

Toward a Hardware-Matrix Photonic-Redox Control Plane

A testable ceLLM hypothesis linking mitochondrial DNA topology, nuclear noncoding hardware, RNA programs, ultra-weak photon emission, and cellular state inference

Hypothesis manuscript draft | Hardware Matrix and RNA Program Revision

Prepared from source materials supplied by John Coates (RF Safe) and expanded into a literature-grounded, experimentally falsifiable framework.

Prepared for scientific discussion, critique, and collaborative experimental design.

Working thesis. The strongest version of the mtDNA-biophoton idea is not a single-channel signal-capture model and not a passive signal-capture theory. The present revision defines ceLLM as a local computing-stack hypothesis: membrane and mitochondrial interfaces act as input sensors; mitochondrial and nuclear DNA topology form the hardware matrix of physical weights and biases; RNA programs function as transient executable scripts; proteins, calcium waveforms, redox state, and bioelectric rhythms form the actuation layer; and feedback from ROS, Ca^{2+} , UPE, and chromatin remodeling updates future state probabilities. The CACNA1C rs7304986 sleep-EEG result is treated as a human biological-eclipse observation: one noncoding letter in native calcium-channel regulatory architecture changed how a controlled 3.6 GHz RF-EMF input was resolved into brain-wide bioelectric rhythm.

Suggested short title: Hardware-matrix photonic-redox control plane

Abstract

Ultra-weak photon emission (UPE) from living systems is an established biophysical phenomenon arising largely from oxidative metabolism and reactive oxygen species (ROS), yet its functional role in cellular signaling remains unresolved [2,5]. Mitochondria are both major generators of UPE and central integrators of redox, calcium, membrane-potential, and transcriptional signals [2,6-8]. Here we extend the original mtDNA-resonance idea into a broader, more biologically plausible framework. Cells may implement a topology-gated photonic-redox control plane in which mitochondrial redox chemistry generates structured optical and electromagnetic fluctuations. In this model, mtDNA topology, TFAM-dependent nucleoid organization, local DNA-protein-water interfaces, and nuclear noncoding topology are treated as components of a geometry-sensitive *hardware matrix*, not as passive signal catchers.

A recently reported CYB5B-dependent EMF-responsive calcium-coding mechanism suggests that mitochondrial nucleoid-centered topology may be complemented by a surface-facing outer-mitochondrial-membrane input sensor [25]. A human 5G sleep-EEG study provides an important nuclear-genome analogue: a single intronic variant in *CACNA1C*, rs7304986, modulated whether 3.6 GHz RF-EMF exposure accelerated NREM sleep-spindle center frequency [45]. Because *CACNA1C* encodes the $\alpha 1C$ subunit of L-type voltage-gated calcium channels, this finding suggests that nuclear noncoding architecture can act as a hardware-matrix variable, shaping how native calcium-channel systems transform electromagnetic and bioelectric inputs into physiological outputs [45].

Within the cellular Latent Learning Model (ceLLM), these processes are interpreted as a multiscale state-inference architecture rather than literal transformer-like computation. The ceLLM stack is defined as follows: input sensors sample local bioelectric, redox, photonic, and electromagnetic variables; DNA topology supplies a physical latent space of weights and biases; RNA programs are transient executable scripts generated by the hardware matrix; proteins, Ca^{2+} rhythms, redox programs, and bioelectric oscillations are outputs; and feedback from ROS, Ca^{2+} , UPE, transcription, chromatin remodeling, and nucleoid compaction updates future probability landscapes.

Human mtDNA provides useful dimensional heuristics for experimental design: a 16,569-bp circular genome has a contour length of about $5.633 \mu\text{m}$, a relaxed-circle diameter of about $1.79 \mu\text{m}$, and is typically packaged into nucleoids near 100 nm, mapping to free-space guideposts near 53 THz, 167 THz, and 3 PHz, respectively [1,9-15]. These values are not asserted as classical in vivo resonant peaks inside hydrated organelles; instead, they define physically motivated search windows for testing whether geometry and topology modulate mitochondrial sensitivity to narrow-band optical or terahertz perturbations [8,14,15].

The paper reframes the central question from same-frequency resonance to cross-scale frequency conversion: optical excited-state chemistry may be converted at hydrated DNA-protein and membrane interfaces into lower-frequency electrochemical envelopes that cells can amplify, integrate, and store as state. The *CACNA1C* sleep-EEG result motivates a parallel nuclear-genome question: whether noncoding sequence, local DNA shape, chromatin topology, and RNA-program deployment function as physical weighting layers for native bioelectric hardware. We present falsifiable predictions spanning TFAM and topoisomerase perturbation, rho0 depletion and rescue, quartz-versus-glass optical isolation, narrow-band THz exposures with thermal controls and Ca^{2+} uptake blockade, cytoskeletal perturbation, MERC disruption, CYB5B perturbation, *CACNA1C* genotype-stratified RF-EEG studies, regulatory profiling, RNA-program profiling, and spectral-entropy analysis of

photon bursts.

Keywords: mitochondrial DNA; nuclear noncoding DNA; CACNA1C; RNA programs; biophotons; ultra-weak photon emission; terahertz; nucleoid topology; TFAM; ceLLM; cytoskeleton; redox signaling; CYB5B; calcium oscillations; sleep spindles; bioelectricity

Box 1. What is established, what is proposed, and what would most directly falsify the model.

What is established	What is proposed here	What would most directly falsify it
UPE exists and is tightly linked to oxidative metabolism. Mitochondria are major UPE sources and signaling hubs. mtDNA is circular, topological, TFAM-packaged, and structurally heterogeneous. MERCs, retrograde signaling, actin dynamics, and mitochondrial subnetworks are real control layers. Nuclear noncoding variants can alter regulatory binding and gene expression, and 3D genome organization shapes transcription and development [46-50].	mtDNA topology and nuclear noncoding topology are related hardware-matrix layers. The aligned atoms of DNA, local DNA shape, chromatin looping, TFAM compaction, and hydration/interfacial structure define physical weights and biases. RNA programs act as transient executable scripts generated after bioelectric queries pass through this hardware matrix. Weak optical/redox events are converted into lower-frequency electrochemical envelopes.	No shift in optical / THz sensitivity after TFAM or topology perturbation. No dependence on mtDNA abundance or organization in rho0/rescue designs. No added predictive value from structured UPE variables beyond standard redox readouts. No association between CACNA1C regulatory/topological state, RNA-program deployment, and RF-induced spindle effects after genotype, CaV1.2 modulation, and chromatin profiling.

1. Introduction

A century after Gurwitsch proposed that dividing tissues might exchange ultraviolet signals, the existence of ultra-weak photon emission from living systems is no longer the main controversy. The unresolved question is whether any biologically useful information is embedded in those emissions, and if so how cells could physically transduce or compute with such weak state variables [2-6]. UPE is now widely interpreted as a consequence of oxidative chemistry, especially excited species generated during ROS-producing metabolism. That baseline interpretation keeps the problem anchored to known chemistry rather than mysticism. At the same time, it leaves open a systems question: whether metabolic light is merely waste, or whether evolution exploited it as one state variable inside existing signaling architectures.

Mitochondria are the natural place to ask that question. They generate and respond to redox signals, shape calcium microdomains, regulate membrane potential, communicate with the nucleus, form extended subnetworks, and sit at the center of many cell-fate decisions. They are also major sources of UPE and have shown both light-responsive behavior in isolated preparations and frequency-selective responses to narrow-band terahertz irra-

diation [2,6-8]. A framework that links mitochondrial organization to weak optical signaling therefore has an empirical foothold, even if stronger historical claims about coherence or long-range photonic control remain unsettled [4,5].

The original mtDNA-geometry formulation is valuable because it treats mitochondrial DNA not only as a sequence archive but as a physical object with contour, circularity, topology, packaging, and hydration. Those properties matter. Human mtDNA is a 16,569-bp circular genome [1]. Its contour length is about 5.633 μm , its relaxed-circle diameter is about 1.79 μm , and in mammalian cells it is usually compacted into TFAM-organized nucleoids near 100 nm that vary in accessibility and activity [9-13]. The key correction made here is that these are not grounds to claim a literal classical resonator operating inside a water-rich organelle. They are grounds to ask a sharper question: can mtDNA geometry and topology change how mitochondria couple to the local optical, terahertz, redox, and electrochemical microenvironment?

The present revision adds an equally important nuclear-genome question. If mtDNA topology can be treated as a physical hardware state, then nuclear noncoding architecture should not be ignored. A randomized, double-blind, sham-controlled human 5G sleep-EEG study found that an intronic *CACNA1C* variant, rs7304986, modulated whether a controlled 3.6 GHz RF-EMF exposure accelerated NREM sleep-spindle center frequency [45]. This result is important because *CACNA1C* encodes native calcium-channel hardware and because the variant lies in the regulatory/noncoding region rather than a straightforward protein-substitution site. In ceLLM language, this is a human example of the broader principle: the same electromagnetic input does not produce a fixed biological output; the output depends on the local hardware matrix through which the input is computed.

To make the question publishable and testable, this paper broadens the scope from mtDNA alone to a pan-genomic topology-gated photonic-redox control plane. mtDNA remains central, but as one DNA-hardware layer embedded in a richer architecture that includes ETC chromophores, hydration-shell physics, cytoskeletal aromatic networks, mitochondrial reticula, organelle contact sites, nuclear noncoding topology, chromatin structure, RNA-program deployment, and slower transcriptional state persistence. The ceLLM concept is used here as a systems metaphor for cellular state inference: cells integrate fast variables through slower structural priors and executable RNA programs, rather than responding as purely local chemical switches. The aim is to define a coherent hypothesis that can be broken by experiment.

2. Empirical foundation

2.1. UPE is real; the signaling question is still open

UPE is experimentally well established. Modern reviews describe it as a low-intensity broadband emission spanning approximately ultraviolet to near-infrared wavelengths and arising from electronically excited species generated during oxidative metabolism and stress [2,5]. Claims that all UPE is coherent or laser-like remain unsupported by the strongest critical reviews [5], but the existence of the emission itself is not in doubt. A candidate signaling variable does not need to be perfectly coherent in order to be biologically sampled; it only needs to covary with internal state strongly enough that a cell can integrate it with other channels.

Mitochondria are unusually well positioned to generate such variables. Oxidative phospho-

rylation produces ROS, excited carbonyls, singlet oxygen, redox oscillations, and membrane-potential fluctuations in a physically confined geometry. Recent isolated-mitochondria experiments suggest that mitochondria can influence one another in non-chemical ways that depend on shielding and ambient-light conditions [6]. Separate photobiomodulation work shows that isolated mitochondria respond directly to 810-nm light [7]. Most strikingly, a 2026 ACS Nano study reported a frequency-selective terahertz effect in which 34.5 THz irradiation promoted mitochondrial biogenesis via calcium influx and the PGC-1 α -NRF1/2-TFAM axis, whereas a nearby tested frequency did not produce the same effect [8]. None of these findings proves photonic computation. Together, however, they justify a serious search for topology-sensitive mitochondrial coupling mechanisms.

The detected external UPE intensity is extremely low, which is precisely why the signaling question remains open [2,5]. External counts need not equal local intramitochondrial flux, because many emitted photons may be reabsorbed or scattered before they escape the cell; that possibility remains a plausible inference rather than a settled measurement. In this regime the relevant question is not whether a single photon triggers a deterministic switch, but whether structured fluctuations in burst timing, spectral mix, or entropy bias an already noisy redox/Ca²⁺ decision landscape. That is the regime in which stochastic resonance becomes physically relevant: in nonlinear threshold systems, added noise can improve detection of weak signals rather than merely degrade it [34].

2.2. mtDNA geometry and nucleoid organization provide a physically meaningful substrate

mtDNA itself is not static packaging. Super-resolution work showed that mammalian nucleoids are relatively uniform in size and often contain a single mtDNA molecule [9]. Subsequent studies showed that only a minority of nucleoids are transcriptionally or replicatively active at a given moment, and that inactivity correlates with high TFAM-to-mtDNA ratios consistent with stronger compaction [10]. More recent structural work found that single-nucleoid architecture is heterogeneous rather than monolithic [11], while mechanistic studies demonstrated sequence-specific DNA bending in TFAM-mediated packaging and transcription initiation [12]. Topology control by mitochondrial topoisomerases provides yet another layer of physical regulation [13]. In other words, the mtDNA system already behaves like a tunable structural state variable before any photonic hypothesis is imposed on it.

Hydration adds an underappreciated physical bridge. Terahertz spectroscopy studies show that the DNA hydration shell differs measurably from bulk water [14], while long-range DNA-water interaction studies indicate that hydrated DNA supports low-frequency collective modes sensitive to temperature and hydration state [15]. This does not demonstrate information-bearing resonance. It does suggest that the relevant coupling problem is not a bare DNA loop in vacuum. The physically interesting object is the DNA-protein-water interface, where excited-state chemistry, local field fluctuations, and structural dynamics coexist.

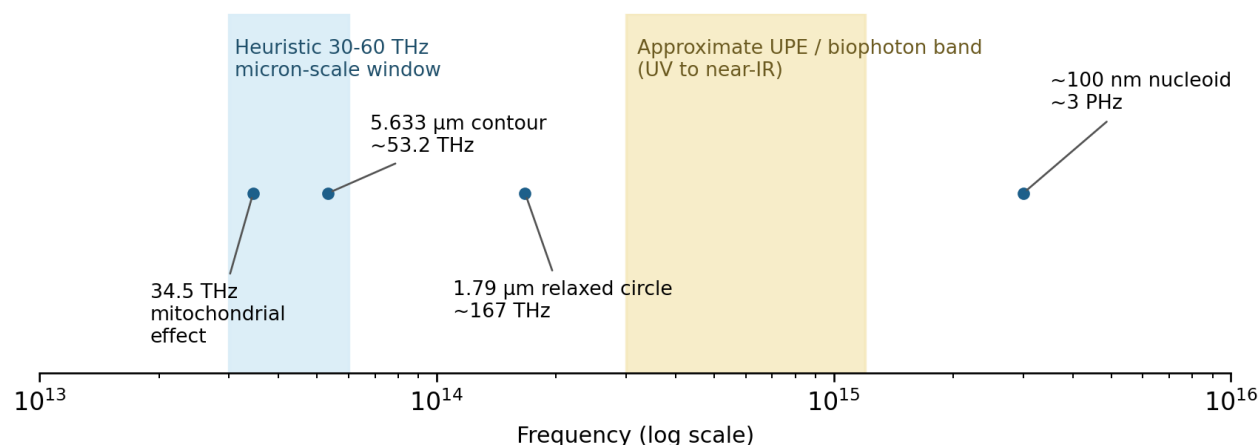


Figure 1: Multiscale frequency markers derived from human mtDNA geometry. The plotted values are dimensional guideposts only: a 5.633 μm contour maps to about 53.2 THz, a 1.79 μm relaxed-circle diameter to about 167 THz, and a 100 nm nucleoid scale to about 3 PHz. These are not asserted as classical in vivo resonant peaks.

2.3. Candidate routing and amplification layers already exist in mainstream cell biology

The intracellular context offers credible routing and amplification partners. Electronic energy migration has been measured in microtubules over a scale comparable to a tubulin dimer [16], and ultraviolet superradiance has been proposed and experimentally probed in large tryptophan-rich biological architectures including tubulin systems [17]. These findings do not prove long-range optical waveguiding inside cells, but they make aromatic cytoskeletal networks plausible short-range relays or sinks for optical excitation.

Actin appears particularly important as a state-distribution system: an interphase actin wave has been shown to promote mitochondrial content mixing, maintain polarization and oxygen consumption, and suppress excess ROS [18]. Mitochondria-ER contact sites regulate exchange, calcium coupling, redox homeostasis, and metabolic transitions [19]. Mitochondria also form sites of contact with the nucleus that help organize retrograde signaling [20]. In muscle, the mitochondrial reticulum behaves like a subcellular energy-distribution network [22], and nanotunnels may provide another route for inter-mitochondrial interaction under constrained motility [23]. Even biomolecular condensates have been shown to reshape electrochemical equilibria and membrane-associated state variables [21].

2.4. CYB5B sharpens the input-sensing layer

A timely addition to this landscape comes from a 2026 Cell study that engineered an EMF-inducible in vivo gene switch and identified cytochrome b5 type B (CYB5B) as an essential mediator, likely acting as an EMF-sensitive input-sensing interface. Activation depended on rhythmic oscillatory calcium dynamics rather than generic calcium influx [25]. This does not prove that CYB5B is the endogenous sensor for UPE or environmental EMF. It does establish that a named mitochondrial-surface protein can sit upstream of structured calcium coding and gene expression under controlled field conditions.

CYB5B is biochemically well placed for such a role. It is a tail-anchored outer-mitochondrial-

membrane hemoprotein with a heme-binding redox domain and established functions in sterol C4-demethylation/cholesterol biosynthesis and the mitochondrial amidoxime-reducing component (mARC) system; related work also links the mARC-CYB5B-CYB5R axis to nitrite/NO chemistry [26-28]. Within the present framework, CYB5B is best treated as an input-sensing and calcium-code-shaping interface that complements, rather than replaces, the DNA hardware matrix.

2.5. The biological eclipse: *CACNA1C* rs7304986 gates field-to-rhythm computation

A randomized, double-blind, sham-controlled human study of 5G RF-EMF exposure in *CACNA1C*-genotyped volunteers provides a striking nuclear-genome analogue to the hardware-matrix hypothesis. Thirty-four participants, including 15 T/C and 19 matched T/T carriers of rs7304986, underwent 30-minute pre-sleep exposure to 700 MHz, 3.6 GHz, or sham. Only T/C carriers showed a significant acceleration of NREM sleep-spindle center frequency after 3.6 GHz exposure, with effects distributed across central, parietal, and occipital cortex [45].

The relevance to ceLLM is not merely that an RF exposure altered a brain rhythm. The critical point is that the response was stratified by a single variant in the third intron of *CACNA1C*, a gene encoding the $\alpha 1C$ subunit of L-type voltage-gated calcium channels. *CACNA1C* is native bioelectric hardware: its channels regulate voltage-dependent calcium entry, neuronal firing, gene transcription, sleep EEG oscillations, learning, memory, and development [45].

Because rs7304986 is intronic, the result points toward regulatory architecture rather than a simple protein-substitution explanation. In ceLLM terms, this is consistent with the idea that noncoding DNA contributes to the physical weights and biases of cellular state inference. It helps determine how a cell or tissue deploys calcium-channel hardware and how the same electromagnetic input is resolved into a physiological output. The *CACNA1C* finding therefore extends topology-gated inference beyond mtDNA nucleoids into nuclear noncoding regulatory architecture.

2.6. Noncoding sequence as atomic hardware, not junk sequence

The *CACNA1C* rs7304986 finding clarifies that topology should not be limited to hydration shells or organelle-scale DNA packaging. A single nucleotide substitution changes the actual atomic structure of DNA at that locus: base chemistry, hydrogen-bonding context, stacking interactions, local groove geometry, electrostatic potential, and the binding grammar available to regulatory proteins. DNA-shape studies show that transcription factors read local DNA geometry, and that shape features such as minor groove width, roll, helix twist, propeller twist, and electrostatic potential contribute to regulatory binding [47-50].

Thus, the noncoding genome can be interpreted as physical hardware architecture. Its letters do not merely encode proteins; they encode local mechanical and geometric constraints that determine how regulatory complexes assemble, how chromatin folds, how RNA programs are spun up, and how bioelectric hardware is deployed. In ceLLM terms, noncoding DNA is a candidate substrate for biological weights and biases: a sequence-defined atomic lattice that shapes the probability with which a given input becomes a cellular or physiological output.

The Biological Eclipse: CACNA1C rs7304986

Same 3.6 GHz probe; different noncoding hardware matrix; different spindle rhythm

Standardized EMF probe: 3.6 GHz + 12.5 Hz amplitude modulation

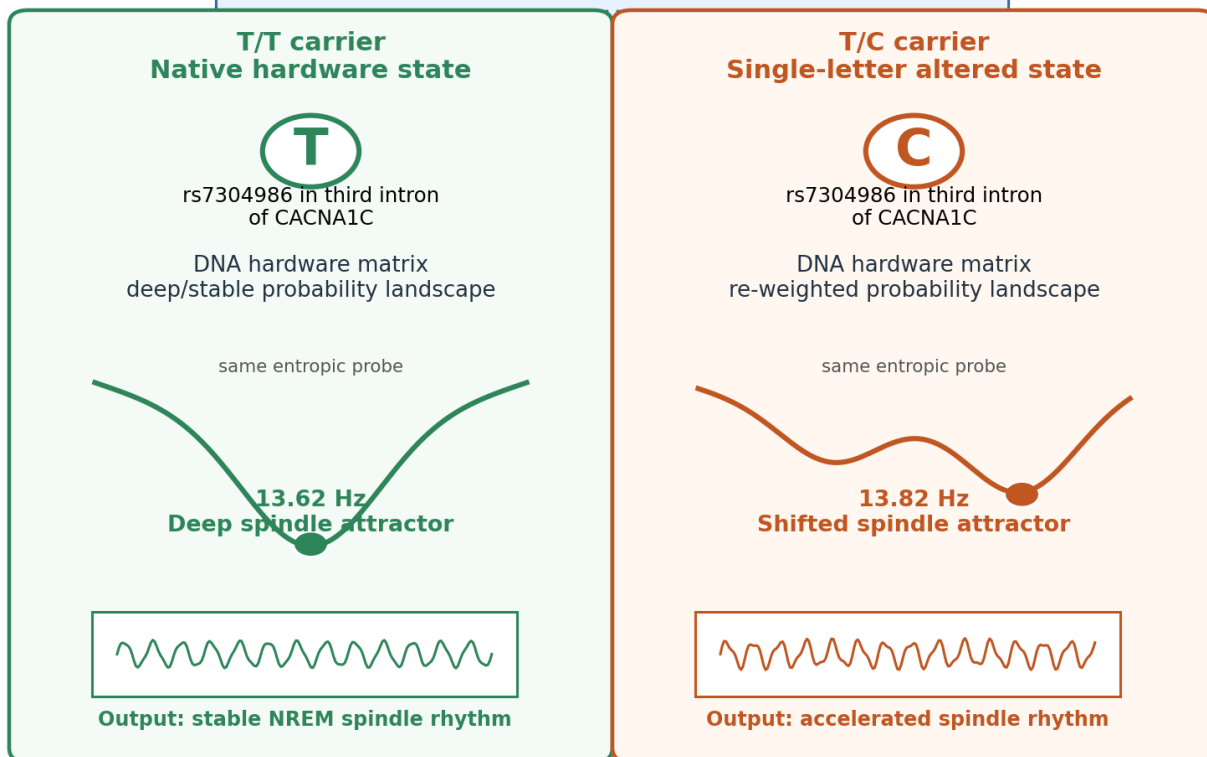


Figure 2: The biological eclipse. A controlled 3.6 GHz RF-EMF probe was resolved differently depending on the *CACNA1C* rs7304986 genomic frame. The study does not require a passive signal-capture metaphor. In ceLLM terms, the noncoding change perturbs the DNA hardware matrix, causing the same input to resolve into a different bioelectric timing output.

Core distinction. The hydration shell is not the weight matrix by itself. It is part of the coupling medium. The aligned atoms of DNA - their base chemistry, shape, electrostatic surface, stacking geometry, and regulatory folding grammar - are part of the mechanical probability architecture.

Table 1: Empirical status of major components used in the hypothesis.

Component	Status	Role in this model	Key uncertainty
UPE from living cells	Strong	Fast optical/redox state variable	Whether UPE is ever functional rather than byproduct
Mitochondria as major UPE sources	Moderate to strong	Primary emitter and integrator	Relative contribution by context and cell type
Circular mtDNA and nucleoid organization	Strong	Mitochondrial DNA hardware matrix for topology-sensitive coupling	How geometry couples in hydrated organelles
TFAM compaction affects nucleoid activity	Strong	Slow prior controlling mitochondrial hardware state	Exact coupling between compaction and field sensitivity
Nuclear noncoding regulatory architecture	Strong biology; ceLLM extension	Physical weights and biases for native bioelectric hardware and RNA-program deployment	Which loci and topologies predict field-to-physiology effects
CACNA1C intronic variant rs7304986	Emerging human evidence	Single-letter biological-eclipse example: noncoding hardware stratifies RF-to-spindle rhythm computation	Whether the causal layer is chromatin topology, expression, splicing, channel density, tissue electrical properties, or combined regulatory architecture
RNA programs	Strong biology; ceLLM reinterpretation	Transient executable scripts: mRNA, lncRNA, miRNA, and splice programs generated after bioelectric queries pass through DNA hardware	How to quantify an RNA program as an executable state variable rather than static expression list
Narrow-band THz mitochondrial effects	Moderate	Experimental foothold for frequency selectivity	Whether mtDNA is the critical mediator
Microtubule energy migration / aromatic networks	Moderate	Possible short-range routing or excitonic relay	Distance, specificity, and biological relevance in vivo

Component	Status	Role in this model	Key uncertainty
Actin-dependent mitochondrial mixing	Strong	Spatial distribution of local perturbations	Whether it carries optical-state information specifically
MERCs / NAMs / retrograde signaling	Strong	Classical amplification layer	Which hubs are necessary for any optical leg
Condensate electrochemistry	Emerging	Persistence / state-stabilization layer	Extent of generalization to mammalian mitochondrial signaling
CYB5B-mediated EMF-responsive calcium oscillations	Emerging	Input-sensing and calcium-code-shaping interface	Exact sensing physics and relevance outside engineered platform
S1-S4 voltage-sensing domains / VGIC route	Strong biology; disputed EMF coupling	Candidate plasma-membrane flood / noise entry route	Whether physiologically relevant weak-field or envelope structure perturbs gating in vivo

3. Hypothesis: from single-channel signal capture to local computing stack

The central claim of this paper is narrower, stronger, and more testable than a literal loop-resonator narrative. The claim is that mitochondrial redox chemistry generates structured optical and electromagnetic microfluctuations; that mitochondrial and nuclear DNA topology form a physical hardware matrix sensitive to those fluctuations through interfacial chemistry and regulatory architecture; that RNA programs are spun up as transient executables; and that downstream mitochondrial, nuclear, and cellular signaling networks amplify the resulting perturbations into stable state updates.

This model is easiest to understand as a stack. The source layer is conventional mitochondrial biophysics: ETC activity, ROS production, excited-state chemistry, membrane-potential oscillation, and UPE generation [2,6-8]. The input-sensing layer includes membrane voltage-gated systems, S1-S4 voltage sensors, outer-mitochondrial interfaces such as CYB5B, and organelle-contact interfaces [25,42]. The hardware matrix includes the mtDNA-nucleoid complex, nuclear noncoding topology, local DNA shape, chromatin looping, TFAM compaction, methylation/accessibility state, and DNA-protein-water interfaces [1,9-15,45-50]. The executable program layer includes RNA outputs: mRNA, lncRNA, miRNA, splice isoforms, and transient transcript sets. The actuation layer includes proteins, calcium waveforms, membrane-potential shifts, ROS programs, UPE statistics, and phenotypic outputs. The feedback/update layer includes ROS, Ca^{2+} , UPE, transcriptional state, chromatin remodeling, nucleoid compaction, and condensate state.

Within this architecture, the most plausible physics is not same-frequency resonance across all components. A more interesting possibility is cross-scale frequency conversion. Optical excited-state chemistry may be converted at hydrated DNA-protein and membrane inter-

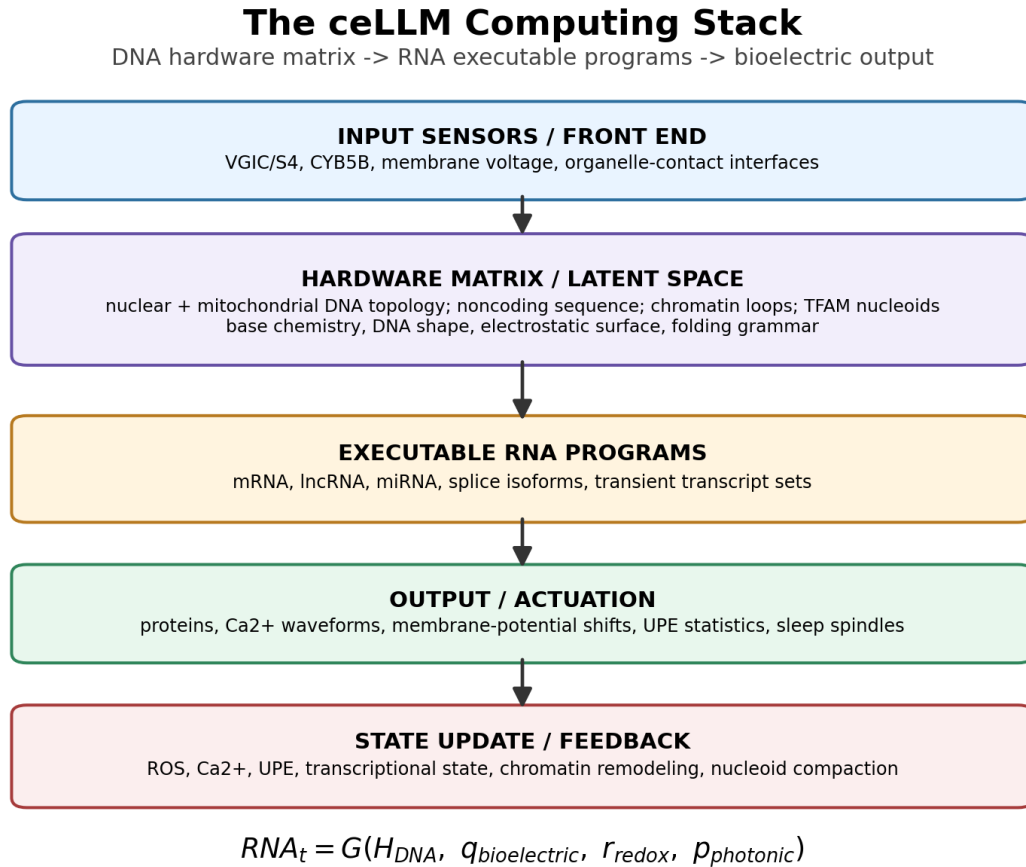


Figure 3: The ceLLM computing stack. Membrane and mitochondrial interfaces form the input-sensing layer. Nuclear and mitochondrial DNA topology form the hardware matrix of physical weights and biases. RNA programs act as transient executable scripts. Proteins, calcium waveforms, redox programs, UPE statistics, and bioelectric rhythms form the output layer. Feedback updates future probability landscapes.

faces into lower-frequency structural or electrochemical envelopes, which then propagate through calcium, redox, RNA-program, and membrane-potential networks. One plausible bridge is non-radiative relaxation: optically excited states produced by ROS chemistry can dump energy through internal conversion and vibrational energy redistribution into low-frequency collective modes and solvent-coupled phonons in the THz band [14,15,37].

The dimensional heuristics still matter. Taking human mtDNA as a circular object with contour length $5.633 \mu\text{m}$ gives a free-space guidepost near 53.2 THz. Treating the relaxed circle diameter as $1.79 \mu\text{m}$ gives a guidepost near 167 THz. Nucleoid compaction near 100 nm maps to around 3 PHz [1,9-15]. In hydrated organelles these values will shift, broaden, and damp. They are useful not as claims of in vivo resonant peaks but as experimental search windows, especially in light of the reported 34.5 THz mitochondrial effect [8]. A serious program would ask whether perturbing nucleoid compaction, topology, mtDNA abundance, or nuclear noncoding topology shifts the sensitivity of cellular readouts within or near those windows.

The ceLLM language offers a disciplined systems interpretation. Let x_t denote latent cellular state, u_t encode input variables sampled by membrane and mitochondrial sensors, r_t encode redox variables, p_t encode structured photon variables such as spectral mix and burst entropy, c_t encode calcium and membrane-potential variables, H_t encode the DNA hardware matrix, and A_t encode active RNA programs. A compact formulation is:

$$A_t = G(H_t, u_t, r_t, p_t, c_t), \quad x_{t+1} = F(x_t, A_t, r_t, p_t, c_t, H_t).$$

This is not a claim that cells implement digital software or transformer-like computation. It states that cells update state by integrating fast fluctuating inputs through slower structural constraints and transient RNA programs. The CACNA1C result is important because it provides a human example in which a one-letter difference in nuclear noncoding hardware changes the relationship between a controlled electromagnetic input and an organismal bioelectric output [45].

4. Intercellular extension and relation to circadian / photobiomodulation narratives

The model suggests a restrained intercellular extension. A pure free-space biophoton-communication thesis is physically difficult because photon counts are low and tissue environments are lossy. A hybrid network is more plausible. Local UPE may provide synchronization, directional bias, or state leakage over short distances, while gap junctions, calcium waves, direct contacts, tunneling nanotubes, mitochondrial transfer, and RNA-program exchange amplify or stabilize the effect [6,19,20,24].

This hybrid framing places the model alongside established external-light pathways rather than against them. In mammals, circadian photoentrainment is initiated primarily by retinal photoreceptors, especially melanopsin-containing intrinsically photosensitive retinal ganglion cells, while cryptochromes remain core clock flavoproteins and blue-light photoreceptors in other taxa [31,32]. Likewise, photobiomodulation literature often treats cytochrome c oxidase as a principal mitochondrial photoacceptor, or at minimum a central node, for red/near-IR responses [7,33].

The present framework operates at a different scale and with different inputs. It asks how endogenous UPE generated by mitochondrial ROS chemistry might be sampled by the nucleoid-cristae-cytoskeletal architecture inside the organelle, how nuclear noncoding

topology may shape bioelectric deployment, and how RNA programs store or actuate the resulting update. External photons and internally generated UPE need not be rival stories; they may converge on the same local computing stack.

5. Falsifiable predictions and experimental program

A credible hypothesis paper must make itself vulnerable. The present model can be falsified, narrowed, or strengthened by an experimental program that distinguishes input sensors, source chemistry, DNA hardware matrix, RNA programs, routing, amplification, and output layers.

First, topology dependence should be tested directly. TFAM overexpression, TFAM depletion, and topoisomerase perturbation should alter nucleoid compaction or topology without necessarily abolishing respiration outright [10-13]. If the model is correct, such perturbations should shift the relationship between optical or terahertz input and mitochondrial readouts such as ROS, membrane potential, calcium handling, RNA-program activation, and transcriptional state. A null result across well-controlled perturbations would weigh against the mitochondrial hardware-matrix claim.

Second, mtDNA itself should be made experimentally dispensable and then restored. rho0 or mtDNA-depleted systems, followed by rescue where feasible, can test whether the candidate coupling layer depends on mtDNA abundance or organization rather than only on membranes and canonical photoacceptors. If narrow-band or optical effects survive complete loss of mtDNA with unchanged magnitude and structure, the strongest version of the mtDNA-centered hypothesis would fail; such persistence would instead implicate alternative chromophores such as flavins, porphyrins, or cytochrome c oxidase [33,35].

Third, RNA programs should be measured as dynamic executables rather than static expression lists. Single-cell RNA-seq, long-read isoform profiling, nascent transcription assays, and noncoding RNA profiling should be synchronized with calcium, ROS, UPE, membrane potential, and topology readouts. The decisive question is whether specific RNA-program signatures predict state transitions better than individual gene counts alone.

Fourth, CYB5B perturbation, rescue, or heme-/anchor-mutant complementation should be combined with controlled low-frequency field stimulation and live-cell imaging of rhythmic calcium dynamics, reporter activation, ROS, membrane potential, MERC organization, TFAM-linked transcriptional readouts, and RNA-program activation [25-28]. If CYB5B functions as a genuine input-sensing and code-shaping interface, disrupting it should abolish patterned field-induced calcium coding more than generic ionophore-driven Ca^{2+} influx.

Fifth, the CACNA1C biological-eclipse observation should be turned into a direct mechanistic program. CACNA1C rs7304986 genotype stratification should be combined with CaV1.2 pharmacological modulation, CACNA1C chromatin accessibility mapping, contact mapping, methylation profiling, splicing analysis, CaV1.2 expression, and sleep-EEG response to controlled RF waveforms. If the hardware-matrix interpretation is correct, spindle shifts should track noncoding regulatory state and RNA-program deployment better than SAR alone.

Sixth, cytoskeletal routing candidates should be separated from sources. Microtubule and actin perturbation should be combined with simultaneous imaging of UPE, ROS, calcium, membrane potential, RNA programs, and mitochondrial morphology [16-18]. The key question is not merely whether the cytoskeleton changes emission intensity, but whether it

changes propagation, anisotropy, synchronization, or the spatial spread of downstream state changes.

Seventh, signal structure should be tested against simple intensity metrics. UPE research often reduces measurements to photon counts, but a control-plane model predicts that burst timing, spectral composition, entropy, and cross-correlation with ROS or calcium events may outperform raw counts as predictors of state transitions [2,29]. State-space models, sequence models, and graph-temporal models can determine whether structured photon variables plus DNA hardware-state variables improve prediction beyond conventional metabolic measurements.

Table 2: Decisive experiments, readouts, and what outcomes would mean.

Perturbation	Core readouts	Prediction if the model is correct	If negative, the model narrows to
TFAM up/down perturbation; topoiso-merase perturbation	UPE spectrum, ROS, membrane potential, Ca^{2+} , RNA programs, transcription, nucleoid imaging	Sensitivity to optical / THz input shifts with nucleoid topology or compaction	Weak support for mitochondrial topology-gated hardware
rho0 depletion and rescue	Same readouts plus mtDNA abundance / organization controls	Specific coupling effects weaken or disappear without mtDNA and recover on rescue	Membranes or protein chromophores instead
RNA-program profiling during controlled bioelectric input	Nascent transcription, mRNA, lncRNA, miRNA, splice isoforms, Ca^{2+} , ROS, UPE	State transitions track executable RNA-program signatures rather than individual gene counts alone	Static expression model is sufficient
CYB5B knockdown / rescue during controlled low-frequency field stimulation	Rhythmic Ca^{2+} oscillations, reporter activation, ROS, membrane potential, MERC imaging, TFAM-linked transcription, RNA programs	CYB5B perturbation abolishes patterned field-induced calcium coding more than generic ionophore responses	Field response points to other sensors or platform-specific effect
CACNA1C rs7304986 genotype stratification $\pm$ CaV1.2 modulation $\pm$ regulatory profiling	Sleep spindle center frequency, CaV1.2 expression, CACNA1C chromatin accessibility/contact mapping, methylation/splicing, RNA programs, EEG response to controlled RF waveforms	RF-induced spindle shifts track noncoding hardware state; regulatory/topological differences and RNA programs predict response better than SAR alone	Effect reflects another tissue/network variable

Perturbation	Core readouts	Prediction if the model is correct	If negative, the model narrows to
34.5 THz exposure $\pm$ mitochondrial Ca^{2+} uptake inhibitors $\pm$ TFAM/-topology perturbation	PGC-1alpha-NRF1/2-TFAM induction, biogenesis markers, UPE statistics, nucleoid imaging, Ca^{2+} microdomains	Ca^{2+} blockade abolishes downstream biogenesis while topology perturbation shifts upstream sensitivity	Purely Ca^{2+} -channel mediated
Microtubule and actin perturbation	Spatial anisotropy, correlation length, timing, mitochondrial mixing, RNA programs	Routing metrics change more than source intensity alone	Transport/mechanical explanation
Quartz-vs-glass optical isolation plus gap-junction / TNT / Cx43 blockade	Intercellular respiration, ROS, UPE, fate markers, RNA programs	Partial separability of optical and contact-mediated components	No optical leg
Sequence or state modeling of photon bursts with topology and RNA variables	Spectral mix, entropy, burst timing, cross-correlation with ROS / Ca^{2+} , RNA programs	Structured UPE variables plus DNA hardware state and RNA programs outperform raw counts or standard redox readouts	Simple redox byproduct model

6. Physical constraints and alternative explanations

Several constraints need to be stated plainly. Hydrated cytoplasm and the mitochondrial matrix are lossy, screened, thermally noisy environments. Early validation will therefore rely on isolated mitochondria, 2D cultures, organoids, and tightly controlled optical-isolation assays, because intact tissues are highly scattering and absorptive media in which photonic effects are difficult to separate cleanly from diffusion, heating, and contact-mediated signaling in vivo [38]. Terahertz fields couple strongly to water and collective vibrational modes, so any in vivo effect is more likely to involve dielectric, hydration-shell, interfacial, DNA-shape, membrane, or channel-gating physics than classical free-space resonator behavior.

Alternative explanations are real. Reported terahertz effects may act primarily through water, membranes, ion channels, or canonical chromophores such as flavins, porphyrins, and cytochrome c oxidase rather than mtDNA specifically [33,35]. Cytoskeletal perturbations may alter mitochondrial behavior through transport or mechanics rather than optical routing. UPE may remain a passive byproduct in many contexts. The coherence question remains unresolved, and the field has a history of overinterpretation [4,5]. These are reasons to design experiments that distinguish mechanisms rather than merely accumulating suggestive correlations.

The CYB5B result sharpens one longstanding objection to weak-field mitochondrial biology - the absence of a named surface mediator - but it does not settle environmental relevance. The Ei platform used controlled low-frequency EMF stimulation and identified CYB5B as an essential mediator of rhythmic calcium coding [25]. It did not demonstrate direct re-

sponses to everyday wireless carriers, nor did it resolve whether the coupling physics arises from heme spin chemistry, altered electron-transfer kinetics, membrane-potential effects, or some combination thereof. The disciplined conclusion is narrow and important: a real outer-mitochondrial-membrane hemoprotein can sit upstream of structured Ca^{2+} and gene-expression change under controlled field conditions.

The same discipline applies to S4/VGIC hypotheses. Modern wireless systems are not defined by carrier frequency alone; they also contain low-frequency temporal structure arising from burst repetition, framing, beaconing, and duty cycle. Reviews advancing an IFO/VGIC account argue that modulation structure and polarization are biologically relevant variables [43], whereas critical reviews conclude that induced currents at guideline limits are far too small and that no validated rectification mechanism has yet been shown for RF-driven Ca^{2+} gating [44]. For this manuscript, the practical implication is methodological: carrier frequency, envelope frequency, waveform structure, and duty cycle should be manipulated explicitly in experiments rather than inferred from carrier frequency alone.

7. Discussion

If the model proves partly correct, the payoff is substantial even without any appeal to exotic coherence. A demonstration that nucleoid topology modulates sensitivity to optical or terahertz perturbations would establish mtDNA organization as an active physical control variable rather than merely a genetic repository. A demonstration that structured UPE variables outperform simple intensity in predicting mitochondrial state would transform biophoton measurements from curiosity to systems-biology readout. A demonstration that RNA programs capture the executable consequences of bioelectric queries would connect the computing metaphor to measurable biology.

7.1. The Biological Eclipse: noncoding DNA as hardware logic

The CACNA1C sleep-EEG result provides a human biological eclipse for ceLLM. In the 1919 solar eclipse, starlight revealed invisible spacetime geometry by bending around the sun. In the CACNA1C study, a controlled 3.6 GHz electromagnetic probe revealed invisible genomic hardware geometry by producing a genotype-gated shift in NREM sleep-spindle frequency [45]. The same external field passed through different genomic frames and produced different bioelectric outputs.

The result is important because the variant is intronic. It is not a simple coding substitution that replaces one calcium-channel part with another. It is a noncoding regulatory difference in a gene whose product is central to voltage-dependent calcium signaling. The observation is therefore naturally interpreted as a change in hardware logic: the same external RF input is transformed differently because the genomic and bioelectric frame of the hardware matrix has changed.

This is precisely the role assigned to genomic topology in ceLLM. Noncoding DNA is not inert filler. It is part of the physical control architecture that determines how channels, redox systems, transcriptional programs, RNA executables, and oscillatory states are deployed. In this sense, the CACNA1C variant functions as a natural perturbation of the cell's probability landscape. It shows that electromagnetic input depends not only on field strength or tissue heating, but on the DNA hardware matrix through which the signal is computed.

7.2. RNA as executable programs

Classical biology often treats RNA as a transcript or messenger. That description is accurate but incomplete in a systems context. In the ceLLM stack, RNA is the executable program layer. When local bioelectric, redox, photonic, and electromagnetic variables are sampled by the input-sensing layer, the DNA hardware matrix computes a probabilistic response by spinning up specific RNA programs: mRNAs, splice isoforms, lncRNAs, miRNAs, and transient transcriptional modules. These RNA programs are temporary scripts that alter the cell's state by changing protein production, chromatin state, translation, degradation, and signaling tone.

This RNA-program layer is the missing bridge between DNA as hardware and proteins as output. The coding region builds the part, but the noncoding architecture helps determine the logic by which RNA programs are deployed. The CACNA1C result is therefore not only about a calcium-channel gene. It is about how one noncoding letter can alter the probability that a particular bioelectric state becomes a measurable organismal rhythm.

7.3. CYB5B, calcium code, and bioelectric dissonance

The newly reported CYB5B pathway changes what can now be asked plausibly. A long-standing mechanistic objection to weak-field mitochondrial biology was the absence of named hardware at the mitochondrial surface. The Ei study does not prove that endogenous CYB5B is the universal sensor of physiological or environmental electromagnetic cues, but it does show that a real outer-mitochondrial-membrane hemoprotein can sit upstream of structured calcium coding and transcriptional change under controlled field conditions [25].

One disease-relevant extension follows naturally. If CYB5B-like interfaces help write structured mitochondrial-linked Ca^{2+} codes while plasma-membrane S4-containing voltage sensors admit bulk Ca^{2+} load, then pathology may arise not from calcium elevation alone but from collapse of calcium signal quality. Patterned oscillatory coding is the signal; unpatterned influx is the noise. When noise dominates, downstream RNA programs and transcriptional state variables may read disordered electrochemical envelopes rather than informative state summaries [25,42]. The framework refers to this potential loss of pattern-to-noise ratio as bioelectric dissonance.

7.4. Morphological memory and structural priors

The multi-timescale architecture of the proposed control plane offers a physical lens for macro-scale phenomena such as morphological memory. Emmons-Bell et al. showed that transient gap-junctional blockade in *Girardia dorocephala* can induce temporary species-specific head morphologies in genetically wild-type animals [39], a result that fits Levin's broader view of bioelectric networks as reprogrammable morphogenetic circuits [40]. Within the present framework, the induced state can be interpreted as a fast, bioelectrically maintained short-term state, whereas reversion toward the native morphology could reflect reassertion of deeper, genetically canalized priors reinforced by coordinated nuclear and mitochondrial signaling.

More broadly, the framework merges literatures that often talk past one another: mitochondrial redox biology, photobiomodulation, biophoton emission, nucleoid organization,

noncoding regulatory architecture, RNA biology, cytoskeletal energy transport, organelle-contact biology, and AI-style state inference. The important discovery may not be that cells communicate by light alone. It may be that cells possess a nested photonic-redox-bioelectric computing stack in which light, redox chemistry, topology, RNA programs, and classical signaling are inseparable parts of one regulatory architecture.

8. Conclusion

This manuscript proposes that the most defensible expansion of the mtDNA-biophoton idea is a hardware-matrix photonic-redox control plane. In this framework, mitochondrial redox chemistry generates weak but structured optical and electromagnetic fluctuations; mtDNA topology, TFAM, and hydration/interfacial structure form a candidate mitochondrial hardware layer; nuclear noncoding regulatory architecture forms a parallel DNA hardware layer for native bioelectric systems; RNA programs serve as transient executable scripts; cytoskeletal and mitochondrial subnetworks distribute local perturbations; organelle contact sites amplify them into canonical signaling variables; and slower transcriptional, chromatin, nucleoid, and condensate states update future probability landscapes.

The CACNA1C rs7304986 sleep-EEG finding provides a human biological-eclipse example of the same principle. A single intronic variant in native calcium-channel regulatory architecture changed how a controlled RF-EMF input was resolved into brain-wide bioelectric rhythm [45]. That observation supports the ceLLM view that DNA is not only a code for parts. It is a physical probability architecture for biological inference.

The paper remains intentionally falsifiable. The decisive issues are whether nucleoid topology changes coupling, whether mtDNA is required, whether noncoding nuclear topology predicts field-to-bioelectric outputs, whether RNA programs capture executable state transitions, whether routing and amplification layers can be separated experimentally, and whether structured UPE variables carry more state information than simple photon counts. Answering these questions cleanly would move the discussion from metaphor to mechanism.

Final axiom.

The coding region builds the part.

The noncoding architecture helps determine the logic.

RNA programs execute the computed response.

The probabilistic logic shapes the bioelectric output.

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