Protect Biological Fidelity

Public Comment of Katie Webb

QuantaX Technology
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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-glial 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.

Katie Webb
QuantaX Technology

References

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

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https://www.law.cornell.edu/uscode/text/21/360ii
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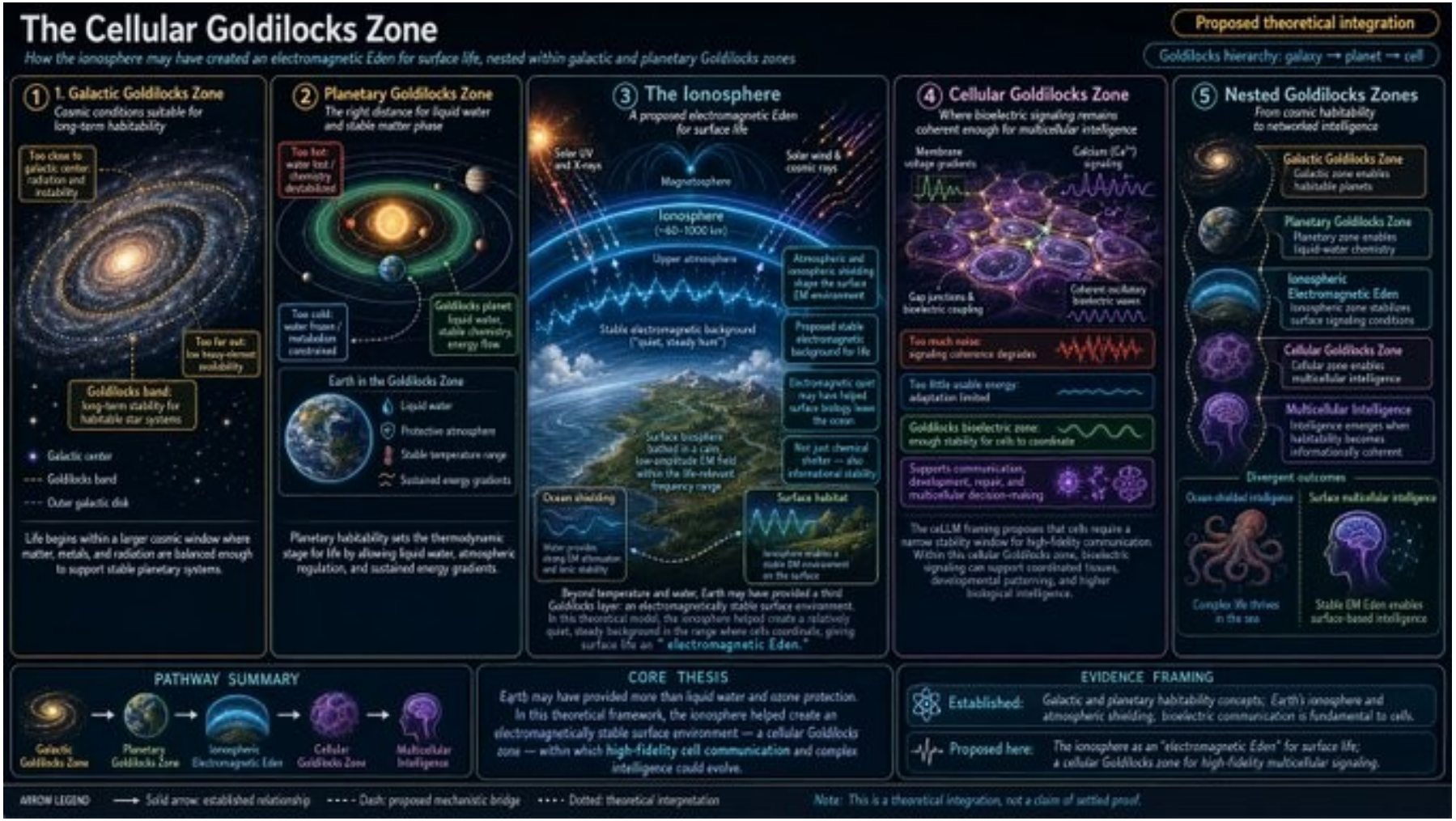
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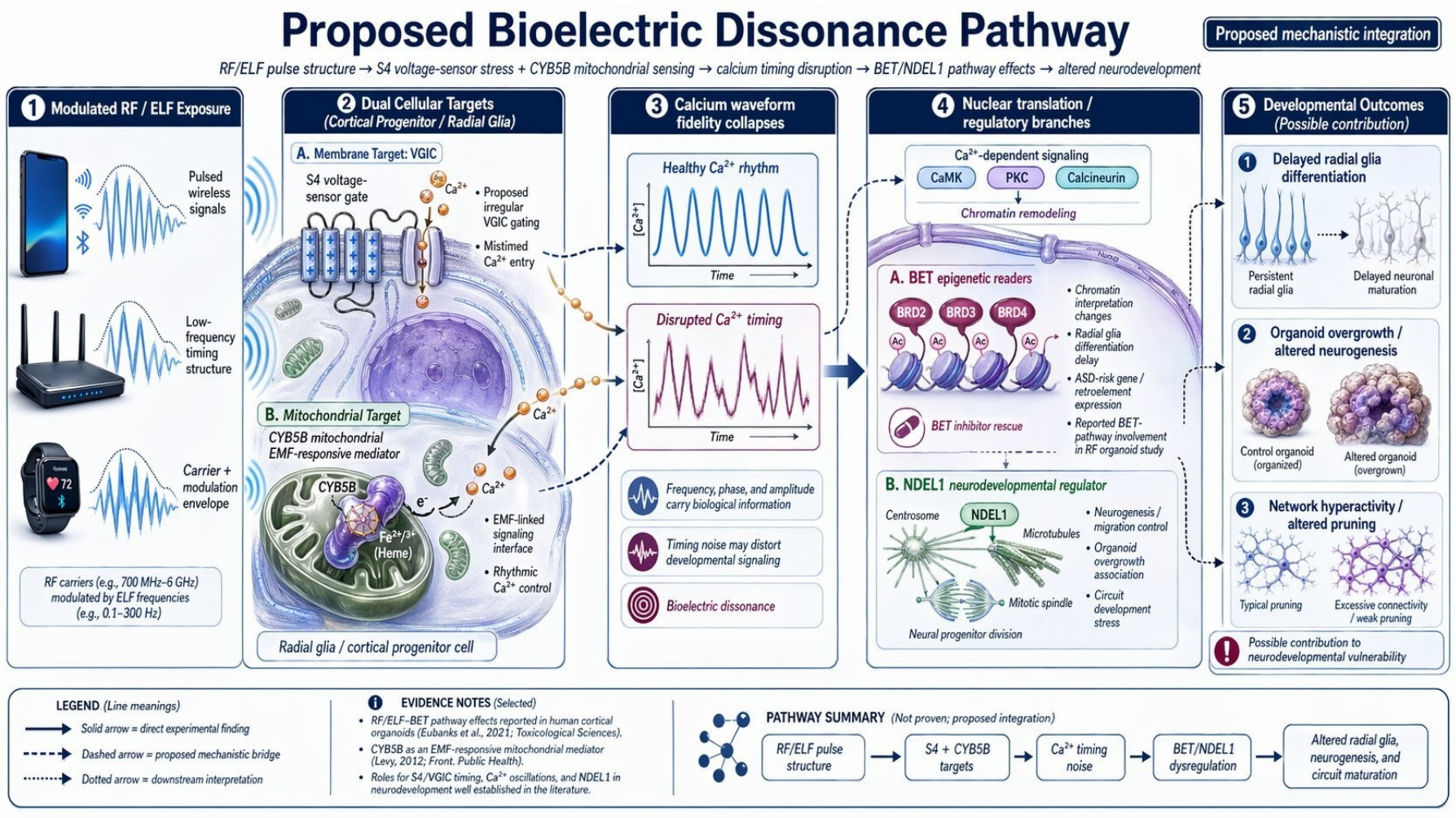
[31] RF Safe. Life Runs on Voltage. S4–Mito–Spin and density-gating framework. Read with the full mechanisms and causal experiments in Attachment B, Question 16.
https://www.rfsafe.com/life-runs-on-voltage/

Attachment A • Pathway 1: The cellular Goldilocks zone



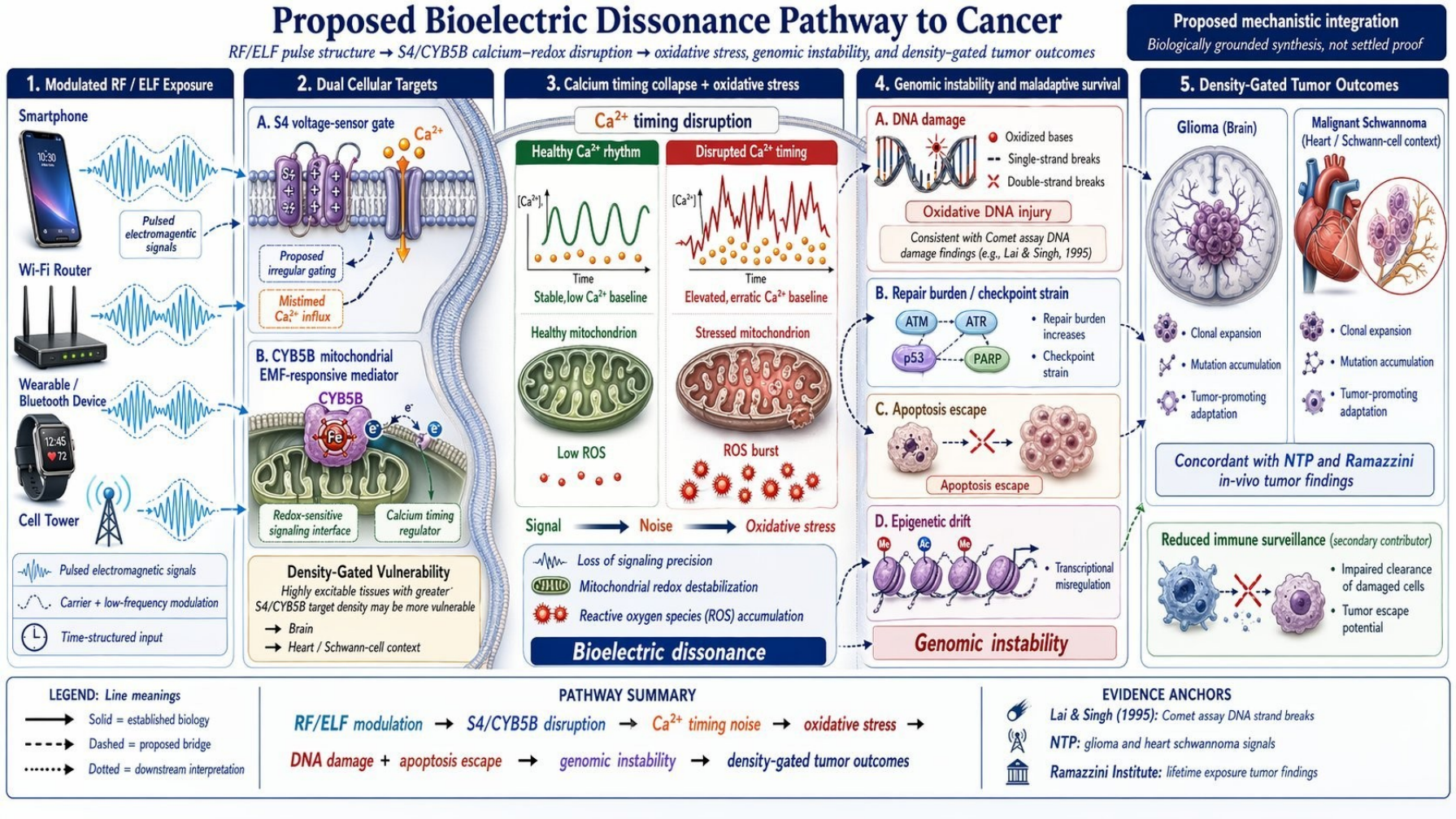
RF Safe's conceptual map connects environmental conditions, cellular signaling, and the operating range required for coordinated development. The research task is to identify which physical inputs change cellular precision, reserve, and recovery. This is the organizing illustration for the proposed biological-fidelity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

Attachment A • Pathway 2: Developmental timing and differentiation



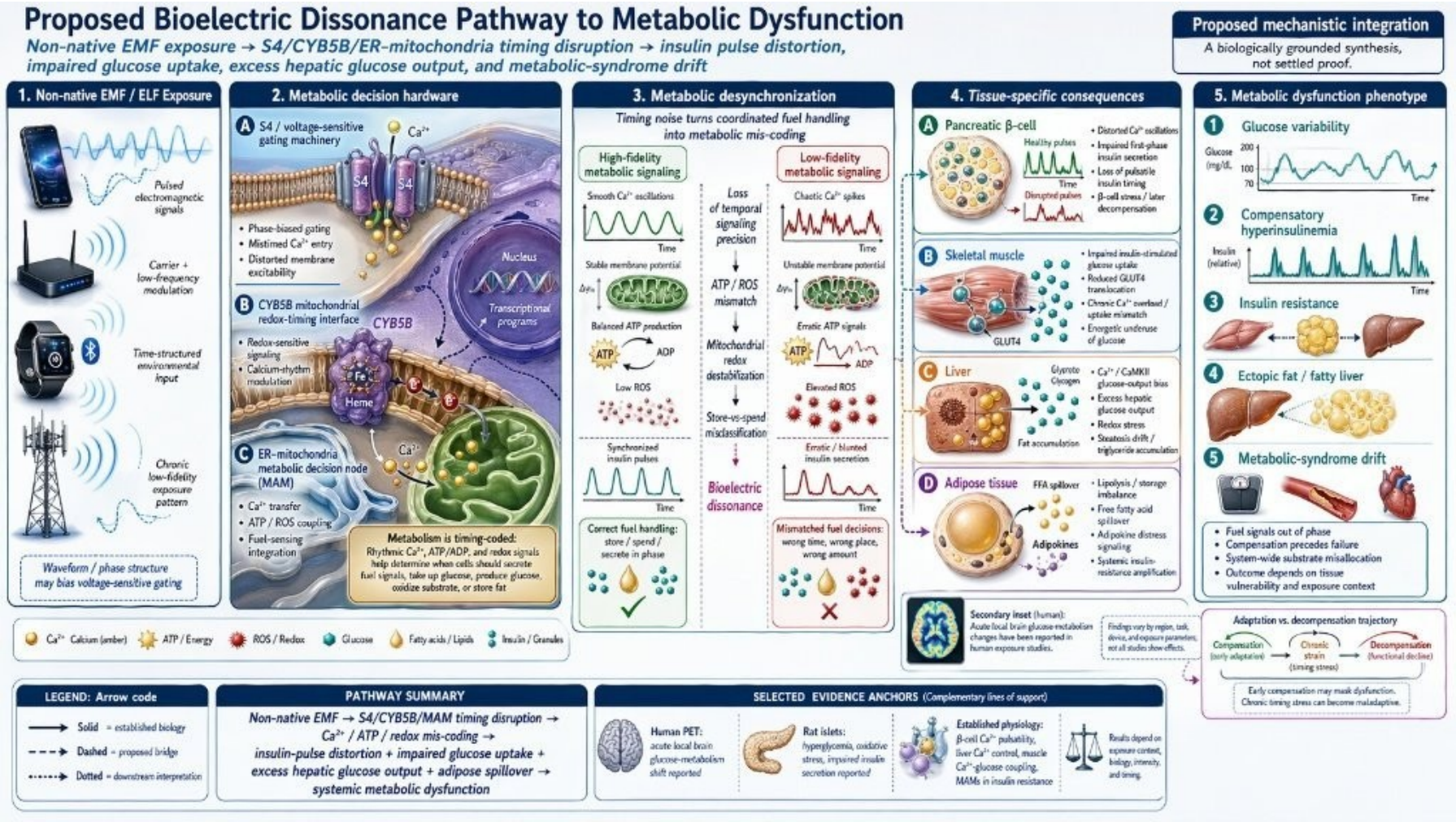
RF Safe's developmental pathway links field-responsive interfaces with calcium timing, mitochondrial and chromatin responses, and differentiation. Citation key for this plate: the cortical-organoid study is Cakir et al. (2025) [9]; the CYB5B gene-switch study is Kim et al. (2026), with its published 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



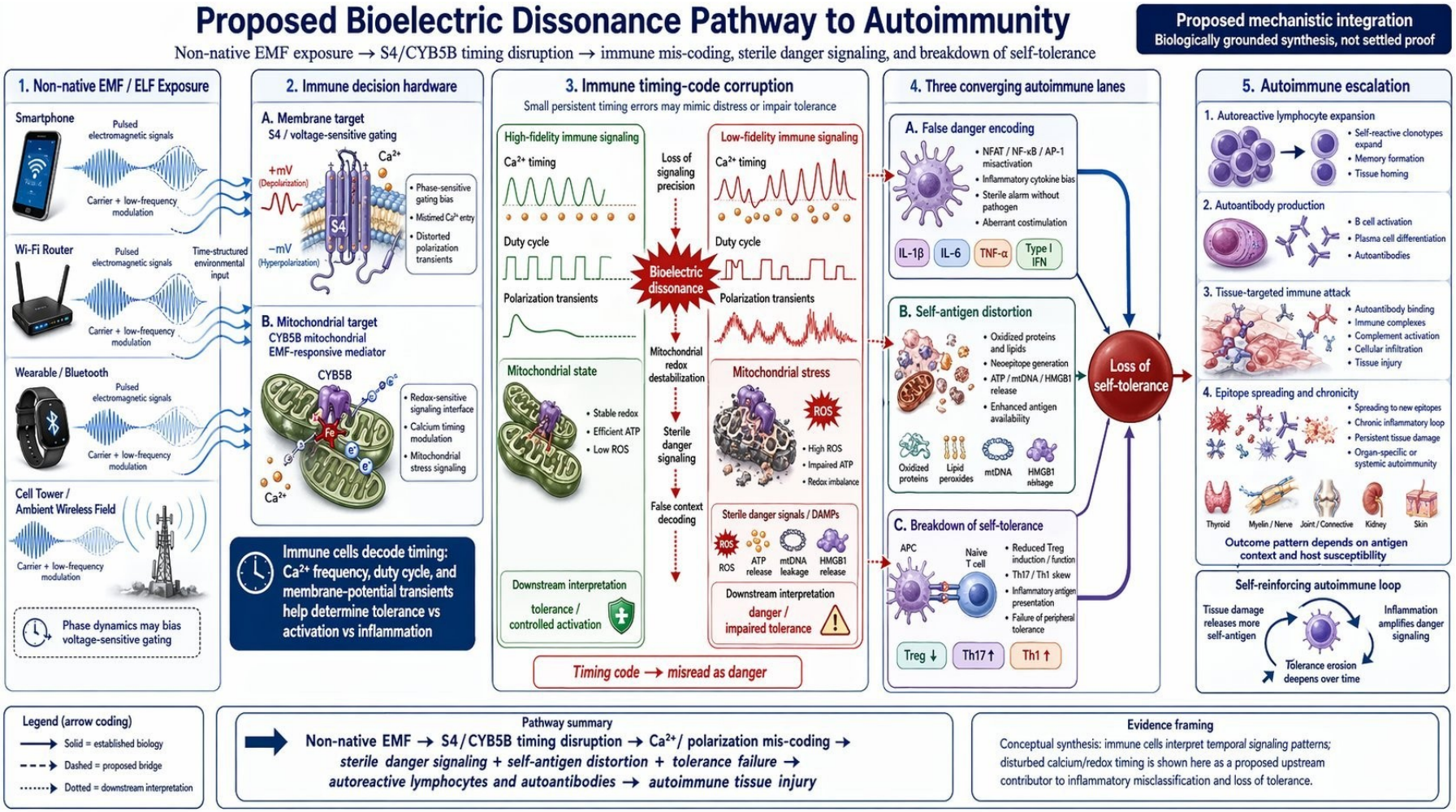
RF Safe's proposed cancer pathway connects initiating interfaces, calcium and redox responses, repair, checkpoint control, apoptosis, and epigenetic regulation. NTP, Ramazzini, and the animal-cancer systematic review supply the tumor evidence; intervention studies must connect the proposed molecular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

Attachment A • Pathway 4: Metabolic coordination and reserve



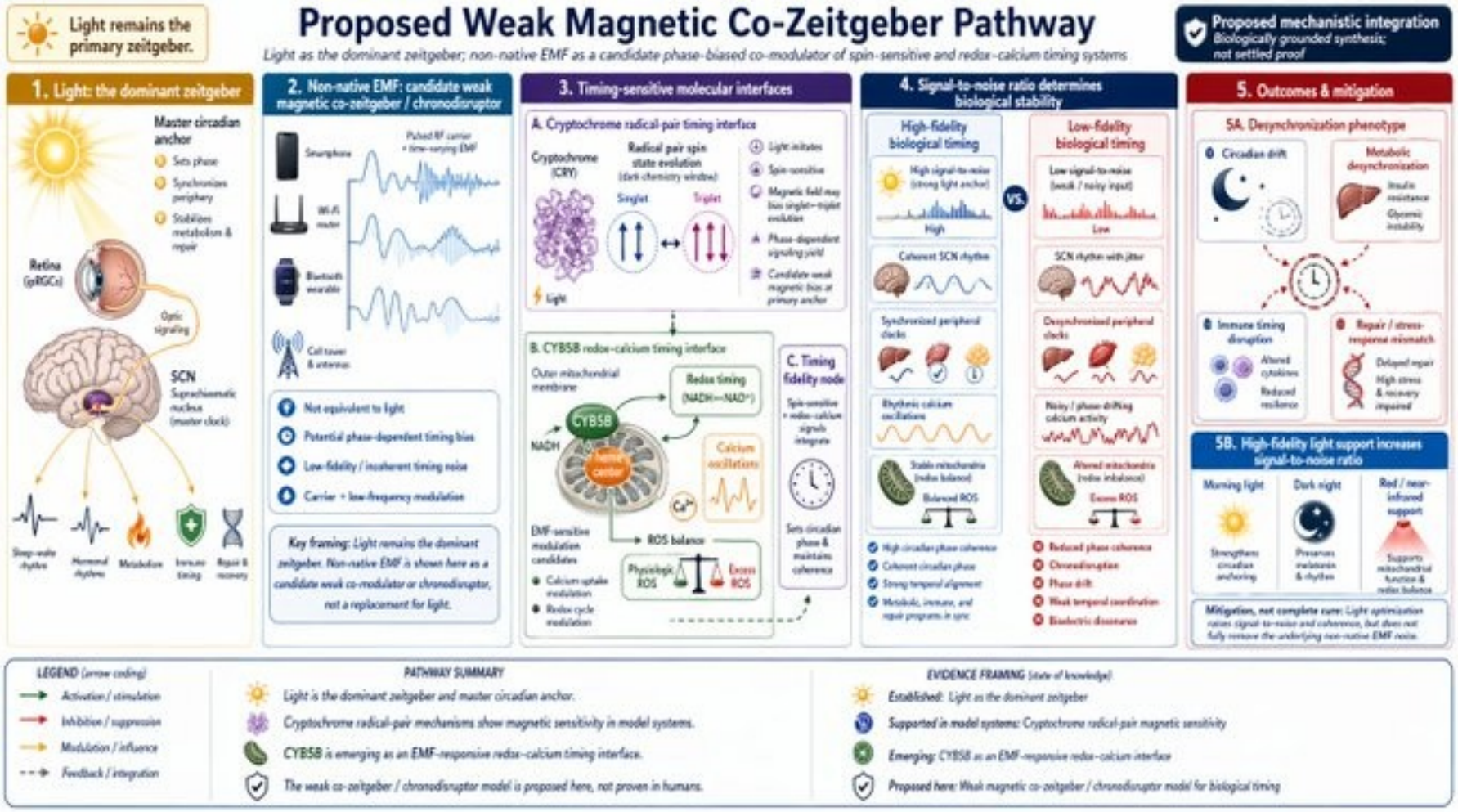
RF Safe's proposed metabolic pathway links calcium and mitochondrial responses with energy regulation across pancreas, liver, muscle, and adipose tissue. The program should measure organ-specific function, compensation, and recovery together. Yan et al.'s offspring findings provide a separate reason 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]