Biophoton-Mitochondrial Resonance within a ceLLM Framework

mtDNA as a candidate electromagnetic transducer: a testable hypothesis manuscript

John Coates
RF Safe

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Abstract

Ultra-weak photon emission (UPE) from living systems is experimentally established, arises largely from oxidative metabolism and reactive oxygen species, and has long been discussed as a candidate non-chemical signaling channel [6-10,14]. Mammalian mitochondrial DNA (mtDNA) is a circular 16,569-bp genome packaged with TFAM into ~100 nm nucleoids whose topology influences transcription and replication [1-5]. Here these literatures are synthesized with the ceLLM (cellular Latent Learning Model) framework, which treats the cell as a probabilistic state-integrating system. The central hypothesis is that mtDNA does not merely encode genes but also behaves as a multiscale electromagnetic transducer: its contour-scale geometry, topological state, and nucleoid-scale compaction may endow mitochondria with frequency-selective sensitivity to local photonic or electromagnetic fields generated by redox chemistry. This article does not claim that classical loop-antenna equations apply literally inside hydrated mitochondria. Instead, free-space resonance estimates are used as heuristics to motivate a concrete physical question: do mtDNA geometry and topology alter how mitochondria respond to ultra-weak optical or terahertz inputs? In this framing, biophotons are not assumed to be proven coherent control signals; rather, they are candidate state variables that could be sampled by mitochondria and folded into cellular decision-making. The article corrects common geometry oversimplifications, explains how the framework reinterprets Gurwitsch's onion-root observations, identifies major physical and evidentiary limitations, and proposes decisive experiments involving mtDNA depletion, TFAM/topology perturbation, quartz-versus-glass optical isolation, and frequency-matched THz exposures.

Keywords: mitochondrial DNA; mitochondria; ultra-weak photon emission; biophotons; terahertz; TFAM; reactive oxygen species; nucleoid topology; ceLLM; mitogenetic radiation

Central claim. The strongest publishable version of this model is that mtDNA should be treated as a candidate multiscale electromagnetic transducer whose geometry and topology may modulate mitochondrial responses to endogenous UPE and carefully controlled optical/THz inputs.

1. Introduction

Ultra-weak photon emission (UPE) from living systems has moved from fringe observation to a reproducible, though still mechanistically contested, biophysical phenomenon. Modern reviews agree that spontaneous emission occurs across bacteria, plants, animals, and human tissues, and that oxidative metabolism or oxidative stress generate the relevant electronically excited species [8-10,14]. At the same time, mitochondria are no longer viewed solely as ATP factories but as signal-integrating organelles whose ROS, metabolites, and calcium handling shape transcription, differentiation, inflammation, and stress adaptation [6,9]. What remains largely unexplored is whether the physical organization of mtDNA contributes to this signaling in a way that is not reducible to sequence alone.

This article advances a testable hypothesis: mtDNA acts as a multiscale electromagnetic transducer embedded within the mitochondrion. The word transducer is used deliberately. The claim is not that mtDNA has already been shown to behave as a classical antenna in vivo, nor that conventional free-space antenna equations apply literally inside a hydrated ionic organelle. Rather, the claim is that the circularity, topology, protein packaging, and compaction state of mtDNA may shape how mitochondria couple to locally generated optical or terahertz microfields and convert those couplings into biochemical state changes.

The hypothesis is organized using the ceLLM (cellular Latent Learning Model) framework. In ceLLM, the cell is treated as a probabilistic state-integrating system: topology, electrostatics, macromolecular geometry, and biochemical context collectively define a latent state space over which the cell updates fate-relevant probabilities. ceLLM is used here as a conceptual language, not as a claim that cells instantiate literal large-language-model machinery. Within this framing, UPE and local electromagnetic structure are candidate inputs into cellular state estimation rather than deterministic switches.

2. Boundary conditions from established biology

Any publication-facing version of the mtDNA-resonance idea has to begin by separating what is already well supported from what remains inferential. The strongest manuscript is therefore one that is explicit about evidence tiers instead of blurring them.

2.1 mtDNA is a dynamic physical structure, not only a gene list

Human mtDNA is a double-stranded circular genome of 16,569 bp [1]. Using the standard B-form rise of 0.34 nm per base pair, its contour length is approximately 5.633 μm. In vivo, however, mtDNA is not a naked ring of that size. It is packaged with TFAM and associated proteins into nucleoids, which in mammals are commonly ~100 nm and often contain a single mtDNA copy [2,3,5].

Topological control is biologically consequential. TFAM loading, topoisomerases, and packing density influence mtDNA transcription, replication, and nucleoid architecture [3-5].

The relevant geometry is therefore inherently multiscale: sequence scale, contour scale, topological scale, nucleoid scale, and whole-mitochondrion scale. That multiscale organization matters because a resonant or geometry-dependent interaction need not live at only one scale.

2.2 Mitochondria, ROS, and ultra-weak photon emission

UPE from living samples is now well documented. Modern reviews place the emission broadly in the UV-to-near-IR range, approximately 300-900 nm depending on system and instrument [8-10]. The underlying excited states arise from oxidative metabolism and oxidative stress, with ROS/RNS chemistry serving as major sources [6-10].

Mitochondria are especially relevant because the electron transport chain is both a major source of cellular ROS and an organellar locus long associated with spontaneous photon emission [6-10]. Crucially, the existence of UPE is on firmer ground than claims about its information content. The best current critical review concludes that UPE itself is experimentally well established, whereas reliable evidence for coherence or nonclassical photon statistics remains lacking [14].

2.3 Gurwitsch and the problem of non-chemical signaling

Gurwitsch's 1923 onion-root experiments remain historically important because they posed a stark question: can cells influence other cells at a distance without chemical contact? Historical reconstructions and later reviews agree on the basic observation pattern - a directional effect on mitosis, transmission through quartz, and loss of effect through ordinary glass - while also emphasizing that the biological effect has proven difficult to reproduce consistently [11,12].

The most defensible modern reading is not that Gurwitsch proved a full biophoton communication theory, but that he opened a still-unsettled problem in non-chemical biological signaling. That problem is precisely where a careful mtDNA-transducer hypothesis can add value: not by claiming victory over the historical controversy, but by proposing sharper mechanistic tests.

2.4 Terahertz bioeffects are relevant but do not remove the caveats

Interest in terahertz interactions with biology has increased because THz fields address collective vibrational, rotational, and hydration-coupled modes of biomolecules. Reviews report both thermal and putatively nonthermal cellular effects, but they also emphasize the need for rigorous dosimetry, thermal control, and attention to the strong role of water [16,17,20].

Recent work has intensified the relevance to the present hypothesis. Wu et al. reported that 34.5 THz, but not 36.1 THz, enhanced mitochondrial biogenesis through $Ca2+$ influx and the PGC-1alpha-NRF1/2-TFAM pathway [18]. This does not prove that mtDNA itself is the

receiving structure, but it does show that narrow spectral changes in the THz regime can map onto mitochondrial outcomes.

Table 1. Evidence map for publication-facing claims

Proposition	Status	Publication-safe wording
Living systems emit UPE.	**Established**	UPE is reproducibly observed across diverse biological systems.
Mitochondrial redox chemistry contributes materially to UPE.	**Supported**	Mitochondria are major contributors to UPE via ROS-linked processes.
Human mtDNA is circular and packaged into ~100 nm nucleoids; topology affects gene expression.	**Established**	mtDNA is a dynamic topological object, not merely a sequence string.
Specific THz exposures can produce frequency-selective mitochondrial effects under controlled conditions.	**Emerging**	Some narrow-band THz exposures map onto mitochondrial readouts, but mechanism is unresolved.
Biological UPE is coherent or laser-like.	**Unresolved**	Coherence remains controversial and should not be treated as established.
mtDNA geometry functions as a biologically meaningful electromagnetic transducer.	**Hypothesis**	This manuscript proposes direct testing of mtDNA as a candidate transducer.
Ambient RF exposure directly detunes mtDNA resonance and drives disease.	**Unproven**	Any exogenous RF-to-mtDNA link remains indirect and experimentally unsettled.

3. Engineering analysis: the scale argument is useful only if it is stated honestly

An engineering treatment of mtDNA should begin by separating length scales rather than conflating them. The common rhetorical error is to treat the 5.633 μm contour length, a hypothetical relaxed-circle diameter, the mitochondrial diameter, and the packed nucleoid diameter as if they were interchangeable. They are not. Each corresponds to a different physical scale and therefore a different heuristic frequency estimate.

The following free-space estimates are intentionally simple. They are not claimed to be measured in vivo eigenmodes. They are first-order scale correspondences intended to

motivate experiments, not to substitute for a full electrodynamic model of a hydrated, protein-loaded, ionic organelle.

Contour length: $C = 16{,}569 \times 0.34\ \text{nm} = 5.633\ \mu\text{m}$
Relaxed-circle diameter: $d = C / \pi \approx 1.79\ \mu\text{m}$
Heuristic mapping: $f \approx c / \lambda$
If $\lambda \approx 5.633\ \mu\text{m}$, then $f \approx 53.2\ \text{THz}$
If $\lambda \approx 1.79\ \mu\text{m}$, then $f \approx 167.2\ \text{THz}$
If $\lambda \approx 100\ \text{nm}$, then $f \approx 3.00\ \text{PHz}$

The contour-scale estimate places the mtDNA polymer in the far-infrared/THz regime. The nucleoid-scale estimate pushes the associated length scale toward the UV region, which is where many UPE measurements begin. This multiscale mapping suggests a bridge rather than a contradiction: different structural levels of the mtDNA-nucleoid-mitochondrion system could, in principle, interact with different spectral windows.

The classical loop-antenna analogy cannot, however, be taken literally. A hydrated mitochondrion is not free space, mtDNA is not bare metal, and the local field is shaped by ionic screening, protein loading, membrane interfaces, conformational disorder, and strong damping. Classical antenna equations therefore overstate precision. The analogy is strongest as a dimensional-analysis tool and weakest as a literal electrodynamic description.

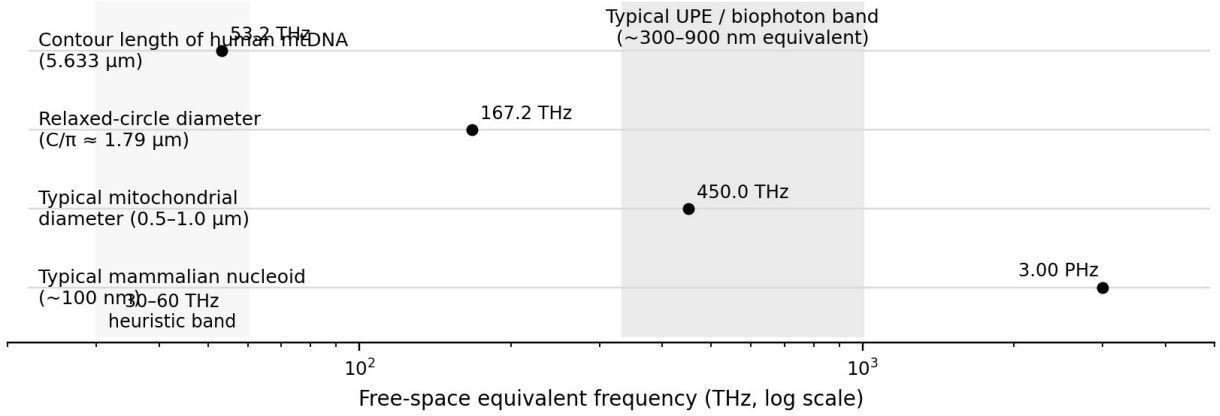

Figure 1. Multi-scale heuristic mapping from characteristic mtDNA / mitochondrial dimensions to free-space equivalent frequencies. These are scale estimates only, not measured in vivo resonance modes.

4. The biophoton-ceLLM resonance hypothesis

The model proposed here has four linked layers. First, endogenous source: electron-transport-chain activity and ROS chemistry generate excited states and UPE in the immediate neighborhood of mtDNA-containing nucleoids [6-10]. Second, geometry-

dependent coupling: circularity, topology, TFAM compaction, and nucleoid architecture modulate local electromagnetic or vibronic coupling. Third, biochemical readout: such coupling perturbs mitochondrial state variables, including membrane potential, Ca^{2+} flux, ROS balance, and mtDNA transcription/replication. Fourth, ceLLM integration: the cell folds those perturbed state variables into probabilistic fate updates.

In this framing, ceLLM does not require coherent, laser-like control signals. Even weak, noisy, or partly incoherent photonic flux can matter if it shifts state distributions in a biased way. The key claim is probabilistic weighting, not binary command-and-control. That distinction is important because it prevents the hypothesis from depending on the strongest and least established version of Popp's coherence claims [13,14].

The biological novelty of the model is therefore not the bare statement that mitochondria emit photons. That is already in the literature [7-10]. The novelty is the proposal that mtDNA topology and packaging are not merely passive substrates located near the source, but candidate structures that help convert local electromagnetic microstructure into mitochondrial state changes. If correct, mtDNA would be both an information-bearing genome and a geometry-bearing sensor interface.

5. Re-reading Gurwitsch's onion-root experiment through this model

Under the present hypothesis, the emitter root tip in Gurwitsch's setup is a metabolically active UPE source. The receiver root experiences an anisotropic UV/visible microfield. Quartz transmits a relevant portion of that field; ordinary glass attenuates it [11,12]. The receiver does not need a dedicated classical photoreceptor at the cell surface for the phenomenon to occur. A mitochondrial-network response inside the receiver meristem could bias local ROS, Ca^{2+}, and transcriptional state on the facing side, increasing the probability of mitosis and elongation there.

This interpretation is intentionally modest. It does not say that Gurwitsch proved mtDNA resonance. It says that the historical emitter-receiver geometry, the quartz-versus-glass contrast, and the side-specific biological response are all consistent with an optical-transduction mechanism that could involve mitochondria. The value of this reinterpretation is that it yields sharper experiments than the original controversy ever had.

The mechanism also does not require plant and human mtDNA to be identical in size or packaging. The shared idea is more abstract: mitochondrial nucleoids are topology-bearing, redox-adjacent structures that may participate in optical-state conversion. If that is false, the relevant experiments should fail cleanly.

6. Decisive experiments and falsifiable predictions

The hypothesis becomes useful only if it can be killed. The following experiments are not confirmatory ornament; they are decision points. Positive findings would not prove the entire ceLLM framework, but they would test the specific claim that mtDNA geometry and topology modulate mitochondrial response to ultra-weak optical or THz perturbation.

Table 2. Experiments that would meaningfully strengthen or weaken the model

Experiment	Prediction if the model is right	Result that weakens the model
mtDNA depletion / rho0 cells with rescue controls	Spectral sensitivity, cross-cuvette signaling, or patterned-light response collapses or shifts when mtDNA is removed, then returns on rescue.	No mtDNA-dependent change beyond generic metabolic failure or gross viability loss.
TFAM, TOP1MT, or TOP3A perturbation	Ordered shifts in optical/THz response track nucleoid compaction or topology, not just ATP depletion.	No topology-sensitive effect after rigorous control for mitochondrial health.
Modern quartz-vs-glass emitter-receiver assay	Signal depends on optical transmission window and on intact receiver mitochondrial signaling.	Effect survives optical blocking or is unaffected by mitochondrial inhibitors and ROS scavengers.
Frequency-matched THz exposures with exact thermal control	Narrow-band response curves emerge and move with mtDNA/topology state.	Only broadband stress or heating effects are seen; no reproducible frequency selectivity.
Correlative UPE imaging plus reporters for ROS, Ca2+, ΔΨm, and mtDNA transcription	Structured correlations appear between UPE microdynamics and mtDNA-dependent state transitions.	No reproducible coupling between UPE statistics and mitochondrial readouts.

Particularly important is the need to separate mtDNA-specific effects from generic mitochondrial collapse. For example, rho0 cells are useful but confounded because they alter respiration broadly. The cleanest study would combine depletion, partial depletion, topology perturbation, and rescue/cybrid strategies so that geometry-sensitive responses can be distinguished from nonspecific bioenergetic failure.

A second priority is reconstitution. Purified or reconstituted nucleoids placed in nanoconfined aqueous environments and interrogated with THz/IR/UV spectroscopy could test whether TFAM-loaded circular DNA differs measurably from linearized or protein-free controls. Such an approach would move the idea from metaphor toward material physics.

7. Major limitations and alternative explanations

Several limitations have to be stated explicitly if this hypothesis is to remain credible. First, UPE may be primarily a byproduct rather than a signaling language. Nothing in the present manuscript proves intentional information transfer. The transducer claim can still be tested even if UPE is partly incidental.

Second, coherence is not established. The hypothesis should therefore be written so that it does not collapse if strong coherence claims fail [14]. A geometry-dependent transducer could respond to flux, spectrum, directionality, intermittency, or local field structure without requiring a biological laser.

Third, any link to exogenous RF exposure must be framed with care. Most ambient RF sources do not operate at tens of THz, so a simple direct-resonance narrative between consumer RF exposure and mtDNA is physically weak. If exogenous electromagnetic effects exist, they are more likely to be indirect, broadband, modulation-dependent, field-redistribution-dependent, thermoregulatory, or stress-mediated than a matter of exact frequency matching [16,19,20].

Fourth, Gurwitsch-type results could in principle reflect other optical receptors, boundary artifacts, or poorly controlled chemical leakage in historical setups. The present model earns its place only if modern isolation experiments, with contemporary imaging and dosimetry, support mitochondrial dependence.

Finally, even a positive result would not establish ceLLM in its broadest philosophical sense. What it would establish is narrower and still important: that mitochondrial genome geometry participates in physical state transduction beyond coding sequence alone.

8. Discussion

Despite these caveats, the framework has value because it creates a concrete bridge between four domains that are usually discussed in isolation: mtDNA topology, mitochondrial signaling, ultra-weak photon emission, and information-theoretic descriptions of cell fate. It also moves the argument away from slogans such as 'DNA as code' versus 'DNA as antenna'. The more useful question is whether genomic molecules simultaneously encode sequence and shape coupling constraints for energetic and electromagnetic microenvironments.

The ceLLM language is useful here because cell-fate changes are rarely binary outputs of a single variable. Cells integrate redox history, membrane state, calcium dynamics, damage signals, topology, and transcriptional context before committing to repair, proliferation, differentiation, senescence, or death. A weak biophysical input could therefore matter not

by overriding chemistry but by biasing a latent decision landscape. That is a more biologically plausible claim than a one-photon, one-outcome caricature.

If the hypothesis is wrong, the proposed experiments will still clarify what UPE is and is not doing in mitochondria. If parts of it are right, mitochondria may emerge as photonic-redox state readers rather than mere ATP engines, and mtDNA may have to be understood as both a genetic polymer and a geometry-bearing interface to the cell's energetic environment.

9. Conclusion

At present, the strongest defensible claim is not that mtDNA is proven to be a living antenna, but that mtDNA is a plausible candidate electromagnetic transducer whose geometry and topology warrant direct experimental testing. Framed this way, the biophoton-ceLLM resonance model becomes publication-ready as a hypothesis article: it rests on established mitochondrial biology and UPE literature, corrects simplistic geometry assumptions, states its limitations openly, and yields falsifiable predictions. That is exactly the posture a serious hypothesis paper should take.

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