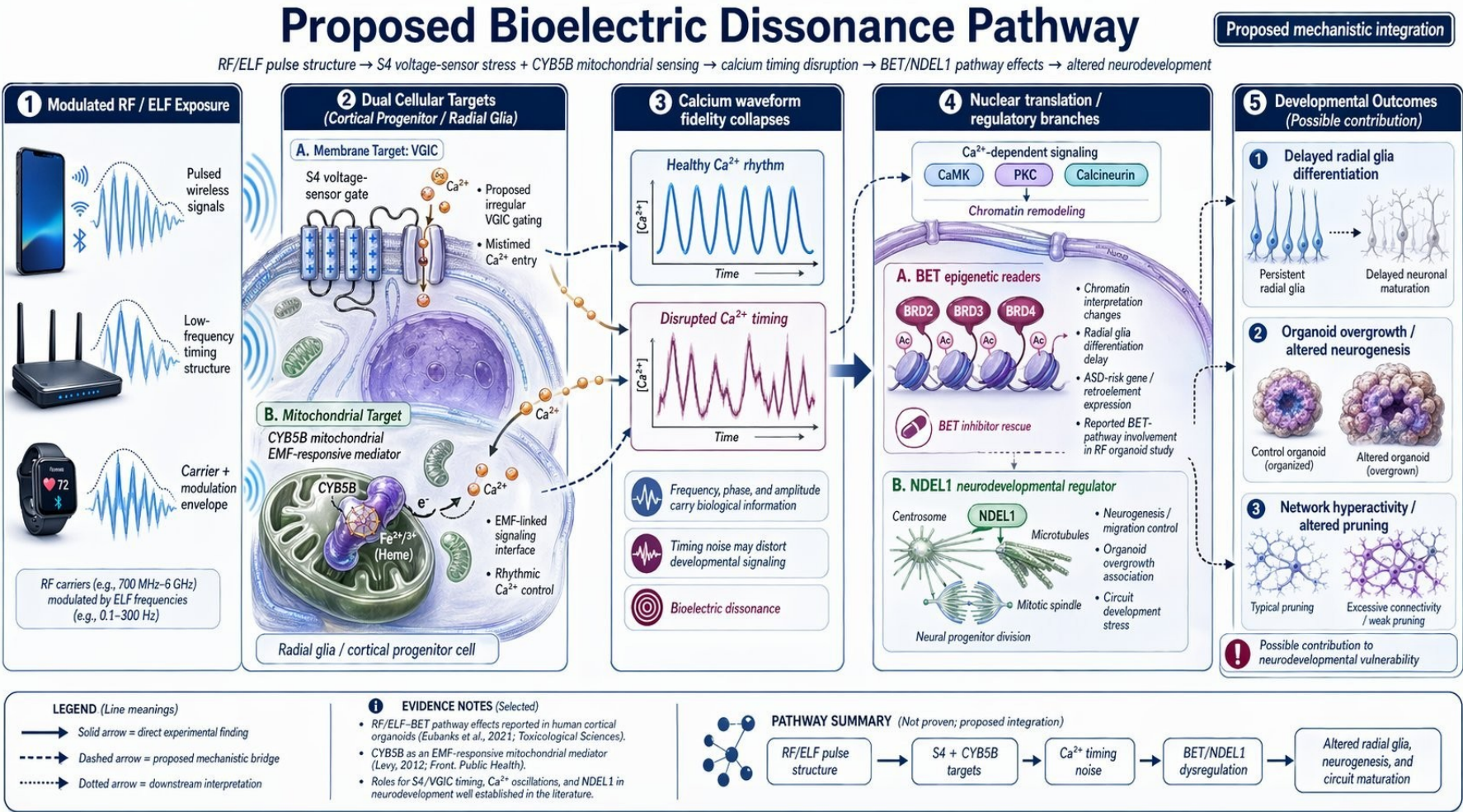
<https://www.rfsafe.com/life-runs-on-voltage/>

Attachment A • Pathway 1: The cellular Goldilocks zone



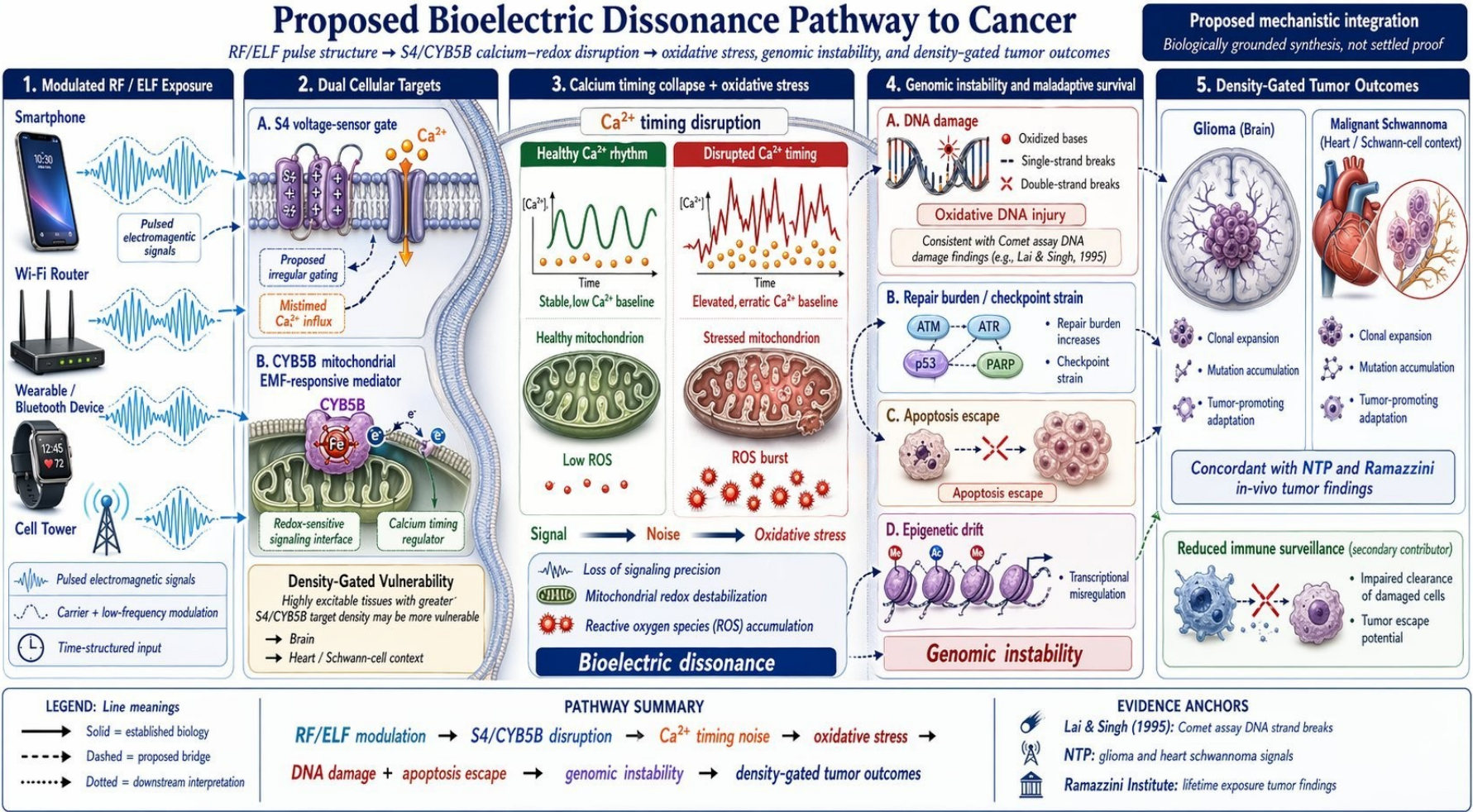
RF Safe’s conceptual map connects environmental conditions, cellular signaling, and the operating range required for coordinated development. The research task is to identify which physical inputs change cellular precision, reserve, and recovery. This is the organizing illustration for the proposed biological-fidelity program. [1,31]

Attachment A • Pathway 2: Developmental timing and differentiation



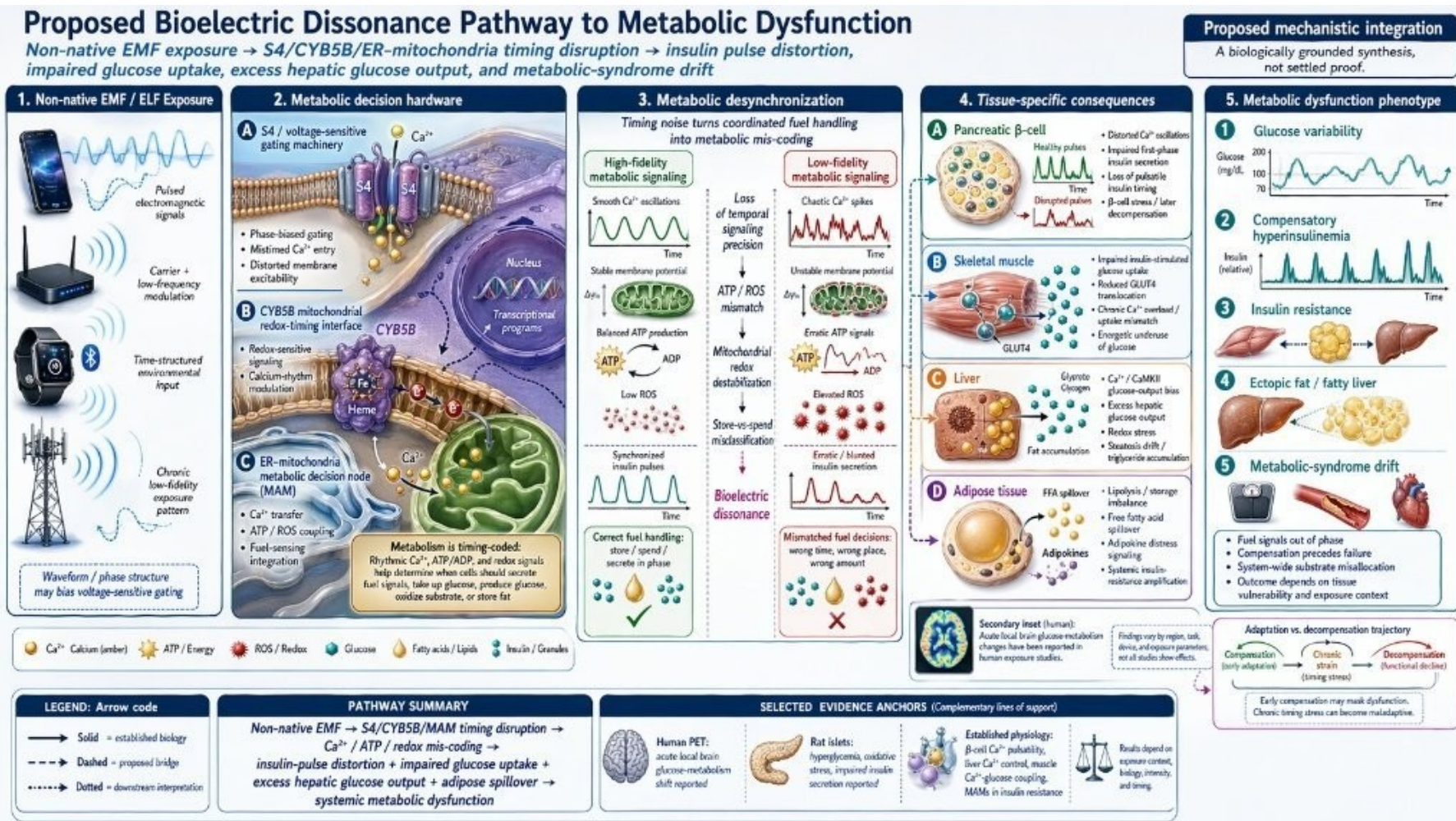
RF Safe’s developmental pathway links field-responsive interfaces with calcium timing, mitochondrial and chromatin responses, and differentiation. Citation key for this plate: the cortical-organoid study is Cakir et al. (2025) [9]; the CYB5B gene-switch study is Kim et al. (2026), with its published correction [13]. These citations supersede the author/year labels printed in the original image. NDEL1 identifies a parallel developmental-control target in the accompanying research program.

Attachment A • Pathway 3: Cancer, repair, and persistent regulatory error



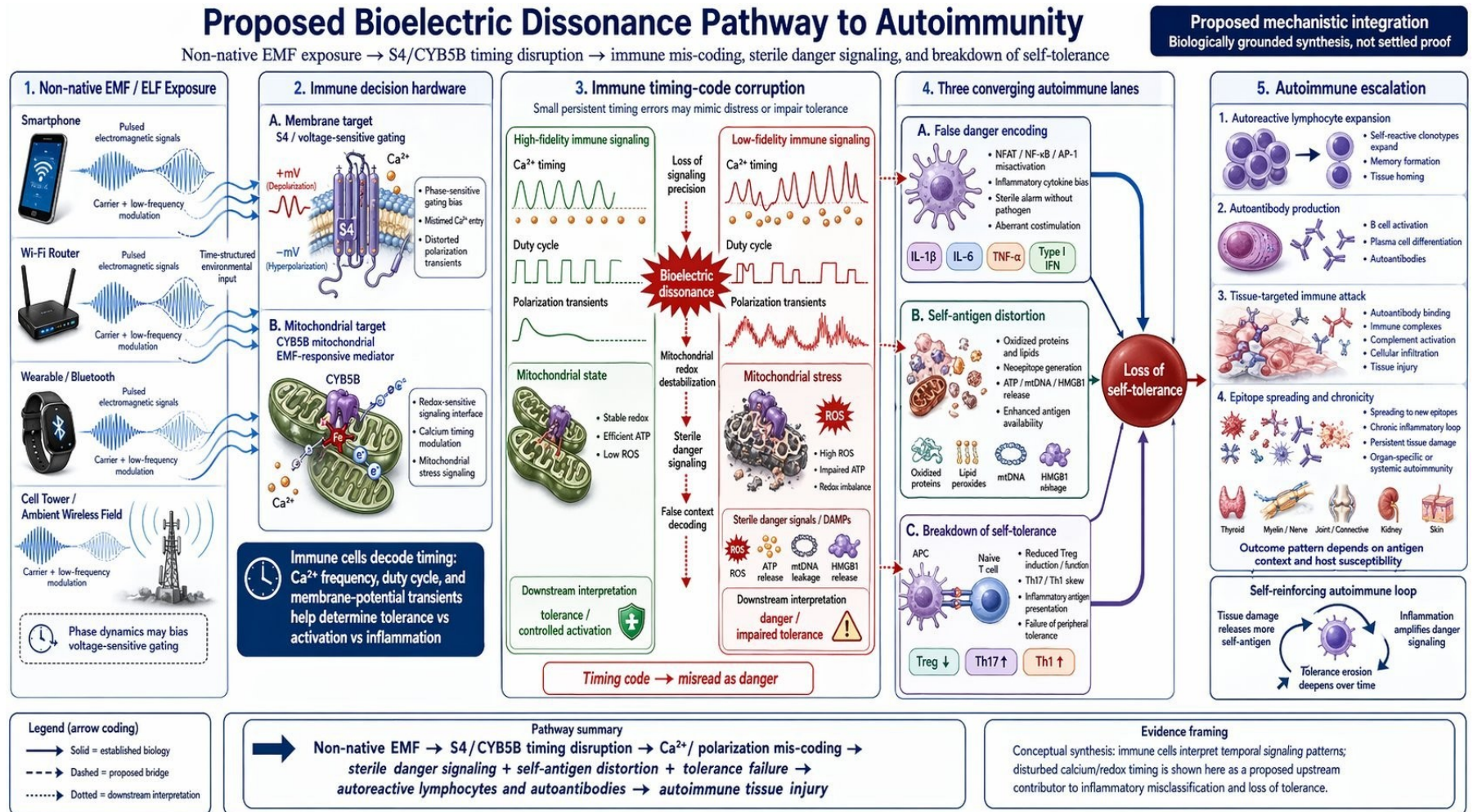
RF Safe’s proposed cancer pathway connects initiating interfaces, calcium and redox responses, repair, checkpoint control, apoptosis, and epigenetic regulation. NTP, Ramazzini, and the animal-cancer systematic review supply the tumor evidence; intervention studies must connect the proposed molecular steps to those outcomes. [1-4,19]

Attachment A • Pathway 4: Metabolic coordination and reserve



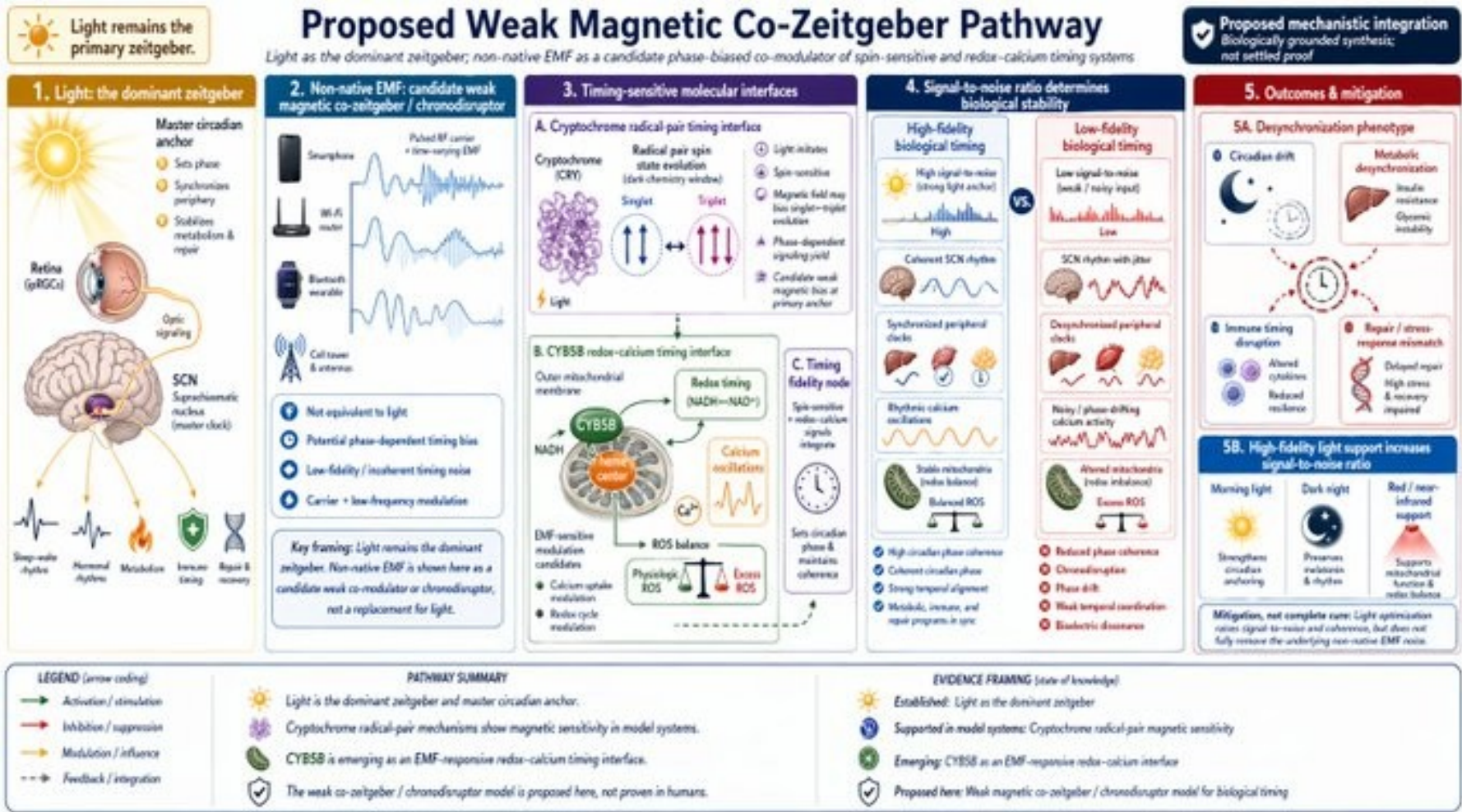
RF Safe's proposed metabolic pathway links calcium and mitochondrial responses with energy regulation across pancreas, liver, muscle, and adipose tissue. The program should measure organ-specific function, compensation, and recovery together. Yan et al.'s offspring findings provide a separate reason to include developmental trajectories in reproductive exposure studies. [1,14,20,21]

Attachment A • Pathway 5: Immune activation and tolerance



RF Safe's proposed immune pathway traces how altered calcium-redox coordination could affect danger signaling, antigen handling, tolerance, and resolution. The required experiments should distinguish an appropriately scaled response from persistent activation and test whether correcting the initiating disturbance restores normal function. [1,31]

Attachment A • Pathway 6: Environmental timing and the co-zeitgeber proposal



RF Safe's timing map places electromagnetic inputs alongside the dominant light-dark cycle and other biological timing cues. The research program should characterize field parameters, illumination, genotype, calcium dynamics, and recovery together. Cryptochrome experiments and genotype-dependent human sleep findings identify testable branches within this proposal. [1,13,16,17]

Attachment B

Public Comment of John Coates

RF Safe

Docket HHS-OASH-2026-0397

Presented with Katie Webb's position statement.

The following 39 pages reproduce John Coates's complete comment, including responses to Questions 2–18, supporting references, pathway figures, and the Clean Ether Act proposal.

Original pagination is retained within the attached comment.

Public Comment of John Coates

Controlled experiments document biological responses to radiofrequency radiation without appreciable heating. The National Toxicology Program and Ramazzini Institute reported the same rare malignant heart tumor in male rats, and a WHO-commissioned systematic review rated the animal evidence for increased glioma and heart schwannoma as high certainty. HHS must act on this record now: test the biological-fidelity framework, reduce unnecessary exposure, and replace assurances based on regulatory compliance with a current, accountable public-health assessment. ^{[1] [2] [3] [4]}

HHS-OASH-2026-0397 • October 21, 2026

Individual response to Questions 2–18 of the HHS Request for Information on Electromagnetic Fields, Radiofrequency Radiation, and Wireless Radiation Exposure. Question numbers and lettered subparts follow the notice. ^[5]

Question 2. Perspective represented

The evidence warrants action from the health agencies responsible for protecting families. I write as an individual, a father, a childhood cancer survivor, and the founder of RF Safe. My work includes public education, research documentation, a public SAR database, and exposure-reduction technology. These are my positions and the actions I ask HHS to take.

RF Safe has dedicated nearly 30 years to open-source design, with product designs developed for the public domain. Our work makes exposure-reduction knowledge and designs available for public use. I am the named inventor on U.S. Patent 11,700,058 B2, which combines optical wireless communication with germicidal light. This work demonstrates the multifunctional use of photons in optical networks. My recommendations draw on this engineering experience and call for public research, independent testing, and accessible wired and optical infrastructure. ^{[6] [7]}

This comment presents RF Safe's research framework: S4–Mito–Spin, density gating, genomic susceptibility, developmental timing, persistence and recovery, and the developmental, cancer, metabolic, and immune pathways. The evidence supports a health-agency program centered on biological coordination and real exposure conditions. Question 16 integrates the developmental, topology-gated photonic-redox, and embodied-cellular-transfer-function components of this framework. Appendix B presents the supplied illustrations, and Appendix C maps the source manuscripts to this submission. ^{[8] [9]}

Question 3. Personal experience

My family's experience is why I demand that HHS investigate preventable hazards before another generation passes through irreversible developmental windows.

(a) Sources; (b) timing and duration. My concern began with childhood exposure while my family lived near a radar installation on a military base. I was diagnosed with kidney cancer at age seven in 1977.

(c) Effects; (d) onset and exposure changes; (e) medical evaluation. My left kidney was removed at age eight. In 1995, my firstborn daughter, Angel Leigh, was born with anencephaly and did not survive. Her neural tube had failed to close during early development. These conditions were medically identified. These experiences have driven my insistence that developmental protection must be a federal priority.

I believe electromagnetic exposures contributed to these tragedies through interference with biological signaling and timing. That conviction has driven my lifelong commitment to understanding the mechanisms and reducing avoidable exposure. HHS must investigate the developmental processes that have to succeed before a child has any opportunity for treatment or recovery.

(f) Reduction; (g) documentation. My response has been decades of exposure-reduction work and public education directed toward preventing harm and protecting other families. The supporting scientific and agency records are cited throughout this comment. Farrell and colleagues' chick-embryo experiments reported increased abnormalities in four of five experimental campaigns using pulsed or sinusoidal low-frequency magnetic fields. That research helped shape my focus on susceptibility and developmental timing. HHS must make prevention a priority before developmental injury occurs. ^[10]

Question 4. Research observations and surveillance

The cancer findings demand investigation of the biological processes connecting exposure, cellular responses, and downstream outcomes. I ask HHS to bring the published findings into a coordinated research and surveillance program.

(h) Effects. Relevant observations include tumors in animal bioassays, altered developmental and reproductive endpoints, sleep EEG and cerebral blood-flow responses, and cellular DNA-repair and redox changes. These span pathology, physiology, and candidate mechanisms; their relationships require investigation. ^{[1] [11] [4] [12]}

These observations connect through functional tasks: developmental cells must divide and differentiate on time; repair systems must recognize and correct errors; metabolic systems must match fuel supply to demand; immune systems must distinguish a challenge and resolve their response. Surveillance should measure deterioration in those tasks and the capacity to recover, alongside diagnoses. A normal population average or a normal systemic marker must not end investigation of a reproducible tissue-specific response.

(i) Onset; (j) populations. Record latency, gestational stage, exposure changes, recovery intervals, age, sex, medical conditions, medications, and consented susceptibility variables. Acute responses, developmental disruptions, and long-latency tumors require different observation schedules.

(k) Exposure patterns; (l) methods. Distinguish near-body transmitters, infrastructure, occupational sources, and ELF fields. Use calibrated dosimetry, blinded outcome assessment, prespecified endpoints, and appropriate diagnostic methods. Self-reported screen time cannot substitute for exposure characterization.

(m) Recommendations. Establish prospective birth and child cohorts, occupational cohorts, and an accessible reporting system with clinical follow-up and appropriate comparison groups. Preserve exposure histories as networks change. Fund independent replication and the early-response experiments in Question 16.

Question 5. Standards and regulatory frameworks

The NTP, Ramazzini, and subsequent review evidence require a fresh examination of what current limits actually protect. A de facto standard is an administrative framework, not a biological law.

Review FCC rules, ICNIRP 2020, IEEE C95.1, IEC/IEEE measurement procedures, European conformity standards, and HHS/FDA's electronic-product radiation-control duties. Separate the adequacy of an exposure limit from the accuracy of testing against it. Preserve the distinction between the U.S. public whole-body limit of 0.08 W/kg and localized SAR of 1.6 W/kg averaged over one gram; a 10-gram value is another measure. ^{[13] [14] [15] [16] [17] [18]}

(n) Scientific, technical, and regulatory changes. Publish a wireless-RF program under 21 USC 360ii and a reasoned performance-standards determination under 360kk. Commission a current assessment of cancer, development, reproduction, neurological function, and susceptibility. Require body-contact testing for foreseeable contact use, independent retail audits, and disclosure of the conditions behind every SAR value. Publish reviewer funding, prior guideline roles, study-level judgments, and conflict-management decisions. ^[18]

The 2021 D.C. Circuit decision required the FCC to address important deficiencies in its explanation concerning non-cancer effects and other issues. HHS must supply the substantive health record needed for an adequate response to that remand. ^[19]

(o) Exposure minimization and research gaps. Use the quantitative risk assessments of Uche and Naidenko and Melnick and Moskowitz to develop health-based limits. Publish the models, exposure-duration assumptions, and uncertainty factors. Prioritize modern waveforms, chronic exposure, prenatal development, recovery, and independent replication. Conduct measured fiber, Ethernet, and optical-network pilots in classrooms, childcare, maternity care, and HHS facilities. ^{[20] [21]}

Quantitative risk assessment demands lower exposure limits

Melnick and Moskowitz (2026) applied EPA benchmark-dose tools and linear extrapolation to NTP and Ramazzini animal tumor data. For one additional cancer per 100,000 exposed people, the public whole-body limit of 0.08 W/kg was **15–900 times** above their modeled levels, depending on daily exposure duration. Applying conventional uncertainty factors to animal male-fertility evidence yielded 3.3–10 mW/kg, which is **8–24 times** below the existing 80 mW/kg limit. ^[21]

EPA’s human-health guidance uses animal data when human dose–response information is insufficient, models the dose–response relationship, and extrapolates toward lower exposures. Its cancer framework generally uses linear extrapolation when mode-of-action information is insufficient; non-cancer assessments use uncertainty factors for cross-species differences, human variability, and data gaps. These are established public-health risk-assessment procedures. ^[21]

HHS and EPA must apply this discipline to RF regulation. I call for a federal quantitative risk assessment that uses the Melnick–Moskowitz analysis as a starting point, publishes the inputs and assumptions, and derives exposure limits that protect against cancer and reproductive harm. Regulatory compliance cannot substitute for a health-protective dose–response assessment. Reduce avoidable exposure while that assessment proceeds.

(p) Exposure classification. Require a multidimensional exposure record: carrier frequency and bandwidth; power density; SAR with spatial distribution, averaging mass and time; electric- and magnetic-field strength; modulation; pulse width and repetition; peaks and duty cycle; beamforming and traffic; simultaneous frequencies; duration; distance and body contact; life stage; cumulative time history; and recovery intervals. Retain these dimensions rather than collapsing them into an unvalidated risk score. An RF modulation envelope and a separately measured ELF field must remain distinguishable.

Question 6. Evidence bearing on current limits

The strongest animal evidence already supplies a reason for action: two independent lifetime programs reported malignant heart schwannomas in male rats. The quantitative assessment in Question 5 turns this tumor evidence into a specific demand for lower public exposure limits. HHS must commission the federal risk assessment and begin exposure reduction while completing it.

NTP reported clear evidence of carcinogenic activity for these tumors and some evidence involving malignant gliomas. Its male GSM glioma counts were 0, 3, 3, and 2 out of 90 across sham, 1.5, 3, and 6 W/kg whole-body groups. Ramazzini studied 2,448 rats with prenatal-to-lifetime exposure; its highest group, approximately 0.1 W/kg whole-body, had 3 heart schwannomas among 207 males versus none among 412 controls. That dose was far below NTP’s lowest. The U.S. public whole-body limit is 0.08 W/kg. ^{[1] [2] [13]}

Brooks and colleagues examined gliomas and cardiac schwannomas from the Ramazzini program using a targeted cancer-gene panel. The rat gliomas shared some alterations with human IDH1-wildtype gliomas, and the schwannomas also contained alterations in queried cancer genes. HHS should extend this translational work with lineage-specific molecular profiling and early non-tumor sampling. The relevant comparison is between defined tumors and pathways, with species-specific findings preserved. ^[22]

The WHO-commissioned review rated the evidence for increased glioma and heart schwannoma in male rats as high certainty. HHS must put this high-certainty animal evidence at the center of its reassessment of human protection. ^[3]

NTP reports whole-body absorption; the FCC's 1.6 W/kg device limit specifies localized absorption averaged over one gram. HHS must preserve these dose definitions when comparing exposures. NTP reports limiting temperature increases to less than 1°C and calls for further investigation of the role of thermal changes. Fund that investigation, temperature-controlled waveform experiments, and lower-dose risk assessment now. ^[23]

Question 7. Available measurement methods

A single averaged SAR value cannot characterize the timing-dependent biological activity that this research asks HHS to investigate. Measuring absorbed energy is necessary; measuring signal structure and biological response is also necessary.

Available methods include laboratory SAR measurements, anatomical computational dosimetry, calibrated electric- and magnetic-field probes, frequency-selective spectrum analysis, personal exposimeters, and time-resolved signal acquisition. IEC/IEEE procedures support device and base-station assessment. Properly configured instruments can capture timing; a scalar compliance result cannot preserve that information by itself. ^[16]

Use complementary methods for individual and population studies: wearable measurements with body-shadowing corrections, fixed monitors, device/network activity records, occupancy diaries, and validated propagation models. Report calibration, bandwidth, sampling rate, uncertainty, near-field limitations, and what the instrument cannot detect. Pair physical measurements with prespecified physiological endpoints.

The Dissonance Protocol adds task-based biological measurements: calcium-event timing and interval variability, phase stability, spatial propagation, membrane potential, mitochondrial reserve, compartment-specific redox signals, transcriptional response, and recovery after a standardized challenge. Record calcium entropy alongside function and coordination. Evaluate whether exposure changes the reliability of the biological task, including when average signal magnitude remains similar. These measurements belong beside dosimetry.

Question 8. Improvements for real-world exposure

The biological evidence makes realistic exposure assessment urgent, and the FCC's own body-proximity records show a concrete testing issue.

The released 2 mm laboratory spreadsheet includes a value of 5.2 W/kg, more than three times the 1.6 W/kg localized limit. The FCC recorded this result while treating the test separation as outside its prescribed guidance. That gap between testing procedure and foreseeable body proximity demands correction. Appendix A reproduces the spreadsheet and the complete agency response. HHS must require testing that represents foreseeable use against the body. ^{[24] [25]}

Require zero-separation testing where contact use is foreseeable, simultaneous-transmitter assessment, realistic traffic modes, and independently purchased retail samples. Publish both typical-use distributions and reasonably foreseeable high-exposure conditions. European changes concerning EN 50566 support this direction; the scheduled withdrawal of the older conformity-standard reference is March 7, 2028. ^[17]

Record peaks, bursts, modulation, and recovery intervals alongside averages. GSM can produce approximately 217 bursts per second in a one-timeslot configuration; common Wi-Fi beacon timing is about ten per second; DECT uses a 10 ms frame. Measure RF modulation envelopes and separate ELF fields as distinct physical exposures, and test their coupling to tissue. ^[26]

Question 9. Public disclosure

People cannot make informed exposure choices when the conditions needed to interpret a compliance number are missing.

Consumers should receive SAR or other applicable exposure values beside test distance, averaging mass and time, body position, active radios, operating mode, and relevant pulse characteristics. Include accessible instructions for disabling unnecessary transmitters and using wired connections. Distinguish tested maxima from typical exposure.

Residents and local governments need installation inventories, bands, antenna patterns, operating-power information, cumulative assessment, and independent measurement results. Workers need task-specific conditions and maintenance controls. Schools and healthcare facilities need occupied-room measurements, child- and patient-relevant assessments, implant/coexistence information, and practical lower-exposure alternatives. Publish a readable summary and machine-readable technical record, with uncertainties visible.

Question 10. Surveillance and data sources

Surveillance must detect early changes in biological regulation alongside later disease. Add detailed exposure histories to the outcome records collected by existing systems.

(a) Registries. Link cancer, birth-defect, and other relevant registries to consented or appropriately governed exposure studies; their strength is population coverage, their limitation is exposure resolution. **(b)**

Occupational systems. Add calibrated task histories to worker cohorts and incident records. **(c)**

Environmental monitoring. Preserve repeated indoor and outdoor measurements, not just installation-time compliance estimates. ^[27]

(d) Longitudinal studies. Follow pregnancy, childhood, and occupational cohorts across network changes.

(e) Biomonitoring. Validate calcium, redox, repair, and recovery measures for prospective research and clinical application.

(f) Adverse events. Establish a structured RF-experience reporting route with clinical follow-up. Use FDA MAUDE to identify device-related safety signals and connect reports with exposure investigation, verified outcomes, and population denominators. **(g) Clusters.** Provide an investigation protocol with verified diagnoses, exposure reconstruction, denominators, and competing explanations. **(h) Other sources.** Use electronic health records, device logs, sleep studies, and community measurements with consent, privacy protection, and calibration. ^[28]

Analyze registry trends by age, histology, tumor site, and molecular subtype, using stable definitions and documented changes in ascertainment. Examine glioma subtypes, meningioma, thyroid, and salivary-gland outcomes in exposure-linked research rather than treating an overall cancer trend as a sufficient answer. The surveillance program should make both exposure history and biologically distinct outcomes more precise.

The benefit is earlier detection and better testable questions. The challenges are selection bias, exposure misclassification, latency, confounding, missing denominators, and privacy. Design the program to address these rather than use them as reasons not to collect the data.

Question 11. Susceptible populations

The same external field need not produce the same absorbed dose or response in every receiver. The cancer and developmental evidence demand explicit susceptibility research.

(a) Children. Study tissue-specific anatomy, developmental timing, near-body use, and lifetime exposure. Dosimetry research identifies age-dependent differences that vary by tissue and configuration. **(b)**

Pregnancy. Study maternal and fetal dose, distinct gestational windows, placental function, and later

offspring outcomes. Cortical-organoid findings provide a model for examining altered differentiation; animal-development reviews provide additional endpoints. A developing child gets only one chance at each developmental window. ^{[29] [30] [11]}

(c) Older adults. Include thermoregulation, medication, sleep, and reduced physiological reserve. **(d) Implants.** Evaluate interference and altered field distribution with device-specific testing; general tissue-exposure compliance does not answer compatibility questions. **(e) Workers.** Require monitoring, training, controlled access, maintenance protection, and exposure histories, including pregnancy and implant needs. ^[31]

(f) Preexisting conditions. Include neurological, cardiovascular, metabolic, immune, and thermoregulatory conditions in justified study designs. Provide respectful assessment and feasible accommodations for people reporting exposure-related symptoms.

(g) Other susceptibility. Investigate genotype and limited control over one's environment. Sousouri and colleagues reported a CACNA1C-genotype-dependent sleep-spindle response after 3.6 GHz exposure in 34 volunteers. Independently replicate this response and determine its biological and clinical significance. Healthy-adult averages must not erase a reproducible subgroup response. ^[32]

The controlled CACNA1C study compared rs7304986 T/C and T/T participants. This makes inherited regulatory variation a concrete study variable. Investigate expression, splicing, channel function, and linked variants, with adequately powered genotype-by-exposure tests. Preserve the identity of each marker and study design when connecting this work to broader CACNA1C biology. ^{[32] [33]}

Palone and colleagues exposed three mouse strains to 27.5 GHz fields for 100 days at 6.67 or 20 W/m². Car-S showed the strongest reproductive changes, CD1 was largely resistant, and C57BL/6J had an intermediate pattern. Study strain-specific absorption and physiological state alongside genetic background. These findings give HHS an animal reproductive example of receiver dependence in addition to the human sleep-timing example. ^[34]

Cell state must also be measured. Durdik's cord-blood work identifies differentiation and redox status as relevant comparisons. The full S4-Mito-Spin program should quantify channel/transducer expression, mitochondrial and redox machinery, and recovery before exposure, then test whether these variables predict response. Developmental deadlines and limited control over exposure deserve explicit protection in policy. ^[35]

Question 12. Evidence below current limits

Low-level biological findings give HHS a direct reason to test whether current protection criteria adequately address signaling, repair, and development.

Huber and colleagues reported modulation-dependent cerebral blood-flow and EEG responses, including comparisons at the same reported 10-gram SAR of 1 W/kg. Belyaev and colleagues reported inhibition of DNA-repair foci in human lymphocytes at approximately 0.037–0.040 W/kg in their experimental system; Markova and colleagues reported stronger effects in stem cells than differentiated cells. Fund replication and adverse-outcome follow-up. Report each experiment's actual dose metric and compare it with the corresponding public-exposure criterion. ^{[4] [12]}

Across the broader RF literature, Henry Lai's June 2026 compilation reports effects in 390 of 438 oxidative studies, 396 of 550 genetic studies, 192 of 228 gene-expression studies, 396 of 507 neurological studies, and 354 of 415 reproductive/developmental studies. Commission a study-level assessment of these findings, including exposure, endpoint, methodological quality, and replication. Their breadth demands an assessment covering all of these biological systems. ^[36]

HHS must identify which experiments satisfy the relevant public-limit comparison, which responses are reproducible, and which are adverse. The benchmark-dose proposals cited in Question 5 show why observed

animal pathology must inform protective extrapolation. Translate these findings into a protective risk assessment and practical exposure reduction.

Question 13. Emerging technologies and cumulative exposure

Evaluate cumulative RF exposure through measured time histories that include repeated exposure, recovery intervals, and co-exposures. Apply this assessment to the entire connected environment.

(a) 5G. Measure sub-6 GHz and millimeter-wave configurations separately, including beamforming, traffic, handset uplinks, and other active radios. **(b) 6G.** Require full waveform and exposure specifications and health-relevant testing as systems develop under the IMT-2030 framework. ^[37]

(c) Wi-Fi. Measure clients and access points, beacons and data traffic, occupied classrooms, and overnight conditions. **(d) Satellite.** Separate downlinks, user terminals, and direct-to-device uplinks. **(e) IoT.** Include gateways, retries, reporting schedules, and aggregate node activity.

(f) Wearables. Prioritize contact dose, location, continuous wear, and child-focused products. **(g) Smart appliances.** Measure always-on connectivity and distinguish communications RF from power-frequency fields; provide usable radio-off options. **(h) Smart cities.** Inventory cumulative communications and sensing sources and preserve exposure histories.

(i) Autonomous vehicles. Distinguish radar, communications, and occupant devices, with measurements inside and outside vehicles. **(j) Wireless medical devices.** Assess contact exposure and coexistence while preserving essential therapeutic and monitoring functions. **(k) Smart meters.** Document transmission peaks, averages, retries, relay duties, meter banks, and occupied-room proximity; evaluate accessible lower-exposure service options.

Compare complete network designs at equivalent service levels. Denser infrastructure can change both ambient fields and handset power. Measure the net exposure change and recovery opportunities before claiming that an installation either increases or reduces total exposure.

My 800 MHz research request is especially relevant to expanding cellular and direct-to-device systems. Rao's laboratory finding supplies a frequency-specific neuronal-calcium result to replicate. Obtain actual network specifications and compare waveform, internal dose, duration, and geometry with the laboratory exposure. The public-health deliverable is a measured deployment comparison and an independently replicated biological assessment. ^[38]

Question 14. Environmental effects

Biological sensitivity is a property of the receiving organism, and a human heating-based limit cannot by itself establish protection for every ecological function.

Engels and colleagues demonstrated disrupted magnetic-compass orientation in migratory birds under characterized anthropogenic electromagnetic noise in the 50 kHz–5 MHz range. This finding demands direct assessment of navigation and other sensitive biological functions. The Levitt reviews assemble a broader flora/fauna record. ^{[39] [40]}

Coordinate with EPA, USDA, DOI, and state agencies on replicated studies of pollinators, migratory species, plants, and livestock. Measure exposure, background variability, reproduction, development, behavior, and fitness. Assess realistic mixtures and competing environmental stressors. Ecological assessment should test whether electromagnetic exposure adds vulnerability to systems already under strain.

Question 15. Infrastructure

The biological evidence justifies reducing unnecessary long-duration exposure where people live, learn, work, and receive care.

(a) Proximity. Require site-specific assessment near homes, schools, childcare, healthcare, and workplaces, including occupied rooms and developmental vulnerability. I call for a prospective 1,500-foot setback for new macro facilities near sensitive uses as a precautionary policy. Combine that safeguard with assessment of antenna patterns, power, terrain, buildings, and cumulative exposure.

(b) Neighborhood exposure. Publish cumulative maps with uncertainty and repeated measurements. **(c) Antenna density.** Evaluate power, direction, traffic, and handset responses rather than equating antenna count with absorbed dose. **(d) Small cells.** Assess windows, sidewalks, occupied rooms, maintenance positions, and aggregate deployments. **(e) Livestock.** Use species-relevant dosimetry and controlled studies of reproduction, development, behavior, and health, accounting for husbandry, infection, nutrition, heat, chemicals, and stray currents.

Require three-dimensional assessment that includes upper-floor rooms, windows, balconies, neighboring roofs, and worker positions. Combine infrastructure downlink with foreseeable device uplink and simultaneous sources. Verify predictions after installation under documented traffic conditions. Preserve meaningful local notice, participation, and review. HHS should ask the FCC to defer further expansion of deployment preemption or deemed approvals until the health record addresses the remanded issues and the mechanisms presented here. ^[19]

Section 704 restricts state and local siting decisions based on RF environmental effects when facilities comply with FCC regulations. That makes the quality of the federal health standard especially consequential. I call for repeal of that restriction, identified in 47 USC 332(c)(7)(B)(iv), so credible current health evidence can inform local protection. ^[41]

Question 16. Priority research: the full S4-Mito-Spin framework

The NTP and Ramazzini findings, the WHO-commissioned animal review, and the signaling experiments described below demand a coordinated investigation of upstream biological disruption. HHS should begin with the accumulated evidence and fund the connections that evidence identifies. My research request brings together nine components: exposure structure, biological transducers, ion channels, calcium timing, mitochondrial and redox integration, spin chemistry, density gating, genomic susceptibility, and functional outcomes. These nine connected components must be investigated together. ^[8]

The meta-disease state: loss of coordination before diagnosis

I use **meta-disease state** for my model of an upstream loss of biological coordination that changes the probability of multiple downstream outcomes. The organizing sequence is structured electromagnetic perturbation; altered channel activity or field-sensitive chemistry; calcium-mitochondrial mismatch; redox and energetic strain; compensatory gene and tissue responses; and reduced regulatory precision. The defining feature is a smaller margin for maintaining normal function under additional stress.

Small disturbances followed by sufficient recovery can be absorbed. Repeated perturbations, poor timing, combined stressors, or a vulnerable receiver can keep compensation active. In this model, endocrine feedback becomes less stable, immune responses less proportional, metabolism less flexible, and tissue repair less exact. The outcome depends on which control system is closest to its limit. A developmental defect, altered metabolic response, immune dysfunction, and a persistent repair error are different downstream expressions to investigate.

Biological dissonance is the process. Low-fidelity biology is the state. Increased disease susceptibility is the downstream consequence. Sleep disruption and air pollution illustrate why public health must investigate upstream burdens across multiple outcomes. HHS should apply that breadth to the electromagnetic environment. An exposure's contribution as a cofactor deserves investigation before every affected person can be assigned the same diagnosis.

The framework makes six recurring features central: waveform sensitivity, frequency dependence, receiver dependence, tissue specificity, adaptive reserve, and nonlinearity. Each requires measurements. A tissue response with a normal blood marker, a genotype-dependent response hidden by the group average, or recovery after one exposure followed by impaired recovery after repeated exposure are distinct results with practical implications. The research program must resolve those patterns.

Calcium timing is biological information

Calcium carries instructions through location, duration, amplitude, and timing. Dolmetsch, Xu, and Lewis experimentally showed that calcium oscillations regulate the efficiency and specificity of gene expression. De Koninck and Schulman showed that CaMKII decodes calcium-spike frequency, with its response also influenced by spike amplitude, duration, and the kinase's previous state. These experiments establish a physical basis for asking whether a mistimed signal changes what a cell does. ^{[42] [43]}

Spatial organization matters as well. Dolmetsch and colleagues' channel-to-nucleus experiments identified a role for channel-associated calmodulin in transmitting an L-type calcium-channel signal through the MAP-kinase pathway. Chawla and colleagues connected neuronal activity and calcium signaling to the location of HDAC4 and HDAC5, proteins involved in gene regulation. A bulk calcium concentration cannot capture all the information in these local signaling arrangements. ^{[44] [45]}

My definition of fidelity is therefore task-specific: how reliably signaling accomplishes a biological function. Measure event timing, phase relationships, propagation between cells, compartment-specific activity, decoding, and return to baseline. Relate those measurements to contraction, differentiation, repair, metabolic control, or immune resolution. A useful fidelity endpoint measures functional precision and recovery; a changed trace alone does not define the task.

Waveform dependence and the historical noise-suppression experiments

The Catholic University of America embryo program belongs near the foundation of this argument. Farrell and colleagues examined more than 2,500 chick embryos across five campaigns and reported increased abnormalities in four campaigns. Their developmental-enzyme companion described stage-dependent changes in ornithine decarboxylase, including opposite directions during gastrulation and neurulation under a 4 μ T, 60 Hz magnetic field. Developmental stage was part of the biological response. ^{[10] [46]}

Litovitz and colleagues also supplied an intervention in temporal structure: adding a spatially coherent but temporally random magnetic noise field, at an amplitude comparable to the applied ELF signal, reduced the excess embryo abnormality rate essentially to the control level. Farrell and colleagues' 1998 companion found that adding an incoherent field at equal RMS strength suppressed the 60 Hz-associated ODC changes during gastrulation and neurulation. These experiments changed the reported response by changing the temporal organization of the exposure. ^[47]

That intervention is crucial to my reasoning. Added external randomness and degraded internal signaling are different things. Randomness can interfere with a receiver's response to a structured external input. The framework must explain the suppression as well as the original response. HHS should recover this experimental line, reproduce its conditions, and identify the molecular and developmental steps responsible.

There are RF observations to explain as well. Blackman and colleagues reported enhanced calcium efflux from chick brain tissue exposed to 147 MHz RF with 16 Hz amplitude modulation at 0.75 mW/cm², within

frequency and intensity windows. Lai and colleagues compared 2.45 GHz pulsed and continuous exposures in rats at a reported average whole-body SAR of 0.6 W/kg for 45 minutes. Pulsed exposure used 2 μ s pulses at 500 per second and reduced hippocampal and frontal-cortex choline uptake; continuous exposure reduced frontal-cortex uptake without a significant hippocampal change. Opioid antagonists blocked the pulsed hippocampal response. Those findings identify waveform, tissue, and pathway variables for direct comparison. ^{[48] [49]}

Human evidence adds another level. Huber's modulation comparisons and the sleep-EEG studies connect characterized exposure to physiological timing and regional response. The carrier, modulation envelope, pulse structure, duty cycle, spatial absorption, and recovery interval belong in the exposure record together. Separately applied ELF fields require their own characterization. ^{[4] [50] [32]}

S4 and the ion-channel branch

The S4 component identifies the charged voltage-sensing machinery of voltage-gated ion channels as a specific initiating target to test. In normal channel physiology, voltage-sensor movement couples changes in membrane electric potential to channel opening. Panagopoulos and colleagues' 2021 and 2025 ion-forced-oscillation models propose that time-varying external fields act through mobile ions near membranes to disturb voltage-gated-channel operation. Their emphasis on polarization, coherence, and slower waveform structure gives this branch concrete physical predictions. ^{[51] [52]}

Rao and colleagues provide a direct RF/calcium anchor. In stem-cell-derived neurons, an 800 MHz, 0.5 W/kg exposure for 60 minutes increased spontaneous calcium spiking from roughly five events in responding control cells to 15.7 ± 0.8 . The authors reported frequency dependence and no SAR dependence over the tested 0.5–5 W/kg range, and implicated N-type channels, phospholipase C, and microfilaments. This directs research toward the receiving machinery and its temporal output. ^[38]

Other experiments supply manipulable channel pathways under ELF conditions. Grassi and colleagues reported changes in calcium-channel expression and current after 50 Hz exposure. Piacentini and colleagues found enhanced neuronal differentiation and Cav1-channel activity in neural progenitors exposed to 50 Hz, 1 mT fields; nifedipine abolished the exposure-associated increase in neuronal differentiation. The mechanistic value is the intervention: changing channel function changed the response. ^[53]

The full channel branch includes sodium and potassium systems as well as calcium. Firing and patch-clamp studies under low-frequency fields include sodium- and potassium-current measurements. HHS should compare channel families, gating currents, expression, and local calcium coupling. Direct S4 tests should alter voltage-sensor charge or structure while preserving suitable channel function, then determine whether field sensitivity changes. That experiment identifies the initiating interface more precisely than a downstream calcium measurement. ^[54]

CYB5B: an identifiable field-to-signal pathway

Kim and colleagues' 2026 Cell study gives the transducer program a molecular starting point. A genome-wide CRISPR-Cas9 screen identified cytochrome b5 type B, CYB5B, as essential to an engineered electromagnetic-field-inducible gene switch. Activation depended on rhythmic calcium dynamics. The work used low-frequency electromagnetic stimulation to control gene expression in experimental systems. This is proof of principle that an external field can engage a molecularly identifiable, calcium-pattern-dependent route to transcription. ^[55]

HHS should map four roles explicitly: the structure detecting the field, the necessary molecular relay, the calcium or biochemical decoder, and the resulting biological function. CYB5B's mitochondrial association makes its relationship to calcium–redox coordination a priority. Replicate the gene-switch result, incorporate

the published correction, and examine endogenous expression and function across cell types. Then compare its operating conditions with characterized telecommunications waveforms.

S4/channel and CYB5B-associated routes are connected branches of the framework. Either can supply an experimental entry point. HHS should determine where they interact, where another interface operates, and which intervention interrupts the response. The objective is a map of biological receivers with measured operating conditions.

Mitochondria: amplification, compensation, and reserve

The Mito arm asks how an initiating disturbance becomes a sustained burden. Mitochondria integrate calcium handling with energy demand and redox signaling. In the framework, a mismatch between calcium timing and energetic demand can change mitochondrial membrane potential, respiratory reserve, ATP availability, and the timing or location of reactive species. Repeated compensation can consume the reserve needed for a later challenge. HHS should measure this sequence over time, including after exposure ends. [56] [57]

Durdik and colleagues' human cord-blood study supplies a cell-state anchor: the acute ROS response to UMTS exposure differed across stem/progenitor and more differentiated populations, while the study did not find the measured DNA damage, preleukemic fusion genes, or apoptosis. This makes recovery and propagation observable parts of the mechanism. Measure baseline redox state separately from the exposure-associated increment, then ask which cells restore control and which retain a functional burden. [35]

Test mitochondrial involvement through simultaneous calcium and bioenergetic measurements, targeted pathway manipulation, and functional rescue. Include endoplasmic-reticulum stress and the unfolded-protein response in the recovery assessment, with IRE1-related signaling among the prespecified experimental targets. An antioxidant effect alone is too broad to locate the initiating event; restoring a defined pathway while maintaining controlled exposure provides a stronger causal test.

Xie and colleagues exposed mouse bone-marrow stromal cells to continuous-wave 900 MHz RF at 120 $\mu\text{W}/\text{cm}^2$ for four hours daily over five days. They reported increased ROS and mitochondrial unfolded-protein-response markers after exposure, with recovery toward sham levels by 24 hours. JNK2 knockdown prevented the reported increase in the response proteins. This experiment supplies exposure, time course, molecular intervention, and recovery in one system. HHS should extend that design to repeated challenges and reduced-reserve states. [58]

Spin chemistry: experimentally controlled reaction pathways

The Spin arm concerns field-sensitive reaction probabilities in molecular chemistry. Sherrard and colleagues reported cryptochrome-dependent ROS accumulation and related cellular responses under a 10 Hz pulsed magnetic exposure. Usselman and colleagues exposed rat pulmonary smooth-muscle cells to a 7 MHz, 10 μT RMS RF magnetic field with a 45 μT static field and observed a redistribution between superoxide and hydrogen-peroxide signals, together with growth changes. These results make the identity, compartment, and timing of reactive species important measurements. [59] [60]

The 2026 molecular spin studies go further in experimentally controlling the relevant chemistry. Meng and colleagues used RF and static magnetic fields to control spin-dependent optical responses in flavoproteins, including cryptochrome and iLOV. Burd and colleagues demonstrated magnetic-resonance control of spin-correlated radical-pair dynamics, including an engineered living-worm system. Abrahams and colleagues demonstrated spin-resonance responses in engineered proteins within living bacterial cells; protein engineering changed response magnitude and speed. The receiving molecular structure and the imposed field were experimental variables. [61]

These are concrete demonstrations of field-controlled spin chemistry. Their optical excitation, static bias fields, prepared proteins, and engineered reporters specify the conditions to reproduce. HHS should identify

endogenous counterparts, determine their chemical products and operating conditions, and connect their responses to calcium timing, redox signaling, and function. Flavins, hemes, iron-sulfur systems, and radical-pair-forming reactions belong in the candidate inventory; each requires its own coupling measurement.

Circadian research connects the same inquiry to biological timing across the organism. Yoshii and colleagues reported light- and cryptochrome-dependent magnetic-field effects on the *Drosophila* clock; Fedele and colleagues examined genetic determinants of related clock responses. Include light, circadian phase, sleep, and recovery as controlled variables in the transducer program and determine which mechanisms operate in mammalian systems. ^[62]

Density gating and the persistence gate

Density gating is my explanation to test for differences among biological receivers. It asks whether the abundance, location, and operating state of responsive machinery determine how strongly an external input is converted into an internal response. It emerged from the tissue and cell-state patterns in the evidence. Its prediction must be made from measurements taken before the outcome is known.

The full architecture has three receiver dimensions and a persistence gate. First, quantify voltage-gated-channel density, excitability, ionic throughput, and timing requirements. Second, quantify mitochondrial abundance, calcium–organelle coupling, energy demand, and reserve. Third, quantify relevant redox cofactors and experimentally supported spin-sensitive reaction systems. The persistence gate then measures cell longevity, turnover, repair, immune clearance, antioxidant capacity, developmental deadlines, and recovery between exposures.

Genomic susceptibility is a separate dimension. A genotype can change regulation, expression, or response without being reducible to the amount of one receptor. The CACNA1C sleep experiment and Palone's strain-dependent reproductive results therefore belong alongside density measurements, with internal dose and cell state characterized. Compare matched and isogenic systems to identify the contribution of each variable. ^{[32] [34]}

The cancer studies make lineage-specific persistence especially important. Malignant heart schwannomas arise from Schwann-cell lineage, so HHS should measure those cells and their microenvironment directly. Map their calcium handling, redox state, repair, turnover, and interaction with neighboring tissue during chronic exposure. The hypothesis earns explanatory value if measured receiver and persistence variables predict response across preparations, including conditions where a response is absent.

The developmental pathway: timing becomes anatomy and function

The developmental branch connects field-responsive machinery to calcium and redox timing, gene-regulatory decoding, progenitor decisions, tissue organization, and later function. Its foundation includes direct experiments on timing: Weissman and colleagues showed that disrupting radial-glia calcium waves reduced proliferation during embryonic neurogenesis. Borodinsky and colleagues changed embryonic neuronal calcium-spike activity and changed neurotransmitter expression. Neural-tube studies connect calcium flashes and intracellular dynamics to pulsed actin contraction, apical constriction, and closure. Each supplies a specific relationship between signal pattern and developmental task. ^{[63] [64] [65]}

Cakir and colleagues supply a directly relevant RF-exposure anchor in human cortical organoids. They reported altered radial-glia differentiation and BET-mediated developmental pathways, including rescue of RF-associated developmental defects with BET inhibition. The important bridge for HHS is to measure calcium patterns, mitochondrial function, chromatin regulation, and differentiation in the same exposed preparation and in the correct time order. ^[30]

Two complementary human-organoid research directions extend this developmental program. Courchesne and colleagues connected cortical-organoid growth and NDEL1-related biology with autism severity. Roh and

colleagues reported restoration of impaired NMDAR function through SLC6A20 suppression in SHANK-mutant models, including engineered human cortical organoids. These developmental and intervention studies identify growth, co-agonist transport, electrophysiological, and phosphorylation endpoints for the RF research program. ^[66]

My integrated developmental experiment brings those endpoints into exposure-controlled systems: calcium waveforms, CYB5B and channel dependence, mitochondrial potential and redox, BET-related regulation, NDEL1, radial-glia persistence, NMDAR currents, glycine availability, SLC6A20, and phosphoproteomic state. Use targeted interventions to determine which upstream changes mediate which downstream outcomes. The prediction is that altered signaling precision precedes the developmental divergence and that correcting the relevant mediator restores the affected function.

Aldad and colleagues' prenatal mouse study adds reported offspring behavioral and electrophysiological outcomes. The historical chick experiments add embryonic morphology, stage dependence, and noise suppression. Together they justify a program spanning organoids, developmental models, and prospective human exposure assessment. A developing child gets only one chance at each developmental window. ^{[67] [10] [47]}

The cofactor branch should examine folate status and transport, MTHFR-related metabolism, maternal inflammation, nutrition, sleep, medication and chemical co-exposures, and mitochondrial reserve. Test interactions explicitly. Match the model to the developmental window: early neural-tube closure and later brain-barrier or choroid-plexus transport are distinct processes. This research should explain how background burdens change the capacity to maintain developmental coordination. ^[68]

The developmental manuscript specifies the calcium-to-chromatin bridge in greater detail. Calcium-responsive kinases and phosphatases alter the activity and location of chromatin regulators. BET proteins read acetylated chromatin and participate in transcriptional regulation. The proposed sequence is a change in calcium timing, altered kinase/phosphatase decoding, a changed acetylation landscape, altered BET occupancy, and a different progenitor-cell decision. Measure each step in the Cakir exposure model instead of inferring the bridge from separate experiments. ^{[45] [69]}

Use a directional rescue test. Restoring the relevant calcium waveform should normalize downstream chromatin and differentiation if that waveform mediates the effect. BET inhibition could rescue the downstream developmental phenotype while leaving the initial calcium/redox disturbance intact. These outcomes distinguish an initiating event from a downstream reader. They also explain why a rescue experiment is more informative than a list of altered genes.

NDEL1 belongs in a parallel developmental-control branch. Its expression, enzymatic activity, phosphorylation, centrosomal localization, and interaction with cytoskeletal machinery are separate measurements. Courchesne's organoid work identifies an association with growth and developmental subgroup; the RF program must test exposure dependence and causal direction in its own preparations. Determine whether restoring NDEL1 changes growth or migration while the upstream signal remains altered, and whether correcting calcium timing restores both branches. ^{[66] [70]}

Study neurulation in a model that actually undergoes closure. Record calcium flashes, actomyosin contractions, apical constriction, tissue strain, closure velocity, and developmental stage together. Follow later cortical differentiation in organoids, and use microglia-containing assembloids for immune-neural interactions and pruning questions. These models address different developmental tasks and windows. Link them to maternal-fetal dosimetry and prospective pregnancy cohorts so the active experimental conditions can be compared with real exposure. ^[65]

The full cofactor program should also examine CYB5B-related sterol handling, immune-cell activation, folate transport and autoantibodies, and mARC-associated metabolic state as separately testable branches. Measure the proposed intermediates in order, with exposure-specific changes and mediator rescue. Ma and colleagues'

CYB5A/CYB5B sterol-oxidation work provides a biochemical anchor for that branch. It is the exposure experiment that must establish whether the pathway connects to altered tolerance or development. ^[71]

The cancer pathway: error persistence and failed correction

The cancer branch connects disrupted coordination to the persistence of genomic and cellular errors. My proposed pathway includes altered repair signaling, oxidative burden, checkpoint control, apoptosis, tissue communication, and immune clearance. Belyaev's repair-foci findings and Markova's cell-type differences identify processes to examine upstream of the tumors observed by NTP and Ramazzini. Measure damage production, repair recruitment, repair completion, and retention of errors separately. ^{[12] [1] [2]}

The experimental question is whether an exposed lineage accumulates a persistent regulatory or repair burden before pathology, and whether intervention at the identified interface prevents that burden. Include lineage tracing, mutation and chromatin analysis, long-term recovery, and the tissue microenvironment. Link early measurements to later pathology in the same experimental program.

Therapeutic RF research reinforces the importance of pathway specificity. Jimenez and colleagues reported tumor-specific amplitude-modulated RF responses involving Cav3.2 channels and calcium influx, including channel-knockdown experiments. FDA authorized TheraBionic P1 under a Humanitarian Device Exemption for a defined advanced liver-cancer indication, assessing safety and probable benefit. Its labeling includes a calcium-channel-blocker contraindication. Field-channel biology already has a clinical research and regulatory context; HHS should use that knowledge in transducer discovery. ^[72]

The metabolic pathway: fuel supply, timing, and compensation

The metabolic branch of my model connects membrane and calcium timing to mitochondrial energy coordination, insulin pulsatility, glucose handling, hepatic output, and adipose signaling. It predicts that repeated compensation can reduce metabolic flexibility and change the relationship between a physiological demand and its response. This supplies a concrete task for fidelity measurement: matching fuel availability and energy production to changing demand. ^[8]

Volkow and colleagues' human crossover study reported a regional glucose-metabolism response after 50 minutes of mobile-phone exposure, without a corresponding whole-brain change. That result makes tissue-resolved metabolic measurement directly relevant. HHS should pair such regional assessments with time-resolved signaling and functional challenge studies. ^[73]

Measure calcium rhythms, insulin secretion dynamics, glucose responses, mitochondrial respiration and reserve, and recovery through repeated standardized challenges. Compare systems with different baseline metabolic burdens. Determine whether an unchanged average glucose or hormone concentration conceals altered timing, greater compensatory effort, or reduced reserve at the next challenge. Use mediator-specific interventions to connect the exposure response to the metabolic task.

The immune pathway: discrimination, tolerance, and resolution

The immune branch addresses the precision with which cells identify a challenge, scale their response, and stop responding. My model connects channel/CYB5B-related calcium signaling and mitochondrial-redox coordination with three routes: inappropriate danger signaling, altered self-antigens, and weakened tolerance. A persistent loop would involve tissue injury, additional antigen exposure, renewed activation, and incomplete resolution. This is a specific framework for investigating autoimmune and inflammatory vulnerability. ^[8]

HHS should measure calcium-dependent transcription, activation thresholds, cytokine timing, mitochondrial danger signals, antigen presentation, regulatory-cell function, and recovery after challenge. Test direct immune-cell responses separately from signals released by exposed tissue. Use pathogen and sterile

challenges with defined doses, follow the resolution phase, then repeat the challenge to assess retained burden. A second challenge can reveal reduced reserve that a resting biomarker misses.

The existing macrophage literature includes experiments measuring responses to bacterial and viral challenges after RF exposure. Such challenge-based designs are relevant to the task, while their actual exposure conditions must remain attached to the findings. For example, Sueiro-Benavides and colleagues used 2.45 GHz at 28 W/m². HHS should extend pathway-specific challenge experiments into lower, public-exposure-relevant conditions and determine the waveform and receiver dependence. ^[74]

ceLLM: the embodied cellular transfer function

The Cellular Latent Learning Model, or ceLLM, is my account of how a living cell integrates its surroundings with its own physical organization and history. S4–Mito–Spin supplies candidate entry points and amplification mechanisms within that larger architecture. The cell receives an input through a particular arrangement of membranes, channels, DNA, RNA, proteins, organelles, cytoskeleton, water, and electrochemical gradients. That arrangement determines its response. The entire energized cell is the receiving system.

The framework connects six operations: an inherited and acquired physical organization; transduction of an input; a changing bioelectric state; metabolic execution; feedback about the consequences; and a persistent update. Those operations recur within organelles, cells, tissues, and the organism. A disturbance becomes consequential when it changes the accuracy of the loop, the cost of compensation, or the structure carried into the next response.

Genome sequence constrains the available components. Noncoding regulatory architecture, chromatin accessibility, mitochondrial-nucleoid organization, RNA processing and localization, channel abundance, organelle position, and prior activity determine which components are available together. RNA participates through messenger transcripts, splice forms, regulatory RNAs, scaffolding, localization, and control of translation. In the hardware-matrix language of my manuscripts, these are physical constraints and transient programs. The broader embodied formulation brings all of them into the same experimentally measurable response function.

Separate relatively slow organization from fast state. Chromatin structure, contact-site arrangement, channel expression, and cell identity change on different schedules from membrane voltage, calcium pulses, pH, phosphorylation, and redox chemistry. Sodium, potassium, chloride, protons, magnesium, and zinc belong in the state description alongside calcium. A biologically useful measurement asks how the organized system responds over time: its gain, delay, phase, threshold, damping, cross-coupling, and return to baseline.

This yields a central research target: the **latent response phenotype**. Two cells or people can have similar resting measurements and respond differently to the same challenge. The controlled CACNA1C sleep study makes that question concrete. HHS should connect genotype-conditioned responses to expression, splicing, channel function, cellular architecture, and internal dose. Isogenic editing and rescue can determine which regulatory changes alter response while leaving baseline function comparatively intact. Susceptibility should be measured during a task, across its recovery, and at the next challenge. ^{[32] [33]}

Photonic-redox feedback: chemistry, geometry, timing, and memory

My photonic-redox extension asks whether the cell also uses excited-state chemistry and ultra-weak photon emission as feedback about its own activity. Oxidative reactions can generate electronically excited species and light. Salari and colleagues imaged differences in ultra-weak emission between living and dead mice and changes in stressed plants. Those measurements establish an accessible optical readout of biological state. I ask HHS to test whether the structure of that readout also contributes to regulation. ^[75]

The proposed information is distributed across spectrum, emission location, timing, burst structure, direction, and the receiver's state. Chemistry supplies the event; geometry determines which structures it can reach; endogenous timing determines the sensitive window; and local molecular state determines the response. The same optical event can therefore have different consequences in cells with different organization or at different phases of a calcium or redox cycle.

This is the meaning of the **whole-cell transfer kernel** in my manuscripts: the physical relationship between an event and the response produced by the living receiver. Absorption, scattering, local excitation transfer, chemical conversion, and downstream amplification all enter that relationship. Test those processes at the relevant distances and photon fluxes. Short-range interactions at membranes and organelle contacts give the program experimentally accessible starting points.

Photonic reafference names the proposed return of activity-generated optical/redox events into the system that generated them. A prior event can leave a temporary local mark through phosphorylation, channel state, oxidation, chromatin accessibility, or organelle positioning. I call the collection of those marks a structural eligibility field. Feedback arriving while a structure remains eligible could selectively reinforce or change that structure. The test is whether prior activation, appropriately timed feedback, and receiver susceptibility jointly determine a persistent update.

The proposed biological lock-in mechanism supplies a further prediction. Endogenous ionic and metabolic rhythms provide a reference phase. Events aligned with that phase should produce a different cumulative response from equally intense events delivered at random phases. This hypothesis concerns biological timing and state-dependent reception. It can be tested with phase-shifted and scrambled replay at matched photon number, spectrum, and energy. If phase does not matter under the specified conditions, that component of the model loses explanatory value.

The resulting research question is whether cells continually calibrate their behavior using feedback from the exact physical organization they currently possess. That would connect immediate signaling to longer-lived adaptation through RNA programs, chromatin state, protein abundance, organelle contacts, and structural remodeling. HHS should test this connection directly through interventions that distinguish a passive optical marker from feedback that changes future behavior.

Topology, hydration, and the physical organization of the receiver

The topology-gated manuscripts identify mitochondrial DNA packaging as one candidate control variable. Brüser and colleagues found distinct nucleoid activity states associated with TFAM-to-mtDNA ratio and compaction. This gives the research program a manipulable structural substrate. Vary compaction, supercoiling, and hydration while measuring respiration, calcium handling, redox state, optical response, and transcription. Determine whether organization shifts sensitivity independently of a general loss of mitochondrial function. ^[76]

The mitochondrial-DNA geometry calculations in my manuscripts define experimental search windows. Converting contour, diameter, or nucleoid size to a free-space frequency is a dimensional heuristic; a measured cellular resonance requires an exposure-response experiment in the hydrated biological structure. HHS should fund that measurement with frequency sweeps, thermal controls, topology perturbation, and rescue. The model's value depends on a predicted shift in response when the receiving structure changes.

Wu and colleagues provide a specific frequency-selective experiment to extend. In their system, 34.5 THz exposure promoted mitochondrial biogenesis through calcium signaling and the PGC-1 α -NRF1/2-TFAM pathway, while 36.1 THz did not produce the same result. Their modeling implicated calcium-channel pore dynamics. Use that defined result to separate a channel-mediated response from any additional topology-dependent contribution, with independently controlled absorption and temperature. ^[77]

The cytoskeleton supplies other measurable physical relationships. Kalra and colleagues measured electronic excitation migration over approximately 6.6 nm in microtubules. Coscia and colleagues showed that actin waves support mitochondrial content mixing and organelle homeostasis. These are distinct experimental anchors for local energy transfer and structural distribution of mitochondrial state. Test whether cytoskeletal perturbation changes the spatial propagation of a photonic-redox response after controlling for transport, mechanics, respiration, and source intensity. ^{[78] [79]}

Zadeh-Haghighi and colleagues' 2026 tubulin experiment adds an isotope-sensitive physical intervention: magnesium isotope and an applied 3 mT magnetic field affected polymerization in a pattern consistent with radical-pair modeling. The reported field and preparation define the starting conditions. Extend the experiment across field strengths and living-cell states, identify the participating chemistry, and determine whether changes propagate into cellular function. This joins the Spin branch to a specific structural endpoint. ^[80]

Organelle contacts, calcium microdomains, mitochondrial networks, and retrograde signaling are candidate amplification routes. At the tissue level, gap junctions and other direct cellular connections can distribute or stabilize a local change. The ceLLM program should measure these ordinary biological amplification steps alongside any proposed optical contribution. A weak initiating event becomes relevant through the response it recruits.

Four kinds of fidelity and the accumulation of burden

HHS should distinguish four functions. **Transduction fidelity** concerns whether the input produces an appropriate intracellular signal. **Execution fidelity** concerns whether metabolism and cellular action match that signal. **Readback fidelity** concerns whether feedback accurately represents the consequence. **Update fidelity** concerns whether the resulting adaptation improves the next response. Each needs a defined task, reference distribution, and measurable outcome.

This distinction explains why more activity is not necessarily better function. Higher calcium, ROS, photon output, or transcription can accompany successful adaptation, compensatory effort, or loss of control. A quiet baseline can coexist with impaired reserve. The proposed bioelectric-fidelity measurements therefore include frequency, amplitude, phase, spatial propagation, heterogeneity, compartmental coupling, and recovery. Validate their ability to predict later function before combining them into an index.

I use **fidelity debt** for the accumulating cost of compensation that preserves present performance while reducing future reserve. I use **somatic overfitting** for adaptation to persistently distorted inputs that leaves a cell less capable of responding to its ordinary physiological environment. These concepts connect repeated perturbation to hysteresis, altered recovery, and the meta-disease state. They demand repeated-challenge experiments and observation after exposure ends.

The model also requires specific interactions with sleep disruption, circadian timing, inflammation, hypoxia, nutrient status, chemical exposure, and inherited susceptibility. Determine which combination changes a defined control loop and when. The proposed Goldilocks zone is a range of coordinated biological operation, to be measured rather than assumed from the label “natural.” Record background magnetic fields and light, then use shielding, restoration, and phase-controlled perturbations to identify their roles.

The co-zeitgeber illustration raises a related timing question: can a characterized field act as an additional timing cue or disturbance alongside light and other established inputs? HHS should measure phase-response curves, amplitude, entrainment, and recovery under controlled light schedules. The cryptochrome experiments supply a molecular research direction; mammalian light entrainment and engineered CYB5B responses must be mapped in their own systems. ^[59]

Nested bioelectric control and AlphaPulse

At tissue scale, Emmons-Bell and colleagues showed that transient disruption of gap-junction communication could alter regenerated head anatomy in genetically wild-type planarians. My exposure extension asks whether a characterized field changes the persistence or subsequent reversion of an induced anatomical state. Pair anatomy with membrane-potential maps, cellular communication, mitochondrial state, and optical measurements. Stabilization, accelerated reversion, and unchanged recovery are experimentally distinguishable outcomes. ^[81]

AlphaPulse is the associated open data and modeling program. Its records should align exposure waveforms with voltage, multi-ion dynamics, redox, mitochondrial function, photon measurements, genotype, developmental stage, and subsequent function. Models should predict held-out responses and recovery trajectories, then be challenged by interventions. A photon-derived variable must add predictive value beyond conventional calcium, ROS, voltage, and respiration measurements before it earns a role in the model. Causal claims require the blocking and rescue experiments as well.

Recovery, aging, and the timing of disease susceptibility

Yang and colleagues investigated loss of epigenetic information in mammalian aging. Debès and colleagues linked age-associated transcriptional changes to longevity outcomes. These experiments make maintenance of biological information and regulatory precision concrete research subjects. HHS should extend that work to environmental inputs, asking whether repeated exposure reduces coordination or recovery earlier in life in susceptible systems. ^[82]

The meta-disease prediction concerns the loss of reserve: an organism faces a later stressor with less capacity to respond. Measure persistence after exposure, restoration during recovery, and interaction with subsequent challenge. In developmental systems, include the deadline itself as an outcome variable. In long-lived tissues, include the retention of a state across months and repeated stress. This addresses the possibility of earlier vulnerability without waiting for visible aging.

Four evidence-linked HHS initiatives

A. Biological transducer discovery. Start from CYB5B, the channel-dependent experiments, and the proposed S4 interface. Independently replicate the existing results, identify sensors and relays, and use genetic perturbation and rescue to map the causal sequence. Deliver a public inventory of supported interfaces, exposure conditions, downstream pathways, and functional consequences.

B. Waveform-specific RF response and the 800 MHz research line. Replicate Rao's neuronal-calcium result, then compare deployment-relevant waveforms, geometry, internal dose, duration, and recovery. Coordinate with FCC and NIST to obtain actual specifications for cellular and direct-to-device systems. Deliver reproducible waveform-response datasets connecting physical exposure to timing, redox, recovery, and function. ^[38]

C. Endogenous spin-sensitive interfaces. Build from the molecular spin-control experiments and cryptochrome-associated responses. Identify endogenous molecules, map static-field, RF, light, and chemical dependencies, and measure downstream effects on coordination. Deliver independently replicated relationships between field parameters, reaction products, and biological performance. ^{[61] [59]}

D. Genetic and genomic determinants. Replicate the controlled CACNA1C finding and examine the strain-dependent reproductive evidence with adequate subgroup power, interaction tests, and internal dosimetry. Use isogenic comparisons and measure channel, transducer, and metabolic state. Deliver supported susceptibility modifiers that can inform protection of people whose responses are concealed by population averages. ^{[32] [34]}

The Dissonance Protocol and decisive causal tests

Compare sham, carrier-only, and realistically pulsed RF at matched average SAR and characterized spatial absorption, with sensitive temporal and spatial temperature monitoring. Vary modulation and recovery intervals independently, and include separately characterized ELF arms. Record calcium-event timing, phase stability, spatial propagation, mitochondrial function, redox pulses, transcriptional variability, repair, and return to baseline before scoring morphological or tumor endpoints.

The record–block–replay–scramble experiment provides a stronger intervention. Record the exposure-associated intracellular pattern; block the candidate interface; replay the measured downstream pattern through an independent calibrated actuator; and compare it with a temporally scrambled pattern matched for relevant amplitude and total activity. Determine whether the original timing reproduces the functional outcome and whether restoring the normal pattern rescues it. Control the actuator's own effects and define each prediction before analysis.

Use blinded analysis, prespecified endpoints, adequate power, and independent replication. A mechanistic intervention should restore the relevant function while exposure and temperature remain controlled. Determine whether early fidelity markers predict later pathology in the same system. If the proposed upstream disturbance is absent where the predicted downstream pathology develops, revise or reject that mechanistic branch. If timing-dependent mediation and rescue are demonstrated, incorporate the result into the protection framework.

A coordinated experiment across the complete loop

The full ceLLM and S4–Mito–Spin program requires the same exposure, receiver, and time axis across measurements. Disconnected positive findings cannot establish the order of events. I ask HHS to fund the following linked experiments with common calibration, open protocols, and independent replication.

1. Receiver and waveform factorial. Cross carrier frequency, modulation envelope, duty cycle, phase, and recovery interval in sham-controlled systems with matched average absorption and measured temperature. Include isogenic CACNA1C comparisons, CYB5B knockdown and wild-type rescue, heme-binding or membrane-localization variants, and functional S4/channel manipulations. Test standalone ELF, RF modulation, and combined exposures as distinct conditions. Record which interface is necessary for which waveform.

2. Time-resolved intracellular sequence. Follow compartment-specific calcium, membrane potential, mitochondrial potential, respiration, redox products, photon emission, transcription, and recovery in the same preparation. Resolve whether an early timing change precedes altered execution and later remodeling. Include positive controls that alter timing without RF, and comparisons that change average calcium without reproducing its temporal pattern.

3. Record, block, replay, and scramble the optical leg. Record natural emission and test whether calibrated replay changes a receiving preparation. Use gas-tight, chemically isolated chambers; characterize quartz, glass, filters, and opaque barriers spectrally. Control shared temperature, vibration, electromagnetic coupling, and gas exchange. Compare faithful replay with time-, phase-, spectrum-, and location-scrambled signals at matched photon number and energy. The response must track optical access and the predicted structure to establish a functional optical contribution.

Mould and colleagues' isolated-mitochondria experiments reported interactions affected by shielding and lighting conditions. That is a concrete starting point for replication with direct photon measurement and separation of the possible transfer routes. The decisive advance is a reproducible receiving-system response that can be blocked and restored through a characterized optical path. ^[83]

4. Eligibility and receiver geometry. Activate one of two distinguishable cellular pathways, vary the delay before feedback, and measure which pathway retains a change at a later challenge. Independently change source position, cell shape, or organelle arrangement while maintaining comparable metabolic state. A spatially and temporally selective update supports the proposed eligibility mechanism; an effect explained entirely by total intensity does not establish it.

5. Topology versus amplification. Combine TFAM and topoisomerase perturbation, mtDNA depletion and feasible rescue, and narrow-band exposure with calcium-uptake or contact-site interventions. Test whether changing topology shifts sensitivity while calcium blockade prevents downstream amplification. Control the substantial metabolic consequences of mtDNA depletion. A double dissociation would locate two distinct steps; persistence without topology dependence would redirect the model to the demonstrated receiver.

6. Routing and tissue memory. Use reversible cytoskeletal interventions to separate source intensity from spatial spread. Extend successful cellular results into communicating tissues, developmental models, and the planarian reversion experiment. A modern optical-isolation replication of the historical Gurwitsch question should directly measure source emission and recipient mitotic timing, using sealed chambers and blinded optical blocking and replay.

7. Persistent burden and later pathology. Repeat standardized physiological challenges across exposure and recovery cycles. Determine whether the early pattern predicts reduced reserve, altered differentiation, impaired repair, metabolic inflexibility, or unresolved immune activation. Follow the relevant lineage into the later outcome. Require the proposed mediator to change before the outcome and require a targeted intervention to modify the predicted sequence.

For photon measurements, publish detector efficiency, dark counts, background subtraction, afterglow controls, calibration, and rejection of cosmic-ray or electronic artifacts. Distinguish spontaneous emission from excitation-induced fluorescence. For every experiment, archive the actual waveform, field geometry, internal dose, temperature, analysis code, raw observations, and all prespecified outcomes. At least three independent laboratories should attempt the core developmental and receiver experiments. These requirements make HHS's investment capable of resolving the model.

Restore long-term NTP research with modern signals, prenatal exposure, both sexes, and doses relevant to public protection. Pair cancer studies with developmental, reproductive, neurological, immune, and metabolic work. NIEHS's statement that no additional RF exposure studies are planned is incompatible with the priority this record demands. HHS should publish the program, responsible offices, funding, milestones, and results. ^[23]

Question 17. Federal coordination

The evidence already crosses agency boundaries; coordination must produce action, ownership, and deadlines.

(a) FDA. Lead a current electronic-product radiation assessment, realistic testing proposals, exposure-reduction evaluation, and the standards determination under existing law. Address cancer and non-cancer outcomes and identify the evidence supporting each decision. **(b) NIH.** Fund modern NTP studies, mechanistic interventions, developmental models, cohorts, and independent replication. **(c) CDC.** Develop surveillance, exposure-linked registry research, occupational coordination through NIOSH, clinician education, and clear public communication. ^[18]

HHS should publish one work plan, budget, responsible office for each deliverable, and annual progress record. Include EPA for environmental-health expertise, FCC and NIST for engineering and measurement, and USDA/DOI for ecological questions. Require open methods, data where appropriate, and transparent

management of financial and institutional conflicts. Health conclusions must be determined through accountable health expertise.

Question 18. Action now and the Clean Ether Act

The NTP, Ramazzini, and WHO-commissioned animal review evidence is already in the public record. HHS should act now on research, exposure reduction, realistic testing, and public accountability.

Reinvigorate Public Law 90-602 through the duties now codified in 21 USC 360hh–360ss. Section 360ii directs the Secretary to carry out an electronic-product radiation-control program, including research to minimize unnecessary radiation and evaluation of exposure-reduction techniques. Section 360kk authorizes performance standards when the required necessity determination is made. Publish an explicit wireless-RF program and that determination. These are HHS responsibilities; collaboration with EPA must strengthen their execution. ^[18]

Ask Congress to transfer primary responsibility for RF health-protection standards from the FCC to HHS and EPA through a clearly assigned statutory framework, while retaining the FCC's spectrum and communications functions. Repeal the Section 704 restriction described above. Health standards should be set by accountable health agencies with the medical, toxicological, developmental, and environmental expertise to evaluate the full record.

Adopt a precautionary goal of reducing unnecessary exposure as far as reasonably achievable, with measured results. Begin with child-priority settings, body-contact testing, transparent disclosures, radio-off options, and fiber/Ethernet procurement. Research should proceed alongside feasible prevention.

Enact a **Clean Ether Act**: a staged transition of suitable indoor connectivity to wired and optical systems, supported by interoperable Li-Fi compatibility in devices, access points, and public procurement. IEEE 802.11bb provides an existing light-communications standard. Fund deployment and independent evaluation in schools, childcare, healthcare, homes, and public buildings. Require actual displacement of avoidable RF transmissions, accessible service, reliable emergency operation, and optical/electrical safety. ^[7]

Legislative program and implementation deadlines

My proposed Clean Ether Act turns the research and engineering agenda into an implementation program. The supplied legislative draft is titled the “Clean Aether Act of 2026.” I ask HHS to support the following provisions and work with Congress on the additional transfer of health-standard authority and repeal of the Section 704 restriction requested in this comment.

Prospective siting protection. Establish a 1,500-foot minimum setback for new macro facilities near residential, school, hospital, and childcare parcels, measured from the equipment-compound boundary or support-structure base to the nearest covered property line. Apply the draft's prospective design to newly sited support structures, with defined treatment of existing infrastructure and collocation. Limit exceptions to specified emergency, public-safety, military, and disaster uses. Require a written record of clear and convincing evidence for a waiver: no feasible alternative, a material gap in critical communications, and the least intrusive means of closing that gap.

Agency action within one year. Require HHS, through FDA and in consultation with NTP, to deliver recommendations on exposure characterization, testing, vulnerable populations, and electronic-product standards. Require NTIA's national alternatives strategy within one year, addressing fiber, optical connectivity, existing infrastructure, and non-terrestrial systems. Compare the resulting device and infrastructure exposures directly. Connectivity architecture must be evaluated through its actual operation.

Standards and interoperability within 18 months. Require completion of the FCC reassessment described in the draft, informed by HHS's health findings, and initiation of a phased optical-interoperability rulemaking.

Address long-term and aggregate exposure, modulation, child protection, realistic device placement, and environmental effects. Build in a five-year cycle for subsequent scientific reassessment. Establish an initial implementation report to Congress at 18 months and annual updates for four more years.

Five-year deployment pilots. Fund fiber and optical systems in public schools and federal buildings, including backhaul, receivers, adapters, installation, staff training, maintenance, cybersecurity, and interoperability testing. Extend child-priority implementation to childcare, maternity and pediatric care, and sleeping environments through appropriate federal programs. Measure RF transmissions displaced, occupied-room exposure, reliability, energy use, cost, accessibility, and service quality. Radios should be readily disabled when wired or optical service meets the task.

Device compatibility and public procurement. Establish a phased requirement for optical-network compatibility in covered smartphones, tablets, laptops, and networking equipment, using published interoperable standards and their successors. The draft sets January 1, 2029 as the federal procurement start date, with reasoned case-specific waivers for mission, availability, accessibility, security, and cost constraints. Support adapters and practical transition periods so the policy expands access to lower-exposure connectivity.

Domestic photonics and multifunctional optical networks. Support optical transceivers, receivers, chipsets, standards implementation, manufacturing readiness, and secure supply chains. Fund Defense, Homeland Security, and NIST research on systems that combine communications with sanitation or protective functions in institutional, transportation, maritime, and defense settings. My optical-network patent demonstrates the engineering direction: photons can perform multiple useful functions within an intentionally designed system. Independent testing must establish performance and suitable optical operating conditions for each deployment.

Voluntary intellectual-property contributions. Establish the draft's authority for Treasury to accept qualifying voluntary donations, with the donation instrument controlling the rights conveyed. Any transfer into a future public investment vehicle would require the relevant congressional authority. Net receipts could support the optical programs subject to appropriations. This provides a route for public-benefit technology contributions consistent with RF Safe's long-standing commitment to public-domain designs.

Fund the transition. The draft proposes \$200 million annually for school and federal-building pilots, \$75 million annually for photonics innovation, and \$50 million annually for dual-use optical research for fiscal years 2027–2031, plus \$5 million in fiscal year 2027 for strategy, reassessment, and reporting. The proposed authorization totals \$1.630 billion. Congress should provide the resources, and agencies should report measured outcomes against them.

The practical sequence is to establish baselines, protocols, and pilots during the first two years; make wired and optical connectivity the default in suitable child-priority settings during years three through five; and scale successful, independently evaluated systems during years six through ten. Preserve reliable emergency service, affordable access, and interoperability throughout the transition. Public research and exposure reduction should advance together.

A developing child's opportunity cannot be restored after the window closes. The federal government should make connectivity compatible with exposure reduction and biological protection. Reinvigorate Public Law 90-602. Repeal Section 704's health-based siting restriction. Put health standards under health agencies. Build the transition through the Clean Ether Act.

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Founder, RF Safe
Submitted as an individual

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Numbered by first citation. Linked titles identify the original studies, agency records, standards, statutes, and stated archives. Corrections are included where relevant.

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Appendix A. FCC source records

The following five pages reproduce the released 2 mm SAR spreadsheet and the complete September 29, 2023 FCC response letter for FOIA Control Nos. 2023-000281 and 2023-000325. The table states that its test separation was outside FCC guidance; the letter supplies the agency’s explanation. Both are included for direct review. Source: pages 36–40 of the supplied FCC packet; see reference [24].

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***NOTE: THE FOLLOWING RESULTS ARE FROM SAR TESTS AT A SEPARATION DISTANCE NOT IN ACCORDANCE WITH FCC GUIDANCE**

FCC ID	Manufacturer	Description	Sample Type	SAR Configuration	FCC Lab Measured SAR Value (W/kg)	Serial Number
BCG-E3091A	Apple	iPhone 7 Portable Handset	Provided by Manufacturer	WCDMA Band 4, RMC, 12.2 kbps, Ch 1513, Body, Bottom, 2 mm	1.440	C6K8HD69HG7N
BCG-E3161A	Apple	iPhone X Portable Handset	Provided by Manufacturer	WCDMA Band 2, RMC, 12.2 kbps, Ch 9262, Body, Bottom, 2 mm	1.350	C39VF007JH2Q
				GP RS 1900, GMSK, 2 Tx Slots, Ch 512, Body, Bottom, 2 mm	0.727	
BCG-E3218A	Apple	iPhone XS Portable Handset	Purchased by FCC	GP RS 1900, GMSK, 2 Tx Slots, Ch 661, Body, Back, 2 mm	2.340	GONZ50M7KPFR
				GP RS 1900, GMSK, 2 Tx Slots, Ch 661, Body, Bottom, 2 mm	1.990	
A3LSMG960U	Samsung	Galaxy S9 Portable Handset	Provided by Manufacturer	CDMA BC1, RC3, TD50 32, Ch 600, Body, Back, 2 mm, Connect then Position	4.520	R38K6095MYL
				CDMA BC1, RC3, TD50 32, Ch 600, Body, Back, 2 mm, Position then Connect	4.380	
A3LSMG960U	Samsung	Galaxy S9 Portable Handset	Purchased by FCC	CDMA BC1, RC3, TD50 32, Ch 600, Body, Back, 2 mm, Connect then Position	No Data Yet	R3M7061T9A
				CDMA BC1, RC3, TD50 32, Ch 600, Body, Back, 2 mm, Position then Connect	No Data Yet	
A3LSMJ337A	Samsung	Galaxy J3 Portable Handset	Provided by Manufacturer	WCDMA Band 4, RMC, 12.2 kbps, Ch 1312, Body, Back, 2 mm, Connect then Position	5.200	RF8KA1J3X6E
				WCDMA Band 4, RMC, 12.2 kbps, Ch 1312, Body, Back, 2 mm, Position then Connect	5.140	
IHDT56XC4	Motorola	Moto e5 play Portable Handset	Provided by Manufacturer	WCDMA Band 2, RMC, 12.2 kbps, Ch 9262, Body, Back, 2 mm, Connect then Position	1.390	359524090350295
				WCDMA Band 2, RMC, 12.2 kbps, Ch 9262, Body, Back, 2 mm, Position then Connect	1.360	
IHDT56XB1	Motorola	Moto g6 Play Portable Handset	Provided by Manufacturer	WCDMA Band 5, RMC, 12.2 kbps, Ch 4132, Body, Front, 2 mm, Connect then Position	1.980	35186409004178
				WCDMA Band 5, RMC, 12.2 kbps, Ch 4132, Body, Front, 2 mm, Position then Connect	1.980	
IHDT56XB1	Motorola	Moto g6 Play Portable Handset	Purchased by FCC	WCDMA Band 5, RMC, 12.2 kbps, Ch 4132, Body, Front, 2 mm, Connect then Position	1.080	3518643090300620
				WCDMA Band 5, RMC, 12.2 kbps, Ch 4132, Body, Front, 2 mm, Position then Connect	1.090	
YHLBLUVIV05MN	BLU	Vivo 5 Mini Portable Handset	Provided by Manufacturer	GP RS 850, GMSK, 4 Tx Slots, Ch 128, Body, Back, 2 mm	2.200	1080021018057240
YHLBLUVIV05MN	BLU	Vivo 5 Mini Portable Handset	Purchased by FCC	GP RS 850, GMSK, 4 Tx Slots, Ch 128, Body, Back, 2 mm	2.750	1080021018069900

SAR Test Data Released under FOIA by the FCC

Above is a screensave of the spreadsheet of measurements from FCC cell phone radiation SAR test data using a seperation distance of 2mm between the phone and body phantom.

Note: the FCC limit for body SAR is 1.6 W / kg.

FCC text is in red above the table.

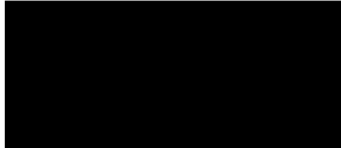


Federal Communications Commission
Washington, D.C. 20554

September 29, 2023

VIA ELECTRONIC MAIL

Theodora Scarato



RE: FOIA Control Nos. 2023-000281 and 2023-000325

Dear Ms. Scarato,

This letter responds to your Freedom of Information Act (FOIA) requests, FOIA Control No. 2023-000281 and FOIA Control No. 2023-000325. Your request FOIA Control No. 2023-000281 seeks “any and all SAR tests or documentation of SAR tests for any cellphones, including but not limited to the BLU phones, that the FCC tested to determine regulatory compliance for SAR. Please send all SAR Reports, as well as all documentation of the SAR test findings. Please also send the emails and attachments between FCC staff related to the findings of the SAR tests for cell phones. Any SAR tests done by the FCC related to cell phones on the US market January 2017 through April 2020.”¹ Your request FOIA 2023-000325 seeks “all SAR tests, the full reports, the measurements, any images and name of staff person doing tests and date done by [the Office of Engineering and Technology (OET)] (not the manufacturer) on cell phones in the year 2019.”²

As a general matter, you can find SAR testing records in the Equipment Authorization Database on our webpage at <https://apps.fcc.gov/oetcf/eas/reports/GenericSearch.cfm>. If you can identify the FCC ID numbers for the equipment you describe, you can look up the details of the grant. Enter the first three characters of the FCC ID Number as the grantee code and the rest including hyphens as the product code. You can also search by the manufacturer. Enter the manufacturer’s name in the Applicant Name field to populate the results.

Specific to your FOIA requests, OET and the Enforcement Bureau (EB) conducted a search and found 18 records responsive to your request. Many of these records relate to an article published by the *Chicago Tribune* on August 21, 2019, claiming several popular cell phones exceed the FCC’s guidelines for RF exposure. The *Chicago Tribune*, through an independent accredited lab, RF Exposure Lab, LLC, had tested devices manufactured by Apple, BLU Products, Motorola, and Samsung to arrive at the conclusion in the article. Because we take seriously any claims of non-compliance with RF exposure standards, the FCC tested the same device models at our Lab. The FCC’s tests confirm that all tested sample devices comply with the FCC’s strict RF exposure guidelines.

¹ FOIA Control No. 2023-000281 (submitted January 30, 2023).

² FOIA Control No. 2023-000325 (submitted February 8, 2023).

We did not have access to the actual phones tested by the test lab used by the *Chicago Tribune*. The FCC Lab, therefore, obtained the same makes and models of these phones (where available), both from suppliers and on the open market. We tested the devices manufactured by Apple, BLU Products, Motorola, and Samsung at a 5mm separation distance, consistent with our published guidance.³ These tests were conducted consistent with the FCC Lab normal practices and the RF exposure compliance report for each phone that is publicly available in the application for certification in the Equipment Authorization System. These devices all complied with the FCC limits for RF exposure, limits that have a significant safety margin. With this letter, we release the test results.

Separately, and only for purposes of comparison with the *Chicago Tribune* results, the FCC Lab tested the devices with a separation distance at which the *Chicago Tribune* tested its devices, i.e., at 2 mm separation distance. We observed that at a 2 mm separation distance, the FCC RF exposure limits were exceeded, and found that the *Chicago Tribune* results at the 2 mm separation distance were even higher than what we observed. With this letter, we release our 2 mm separation distance test results with the caveat that the 2 mm separation distance test results are inconsistent with FCC practice,⁴ and are misleading because they reflect extreme conditions. Notwithstanding the foregoing, given that the RF exposure limit includes a significant safety margin, none of the results suggest there is any RF safety issue with the devices tested.

We also stress that cell phones operate under the control of the wireless networks to which they are connected. We believe that the *Chicago Tribune* tests did not use the appropriate wireless network control codes to set the phone in the proper modes for operation for the U.S. This may have disabled the various sensors or caused the network simulator to not control the phone's transmitters properly for operation in the U.S.

With that explanation, in response to your FOIA requests, out of the 18 documents located, we are releasing 11 records in full with no redactions. We are withholding the remaining seven records in full under FOIA Exemption 5. Exemption 5 protects certain inter-agency and intra-agency records that are normally considered privileged in the civil discovery context.⁵ Exemption 5 encompasses a deliberative process privilege intended to "prevent injury to the quality of agency decisions."⁶ To fall within the scope of this privilege the agency records must be both pre-decisional and deliberative.⁷ Pre-decisional records must have been "prepared in order to assist an agency decision maker in arriving at his decision."⁸

³ KDB 447498 D01 General RF Exposure Guidance v06 specifies the 5 mm minimum test separation distance. https://apps.fcc.gov/kdb/GetAttachment.html?id=f81QgJxTTL5y0oRi0cpAuA%3D%3D&desc=447498%20D01%20General%20RF%20Exposure%20Guidance%20v06&tracking_number=20676 at 11 ("Devices that are designed to operate on the body of users ... without requiring additional body-worn accessories must be tested for SAR compliance using a conservative minimum *test separation distance* $\leq$ 5 mm to support compliance" (emphasis in original); see also KDB 648474 D04 Handset SAR v01r03, at 3, https://apps.fcc.gov/kdb/GetAttachment.html?id=zCDu9bDeV8fcsumpj%2Bef3w%3D%3D&desc=648474%20D04%20Handset%20SAR%20v01r03&tracking_number=33853 (citing KDB 447498 D01 General RF Exposure Guidance v06).

⁴ See, e.g., KDB 447498 D01 General RF Exposure Guidance v06, *supra* note 3.

⁵ 5 U.S.C. § 552(b)(5).

⁶ *NLRB v. Sears Roebuck & Co.*, 421 U.S. 132, 151 (1975).

⁷ *Id.* at 151-52.

⁸ *Formaldehyde Inst. v. Dep't of Health and Human Servs.*, 889 F.2d 1118, 1122 (D.C. Cir. 1989); see also *Coastal States Gas Corp. v. Dep't of Energy*, 617 F.2d 854, 866 (D.C. Cir. 1980) ("In deciding whether a document should

Deliberative records must be such that their disclosure “would expose an agency’s decision-making process in such a way as to discourage candid discussion within the agency and thereby undermine the agency’s ability to perform its functions.”⁹

The records withheld under Exemption 5, constituting internal documents and emails, contain pre-decisional internal deliberations among Commission staff. These records include staff summaries, pre-decisional discussion of the merits of information provided by third parties, preliminary thoughts of agency staff regarding policy, and general discussion of internal staff questions and viewpoints. Particularly, the withheld records implicate sensitive matters that require particular candor in the advice given to decision makers, which would be discouraged by the public release of the advice. We have determined that it is reasonably foreseeable that disclosure would harm the Commission’s deliberative processes, which Exemption 5 is intended to protect. Release of this information would chill deliberations within the Commission and impede the candid exchange of ideas.

We are required by both the FOIA and the Commission’s own rules to charge requesters certain fees associated with the costs of searching for, reviewing, and duplicating the sought-after information.¹⁰ To calculate the appropriate fee, requesters are classified as: (1) commercial use requesters; (2) educational requesters, non-commercial scientific organizations, or representatives of the news media; or (3) all other requesters.¹¹

Pursuant to section 0.466(a)(5)-(7) of the Commission’s rules, you have been classified as category (2), “educational requesters, non-commercial scientific organizations, or representatives of the news media.”¹² As an “educational requester, non-commercial scientific organization, or representative of the news media,” the Commission assesses charges to recover the cost of reproducing the records requested, excluding the cost of reproducing the first 100 pages. The production in response to your request did not involve more than 100 pages of duplication and is being provided in electronic form. Therefore, you will not be charged any fees.

If you consider this to be a denial of your FOIA request, you may seek review by filing an application for review with the Office of General Counsel. An application for review must be *received* by the Commission within 90 calendar days of the date of this letter.¹³ You may file an application for review by mailing the application to Federal Communications Commission, Office of General Counsel, 45 L Street NE, Washington, DC 20554, or you may file your application for review electronically by e-mailing it to FOIA-Appeal@fcc.gov. Please caption the envelope (or subject line, if via e-mail) and the application itself as “Review of Freedom of Information Action.”

be protected by the privilege we look to whether the document is . . . generated before the adoption of an agency policy and whether . . . it reflects the give-and-take of the consultative process. The exemption thus covers recommendations, draft documents, proposals, suggestions, and other subjective documents . . .”).

⁹ *Formaldehyde Inst.*, 889 F.2d at 1122 (quoting *Dudman Commc’ns Corp. v. Dep’t of the Air Force*, 815 F.2d 1565, 1568 (D.C. Cir. 1987)).

¹⁰ See 5 U.S.C. § 552(a)(4)(A); 47 CFR § 0.470.

¹¹ 47 CFR § 0.470.

¹² 47 CFR § 0.466(a)(5)-(7).

¹³ 47 CFR §§ 0.461(j), 1.7 (documents are considered filed with the Commission upon their receipt at the location designated by the Commission).

If you would like to discuss this response before filing an application for review to attempt to resolve your dispute without going through the appeals process, you may contact the Commission's FOIA Public Liaison for assistance at:

FOIA Public Liaison
Federal Communications Commission
Office of the Managing Director
Performance Evaluation and Records Management
45 L Street NE, Washington, DC 20554
202-418-0440
FOIA-Public-Liaison@fcc.gov

If you are unable to resolve your FOIA dispute through the Commission's FOIA Public Liaison, the Office of Government Information Services (OGIS), the Federal FOIA Ombudsman's office, offers mediation services to help resolve disputes between FOIA requesters and Federal agencies. The contact information for OGIS is:

Office of Government Information Services
National Archives and Records Administration
8601 Adelphi Road-OGIS
College Park, MD 20740-6001
202-741-5770
877-684-6448
ogis@nara.gov
<https://www.archives.gov/ogis>

Sincerely,



Ronald T. Repasi
Chief
Office of Engineering & Technology

Attachments

cc: FOIA Office, EB

B1. The cellular Goldilocks zone



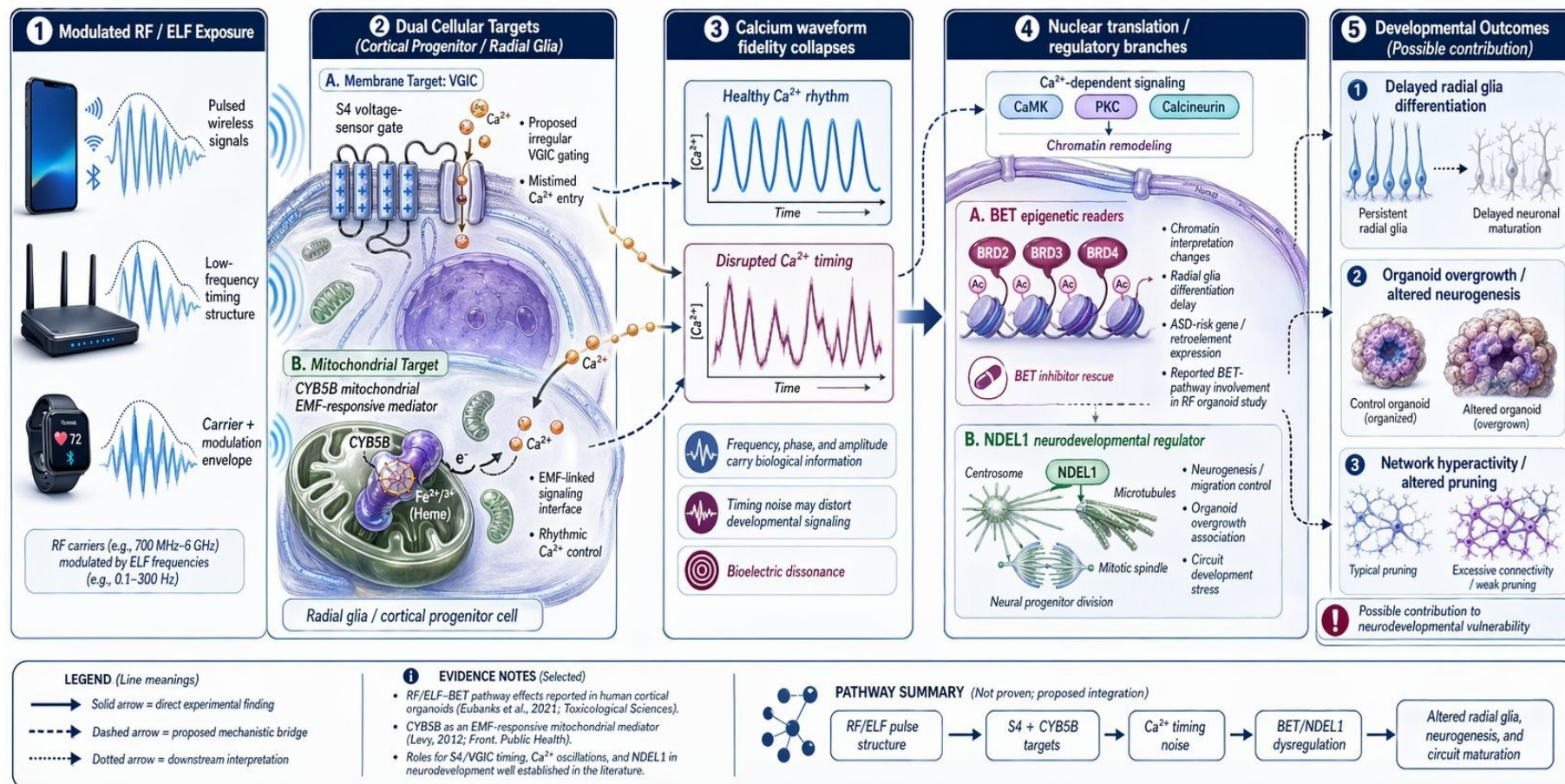
RF Safe conceptual illustration. The testable cellular question is whether specified environmental inputs shift coordination, response thresholds, or recovery. The broader nested-zone image supplies context; the research measurements are field strength, light, waveform, receiver state, and biological function. See Question 16, Four kinds of fidelity and the accumulation of burden.

B2. Developmental signaling and critical windows

Proposed Bioelectric Dissonance Pathway

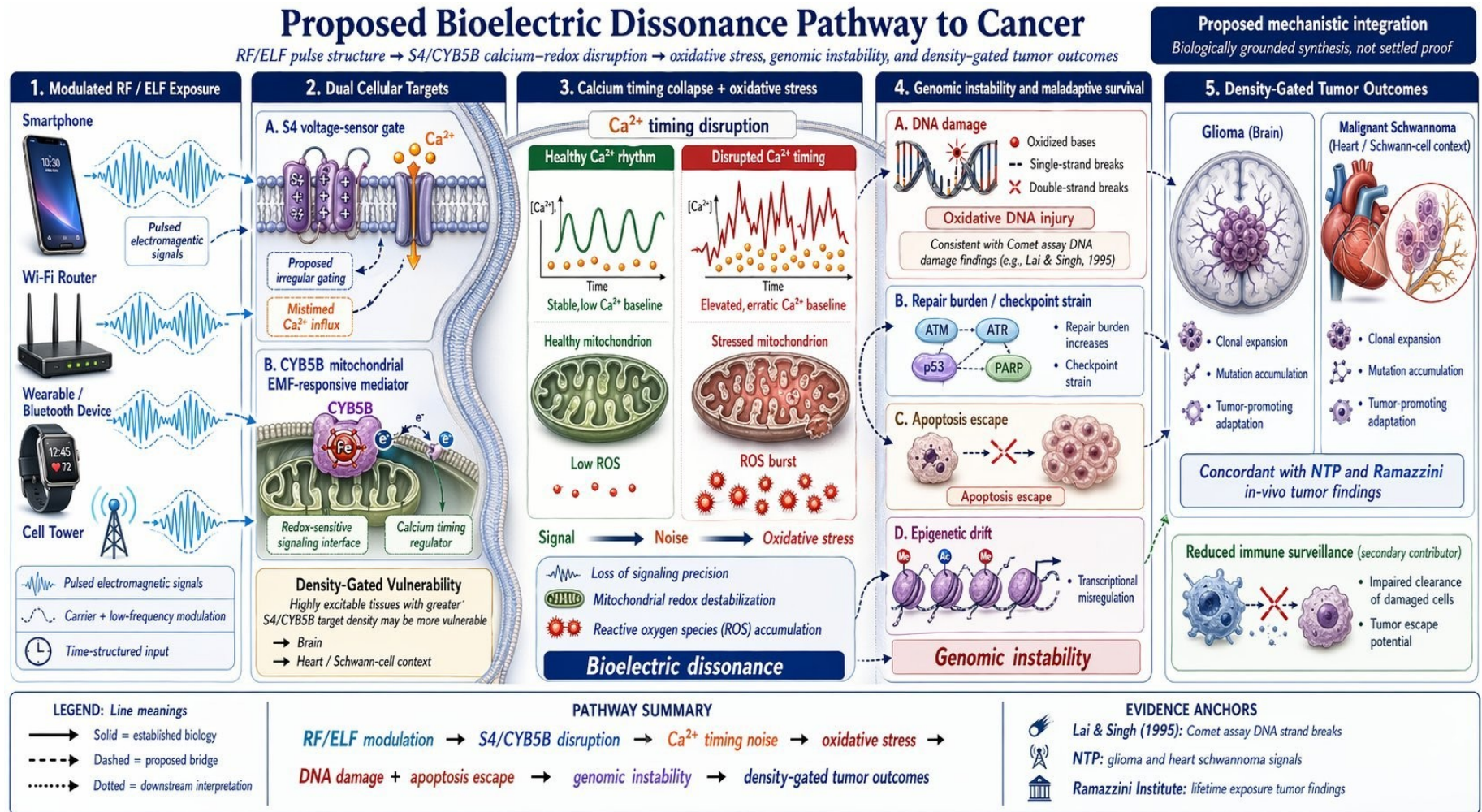
RF/ELF pulse structure → S4 voltage-sensor stress + CYB5B mitochondrial sensing → calcium timing disruption → BET/NDEL1 pathway effects → altered neurodevelopment

Proposed mechanistic integration



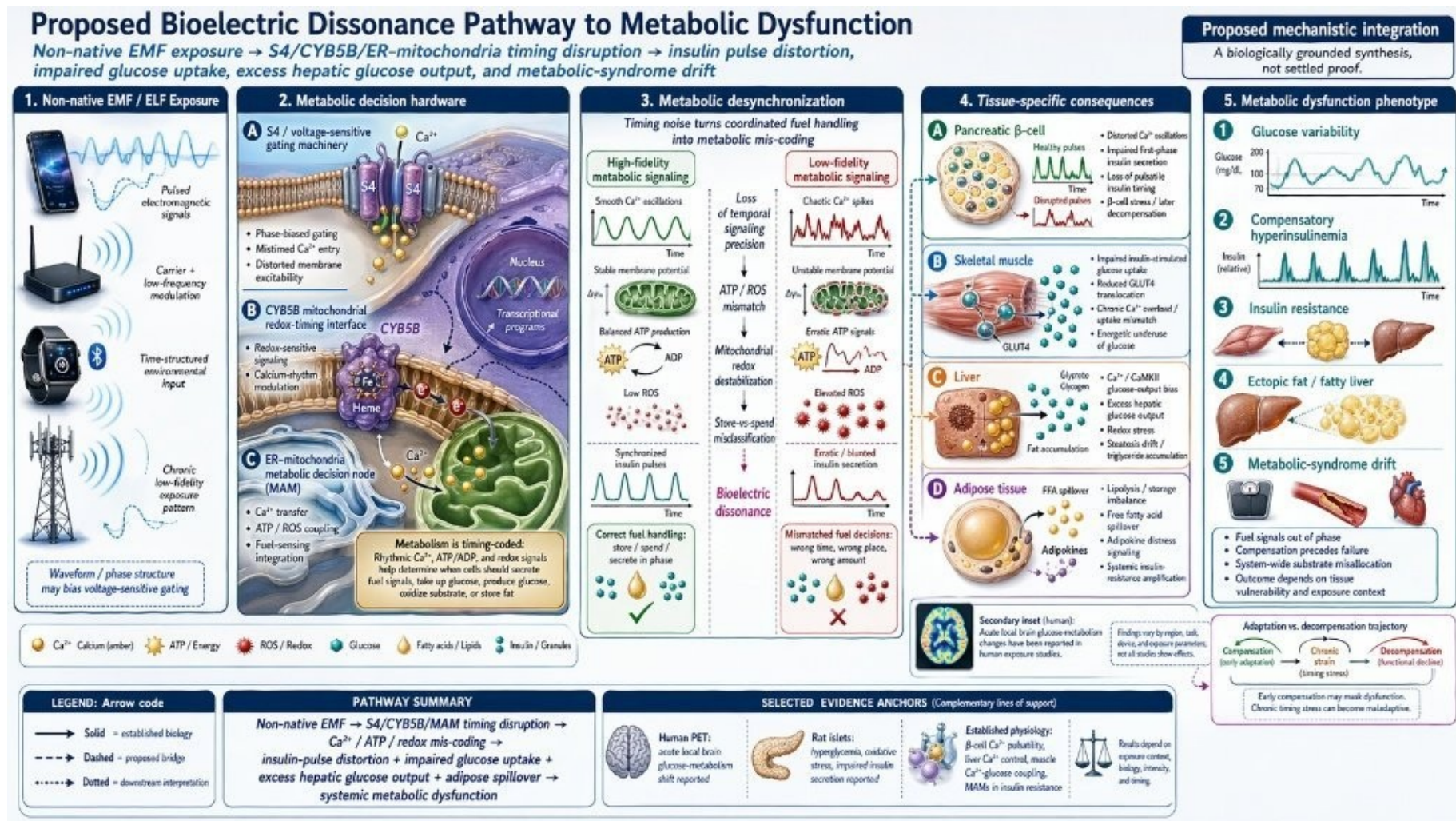
RF Safe pathway illustration, read with the developmental research program in Question 16. Citation correction: the RF cortical-organoid study is Cakir et al. (2025), and the CYB5B gene-switch study is Kim et al. (2026), including its correction; these replace the author/year labels printed in this illustration. NDEL1 is a separate developmental branch to test through exposure, time ordering, and rescue. [30] [55] [66]

B3. Cancer and the persistence of cellular errors



RF Safe conceptual pathway. Question 16 specifies the mediator and lineage experiments; Question 6 presents the animal tumor evidence. Test damage generation, repair completion, checkpoint control, apoptosis, and immune clearance separately, then connect early changes to later pathology in the same program. Molecular profiling provides a further translational comparison. ^{[1] [2] [22]}

B4. Metabolic coordination and reserve



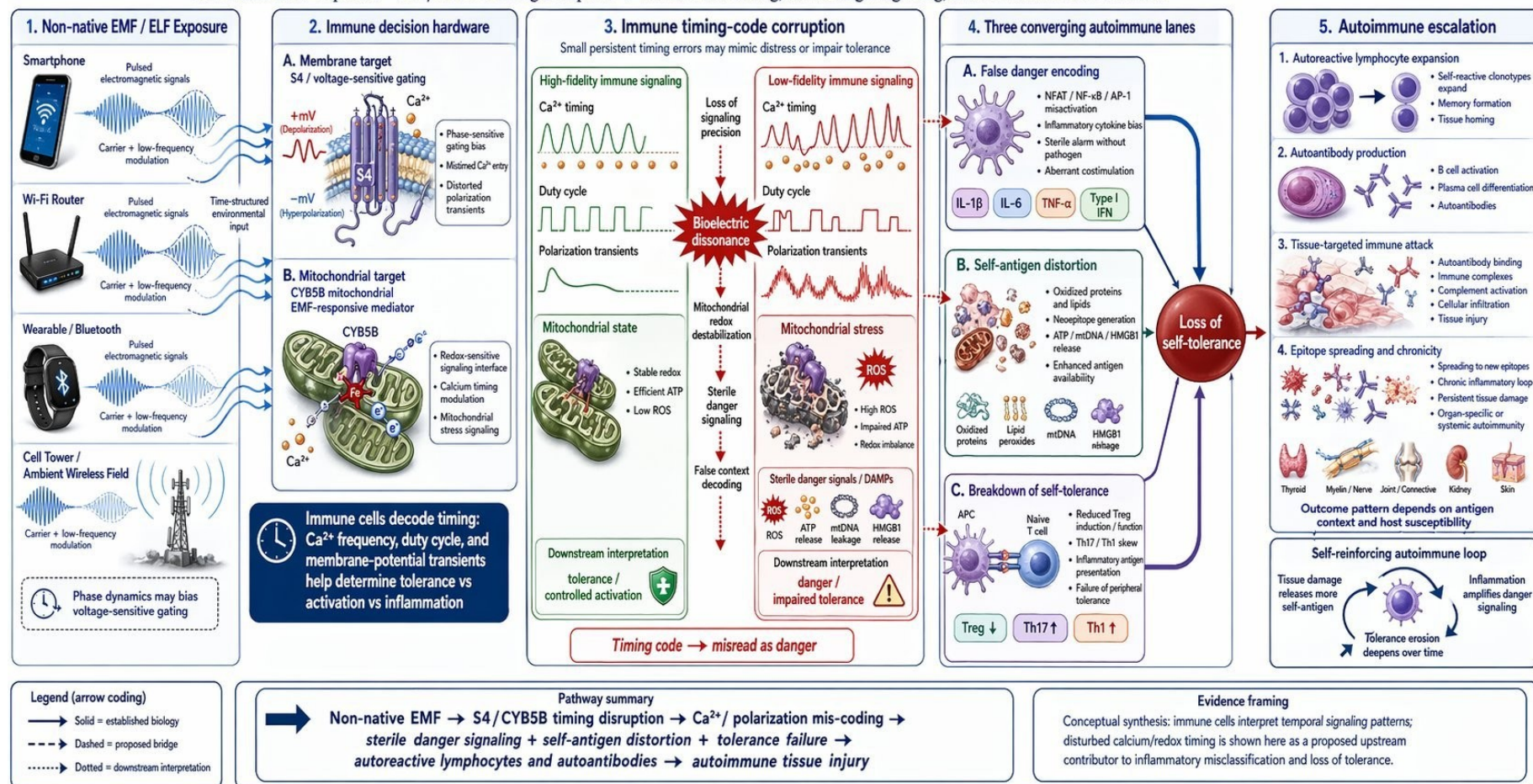
RF Safe conceptual pathway. The proposed outcome is reduced precision in matching physiological demand to fuel delivery and energy production. Measure insulin pulsatility, regional metabolism, mitochondrial reserve, challenge response, and recovery. Volkow et al. provide the regional human brain glucose-metabolism experiment cited in Question 16. ^[73]

B5. Immune discrimination and resolution

Proposed Bioelectric Dissonance Pathway to Autoimmunity

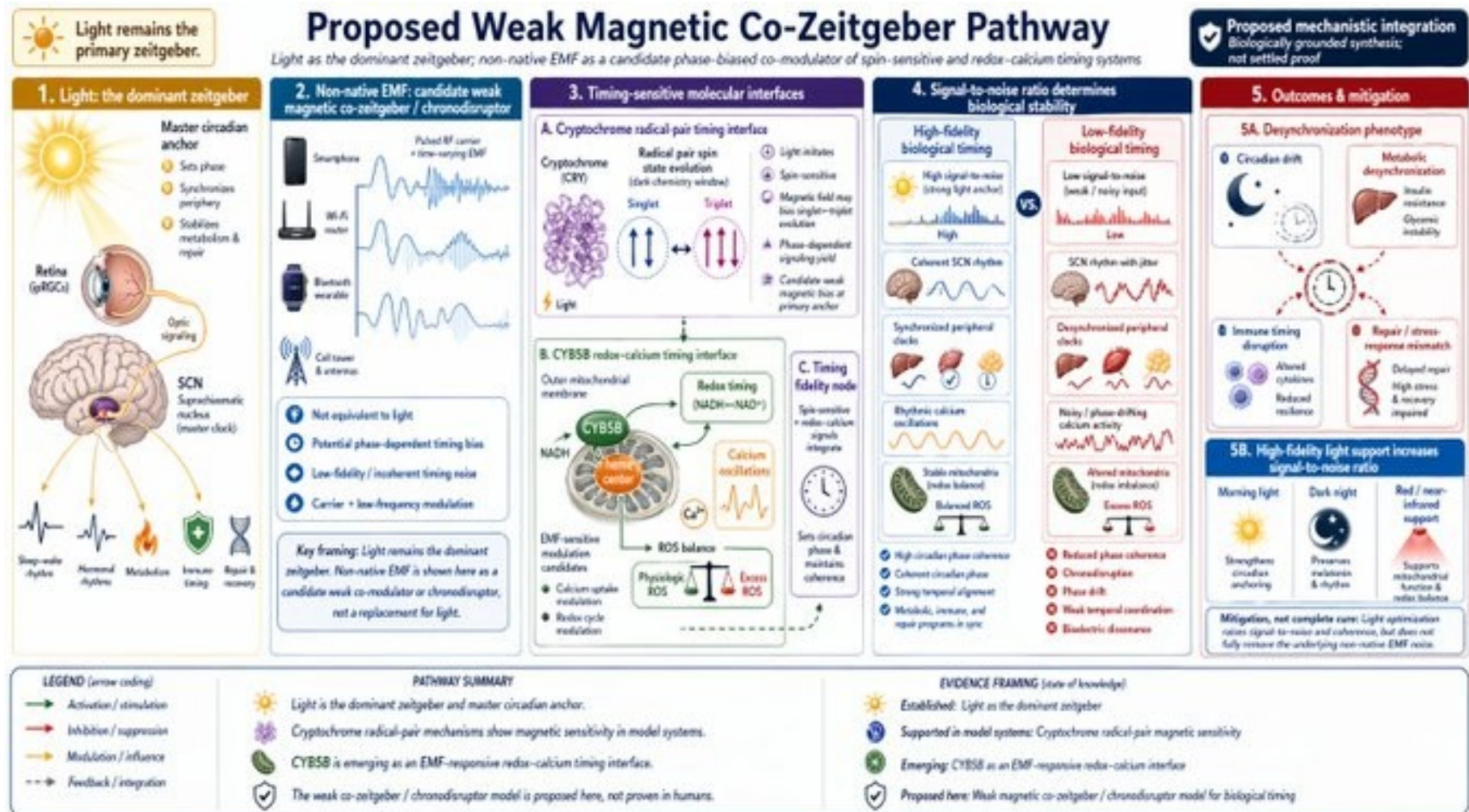
Non-native EMF exposure → S4/CYB5B timing disruption → immune mis-coding, sterile danger signaling, and breakdown of self-tolerance

Proposed mechanistic integration
Biologically grounded synthesis, not settled proof



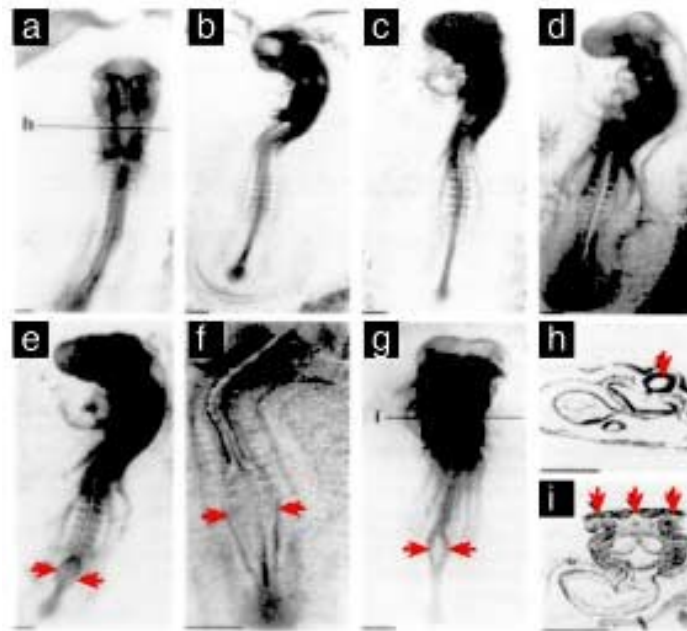
RF Safe conceptual pathway. The three branches guide separate experiments on danger signaling, antigen presentation, and tolerance. Measure activation thresholds and the resolution phase, then repeat the challenge to test retained burden. The macrophage study cited in Question 16 supplies a challenge-based design to extend across dose, waveform, and receiver state. ^[74]

B6. Additional timing cues and circadian coordination



RF Safe conceptual illustration. Test whether a characterized magnetic or RF exposure changes phase, rhythm amplitude, entrainment, or recovery under controlled light schedules. Keep the physical exposure and species-specific receiver explicit. Cryptochrome-related experimental responses and the engineered CYB5B pathway provide distinct starting points. [59] [55]

B7. Embryonic morphology and developmental endpoints



Author-supplied reference montage. The original study and exposure conditions of these panels are unverified, so no experimental comparison is assigned to the image. The developmental claims in this comment rest on the numbered studies. HHS should pair independently scored morphology with live calcium, actomyosin, tissue-mechanics, and closure measurements during defined developmental windows. ^[10] ^[65]

Appendix C. Source synthesis map

Author manuscripts and published evidence. The following RF Safe manuscripts supply the integrated framework and policy proposals. The numbered references identify the published experiments, agency records, and legal provisions supporting specific factual statements. Question 16 presents the complete proposed model and the experiments needed to test its connections.

The Embodied Cellular Transfer Function ceLLM, 50 pages. Contributes the whole-cell response function; fast state and slower organization; latent response phenotypes; geometry-addressed photonic reafference; biological lock-in; structural eligibility; fidelity debt; somatic overfitting; and experiments across genotype, phase, source position, and persistent adaptation. Integrated principally in Question 16 and susceptibility measures in Question 11.

Topology-Gated Photonic-Redox Control Plane ceLLM Hardware Matrix / RNA Revision, 22 pages. Contributes DNA and RNA organization, mitochondrial-nucleoid topology, hydration, optical/redox coupling, receiver-dependent responses, cytoskeletal routing, and topology-versus-amplification experiments. The two supplied copies contain the same text and are represented once. Integrated in Question 16.

Topology-Gated Photonic-Redox Control Plane ceLLM Manuscript Bioelectricity Integrated, 13 pages. Contributes organelle contacts, tissue-level bioelectric communication, planarian state and reversion, optical isolation, and the distinction between source, transducer, routing, amplification, and persistent update. Integrated in Question 16.

Bioelectric Fidelity in Early Human Development, 35 pages. Contributes calcium waveform decoding, the CYB5B/channel-to-chromatin research sequence, BET directionality, the parallel NDEL1 branch, neural-tube closure mechanics, developmental-window matching, cofactor experiments, and reproducibility requirements. Integrated in Questions 4, 7, 11, and 16.

RF Safe Clean Ether Flagship. Contributes the synthesis of hazard evidence, biological fidelity, regulatory accountability, engineering alternatives, and staged public implementation. Integrated across Questions 5–18, with concrete legislation in Question 18.

Proposed Comments Section: Non-Thermal, Nonlinear, and Tissue-Specific EMF Mechanisms, 15 pages. Contributes S4/VGIC and CYB5B research targets, the TheraBionic regulatory precedent, density gating, Ramazzini tumor profiling, exposure reporting, federal research duties, and the requested connection between health review and deployment policy. Integrated in Questions 5, 6, 8, 15–18.

Letter to Scott McCollough concerning RF Safe's regulatory research position. Contributes the policy requests concerning preemption, the remand record, independent mechanism research, and HHS responsibilities. These substantive requests are integrated in Questions 5 and 15–18; the correspondence is not presented as independent scientific evidence.

Clean Aether Act of 2026, legislative discussion draft, 8 pages. Contributes prospective setbacks; agency review and reporting deadlines; school and federal-building pilots; interoperability and procurement; domestic photonics; dual-use optical research; voluntary intellectual-property contributions; and proposed appropriations. Integrated in Question 18 as requested legislation.

Earlier HHS comment versions, 16 and 25 pages. Supply the numbered-question structure, personal account, evidence record, and FCC exhibits. This integrated submission preserves the corrected optical-network patent statement and the author's stated open-source history.

Seven supplied images. Reproduced in Appendix B with captions connecting them to the research program. The developmental diagram's study citations are corrected in its caption. The standalone embryo montage carries no inferred exposure comparison.