HYPOTHESIS AND THEORY

THE EMBODIED CELLULAR TRANSFER FUNCTION

Noncoding Response Architecture, Bioelectric State Inference, and Geometry-Addressed Photonic Reafference in Living Systems

A Cellular Latent Learning Model (ceLLM) hypothesis of cellular intelligence and low-fidelity biology

The biological message is not contained in a field, molecule, ion, or photon alone. It emerges from the interaction between an event and the living transfer function that receives it. The cell's structure is not merely where computation occurs; the energized, changing structure is the computation.

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Abstract

Living systems display capacities that are legitimately described in control-theoretic terms: they sense, integrate, select among alternative states, preserve identity, repair damage, learn from repeated perturbations, and coordinate local actions with larger anatomical goals. Yet the physical unit that implements this intelligence remains incompletely specified. Sequence-centric accounts identify indispensable molecular components but do not, by themselves, explain how the same genome can instantiate distinct cell identities, how nominally similar cells acquire different susceptibilities, or how a weak event acquires different meanings in different cellular states.

This paper proposes the **Embodied Cellular Transfer Function** hypothesis, a mechanistic extension of the Cellular Latent Learning Model (*ceLLM*). The core claim is that the fundamental computational object in a living cell is not DNA, RNA, a membrane, a mitochondrion, or a cytoskeletal filament in isolation. It is the time-varying transfer function produced by their energized integration. Genome sequence and noncoding regulatory architecture constrain a manifold of possible cellular networks. RNA localization and folding, chromatin and mitochondrial-nucleoid topology, protein conformations, membranes, ion gradients, organelle placement, cytoskeletal order, hydration, redox state, and developmental history select the particular network that is physically instantiated at a given moment.

This framework introduces six linked hypotheses. First, biological weights are frequency- and state-dependent transfer functions that encode gain, phase delay, threshold, and cross-coupling, not merely static distances. Second, noncoding variation can alter the *response Jacobian* of a cell—its derivative of response to perturbation—while leaving baseline phenotype comparatively intact. The genotype-conditioned sleep-electroencephalogram response reported for the intronic *CACNA1C* variant rs7304986 is treated as an example of such a latent response phenotype, not as proof of a particular molecular mechanism or health consequence. Third, environmental and endogenous inputs are converted into multidimensional bioelectric state trajectories involving membrane potential and many ions, with calcium serving as a well-characterized but non-exclusive channel. Fourth, mitochondrial execution and redox chemistry generate electronically excited states and ultraweak photon emission (UPE), providing a chemistry-tagged optical readback of metabolism, stress, and recovery. Fifth, the biological meaning of a photon is relational: source coordinate, wavelength, arrival time, burst structure, direction, local attenuation, and receiver state interact with the whole-cell transfer kernel. Sixth, recently active structures form a distributed eligibility field. A returning photonic-redox event can therefore be locally interpreted by matter already marked by the preceding event, supplying a physically plausible route to cellular credit assignment.

A key advance is the proposal of **biological lock-in detection**: endogenous ionic, redox, and metabolic oscillations provide a slow reference phase that transiently sensitizes particular receivers. Correlated photon events can accumulate biological effect, whereas uncorrelated background events average toward noise. This does not require optical phase coherence, laser-like emission, or a cell-scale fiber-optic bus. It requires only state-dependent reception, temporal coincidence, local amplification, and persistent remodeling. The resulting concept—**geometry-addressed photonic reafference**—treats UPE as a candidate means by which the cell reads the consequences of its own activity through the exact struc-

ture that performed it.

Low-fidelity biology is then defined operationally as declining reliability in the coupling among input transduction, bioelectric state, metabolic execution, photonic-redox readback, and persistent update. External electromagnetic fields are one candidate perturbation class among circadian disruption, sleep loss, hypoxia, pollutants, inflammation, metabolic toxicants, and electrolyte imbalance. The framework makes discriminating predictions and specifies record-block-replay, phase-scramble, source-coordinate, eligibility-window, isogenic-genotype, transducer-knockout, topology-perturbation, cytoskeletal-routing, and morphogenetic experiments capable of supporting, narrowing, or breaking the theory.

Keywords: cellular intelligence; bioelectricity; noncoding DNA; RNA; response Jacobian; mitochondria; reactive oxygen species; ultra-weak photon emission; biophotons; microtubules; photonic reafference; eligibility trace; low-fidelity biology; electromagnetic fields

The cell does not read a photon as a self-contained message. It reads the change that the photon or excited-state event produces in a structure already configured by the preceding biological event. Chemistry supplies the event; geometry supplies the address; endogenous timing supplies the reference; receiver state supplies the meaning; remodeling stores the learning.

Conceptual contributions

C1. Embodied transfer function. The whole living cell is a state-dependent response operator.

C2. Response Jacobian. Noncoding architecture can tune response gain, phase, threshold, and cross-coupling.

C3. Complex biological weights. Effective couplings contain amplitude and delay shaped by geometry, hydration, redox, conformation, and voltage.

C4. Relational photon semantics. Meaning belongs to the interaction between a photonic event and the receiver's current transfer kernel.

C5. Biological lock-in. Endogenous oscillations and temporary susceptibility distinguish correlated events from background.

C6. Structural eligibility field. The forward event writes a temporary address into matter; feedback modifies structures that remain eligible.

C7. Endogenous system identification. Self-generated excited-state events may continuously calibrate the exact cell that exists.

C8. Low-fidelity biology. Degradation is loss of reliable coupling across a recurrent loop, creating fidelity debt and somatic overfitting.

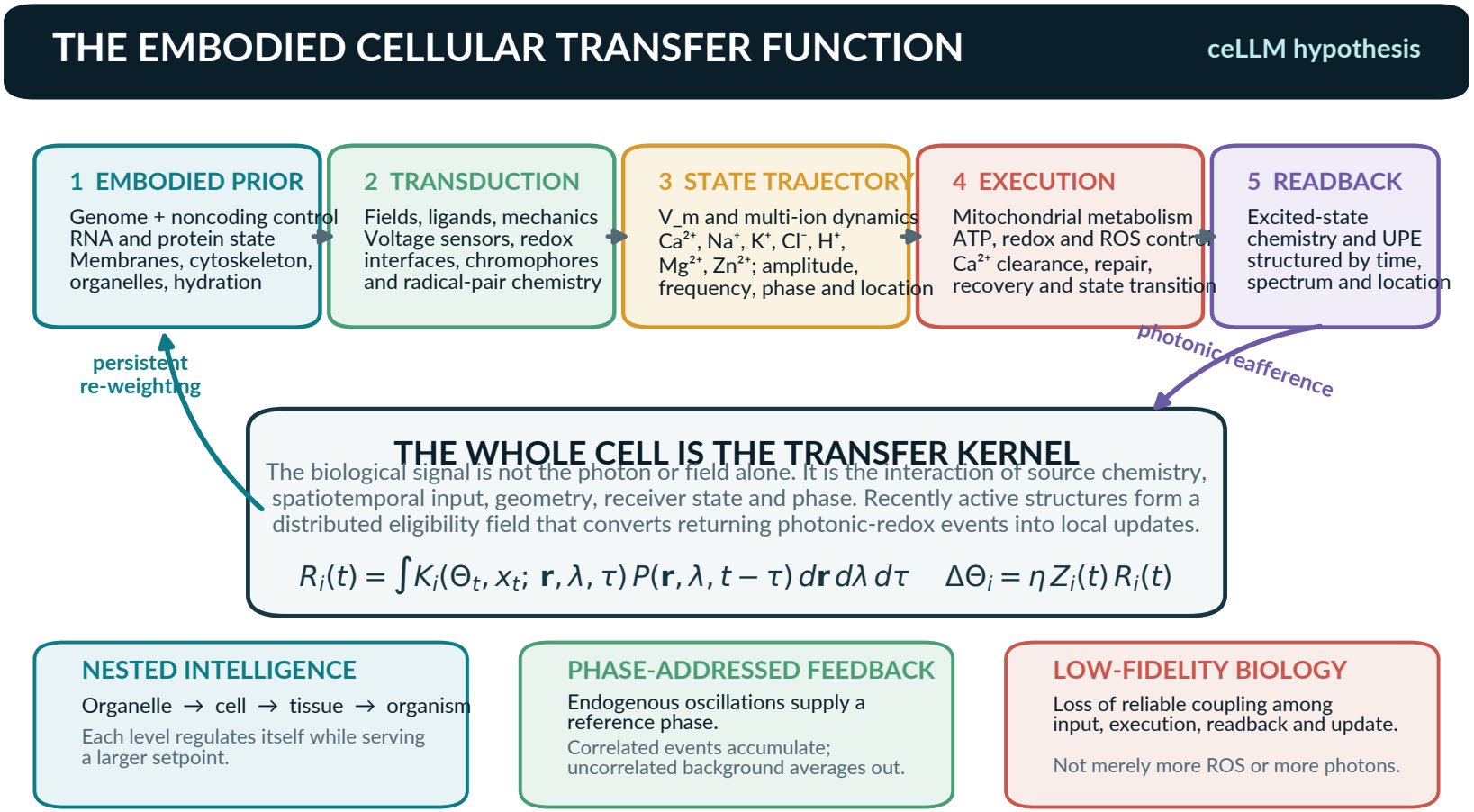


Figure 1: Graphical abstract of the Embodied Cellular Transfer Function hypothesis. Slow molecular and structural priors shape transduction of environmental and endogenous inputs into a multidimensional bioelectric state trajectory. Metabolic execution produces redox-linked excited states and UPE. The whole-cell transfer kernel and a distributed eligibility field determine how that readback is converted into persistent structural and regulatory updates. The recurrent loop is nested within tissue- and organism-level control.

Contents

1. Introduction: locating the physical unit of cellular intelligence

The term *cellular intelligence* is most useful when defined operationally rather than anthropomorphically. A system displays a minimal form of intelligence when it can detect relevant differences, integrate incomplete evidence, select among alternative actions, maintain preferred states, learn from perturbation, and coordinate local behavior with higher-level goals. Cells do all of these things. They regulate internal variables, distinguish self from non-self, navigate chemical and electrical gradients, repair structures, alter future sensitivity after prior exposure, cooperate in tissues, and participate in anatomical pattern homeostasis. Developmental bioelectricity has made especially clear that membrane potential and gap-junctional networks can store and reprogram large-scale pattern information without changing genomic sequence [1–4].

The unresolved question is not whether cells process information. It is *what physical object performs the processing*. The conventional answer often begins with genes and biochemical pathways. Those are indispensable, but the answer remains incomplete for the same reason that a list of artificial-neural-network parameters is not equivalent to a running model. Printed weights do not compute until they are embedded in an architecture with energy flow, activation dynamics, memory, input-output boundaries, and feedback. Likewise, a DNA sequence does not instantiate a living cellular intelligence until it is embedded in chromatin, RNA, proteins, membranes, ionic gradients, organelles, cytoskeleton, solvent, and developmental history.

This paper develops the proposition that the relevant physical unit is the *embodied cellular transfer function*. The cell's active architecture determines which components are coupled, with what gain and delay, at what frequencies, under which thresholds, and for how long. The same external perturbation can therefore be transformed into different internal state trajectories in different cells or in the same cell at different times. The *receiver is part of the biological signal*.

This perspective becomes especially important for UPE, also called biophoton emission. UPE is now an established consequence of oxidative and excited-state chemistry across living systems [5–8]. The July 2026 *Nature* feature on the field captured its current inflection point: the existence of the glow is no longer the principal controversy; the frontier is whether the glow is useful as telemetry and whether living systems themselves exploit it [9]. Early diagnostic work already reports state-dependent emissions from living and dead mice, stressed plants, brain tissue, astrocytes, glioblastoma, melanoma, and other systems [8, 10–13]. The stronger signaling question remains open.

The usual framing—metabolic waste *versus* biological signal—is too binary. An unavoidable byproduct can become a signal when evolution produces a receiver whose behavior benefits from conditioning on it. Oxygen itself, carbon dioxide, ROS, metabolites, heat, and mechanical strain all acquire biological meanings through evolved reception. The source process does not need to have originated for communication. Function is created by the relation between source, channel, receiver, and adaptive consequence.

The framework developed here therefore does not ask whether a photon carries a complete instruction. It asks whether excited-state events participate in a recurrent control architecture whose informational specificity is supplied largely by the receiving cell. This shift

resolves several persistent objections. A sparse signal need not encode the entire cell state. A photon need not contain a molecular address. Global optical coherence is unnecessary. A wet, lossy medium can provide spatial localization rather than merely attenuation. Microtubules need not function as literal long-distance fiber-optic cables. The whole cell can supply the channel, the address, the context, and the gain.

> *The central scientific object is not the photon. It is the conditional response of a living architecture to the photon.*

The paper proceeds from the general theory of embodied cellular computation to specific mechanisms. It formalizes the whole-cell transfer function, develops the concept of noncoding response architecture, treats bioelectric signaling as a multi-ion state trajectory, connects metabolic execution to UPE, identifies cytoskeletal and nucleic-acid geometry as state-dependent routing and memory layers, proposes phase-addressed photonic reafference and structural eligibility, places the loop within nested morphogenetic intelligence, and defines low-fidelity biology as a measurable loss of control-loop reliability. It concludes with an experimental program designed not merely to accumulate suggestive correlations, but to force the central causal claims into decisive competition.

2. Twelve postulates of the ceLLM framework

The Cellular Latent Learning Model (*ceLLM*, pronounced “cell-M”) treats the cell as an active inference and control system. “Latent” refers to the internal variables that are not directly observed but organize future response: developmental identity, energetic reserve, stress history, organelle topology, receptor availability, chromatin accessibility, and tissue-level setpoints. “Learning” refers to persistent change in the mapping between input and future response. It does not require consciousness, language, or a software transformer.

Table 1: Core postulates of the Embodied Cellular Transfer Function hypothesis.

No.	Postulate	Operational meaning
1	Intelligence is a closed-loop property.	Sensing, action, feedback, and persistent update must be analyzed together; no isolated molecule is intelligent by itself.
2	Sequence constrains; structure instantiates.	DNA and RNA encode indispensable constraints, but the running response function is realized by their physical integration with proteins, membranes, ions, organelles, solvent, and tissue context.
3	Biological weights are transfer functions.	Effective coupling includes amplitude, delay, frequency response, threshold, saturation, and state dependence—not distance alone.
4	Noncoding architecture can tune derivatives.	Regulatory variation may leave baseline output similar while changing the gain, phase, or threshold of response to a perturbation.

No.	Postulate	Operational meaning
5	The receiver is part of the biological dose.	Physical exposure metrics describe the incident or absorbed field; the internal trajectory additionally depends on genotype, cell state, geometry, timing, and transducer abundance.
6	The forward code is multidimensional.	Membrane potential and fluxes of Ca^{2+}, Na^{+}, K^{+}, Cl^{-}, H^{+}, Mg^{2+}, Zn^{2+}, and other species carry spatial and temporal structure.
7	Metabolic execution leaves a redox audit trail.	Mitochondrial work, repair, clearance, and stress generate redox changes and electronically excited species that can be sampled as UPE.
8	Photon meaning is relational.	Source coordinate, spectrum, event timing, direction, attenuation, receiver state, and endogenous phase jointly determine biological consequence.
9	The whole cell is the transfer kernel.	Membranes, cytoskeleton, organelles, nucleic acids, condensates, and hydration collectively route, absorb, convert, and amplify events.
10	Forward activity writes a temporary address.	Participating structures remain transiently altered; this distributed eligibility field makes later feedback pathway-specific.
11	Learning is local overlap plus persistence.	A lasting update occurs where prior activation, returning feedback, and local susceptibility coincide.
12	Low fidelity is a coupling failure.	Pathology-relevant decline is measured as reduced reliability among transduction, execution, readback, and correct update, not simply as more ROS, more photons, or more entropy in an undefined sense.

The postulates define a minimal core. Additional extensions—for example, specific mtDNA resonances, long-range optical waveguiding, nonclassical photon statistics, or quantum-coherent neuronal computation—may be investigated, but the central theory does not depend on them. This separation is important because it allows individual mechanisms to fail without collapsing the entire research program.

3. The embodied cellular transfer function

3.1 State, architecture, input, and output

Let the fast cellular state at time t be $\mathbf{x}_t$. It includes membrane potential, ionic concentrations, phosphorylation, redox ratios, metabolite pools, conformational states, organelle potentials, and other rapidly changing variables. Let Θ_t denote slower architecture: genomic and noncoding regulatory configuration, RNA localization, chromatin and nucleoid topology, protein abundance, channel composition, cytoskeletal organization, organelle place-

ment, contact-site density, condensate state, hydration, and developmental history. Let $\mathbf{u}_t$ denote external and endogenous inputs.

A minimal dynamics model is

$$\dot{\mathbf{x}}_t = F(\mathbf{x}_t, \mathbf{u}_t; \Theta_t), \qquad \mathbf{y}_t = G(\mathbf{x}_t; \Theta_t), \tag{1}$$

where $\mathbf{y}_t$ denotes measurable output: transcription, metabolism, motion, repair, secretion, division, apoptosis, or contribution to a tissue-level pattern.

The crucial point is that the input is not biologically interpreted before it passes through the receiver. Define the internal query

$$\mathbf{q}_t = \mathcal{T}_{\Theta_t, \mathbf{x}_t}[\mathbf{u}_{0:t}], \tag{2}$$

where $\mathcal{T}$ is a history-dependent transfer operator. A free-space waveform, chemical concentration, mechanical force, or photon field is therefore not yet a biological prompt. The prompt is the trajectory produced after the living receiver has filtered it.

This distinction is not semantic. It predicts that equal physical inputs can produce unequal biological trajectories when receptor density, membrane geometry, developmental stage, metabolic reserve, genotype, or endogenous phase differs. Conversely, distinct external inputs can converge on the same internal query when the receiver compensates. Biological identity is therefore expressed partly as a family of transfer functions.

3.2 Biological weights are complex, state-dependent couplings

Artificial neural networks usually present weights as scalar numbers. A living network is oscillatory, dissipative, spatially extended, nonlinear, and state dependent. A more appropriate local representation is a frequency-dependent complex coupling:

$$W_{ij}(\omega, t) = A_{ij}(\omega, t) \exp\left[-\mathrm{i}\,\omega \tau_{ij}(t)\right], \tag{3}$$

where A_{ij} is effective gain and τ_{ij} is delay. Complex notation is used here as a compact description of amplitude and phase; it does not imply a coherent quantum wavefunction extending across the cell.

The coupling W_{ij} can depend on

$$W_{ij} = f\left(r_{ij}, \theta_{ij}, \varepsilon_{ij}, H_{ij}, R_{ij}, C_{ij}, V_{\mathrm{m}}, S_{ij}, t\right), \tag{4}$$

where r is distance, θ relative orientation, ε dielectric environment, H hydration, R redox state, C conformation, V_{m} membrane potential, and S spectral overlap. Chemical bonds and tertiary structure set local modes; folding, compaction, membrane curvature, organelle motion, and cytoskeletal remodeling continuously reweight the graph.

This formalism captures the valid intuition behind the phrase “structure is the weights and biases” while avoiding the claim that every atom is literally a neuron. The effective nodes are molecular states and organized complexes—channels, chromophores, redox centers, nucleoid domains, membrane microdomains, cytoskeletal segments, contact sites, and condensates. Atomic geometry determines their coupling coefficients.

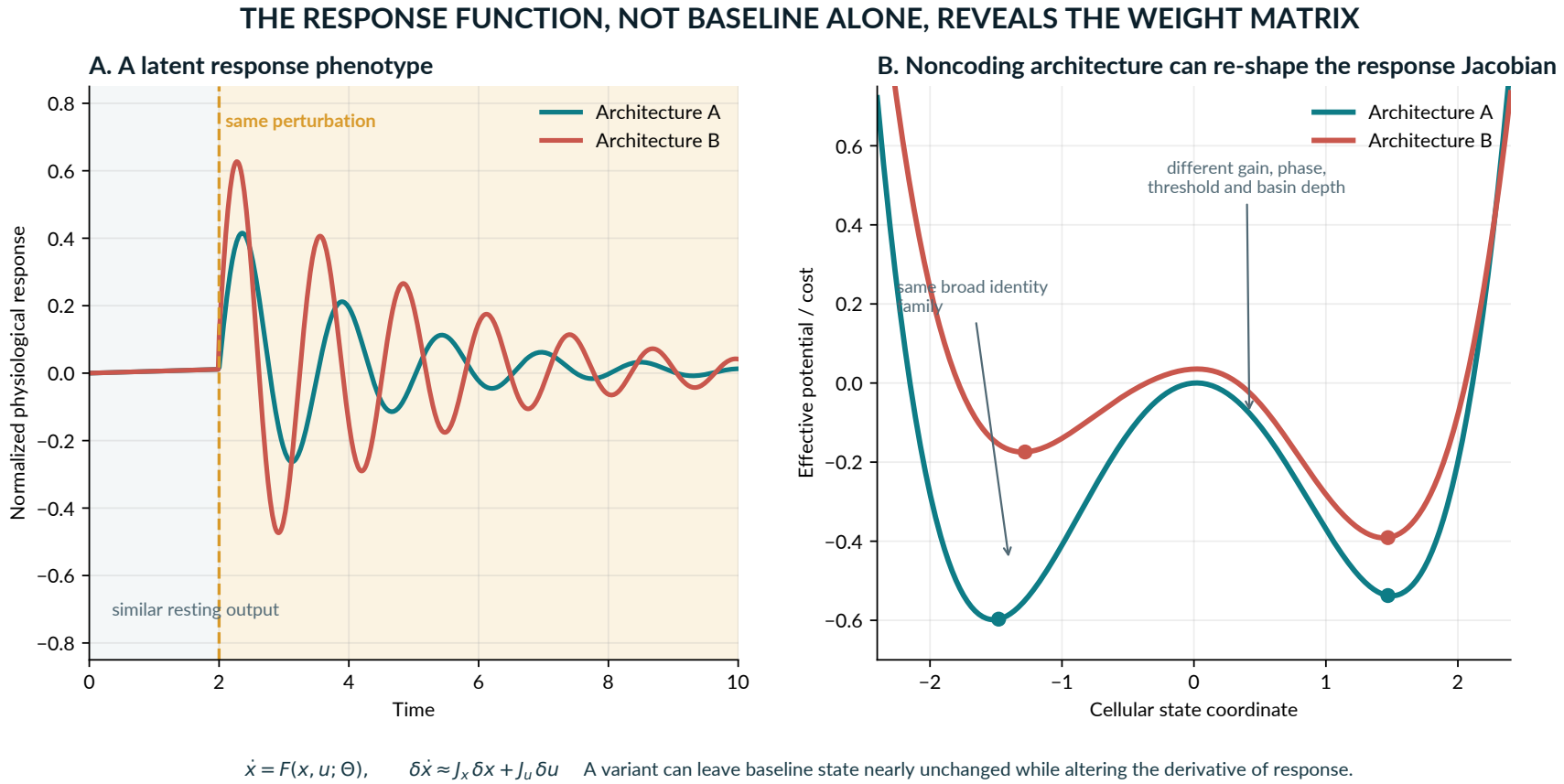


Figure 2: The response function can reveal biological architecture that is not apparent at baseline. Two systems can occupy similar resting states yet differ in transient gain, phase, damping, threshold, and attractor geometry. In the *ceLLM* framework, noncoding regulation and embodied structure can alter the response Jacobian without requiring an obvious resting phenotype.

3.3 The response Jacobian and latent response phenotypes

Linearizing equation (1) around a state gives

$$\delta\dot{\mathbf{x}} \approx J_x\,\delta\mathbf{x} + J_u\,\delta\mathbf{u}, \qquad J_x = \frac{\partial F}{\partial \mathbf{x}}, \quad J_u = \frac{\partial F}{\partial \mathbf{u}}. \tag{5}$$

The matrices J_x and J_u summarize local stability, gain, cross-coupling, and sensitivity. Their eigenmodes determine which perturbations decay, oscillate, amplify, or push the cell toward another attractor.

A regulatory variant, epigenetic state, prior stressor, or change in geometry can satisfy

$$\mathbf{x}^{(A)}_{\text{baseline}} \approx \mathbf{x}^{(B)}_{\text{baseline}}, \qquad J_u^{(A)} \neq J_u^{(B)}. \tag{6}$$

The two systems look similar until they are challenged. This is a **latent response phenotype**. It is more informative than a simplistic division between "normal" and "mutant," because it describes a conditional difference in network operation.

In practical terms, biology should increasingly be phenotyped with controlled perturbations. A resting transcriptome, metabolome, or photon count is a snapshot. An impulse response reveals the operating architecture. This principle applies to environmental exposure, drug response, disease susceptibility, regenerative capacity, and aging.

4. Noncoding response architecture: sequence as constraint, topology as runtime

4.1 Noncoding DNA and RNA as control architecture

Protein-coding sequence specifies indispensable parts, but much of the temporal, spatial, and cell-type specificity of gene use is implemented through noncoding regulatory elements, three-dimensional chromatin contacts, alternative splicing, noncoding RNAs, RNA localization, and RNA-protein assemblies [14–16]. The same coding sequence can therefore participate in different transfer functions depending on enhancer activity, promoter contact, chromatin accessibility, transcript isoform, localization, translation kinetics, and protein complex formation.

Within the present framework, noncoding architecture can alter cellular computation through at least five routes:

1. **Gain control:** changing expression or availability of channels, receptors, pumps, enzymes, and scaffolds.
2. **Phase control:** changing delays through splicing, localization, degradation, trafficking, and feedback kinetics.
3. **Topology control:** changing chromatin contacts, nucleoid compaction, RNA scaffolding, condensate composition, and spatial co-localization.
4. **Threshold control:** changing promoter accessibility, channel abundance, buffering capacity, or nonlinear activation conditions.

5. **Memory control:** stabilizing a prior state through chromatin marks, RNA feedback, protein modifications, or structural persistence.

RNA deserves particular emphasis. RNA is not merely a transient text copied from DNA. Folded RNAs are charged, mobile, structure-sensitive components that bind proteins, regulate translation, shape condensates, localize machinery, and modify reaction kinetics. Long and small noncoding RNAs can act as guides, scaffolds, decoys, catalysts, and feedback elements [16]. Biomolecular condensates can, in turn, reshape cellular electrochemical equilibria, providing a direct bridge between molecular organization and bioelectric state [17, 18].

A concise hierarchy is therefore:

Sequence supplies evolutionary constraints. Noncoding DNA and RNA parameterize context. Three-dimensional molecular organization instantiates the runtime. Bioelectric and metabolic dynamics perform the state transition. Feedback changes the next transfer function.

4.2 The CACNA1C rs7304986 study as a response-function experiment

The 2025 randomized, double-blind, sham-controlled study of rs7304986 in *CACNA1C* provides an unusually clear example of why response architecture matters [19]. Thirty-four genotyped volunteers—15 T/C and 19 matched T/T carriers—received 30 minutes of pre-sleep exposure to 3.6 GHz, 700 MHz, or sham. Only the 3.6-GHz condition in T/C carriers produced the reported faster spindle center frequency across central, parietal, and occipital regions. The study was small, acute, and not designed to establish disease. The molecular function of rs7304986 itself remains unresolved.

Its theoretical importance is nevertheless substantial. The same nominal exposure was transformed differently by different receivers. Moreover, the 3.6-GHz and 700-MHz signals shared a 12.5-Hz power-control component and a dominant time-slot-related modulation near 200 Hz, yet did not produce interchangeable outcomes. This argues against a simple one-envelope-frequency/one-output model. The biological response likely depended on the combined carrier, modulation, spatial absorption, tissue state, genotype, and endogenous network dynamics.

The result is best represented as

$$\text{response} = \mathcal{R}(\text{carrier}, \text{envelope}, \text{dosimetry}, \text{genotype}, \text{cell state}, \text{network phase}), \quad (7)$$

not as a claim that one noncoding letter directly acts as a radio antenna. Other intron-3 variants in *CACNA1C* have been shown to reside in regulatory regions with allele-dependent activity and promoter interactions, establishing the general plausibility of regulatory control in this locus [14, 15]. Whether rs7304986 is causal, linked to another functional variant, or simply marks a broader regulatory haplotype is an experimental question.

A noncoding variant can alter the response Jacobian by changing channel abundance, isoform balance, chromatin contacts, timing, or network compensation. The variant need not produce a large resting difference. The controlled perturbation reveals the re-weighting.

The decisive next step is isogenic editing. T-to-C and C-to-T editing in otherwise matched human neuronal systems would distinguish direct variant effects from linked genetic background. Simultaneous measurement of Cav1.2 expression and splicing, membrane electrophysiology, multi-ion dynamics, mitochondrial state, UPE, and network oscillation would reveal where the response diverges.

5. Transducer pluralism and the multi-ion bioelectric state trajectory

5.1 No single universal receptor is required

A whole-cell theory should not depend on one universal electromagnetic or photonic sensor. Multiple transducer classes can coexist, dominate in different frequency regimes, and converge on common state variables. Candidate classes include:

The 2026 *Cell* study is particularly valuable because it used an unbiased CRISPR screen to identify CYB5B as essential to an engineered electromagnetic-field-inducible gene switch and showed that activation depended on rhythmic calcium oscillations rather than a generic increase in bulk calcium [21]. The field was a defined low-frequency magnetic exposure, not 5G or Wi-Fi, and the result is not evidence of harm. Its importance is mechanistic: a field-to-redox-protein-to-rhythmic-calcium-to-transcription pathway can exist under controlled conditions.

The 2026 terahertz study provides a different scale of transduction. Irradiation at 34.5 THz, but not 36.1 THz, increased calcium influx and activated the PGC-1α-NRF1/2-TFAM mitochondrial-biogenesis pathway [23]. The authors' modeling pointed to a bending mode in a channel-pore glutamate, not demonstrated mtDNA absorption. The result supports frequency-selective biological coupling while simultaneously warning against assigning the receiver in advance.

Likewise, the 2026 tubulin study found isotope-dependent polymerization effects enhanced by a 3-mT magnetic field, with a radical-pair model matching the observations [24]. This is a defined in vitro result, not evidence that environmental RF broadly reorganizes microtubules in vivo. It nevertheless demonstrates that field-sensitive spin chemistry can influence a structural cellular polymer under specific conditions.

5.2 The forward code is a state trajectory, not a calcium flood

Calcium is a uniquely important intracellular signal because its amplitude, location, duration, and oscillatory structure can selectively control transcription, secretion, contraction, metabolism, and death. Calcium oscillations can increase the efficiency and specificity of gene expression, while amplitude and duration can differentially activate transcription

Table 2: Candidate transducer classes and their role in the hypothesis. "Established" refers to the underlying biological function, not to universal sensitivity to ordinary wireless exposure.

Class	Established function	Potential role in ceLLM	Discriminating experiment
S4-containing voltage-sensing domains	Convert transmembrane voltage changes into channel gating [20]	Transform field-induced or endogenous membrane changes into ion flux; direct RF coupling is not assumed	Channel-specific knockout, voltage-clamp, waveform and thermal controls
CYB5B and redox interfaces	Outer-mitochondrial-membrane redox protein; required in a defined EMF-inducible gene switch [21]	Candidate field-sensitive redox-to-calcium interface in the specific engineered system; broader generalization must be tested	CYB5B knockout/rescue under the original field parameters and under distinct exposure classes
Radical-pair chemistry	Spin-dependent reaction yields are physically established; implicated in magnetoreception [22]	Route by which weak magnetic fields may influence chemical branching or structural assembly	Isotope substitution, field orientation, scavenger and spin-state controls
Canonical chromophores	Flavins, porphyrins, cytochromes, aromatic residues, and related molecules absorb light	Convert absorbed photons into redox, conformational, or electrochemical change	Action spectrum plus molecular knockout/rescue
Membrane and hydration interfaces	Dielectric boundaries, collective water modes, and protein-lipid interfaces shape local fields	Frequency conversion, envelope detection, channel modulation, and local amplification	Matched heating, isotope/water substitution where feasible, membrane composition perturbation
Mechanosensitive and electrochemical structures	Couple membrane tension, curvature, charge, and ionic gradients to signaling	Cross-modal transduction of electromagnetic, mechanical, and metabolic perturbations	Mechanical clamping, channel perturbation, multimodal state measurement

factors [25–27]. Cytosolic, endoplasmic-reticulum, and mitochondrial calcium signals maintain structured phase relationships [28]. Oscillations can also improve the energetic efficiency of mitochondrial metabolism [29].

But cellular bioelectricity is not a calcium-only language. A more complete state vector is

$$\mathbf{i}(t) = \left[V_{\mathrm{m}}, [\mathrm{Ca}^{2+}], [\mathrm{Na}^{+}], [\mathrm{K}^{+}], [\mathrm{Cl}^{-}], [\mathrm{H}^{+}], [\mathrm{Mg}^{2+}], [\mathrm{Zn}^{2+}], \ldots\right], \tag{8}$$

with each component carrying information in concentration, location, flux direction, amplitude, frequency, phase, and duration. A transient sodium current can, for example, initiate a regenerative program in a vertebrate model [4]. Proton gradients drive mitochondrial energy conversion. Magnesium shapes ATP chemistry and channel function. Chloride and potassium help set membrane potential and cell volume. Zinc can act as a signaling ion and transcriptional regulator.

The forward pass is therefore better described as a **bioelectric state trajectory**. External and endogenous inputs alter a coupled ion-voltage-redox system; the cell's embodied transfer function converts that trajectory into action.

Physical dosimetry remains indispensable, but it is not a complete biological state descriptor. A receiver-aware dose model records the incident and absorbed field *plus* carrier, modulation, duty cycle, orientation, tissue geometry, developmental state, genotype, transducer abundance, endogenous phase, and adaptation history.

6. Mitochondrial execution, redox recovery, and the photonic audit trail

6.1 Execution and reset

Mitochondria are not merely ATP generators. They integrate calcium, redox, membrane potential, metabolism, innate immunity, apoptosis, biosynthesis, and retrograde signaling. They form networks, contact the endoplasmic reticulum and nucleus, exchange components, and remodel in response to demand [28, 30–33].

The execution phase of a cellular event can include calcium uptake and release, ATP production and consumption, membrane repolarization, receptor deactivation, dephosphorylation, restoration of NADH/NAD^{+}, repair of oxidized molecules, organelle repositioning, and transcriptional change. Calcium ions are cleared through pumps, exchangers, buffers, uptake, and sequestration. ROS does not "neutralize spent calcium." ROS instead occupies a dual role: controlled ROS can act as a signal, while excessive or prolonged ROS damages lipids, proteins, and nucleic acids [34].

This distinction strengthens the theory. The photonic event need not represent a binary "cache cleared" acknowledgement. It can report the chemistry, timing, and energetic cost of execution and recovery. A larger burst may mean successful high-throughput metabolism, acute stress, incomplete recovery, lipid oxidation, mitochondrial dysfunction, or injury. The receiver must interpret structure, not brightness alone.

6.2 Self-encoding photochemistry

During oxidative metabolism and stress, ROS-linked reactions can produce electronically excited carbonyls, singlet oxygen, and other excited species. Relaxation toward lower-energy states can emit photons across ultraviolet, visible, and near-infrared bands [5–7]. The source chemistry therefore partly encodes the emitted point process:

$$P(\lambda, t, \mathbf{r}) = \sum_j a_j(t, \mathbf{r})\, k_j(\lambda; \mathbf{x}_t) + \epsilon, \tag{9}$$

where a_j is the activity of excited-state pathway j, k_j its spectral-temporal fingerprint, and ϵ detector and biological noise.

This is **self-encoding photochemistry**. An engineered optical transmitter uses a separate encoder to modulate a carrier. In a redox-generated UPE process, the chemistry that occurred determines the distribution of emitted events. The signal is not a complete transcript of the biochemical transaction; it is a compressed audit trail.

A photon event can be represented as

$$e_k = \left(\mathbf{r}_k, t_k, \nu_k, \hat{\mathbf{k}}_k, \hat{\mathbf{p}}_k\right), \tag{10}$$

where source coordinate, time, frequency, direction, and polarization are explicit. For a burst, photon number, inter-event intervals, and envelope must also be represented. Frequency and photon energy are not independent payloads because $E = h\nu$; information is carried by the organization of a stream or by its relation to the receiver.

6.3 From metabolic exhaust to recruited feedback

The historical Gurwitsch onion-root experiments are important not because they settle the signaling question, but because their reported architecture was conditional. A growing root tip reportedly altered mitosis in a neighboring root; the effect was blocked by glass or opaque barriers but not quartz, which transmits ultraviolet light. Replication was inconsistent, and later reviews appropriately emphasize methodological difficulty [9, 35]. Yet the logic is instructive: an optical event appeared to act only on competent tissue in a particular spatial and developmental state.

Modern isolated-mitochondria experiments provide a more controlled but still preliminary parallel. Stressing one mitochondrial population altered respiration in a chemically separated neighboring population; opaque shielding and ambient-light conditions affected the result, and mitochondria of cancerous and non-cancerous origin responded differently [36]. The source light was not directly measured in that experiment, so the mechanism remains open. The receiver dependence is nevertheless exactly what a relational signaling theory predicts.

Evolution need not have invented UPE for communication. It needed only to recruit photoreceptive or excited-state-sensitive structures that could use a pre-existing metabolic emission. The same event can be exhaust at the source and information at the receiver.

UPE is proposed to be a chemistry-tagged readback whose useful variables may include spectrum, burst timing, source location, event correlation, and relation to endogenous state. The theory does not predict that every UPE event is functional or that all cell types use the same variables.

7. The whole cell as a geometry-dependent transfer kernel

7.1 The biological signal is the interaction of event and transfer kernel

Let $P(\mathbf{r}, \lambda, t)$ describe the photonic point process or coarse-grained field generated by excited-state chemistry. Let K_i be the state-dependent transfer kernel from source coordinate $\mathbf{r}$ and wavelength λ to receiving site i. The local response is

$$R_i(t) = \int K_i(\Theta_t, \mathbf{x}_t; \mathbf{r}, \lambda, \tau)\, P(\mathbf{r}, \lambda, t - \tau)\, d\mathbf{r}\, d\lambda\, d\tau. \tag{11}$$

The kernel can include absorption, scattering, local re-emission, excitonic transfer, near-field coupling, conformational gating, redox conversion, and classical biochemical amplification. Its state variables include membrane voltage, chromophore oxidation, protein conformation, hydration, local geometry, and proximity to amplifying machinery.

The biological signal is therefore not P alone. It is $K \times P$. The same photon can produce different consequences when a mitochondrion moves, a microtubule polymerizes, a membrane changes potential, a protein binds calcium, chromatin opens, a contact site forms, or a chromophore changes redox state.

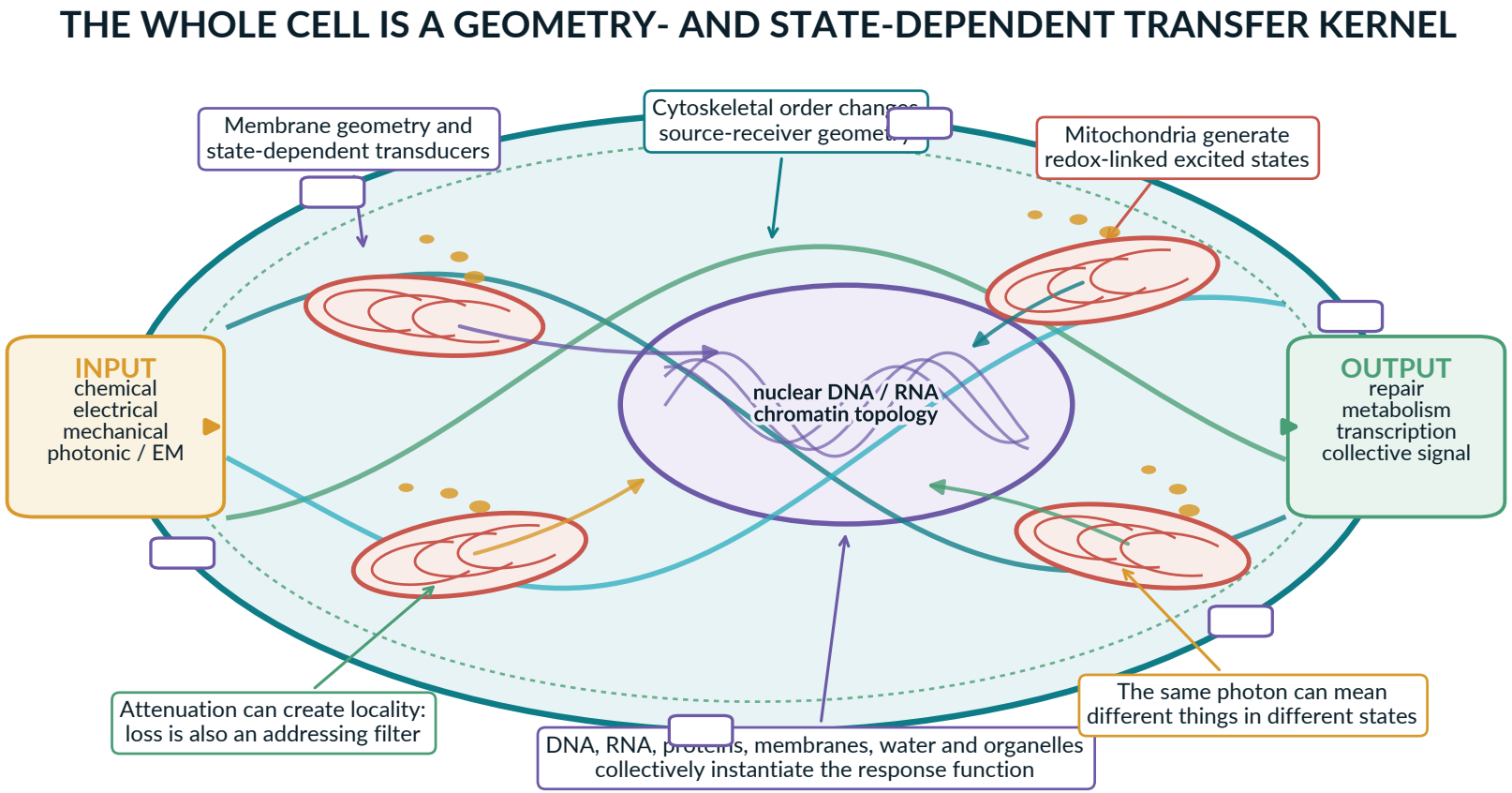


Figure 3: The entire cellular architecture contributes to the transfer kernel. The figure does not assign a permanent sender, wire, receiver, or memory element. Those roles change with geometry and state. Microtubules and actin can alter organelle placement and local transfer; DNA, RNA, proteins, membranes, chromophores, condensates, and hydration determine absorption and amplification; attenuation can restrict action to the neighborhood in which an event was generated.

7.2 Loss can create locality

The intracellular environment is wet, thermally active, and optically lossy. This is often treated as a decisive objection to intracellular optical signaling. It is a decisive objection to a high-bandwidth, long-distance, free-space optical bus. It is not an objection to local, state-gated feedback.

A cellular control loop usually needs to couple structures separated by nanometers to micrometers. In that regime, attenuation can function as an addressing filter. A photon or excited state absorbed near its source preferentially affects the closest compatible membrane, chromophore, cytoskeletal segment, contact site, or nucleoid. Limited range reduces cross-talk. The relevant relation is approximately

$$\Pr(\text{target interaction}) \propto \text{spectral compatibility} \times \text{geometric proximity} \times \text{receiver eligibility}. \tag{12}$$

An external air-gap experiment and an intracellular loop need not use the same transport regime. Across a quartz-separated air gap, propagating photons may dominate. Inside a cell, local reabsorption, near-field coupling, excitonic migration, non-radiative relaxation, and classical amplification can coexist.

7.3 Microtubules as adaptive routing geometry

Microtubules are central to cellular architecture, transport, polarity, organelle placement, and dynamic organization. Recent work adds optical and spin-sensitive possibilities. Electronic excitation migration has been measured over approximately 6.6 nm in microtubules, with dependence on polymerization state [37]. Cooperative ultraviolet behavior has been analyzed in large tryptophan networks within biological architectures [38]. A 2026 open-quantum-system model found that chromophore positions, dipole orientations, initial excitation, lattice order, and disorder altered the direction, retention, and dissipation of information in microtubule tryptophan networks [39]. A separate 2026 experiment linked magnesium isotope nuclear-spin properties and a weak applied magnetic field to tubulin polymerization dynamics [24].

These findings do not demonstrate cell-scale optical waveguiding or conscious quantum computation. They support a more general and more useful proposition: microtubule geometry can change how energy, correlation, organelles, and signals are routed. In *ceLLM*, microtubules can contribute through four roles:

1. **Architecture:** positioning mitochondria, nuclei, vesicles, and membrane domains.
2. **Local transfer:** supporting short-range excitation migration or acting as an anisotropic sink.
3. **Adaptive routing:** changing the transfer kernel through growth, shrinkage, orientation, and post-translational modification.
4. **Eligibility:** retaining local conformational or binding-state changes after a forward event.

Actin provides a complementary layer. An interphase actin wave can promote mitochondrial

content mixing, maintain polarization and oxygen consumption, and limit excess ROS [40]. Cytoskeletal perturbation can therefore alter photonic-redox behavior through transport and mechanics even if no optical routing exists. A decisive experiment must distinguish those alternatives by measuring spatial correlation and anisotropy, not merely total UPE.

The 2026 Arendash review independently proposes a neuronal microtubule/RF/photon "vibrational fabric" related to memory and consciousness [41]. It is a useful convergence of themes, but the present theory is broader and requires neither Orch OR, objective reduction, crystalline-water memory, nor a consciousness-specific mechanism. Its minimal commitments are whole-cell geometry, state-dependent coupling, local excited-state transfer, classical amplification, and persistent update.

7.4 DNA and mtDNA topology as tunable physical variables

DNA is not a bare sequence. Nuclear DNA is folded into chromatin; mtDNA is circular, supercoiled, hydrated, and packaged with TFAM into nucleoids. Human mtDNA contains 16,569 base pairs [42]. Mammalian nucleoids are compact and heterogeneous, frequently contain a single mtDNA molecule, and display activity states related to TFAM-to-mtDNA ratio and packaging [43–47]. DNA hydration shells differ from bulk water and support collective dynamics sensitive to hydration and temperature [48, 49].

These properties justify treating topology as a control variable. They do not justify treating mtDNA as a simple metal loop antenna in vacuum. Dimensional guideposts—the approximately 5.633-μm mtDNA contour, approximately 1.79-μm relaxed-circle diameter, and approximately 100-nm nucleoid scale—can motivate search windows near 53 THz, 167 THz, and 3 PHz, respectively, but hydrated in vivo modes will shift, broaden, mix, and damp. The scientifically decisive question is not whether a free-space formula gives a numerically suggestive frequency. It is whether changing topology, TFAM compaction, hydration, or mtDNA abundance changes the biological transfer function under controlled optical or terahertz perturbation.

8. Phase-addressed photonic reafference

8.1 The background-light problem points to timing, not brightness

UPE escaping a biological sample is extremely weak, often only tens to hundreds of photons per square centimeter per second [7, 9]. Background photons, detector noise, thermal fluctuations, and ordinary chemistry create a serious signal-to-noise problem. A model that depends on every emitted photon being individually decoded at long range is physically implausible.

A different possibility is that the cell detects conditional correlation rather than intensity. Endogenous calcium, voltage, redox, membrane-potential, and metabolic oscillations provide internal timing references. The forward event can transiently change the absorption, conformation, redox state, or amplification gain of the structures that participated. A subsequent photon arriving while those structures are receptive has a different probability of producing a state change than the same photon arriving at another phase.

This is analogous to lock-in detection. An electronic lock-in amplifier multiplies a noisy input by a reference waveform and integrates; uncorrelated noise averages toward zero while the phase-correlated component accumulates. A cell need not perform explicit multiplication. State-dependent absorption, coincidence chemistry, refractory periods, conformational gating, and nonlinear thresholds can implement the same logic physically.

Crucially, the relevant phase is the slow biological envelope phase, not the optical carrier phase. Spontaneous UPE need not be laser-like or globally coherent. The receiver need only be sensitive during reproducible windows relative to calcium, redox, or metabolic cycles.

A schematic detector is

$$D_i = \int Z_i(t)\, P_i(t)\, \Gamma[\phi_P(t) - \phi_i(t)]\, dt, \tag{13}$$

where Z_i is local susceptibility, P_i local photon-event density, ϕ_i the biological reference phase, and Γ a phase-sensitive coupling function. The model predicts that equal photon counts delivered in phase, antiphase, and phase-scrambled conditions can produce different effects.

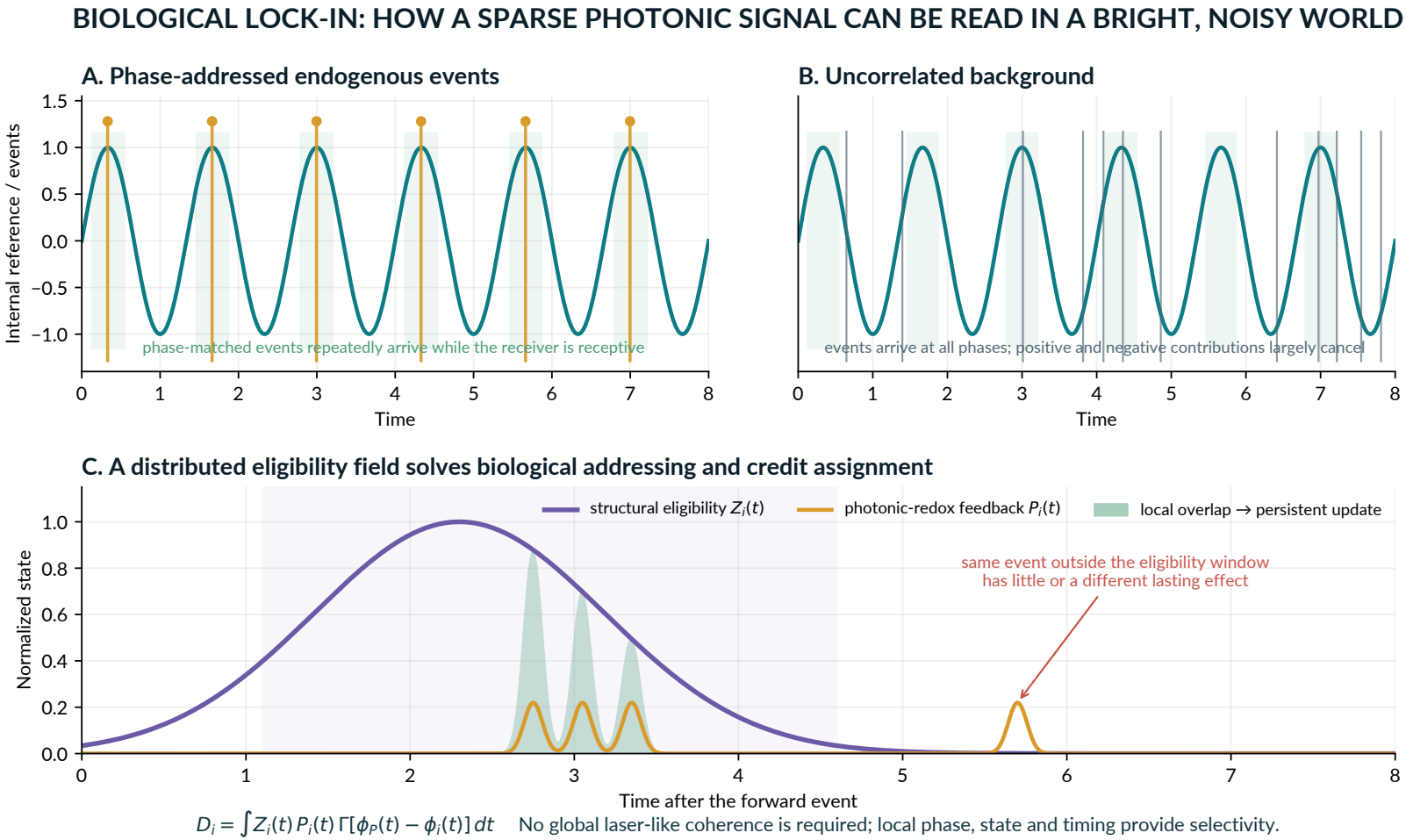


Figure 4: Biological lock-in and structural eligibility. Endogenous oscillations can supply a reference phase, allowing correlated events to accumulate preferentially while uncorrelated background is attenuated by averaging and state gating. The forward event leaves a finite eligibility window. A persistent update requires overlap among prior activation, returning photonic-redox feedback, and local susceptibility.

8.2 The structural eligibility field

During a forward event, the participating pathway is physically changed. Channels open or inactivate; calcium-binding proteins change conformation; kinases phosphorylate targets; membranes change voltage; organelle contacts form; microtubule-associated complexes bind cargo; local redox pairs shift; chromatin becomes accessible; RNA-protein condensates reorganize. These changes are temporary records of participation.

Let $Z_i(t)$ denote the eligibility state of structure i. A persistent update can be written

$$\Delta\Theta_i = \eta\, Z_i(t)\, \delta_i(t), \tag{14}$$

where δ_i is a local photonic-redox or multimodal residual and η is effective learning gain. The residual need not be a digital error number. It can be embodied as mismatch among expected calcium phase, redox state, membrane potential, and incoming excited-state event.

This is a biological three-factor learning rule:

$$\begin{aligned}&\text{prior local activation} \times \text{returning feedback}\\ &\qquad \times \text{current susceptibility} \longrightarrow \text{persistent local change.}\end{aligned} \tag{15}$$

Eligibility traces are well established in neural learning, where a delayed reinforcement signal acts selectively on synapses that remain marked by recent activity [50]. Their existence in neural circuits does not prove a photonic cellular mechanism. It establishes the general biological logic needed to solve temporal credit assignment.

The key conceptual advance is that the photon does not need to carry an address. The forward event writes the address into matter. The whole recently active cell becomes a distributed eligibility field.

8.3 Physical backpropagation without software gradients

The phrase “biophoton backpropagation” can be retained as a public-facing analogy if defined carefully. Literal machine-learning backpropagation computes derivatives through a network. No evidence shows that a cell implements the chain rule in software. But physical systems can propagate fields in ways that reveal local gradients. In situ photonic neural-network training uses forward and adjoint fields so that the medium itself contributes to gradient measurement [51].

The cellular proposal is an *adjoint-like* physical process, not an identity with that engineering method. The forward event changes local susceptibility; returning feedback traverses the changed medium; local overlap determines where persistent modification occurs. The whole-cell transfer function plays the role that an explicit Jacobian plays in a computational model.

The photon carries no molecular mailing label. The preceding event makes the relevant structures temporarily addressable.

8.4 Endogenous system identification: the cell reads itself through light

Every cell is a unique physical instantiation. Even clonally related cells differ in mitochondrial topology, organelle position, cytoskeletal geometry, chromatin conformation, protein abundance, redox state, and damage history. Yet they remain within robust identity attractors. The genome cannot precompute every microscopic contingency of every realized cell.

A self-generated excited-state event automatically probes the cell that actually exists. The source chemistry writes the event; the current geometry filters it; the resulting state transition contains information about both execution and structure. In engineering terms, this is endogenous system identification.

The cell is not passively illuminated by a generic signal. It produces a chemistry-tagged probe whose path and absorption are conditioned by the same architecture that performed the work. This suggests the paper's most compact formulation:

> *The cell does not read a photon. The cell reads itself through the photon.*

The hypothesis predicts that source coordinate matters. An otherwise identical low-flux replay delivered near a nucleus, an ER-mitochondrial contact, a microtubule-aligned mitochondrion, or a plasma-membrane domain should not be biologically interchangeable. It also predicts that controlled changes in cell shape or organelle placement alter the impulse response even when total photon number and spectrum are held constant.

9. Nested intelligence and morphogenetic control

9.1 Organelle, cell, tissue, and organism

A cell is neither an isolated computer nor a passive component. It is simultaneously an agent maintaining its own viability and a node serving tissue-level anatomical goals. Mitochondria and other organelles maintain local setpoints; cells maintain identity and repair; tissues maintain boundaries and morphology; organisms maintain physiology and behavior. Information flows upward and constraints flow downward.

A tissue-level response matrix can be represented as

$$J_{\text{tissue}} = \begin{bmatrix} J_1 & C_{12} & \cdots \\ C_{21} & J_2 & \cdots \\ \vdots & \vdots & \ddots \end{bmatrix}, \tag{16}$$

where diagonal terms J_i are embodied cellular transfer functions and off-diagonal terms C_{ij} are gap-junctional, chemical, mechanical, electrical, and candidate optical couplings.

Michael Levin's work on developmental bioelectricity provides strong evidence that tissue-scale physiological networks can store and select anatomical outcomes. Transient gap-junctional blockade can induce alternative species-like head morphologies in genetically wild-type planaria, and transient bioelectric interventions can produce persistent changes

in regenerative anatomy [1–3]. These findings do not establish photonic control. They establish the larger informational architecture within which a subcellular photonic-redox feedback layer could operate.

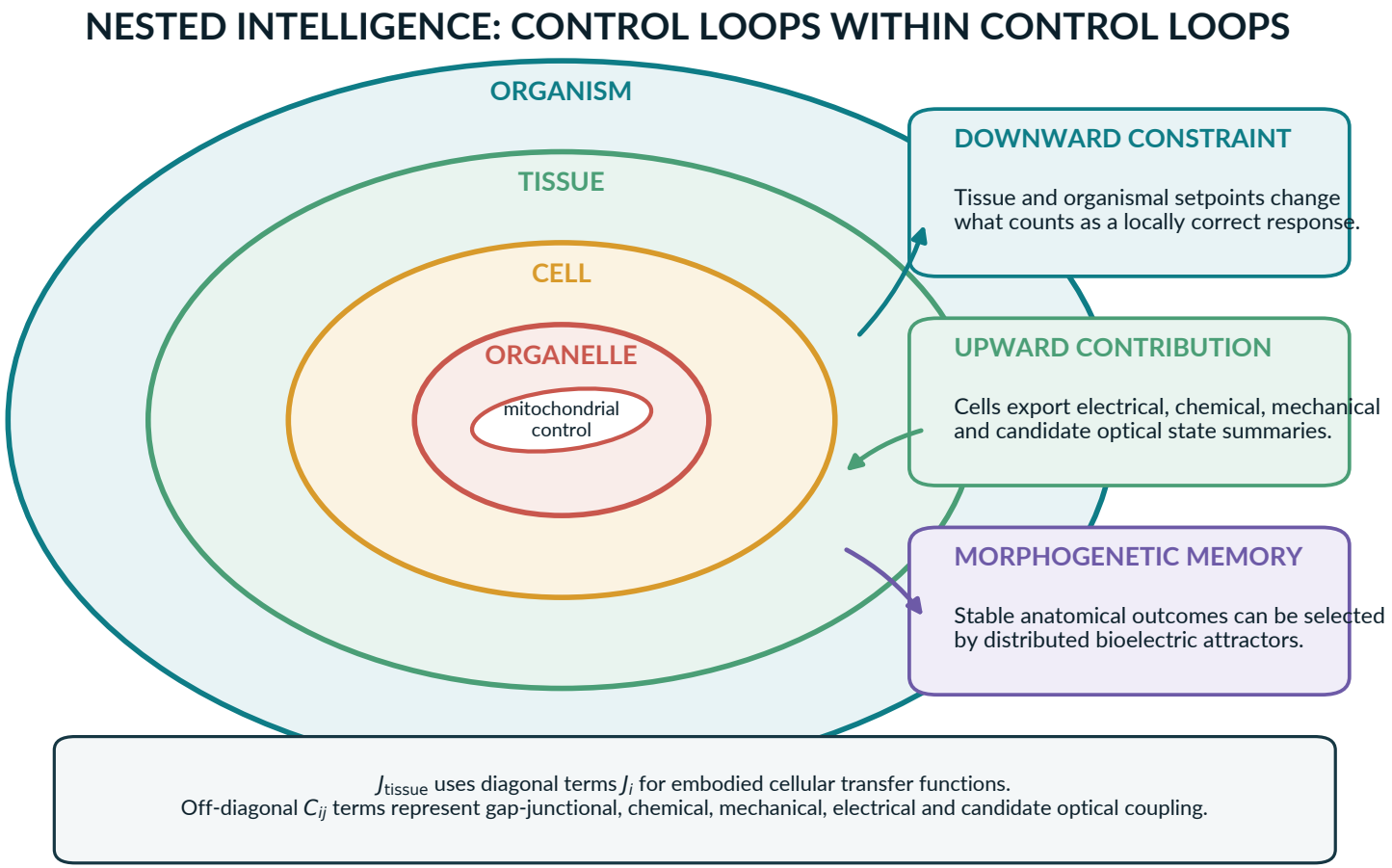


Figure 5: Nested intelligence. Each level maintains local setpoints while contributing to larger-scale goals. Tissue-level bioelectric attractors constrain what counts as a correct cellular response; cells export state summaries that help maintain those attractors. The proposed photonic-redox loop is one subcellular component of this hierarchy, not a replacement for classical electrical, chemical, mechanical, or gap-junctional signaling.

9.2 A cellular self-model without a homunculus

The language of inference can sound as if a hidden observer inside the cell interprets signals. No such homunculus is required. A biological self-model can be implicit in the architecture of control: preferred voltage ranges, membrane integrity, redox balance, organelle organization, cell identity, tissue position, and repair setpoints. Deviations alter dynamics; corrective pathways are activated; successful states are stabilized.

This is compatible with predictive-processing or free-energy descriptions, which model living systems as minimizing deviations from expected viable states [52]. The present hypothesis adds a physical candidate for one type of reafferent readback. It does not claim that cells consciously predict or explicitly calculate probabilities. The probabilistic description is a model of state-dependent transitions.

9.3 Robust identity with microscopic individuality

No two cells need have identical microscopic geometry to share a functional identity. Many distinct microstates can lie within the same attractor basin. A skin cell at one fingertip and a corresponding cell at another may differ in organelle placement, local stress history, and chromatin details while remaining equivalent at the tissue level.

This is why perturbation response can be more revealing than static state. Cells within the same identity family may have different margins to instability. A genotype, prior exposure, or age-related structural change can shallow an attractor basin or alter phase response without immediately changing the visible phenotype. The latent response phenotype is therefore a bridge from molecular individuality to population-level susceptibility.

10. Low-fidelity biology

10.1 An operational definition

“Low-fidelity biology” should not be used as a synonym for oxidative stress, damage, thermodynamic entropy, or disease. It is a control-theoretic property:

Low-fidelity biology is a measurable decline in the reliability with which biological inputs are transformed into appropriate state trajectories, metabolic execution, feedback, and persistent updates across nested control loops.

Four conditional fidelities can be defined:

$$F_T = I(\mathbf{U}; \mathbf{Q} \mid \Theta), \quad \text{transduction fidelity,} \tag{17}$$

$$F_E = I(\mathbf{Q}; \mathbf{M} \mid \Theta), \quad \text{execution fidelity,} \tag{18}$$

$$F_R = I(\mathbf{M}; \mathbf{P} \mid \Theta), \quad \text{readback fidelity,} \tag{19}$$

$$F_L = I(\mathbf{P}; \Delta\Theta \mid \mathbf{Z}, \Theta), \quad \text{learning/update fidelity,} \tag{20}$$

where I is conditional mutual information, $\mathbf{U}$ input, $\mathbf{Q}$ internal query, $\mathbf{M}$ metabolic execu-

tion, **P** photonic-redox readback, and **Z** eligibility. These are conceptual definitions; experiments can estimate them with transfer entropy, coherence, causal state-space models, out-of-sample prediction, and intervention.

The loop fidelity is a vector, not a single number:

$$\mathcal{F}_{\text{loop}} = (F_T, F_E, F_R, F_L). \tag{21}$$

Different diseases or stressors can degrade different components. A channelopathy may impair transduction. Mitochondrial dysfunction may impair execution. Excess oxidative damage may distort readback. Chronic inflammation or maladaptive epigenetic remodeling may impair credit assignment and update.

10.2 Photon load is not photonic fidelity

Total photon load can be written

$$L_P = \int P(t)\, dt. \tag{22}$$

A stressed cell can have $L_P \uparrow$ while the structured relationship between execution and readback deteriorates. Define a photonic-loop-fidelity metric

$$\text{PLF} = I\left(\Phi(P_t); Y_{t+\Delta} \mid \text{Ca}_t^{2+}, \text{ROS}_t, \Delta\psi_{\text{m},t}, \Theta_t\right), \tag{23}$$

where $\Phi(P_t)$ extracts spectrum, burst timing, source location, inter-event intervals, entropy, and phase coupling. PLF asks whether photon structure predicts a future cellular state beyond conventional metabolic variables.

This yields a strong prediction:

$$L_P \uparrow \quad \text{while} \quad \text{PLF} \downarrow \tag{24}$$

can occur in low-fidelity states. More light can mean less usable information.

10.3 Bioelectric dissonance as task-specific phase error

"Bioelectric dissonance" is best defined without assuming that artificiality is intrinsically harmful:

> Bioelectric dissonance is a perturbation-induced loss of task-relevant timing, phase, or causal coupling among membrane/ion dynamics, metabolic execution, redox recovery, and feedback. A waveform is noise only relative to a biological task whose fidelity it measurably reduces.

This definition accommodates neutral, adaptive, therapeutic, and disruptive field effects. The same nominal field can have different signs in different states. Frequency, waveform, duty cycle, orientation, dose, developmental stage, genotype, and endogenous phase can all matter. A beneficial 1-GHz treatment hypothesis, a low-frequency gene switch, a terahertz channel effect, and a disruptive exposure hypothesis are not mutually exclusive; they

occupy different regions of a high-dimensional response function.

10.4 Fidelity debt and somatic overfitting

Cells compensate for perturbation by changing channel expression, antioxidant capacity, chromatin state, mitochondrial topology, cytoskeleton, inflammation, and metabolism. Compensation can preserve short-term viability. But every persistent patch changes the future transfer function.

Fidelity debt is the accumulating cost of maintaining current function through compensatory remodeling that narrows future robustness. If the update is

$$\Delta\Theta_i = \eta Z_i \left(\delta_i^{\text{true}} + \epsilon_i^{\text{perturbation}}\right), \tag{25}$$

then recurrent perturbation can train the cell on a contaminated residual. The cell becomes locally adapted to the distorted condition but less capable in its native environment. This is **somatic overfitting**.

The compounding loop is

$$\begin{aligned} &\text{perturbation} \rightarrow \text{compensation} \rightarrow \text{altered transfer function} \\ &\qquad \rightarrow \text{new interpretation error} \rightarrow \text{further compensation.} \end{aligned} \tag{26}$$

This mechanism can contribute to aging and disease susceptibility without claiming that every age-related change is error or that all diseases share one cause. The proposed metastate is declining control fidelity, which broadens access to multiple disease-specific attractors.

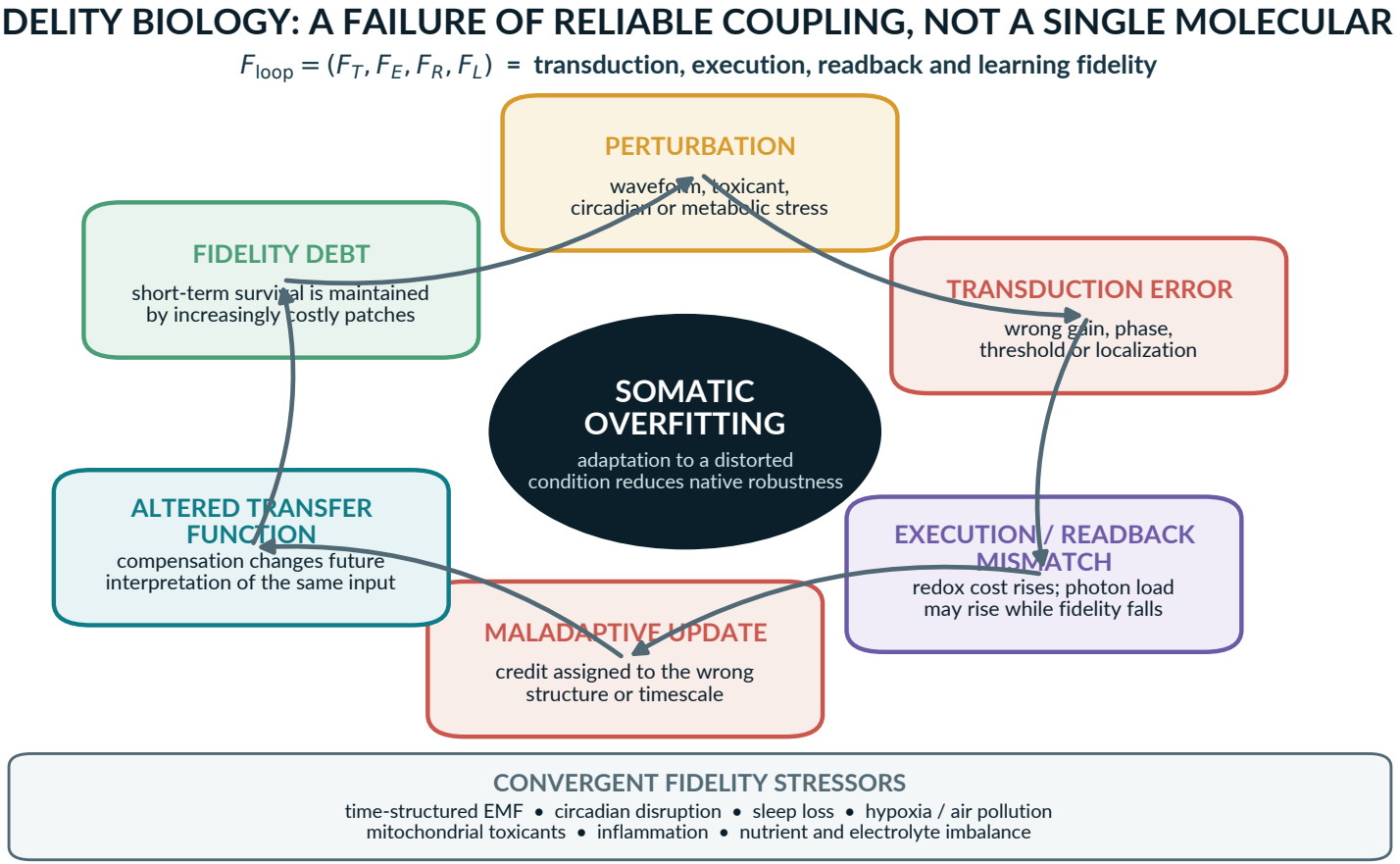


Figure 6: Low-fidelity biology as a recurrent degradation process. Different stressors can enter at different points, but repeated compensation may alter the transfer function itself. Fidelity debt preserves short-term performance at increasing cost; somatic overfitting adapts the cell to a distorted condition, reducing robustness when conditions change.

10.5 Convergent fidelity stressors

The framework is perturbation-agnostic. Multiple exposures can lower different components of loop fidelity:

Table 3: Perturbation classes and predicted entry points into the ceLLM loop. These are mechanistic hypotheses, not assertions that every exposure produces harm.

Perturbation class	Likely entry point	Primary measurable signature
Time-structured electromagnetic fields	Transducer activation, membrane timing, ion dynamics, radical-pair chemistry, redox, or receiver coupling	Changed impulse response, phase coupling, genotype interaction, or waveform-specific effect at matched dosimetry
Circadian light disruption	Phase reference, clock transcription, metabolic synchronization	Phase drift, reduced recovery, altered time-of-day response
Sleep deprivation	Repair, redox recovery, proteostasis, network recalibration	Slower return to baseline, increased response variance, altered PLF
Hypoxia and air pollution	Mitochondrial execution, oxygen chemistry, inflammation	Increased execution cost, altered UPE spectrum, reduced energetic reserve
Mitochondrial toxicants or biologically active chemicals	Enzymes, membranes, redox, chromatin, ion handling	Stage-specific failure localized by perturbation/rescue
Chronic inflammation	Tissue coupling, cytokine state, redox and persistent remodeling	Off-target pathway activation and reduced attractor stability
Nutrient or electrolyte imbalance	Ion state space, buffering, cofactor availability, membrane potential	Altered thresholds, oscillation geometry, and metabolic efficiency
Mechanical injury	Membrane tension, cytoskeleton, wound fields, repair attractors	Spatially organized voltage, calcium, redox, and UPE changes

RF-EMF is especially important to test rigorously because exposure is widespread and difficult to avoid, but the scientific strategy should not begin by assigning a universal sign. A 2024 systematic review found RF-EMF oxidative-stress evidence heterogeneous and of very low certainty overall [53]. The appropriate response is better mechanistic design: preserve waveform, developmental state, genotype, phase, geometry, and simultaneous readouts rather than collapsing exposure to a single average scalar.

11. What the theory predicts

A useful theory should create predictions that would not automatically follow from conventional "UPE is a byproduct" or "RF causes oxidative stress" models.

P1. Same baseline, different impulse response. Isogenic or naturally variant cells can show similar resting outputs but different gain, phase, damping, or threshold after a controlled perturbation.

P2. Carrier-envelope interaction. Equal average absorbed energy and shared modulation do not guarantee equal biological output; carrier, spatial absorption, and receiver architecture interact nonlinearly.

P3. Source-coordinate specificity. The same spectral-temporal photon pattern delivered at different subcellular coordinates produces different local and persistent responses.

P4. Geometry dependence. Reversible changes in cell shape, organelle placement, cytoskeletal order, or nucleoid topology change the transfer function and restore when geometry restores.

P5. Phase-sensitive replay. In-phase, antiphase, and phase-scrambled photon replays with identical count and energy produce different effects relative to an endogenous calcium or redox reference.

P6. Finite eligibility window. A persistent update occurs only when feedback overlaps a recently active pathway within a measurable temporal window.

P7. Receiver knockout and rescue. Removing the relevant chromophore, redox protein, channel, or structural component abolishes a replay effect; molecular rescue restores it with a corresponding action spectrum.

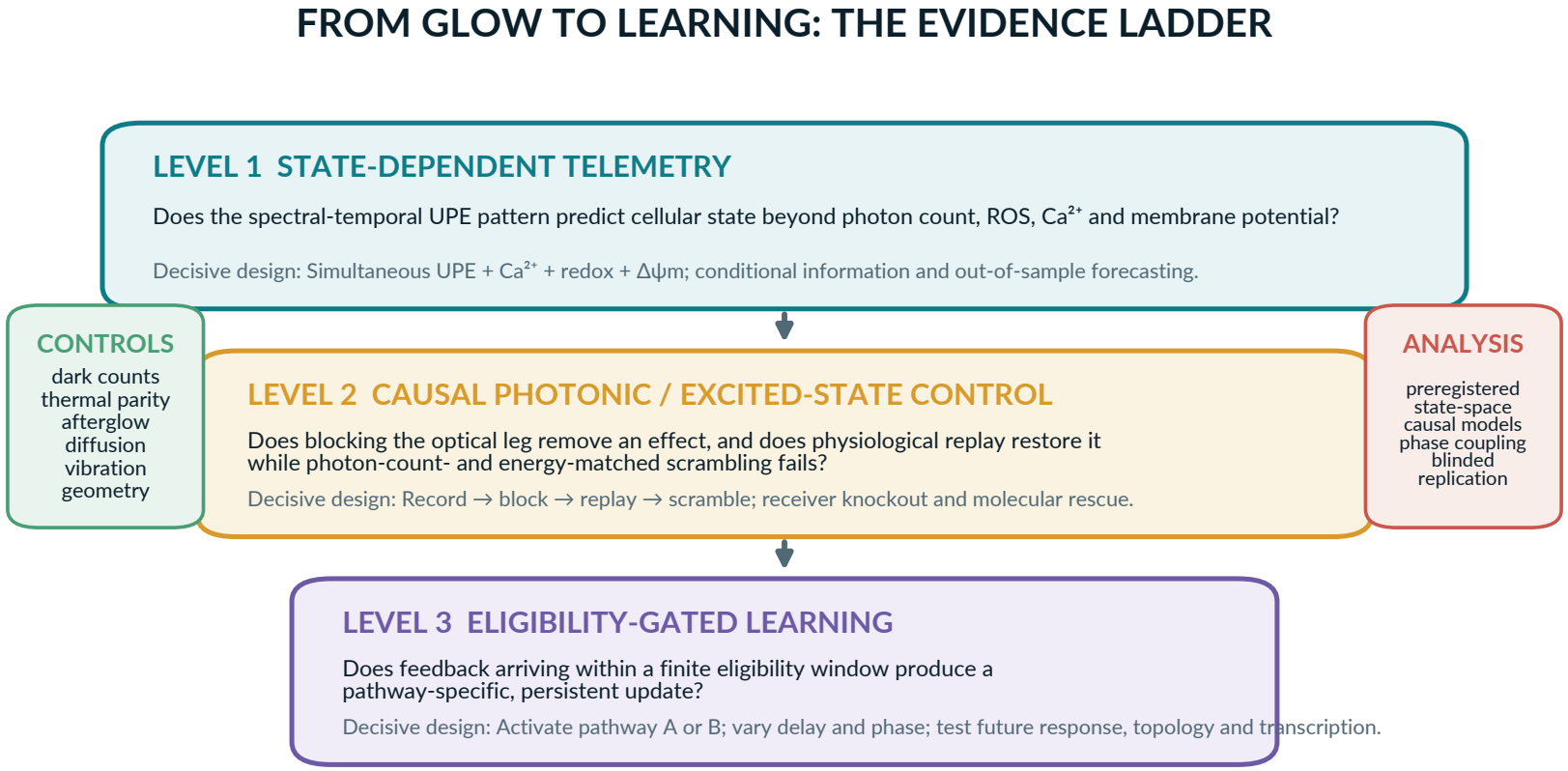
P8. Routing changes exceed source changes. Cytoskeletal perturbation alters spatial anisotropy, correlation length, and synchronization more strongly than mean photon count if it contributes primarily to routing.

P9. Topology shifts sensitivity. TFAM, topoisomerase, hydration, or mtDNA-abundance perturbation shifts an optical/THz response function if nucleoid topology contributes to transduction.

P10. Photon load and fidelity dissociate. Oxidative stress can increase UPE counts while reducing structured predictive information and phase coupling.

P11. Challenge-response diagnostics outperform resting brightness. The impulse response and recovery trajectory discriminate cell states better than a static photon count.

P12. Nested-scale coupling. Cellular photonic-redox variables predict or alter transitions in tissue-level bioelectric attractors only when classical intercellular amplifiers remain available.



A signal becomes a learning mechanism only after structure-sensitive causality and persistent credit assignment are demonstrated.

Figure 7: The evidence ladder. State-dependent telemetry is scientifically useful but does not establish function. Causal control requires blocking and replay with energy-matched scrambling. A learning claim requires a finite eligibility window, pathway specificity, and a persistent change in future response.

12. A decisive experimental program

12.1 Principle: record, block, replay, scramble, rescue

The central experimental logic is simple:

$$\text{record} \rightarrow \text{block} \rightarrow \text{replay} \rightarrow \text{scramble} \rightarrow \text{molecular rescue}. \tag{27}$$

Correlation between UPE and state establishes telemetry. Blocking the optical band while preserving source chemistry tests necessity. Replaying the measured physiological pattern tests sufficiency. Time-, phase-, wavelength-, or location-scrambling at matched photon number and energy tests whether structure matters. Knockout and rescue identify the receiver.

12.2 Experiment 1: isogenic noncoding response architecture

Create human induced-pluripotent-stem-cell lines or neuronal co-cultures that differ only at rs7304986 by base editing. Include T/T, T/C, and where feasible C/C genotypes with multiple independently derived clones. Characterize baseline Cav1.2 abundance, isoforms, chromatin contacts, membrane electrophysiology, and network dynamics. Apply the published 3.6-GHz, 700-MHz, and sham protocols, plus continuous-wave, envelope-scrambled, and matched-thermal controls.

Readouts should include high-speed voltage and multi-ion imaging, calcium-channel currents, mitochondrial calcium, $\Delta\psi_m$, oxygen consumption, NADH/FAD, ROS, UPE, transcriptomics, and persistent post-exposure response. The strongest result would be a genotype-specific difference in gain or phase that survives clonal replication and is reversed by editing the allele back.

A null result would suggest that rs7304986 marks linked background variation in the human study or that the phenotype requires whole-organism sleep circuitry. Either outcome narrows the model productively.

12.3 Experiment 2: factor carrier, envelope, phase, and dose

Use a factorial exposure matrix:

- carrier: 700 MHz, 3.6 GHz, and appropriate controls;
- temporal structure: original, continuous, time-scrambled, phase-randomized, and envelope-only surrogate where physically meaningful;
- absorbed energy: matched across conditions;
- endogenous phase: exposure initiated at controlled calcium, circadian, or network phases;
- receiver state: genotype, cell identity, metabolic state, and channel blockade.

The prediction is an interaction, not a universal monotonic dose response. A purely thermal model predicts convergence under matched temperature and absorbed energy. A

waveform-sensitive transduction model predicts residual differences associated with temporal structure and receiver state.

12.4 Experiment 3: CYB5B pathway localization

Reproduce the original electromagnetic-field-inducible gene-switch system under its published field conditions [21]. Use CYB5B knockout and rescue, calcium-channel perturbation, redox-state manipulation, and time-resolved calcium imaging. Then ask whether the field changes bulk calcium, oscillatory structure, phase relationships, or only reporter output.

Only after reproducing the defined mechanism should the same molecular components be tested under other field classes. This prevents inappropriate extrapolation from a therapeutic engineered system to ordinary wireless exposure.

12.5 Experiment 4: single-organelle temporal alignment

Simultaneously measure UPE, mitochondrial flashes, calcium, $\Delta\psi_m$, NADH/FAD, ROS, and oxygen consumption in isolated mitochondria or optically accessible cells. Because fluorescence excitation can overwhelm UPE, use time-multiplexed measurement, label-free sensors where possible, or alternating dark acquisition windows with carefully quantified afterglow.

The first question is temporal: does a reproducible sequence occur?

$$Ca^{2+} \rightarrow \Delta\psi_m \rightarrow \text{redox transition} \rightarrow \text{UPE} \rightarrow \text{recovery}. \tag{28}$$

The second question is informational: do spectral-temporal UPE variables add out-of-sample predictive value beyond standard redox measurements?

12.6 Experiment 5: record-block-replay in isolated mitochondria

Use two sealed mitochondrial populations separated by a controlled optical path. Record source emission with calibrated single-photon detectors or spectral channels. Include quartz, ordinary glass, opaque, and narrow-band filter conditions; gas-tight chemical separation; vibration isolation; matched temperature; and ambient-light control. Stress the source population, then replay the recorded photon point process to the receiver after blocking the natural path.

Compare:

- exact replay;
- photon-count- and energy-matched Poisson replay;
- time-scrambled replay;
- wavelength-scrambled replay;
- phase-shifted replay relative to a receiver reference;
- spatially shifted replay;

- dark control.

A structured replay effect that survives strict chemical and thermal controls and fails under matched scrambling would move the field from suggestive coupling toward causal optical information.

12.7 Experiment 6: biological lock-in

Induce a stable calcium or metabolic oscillation in a receiver preparation. Deliver identical low-flux photon patterns at fixed phases of the oscillation. Compare in-phase, antiphase, and random-phase conditions. Measure immediate local state change and persistent response.

The critical result is not a difference in average ROS. It is a phase-response curve: biological effect as a function of delay relative to the endogenous reference. If the curve is flat, the lock-in hypothesis is weakened. If it is structured and receiver-state dependent, the background-light objection acquires a concrete physical answer.

12.8 Experiment 7: geometry and source coordinate

Use micropatterned substrates to control cell shape and cytoskeletal orientation. Reposition mitochondria through motor-protein or tethering tools. Deliver the same calibrated optical point process at distinct coordinates: near nucleus, plasma membrane, ER-mitochondrial contact, microtubule-aligned mitochondrion, and geometrically isolated mitochondrion.

The prediction is that response follows the realized geometry. Reversible restoration of geometry should restore the original impulse response. A response determined only by total delivered energy would argue against geometry-addressed signaling.

12.9 Experiment 8: eligibility-window credit assignment

Activate pathway A in one spatial region and pathway B in another, using orthogonal receptors or optogenetic/chemogenetic tools whose excitation is separated in time from UPE acquisition. Deliver the same feedback pattern after delays spanning milliseconds to minutes. Test whether future response, channel abundance, chromatin accessibility, cytoskeletal organization, or transcription changes selectively in the recently active pathway.

The defining result is a three-way interaction:

$$\text{pathway identity} \times \text{feedback timing} \times \text{persistent update}. \tag{29}$$

General phototoxicity would not show pathway-specific eligibility.

12.10 Experiment 9: mtDNA topology and THz double dissociation

Combine 34.5- and 36.1-THz exposure with:

- TFAM overexpression and depletion;

- mitochondrial topoisomerase perturbation;
- rho-zero or mtDNA-depleted cells with rescue where feasible;
- mitochondrial-calcium-uptake inhibition;
- matched thermal dosimetry;
- UPE, nucleoid imaging, calcium, $\Delta\psi_m$, respiration, and biogenesis readouts.

If calcium is the downstream amplifier, calcium blockade should abolish PGC-1α-NRF1/2-TFAM induction. If topology contributes upstream, TFAM/topology perturbation should alter the sensitivity function even when calcium influx machinery remains available. Persistence without mtDNA would shift the model toward membranes or protein chromophores.

12.11 Experiment 10: cytoskeletal routing

Perturb microtubules and actin reversibly while measuring total UPE, source location, spatial anisotropy, correlation length, calcium propagation, mitochondrial synchronization, and persistent state. A routing role predicts altered spatial and temporal organization disproportionate to changes in total photon production. A purely mechanical or transport explanation predicts changes that track organelle motion and metabolism without an independent optical signature.

12.12 Experiment 11: modern Gurwitsch assay

A rigorous onion-root replication should measure, not infer, the source emission. Use automated mitotic mapping, quartz/glass/opaque/narrowband barriers, gas-tight separation, matched temperature/humidity, vibration control, source metabolic inhibition, dead-root controls, and recipient cell-cycle staging. Record the source point process and perform replay after blocking.

The most informative result would be an effect in competent recipient tissue produced by the natural and faithfully replayed pattern but not by matched scrambling. Failure under these conditions would substantially weaken the long-range inter-organism optical branch while leaving local intracellular reabsorption as a separate question.

12.13 Experiment 12: nested morphogenetic coupling

In planaria or another regenerative model, simultaneously map membrane voltage, calcium, mitochondrial state, and UPE during transitions among bioelectric attractors. Perturb the candidate optical leg while preserving gap-junctional and metabolic function as much as possible. Test whether UPE variables merely report state or causally shift the probability of a regenerative outcome.

A positive result must show that optical intervention changes anatomy through an identified cellular receiver and that classical intercellular coupling is required to amplify the local effect. This would connect subcellular reafference to tissue-level goal states.

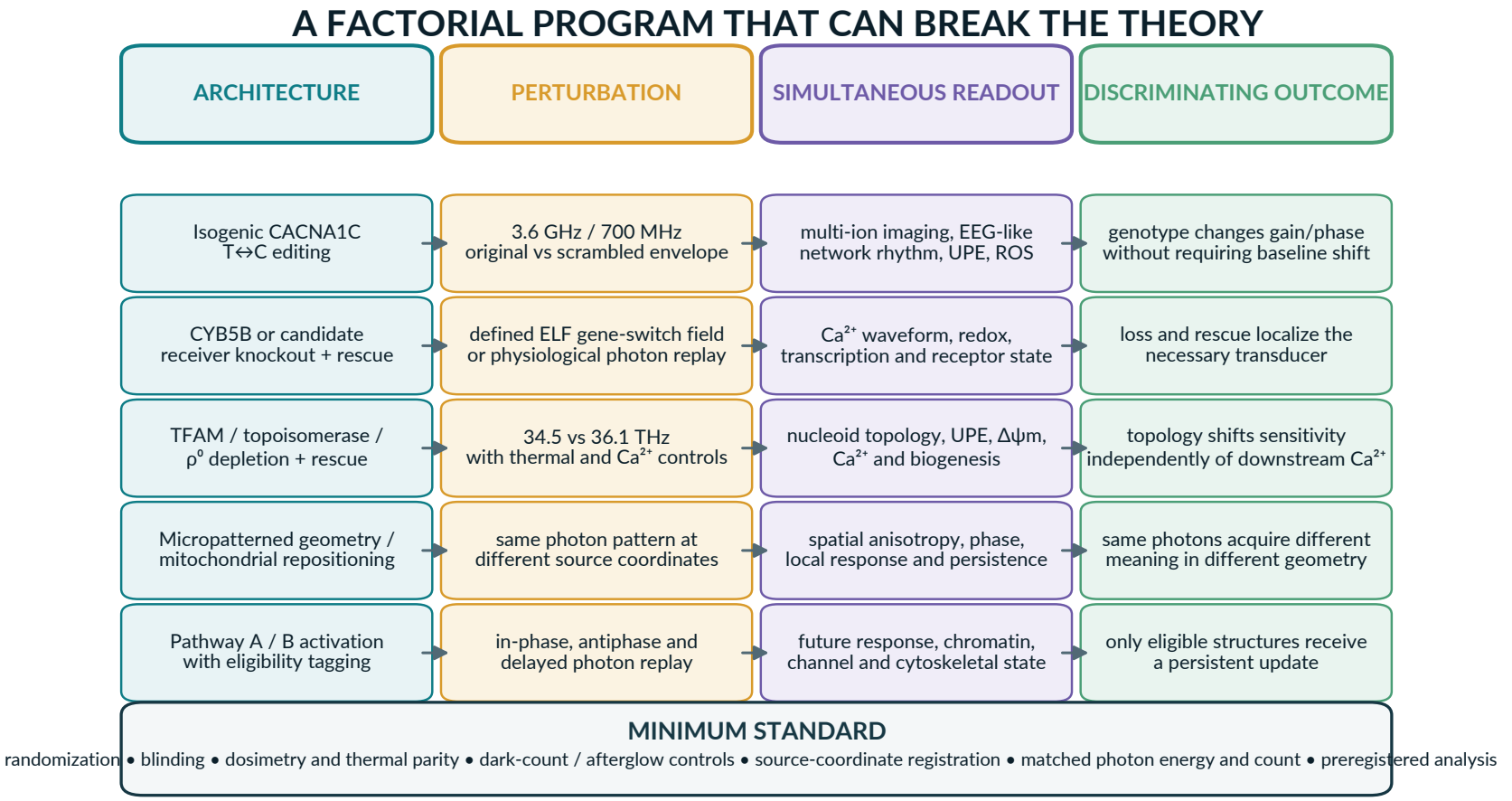


Figure 8: A factorial experimental roadmap. Architecture, perturbation, simultaneous readout, and discriminating outcome must be varied together. The theory is strongest when a specific mechanism can be removed and rescued, when equal-energy scrambling fails, and when geometry or phase changes the response.

12.14 Minimum reporting standard

- preregistration of primary endpoint and analysis;
- randomization and blinded acquisition/analysis;
- calibrated dosimetry, temperature, and environmental monitoring;
- detector dark counts, cosmic-ray rejection, afterglow, spectral calibration, and ambient-light characterization;
- strict diffusion, gas, vibration, and mechanical controls for separated preparations;
- simultaneous or time-multiplexed measurement of UPE, ROS, calcium, $\Delta\psi_m$, respiration, and geometry;
- registration of source coordinate and receiver state;
- matched photon number and delivered energy for scrambled controls;
- multiple independent clones, preparations, days, and laboratories;
- publication of raw photon-event streams and exposure waveforms.

12.15 Computational and AI analysis

AI can contribute immediately, but prediction must not be confused with mechanism. The appropriate sequence is:

1. fit state-space or point-process models to simultaneous ion, redox, UPE, and geometry data;
2. test whether UPE-derived features improve preregistered out-of-sample forecasting;
3. estimate conditional information, phase-amplitude coupling, transfer entropy, and graph-temporal dependencies;
4. compare models with and without topology, genotype, or source-coordinate variables;
5. use interventions to orient causal edges;
6. derive the smallest experiment that distinguishes leading mechanisms.

Graph neural differential equations can represent dynamic cellular topology; hidden-state models can infer latent execution phases; causal representation learning can identify which photon features survive control for ROS and $\Delta\psi_m$. The decisive evidence, however, remains experimental block, replay, scramble, knockout, and rescue.

13. Implications

13.1 Diagnostics should measure impulse response

Biophoton diagnostics are often imagined as passive brightness measurements. The present framework predicts that the more informative assay will be a controlled challenge-response test. Apply a standardized metabolic, electrical, mechanical, or pharmacological perturbation; record the spectral-spatial-temporal UPE impulse response; measure recovery; and estimate loop fidelity.

A diagnostic signature might include:

- latency from calcium or redox event to UPE burst;
- spectral mixture and burst entropy;
- source anisotropy and propagation length;
- phase coupling to calcium or $\Delta\psi_m$;
- recovery time and hysteresis;
- added predictive value beyond conventional metabolic measurements.

This approach could explain why resting counts sometimes discriminate cell states but fail in intact tissue: the informative variable may be the transfer function, not the baseline emission.

13.2 Therapy should target state and phase, not wavelength alone

Photobiomodulation demonstrates that mitochondria and other cellular structures can respond to external light under appropriate conditions [54, 55]. The present theory predicts that therapeutic specificity may improve when wavelength, pulse timing, source geometry, metabolic state, and endogenous phase are jointly controlled. A phase-targeted low-flux intervention could, in principle, produce a different result from the same average dose delivered continuously.

This is a research implication, not a clinical recommendation. The same state dependence that permits beneficial tuning can create unanticipated effects. Mechanism-specific action spectra, phase-response curves, and receiver identification are prerequisites.

13.3 Receiver-aware exposure science

The CACNA1C and CYB5B studies make a general point: biological activity can be genotype dependent, waveform selective, and mediated by structured calcium dynamics [19, 21]. Exposure science should therefore record more than carrier and average power. It should preserve the full waveform and test interaction with receiver variables.

This does not invalidate SAR, temperature, or conventional dosimetry. It adds a second layer:

$$\text{biological trajectory} = \text{physical exposure} \otimes \text{receiver transfer function}. \tag{30}$$

The tensor-product symbol emphasizes interaction, not multiplication in a literal dosimetric formula.

13.4 Fidelity preservation as a unifying health principle

The framework suggests a general aim for longevity and prevention: preserve the reliability of control loops. Good sleep, circadian alignment, clean air, metabolic health, adequate nutrients, and reduction of avoidable toxic exposures can all be understood as protecting different components of the same control architecture. Electromagnetic exposure belongs within this broader fidelity ecology rather than above all other stressors.

The theory does not imply that every non-native input is harmful or that natural inputs are automatically safe. The relevant question is whether a condition measurably degrades or improves a defined biological task, over a defined timescale, in a defined receiver.

14. Minimal commitments, optional extensions, and failure modes

A rigorous theory should distinguish what must be true from what is merely compatible.

Table 4: The minimal theory and optional extensions.

Minimal commitments	Optional extensions not required by the core
Cells implement state-dependent, recurrent control through integrated molecular and bioelectric architecture.	Global or long-lived quantum coherence across an entire cell or organism.
Noncoding and structural variables can alter response derivatives.	A particular rs7304986 molecular mechanism before isogenic validation.
Metabolism generates structured UPE and excited-state chemistry.	Every UPE event is functional or intentionally emitted.
The whole-cell geometry can condition absorption, conversion, and amplification.	Microtubules are cell-scale fiber-optic cables.
A distributed eligibility field can make delayed feedback pathway-specific.	Literal chain-rule backpropagation in a cell.
Local timing and state gating can distinguish correlated events from background.	Optical carrier-phase coherence or laser-like emission.
Low fidelity is measurable as degraded coupling among stages.	One universal stressor or one universal disease mechanism.

The core model would be substantially weakened if:

- structured UPE variables add no predictive information beyond conventional redox measures across well-powered systems;
- optical blocking never changes a candidate effect under strict chemical and thermal controls;
- physiological replay cannot restore an effect, or matched scrambling is equally effective;
- source coordinate, geometry, and phase do not influence response;
- no finite eligibility window or pathway-specific persistent update exists;

- receiver knockout/rescue fails to localize any mechanism;
- topology perturbation never changes a proposed topology-dependent response.

Negative results need not eliminate all photonic biology. They can narrow the theory from learning signal to telemetry, from mtDNA to membrane chromophores, from radiative propagation to local non-radiative transfer, or from tissue-scale communication to intracellular state readback. A modular theory improves through such failures.

15. Discussion: where intelligence research should focus

The central mistake in many accounts of biological intelligence is the search for a privileged object that "contains" the intelligence: DNA, a brain, a microtubule, a mitochondrion, a membrane, or a quantum state. Intelligence is not a substance that can be extracted from one component. It is a property of a closed, adaptive organization in which components constrain one another across time.

The most productive research focus is therefore the **mapping among scales and timescales**:

1. How does molecular sequence constrain a manifold of possible structures?
2. How do noncoding DNA and RNA select context-specific gain, phase, and topology?
3. How does the present geometry transform environmental input into a multi-ion trajectory?
4. How does metabolism execute that trajectory and report its cost?
5. How does a sparse feedback event become locally addressable?
6. How is a local update accepted, rejected, or overridden by tissue-level goals?
7. How does repeated compensation change future interpretation?

The proposed breakthrough is that one mechanism can connect several of these questions. A forward bioelectric event marks the structures that participated. Metabolic execution generates a chemistry-tagged excited-state field. The field re-enters the same geometry. State and phase determine where it is converted. Local overlap updates the structures that remain eligible. Tissue constraints determine whether the update is retained. The next input is then processed by a slightly different transfer function.

This loop is neither purely chemical nor purely electromagnetic. It is a hybrid physical controller in which chemical identity, electric potential, ionic motion, mechanical geometry, hydration, redox, and light are different coordinates of the same embodied state.

15.1 The dual-purpose photon as an engineering analogy

US Patent 11,700,058 B2 describes an engineered Far-UVC system in which modulated light can carry data while the absorbed photon energy simultaneously performs germicidal photochemistry [56]. The patent does not prove that cells use an analogous network. It

provides an engineering existence proof for a general principle: one optical field can carry structured information and perform physical work.

The biological proposal is more integrated. The chemistry is both source and encoder. The emitted or transferred excitation is both readback and potential catalyst. The receiver is not a separate detector bolted onto the system; it is the living structure whose state created the event.

Engineered network: data source → modulator → optical field → detector → feedback.
Proposed cellular network: bioelectric state → metabolic chemistry → UPE/excited-state event → whole-cell transfer kernel → persistent re-weighting.

15.2 The central synthesis

The framework can now be stated in one continuous argument:

The genome specifies a space of possible cellular networks. Noncoding DNA and RNA help parameterize how those networks respond to change. The whole living structure instantiates the effective weights and delays. Membranes and transducers convert environmental events into a multidimensional ionic trajectory. Metabolism executes that trajectory and generates a redox-linked photonic audit trail. The emitted or transferred excited-state field re-enters the same geometry that generated it. Endogenous phase and temporary structural eligibility make sparse events locally meaningful. Their overlap changes the next transfer function. Cellular intelligence is the recurrent integrity of this loop; low-fidelity biology is its progressive loss of reliable coupling.

16. Conclusion

The Embodied Cellular Transfer Function hypothesis relocates cellular intelligence from any single molecule to the energized, recurrent organization of the whole living system. DNA and RNA remain central, but as sequence, topology, scaffolding, regulation, and memory within a distributed architecture. Membrane potential and ion flux provide a multidimensional state trajectory. Mitochondria execute and evaluate that trajectory through energy and redox chemistry. UPE provides a measurable optical trace and, potentially, a geometry-addressed reafferent signal.

The strongest new claim is that a forward event changes the receiving structure before the photon arrives; photonic or excited-state feedback is therefore interpreted by matter already configured by the event it is evaluating. The active cell becomes the address. Endogenous oscillations provide a reference phase. Geometry and attenuation provide locality. Classical biochemical networks provide amplification. Persistent remodeling provides

memory.

This architecture solves the low-photon, background-light, addressing, and credit-assignment problems without requiring global coherence or a hidden controller. It also yields a disciplined definition of low-fidelity biology and a research program capable of decisive failure.

The final thesis is therefore:

Bioelectric dynamics initiate the action.
The whole cell executes it.
Metabolism generates a photonic-redox reafferent field.
The whole cell is the eligibility field.
Geometry provides the address.
State and phase provide the meaning.
Local overlap assigns the credit.
Remodeling stores the learning.

The cell does not merely read its environment. Through metabolism, structure, and light, it may continually read—and rewrite—the physical model of itself.

Glossary of proposed terms

Term	Definition
Embodied cellular transfer function	The time-varying operator by which the integrated physical cell converts input history into state transitions and outputs.
Latent response phenotype	A difference in response gain, phase, threshold, damping, or attractor stability that is not obvious at baseline.
Response Jacobian	The local derivative of cellular dynamics with respect to state and input; a compact representation of sensitivity and coupling.
Complex biological weight	A frequency-dependent coupling described by gain and delay/phase, shaped by geometry and state.
Self-encoding photochemistry	Excited-state chemistry in which the reaction that occurred determines the statistical structure of the emitted photon process.
Photonic-redox audit trail	UPE and associated excited-state variables considered as a compressed readback of metabolic execution and recovery.
Geometry-addressed photonic reafference	Self-generated optical/excited-state feedback whose consequence depends on source coordinate and the cell's current transfer kernel.
Biological lock-in	State- and phase-dependent reception that preferentially integrates correlated events while attenuating uncorrelated background.
Structural eligibility field	The distributed, temporary change in susceptibility left in structures that participated in a preceding event.
Photonic loop fidelity	The structured predictive or causal relationship among execution, photon-event features, and future cellular state beyond raw photon count.
Bioelectric dissonance	Task-specific loss of timing or causal coupling among ion dynamics, execution, recovery, and feedback.
Fidelity debt	The accumulating cost of compensatory remodeling that preserves short-term performance while reducing future robustness.
Somatic overfitting	Persistent adaptation to a distorted local condition that impairs performance when the cell returns to or encounters its native environment.
Low-fidelity biology	Declining reliability among transduction, execution, readback, and correct update across nested biological control loops.

Author and disclosure statement

Author: John Coates, RF Safe, Seminole, Florida, USA.

Conceptual origin: This manuscript develops the author's Cellular Latent Learning Model, photonic-redox control-plane hypothesis, low-fidelity biology framework, and related RF Safe concepts into an integrated and experimentally falsifiable theory.

Competing interests: The author is founder of RF Safe and is the named inventor on US Patent 11,700,058 B2, discussed as an engineering analogy in this manuscript. The author advocates research and policy attention to potential biological effects of electromagnetic exposure.

Use of generative AI: Generative AI was used for literature organization, conceptual stress-testing, drafting assistance, equation and figure development, and editorial refinement. Scientific interpretations, hypotheses, and responsibility for the final manuscript remain with the author.

Article status: Hypothesis and theory manuscript. It has not been peer reviewed and is intended to motivate critical discussion, preregistered experimentation, and independent replication.

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