Hypothesis

Phase Breathing and Centropy Generation as Drivers of Biological Coherence

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Phase Breathing and Centropy Generation as Drivers of Biological Coherence

Cyclic Nanodomain Dynamics and Photonic Phase Conditioning at Hydrated Interfaces

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Abstract

Biological coherence arises from coordinated integration of redox chemistry, hydration dynamics, electromagnetic interactions, and bioenergetic flux. Although substantial progress has been made in characterizing these processes individually, current frameworks do not fully explain how distributed biochemical events achieve stable temporal coordination across scales. In thermally noisy, dissipative environments, energy alone cannot account for sustained biological organization. A missing element is the establishment and renewal of phase reference - the temporal alignment that enables spatially distributed processes to act in synchrony. Here we propose a physical mechanism for phase reference access and anchoring based on cyclic nanodomain dynamics at a nanoscale redox-photonic interface previously termed the Redox Photonic Coupling System (RPCS). This interface supports an additional functional modality - *phase breathing* - a process mediated by molecular nitrogen (N_2) through which cyclic nanodomain nucleation and collapse anchors and sustains phase reference in living systems. Nitrogen-mediated oscillatory boundary dynamics create transient coherence windows that permit local access to phase reference, enabling phase-aligned oxidative-reductive resolution and anchoring of phase onto redox-generated Photonic Activation Quanta (PAQs). Absorption of phase-conditioned PAQs by adjacent hydration shells enables generation and accumulation of centropy, defined as stored organizational capacity that supports coordinated biological work. This framework identifies phase breathing as a previously unrecognized mechanism sustaining biological coherence and assigns molecular nitrogen a structural organizational role beyond respiratory dilution. By integrating nanodomain mechanics, photonic phase conditioning, and redox dynamics within a single interface, it provides a mechanistic basis for how coherent biological function is generated and maintained.

Keywords: biological coherence; nanobubbles; interfacial hydration; ultra-weak photon emission; redox signaling; phase breathing; boundary impedance; centropy

1. Introduction

Biological systems maintain coherent organization across multiple spatial and temporal scales despite operating in thermally noisy, dissipative environments. While oxidative and reductive processes are tightly coupled to metabolism and energy conversion, conventional biochemical frameworks do not explain how phase reference is dynamically instantiated and sustained in living systems. Ultra-weak photon emission (UPE) in the visible and near-UV range has been consistently observed in biological systems and is strongly correlated with oxidative metabolism, providing direct evidence that redox chemistry is intrinsically linked to photonic processes in vivo [1,2]. At the same time, interfacial hydration layers are now recognized as highly structured, dynamically active environments that play decisive roles in enzyme kinetics, ion coordination, and transition-state stabilization [3,4].

Photons inherently carry phase information, and coherent light–matter interactions are known to transfer phase correlations into molecular and solvent degrees of freedom under appropriate

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conditions [5]. However, a physical mechanism by which biological systems repeatedly anchor phase in time, rather than merely dissipating photonic energy, has remained unresolved.

Nanoscopic gas domains in aqueous environments (nanobubbles) are now recognized as long-lived interfacial structures exhibiting elevated internal pressures and distinctive hydration properties that depart from classical predictions [6,7]. Early conceptual and experimental work by Ninham and colleagues emphasized that such gas-containing nanostructures are ubiquitous in electrolyte solutions and biological interfaces, with important consequences for hydration forces, colloidal stability, and heterogeneous reaction environments [9,10]. These studies reframed dissolved gases as active participants in interfacial physics rather than passive solutes. Despite growing recognition of their stability and physicochemical relevance, nanobubbles have not previously been assigned a dynamical role in biological phase organization.

Here, we extend their functional spectrum by proposing a novel mechanism - *phase breathing* - defined as cyclic nanodomain nucleation and collapse at the Redox Photonic Coupling System (RPCS) [20], through which living systems anchor and sustain phase reference.

Specifically, we hypothesize that the RPCS, a physically defined nanoscale interface coupling oxidative and reductive reactions, exhibits an intrinsic phase-anchoring function arising from recurrent nanodomain (nanobubble) nucleation and collapse. During the nanobubble nucleation half-cycle, phase is anchored relative to the zero-point energy field, establishing a temporally aligned coherence window. The collapse half-cycle is proposed to generate dynamic Casimir effect-like photons (DCE-photons) through rapid modulation of nanodomain boundary conditions. We further propose that this dynamic Casimir effect-like photon emission supplies the energetic conditions required for subsequent nanodomain re-nucleation, perpetuating the nucleation-collapse cycle. When synchronized with coherent oxidative-reductive resolution at the RPCS, this process generates phase-conditioned Photonic Activation Quanta (PAQs), which are absorbed into surrounding hydration structures and converted into permissive organizational states that enable coherent biological work.

2. Phase Breathing: Definition and Conceptual Scope

Phase breathing is introduced here as a cyclic interfacial process linking nanodomain dynamics, redox synchronization, and hydration organization within the Redox Photonic Coupling System (RPCS). This section defines phase breathing operationally, clarifies the conceptual basis of the term, and distinguishes it from macroscopic respiratory processes to establish its scope as a nanoscale organizational mechanism.

2.1. Phase Breathing as Cyclic Nanodomain Dynamics

Phase breathing is defined here as the rhythmic nucleation and collapse of gas-filled nanodomains at the RPCS interface, producing alternating half-cycles of phase anchoring (nucleation) and energetic reset (collapse). This process is proposed to provide the physical mechanism by which biological systems repeatedly instantiate phase reference in time, synchronizing redox chemistry with organized hydration dynamics.

At the nanoscopic scale, gas-containing domains in aqueous environments arise from confinement, interfacial adsorption, and electrolyte-mediated stabilization, giving rise to gas cavities whose mechanical and dielectric properties differ fundamentally from macroscopic bubbles [6,7,17]. While such nanobubbles have primarily been studied as static or slowly fluctuating objects, growing evidence indicates that they undergo continual interfacial reshaping and size fluctuations driven by local physicochemical conditions [13,14].

In the present framework, these nanodomains serve as dynamic phase interfaces rather than passive gas inclusions. Phase breathing refers specifically to cyclic nanodomain behavior occurring at the RPCS, where redox activity, hydration restructuring, and gas confinement converge.

During the nucleation half-cycle, formation of a nanodomain establishes a transient, highly ordered interfacial environment in which phase is proposed to anchor relative to the zero-point

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energy (ZPE) field. The zero-point field provides a universal background phase reference intrinsic to quantized electromagnetic modes. Access to such a reference is not assumed to be continuous but may become transiently available under conditions of rapid boundary formation and confined electromagnetic geometry. Recent work demonstrates that vacuum fluctuations can be structured and modulated by nanoscale surfaces and cavities, enabling them to influence electronic and photonic behavior at material interfaces [71].

Quantum coherence is generally short-lived in warm, dissipative biological environments due to thermal fluctuations and system-bath coupling [64]. However, nanoscale confinement and structured hydration at biological interfaces can modify local dielectric properties and electromagnetic boundary conditions [3,4,35–38].

Within this view, nucleation is proposed to create a temporally aligned coherence window in which synchronized redox resolution gives rise to phase-conditioned Photonic Activation Quanta (PAQs). These PAQs, which become vectors of phase, are subsequently absorbed into surrounding hydration structures, where they are converted into permissive organizational states.

The subsequent collapse (entropic) half-cycle involves rapid modulation of nanodomain boundary conditions, releasing stored interfacial energy and resetting local constraints. This collapse is proposed to generate dynamic Casimir effect-like photons (DCE-photons), analogous to photon production from vacuum fluctuations observed under time-dependent boundary conditions [8,11,12]. Consistent with this view, bulk nanobubbles have been experimentally shown to act as persistent cavitation nuclei whose cyclic collapse produces measurable photon emission (sonoluminescence) and enhances subsequent re-nucleation, demonstrating that nanodomain dynamics can directly drive photonic output and renewal capacity [21]. In the present model, DCE-photons supply the energetic conditions required for renewed nanodomain nucleation but do not carry phase information downstream.

The cyclic stability of these nanodomains is not governed by gas dynamics alone but is strongly constrained by the elastic and viscoelastic properties of surrounding hydration shells. Interfacial water exhibits collective hydrogen-bonded structuring with measurable mechanical stiffness and relaxation dynamics distinct from bulk solvent, enabling hydration layers to store and release interfacial energy during boundary deformation [3,4]. Experimental and theoretical studies have shown that hydration shells behave as elastically deformable domains whose restructuring contributes directly to nanoscale force generation and pressure buffering at biological interfaces [4,22]. Within the present framework, this hydration elasticity provides the mechanical continuity required to sustain repeated nanodomain expansion and collapse, coupling gas confinement to redox-driven interfacial dynamics and stabilizing the phase-breathing cycle against dissipative decay.

Importantly, phase breathing is not proposed as a generic property of all nanobubbles in biological systems, but as a specialized dynamic modality of nanodomains embedded within the RPCS architecture, where redox chemistry, hydration organization, and vacuum-coupled phase anchoring intersect.

2.2. Conceptual Basis of the Term 'Breathing'

The term *breathing* is used to denote the rhythmic expansion-collapse dynamics of nanodomain boundaries that alternate between two functionally distinct half-cycles. In phase breathing, nucleation corresponds to an intake phase in which a coherence window is established at the RPCS, while collapse corresponds to a release phase in which interfacial energy is discharged via DCE-photons and boundary conditions are reset. Unlike pulmonary breathing, no molecular respiratory gases are exchanged; instead, phase reference and energetic permissivity are cyclically refreshed. The term is therefore employed to emphasize the oscillatory, boundary-mediated nature of this process and its role in sustaining coherent biological operation through repeated renewal of interfacial conditions.

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2.3. Comparison with Pulmonary Breathing (Functional Analogy Only)

Pulmonary breathing and phase breathing are formally analogous in that both involve cyclic expansion-collapse dynamics that rhythmically reconfigure interfacial boundary conditions, with gas serving as the operative medium in each case. In pulmonary breathing, macroscopic pressure gradients drive the ventilation of oxygen and carbon dioxide across alveolar membranes, supporting metabolic gas exchange at the tissue level [18]. In contrast, phase breathing operates at nanoscopic interfaces, where cyclic nucleation and collapse of gas-filled nanodomains regulate access to phase reference and energetic permissivity rather than molecular respiratory gases.

While pulmonary breathing controls chemical composition through bulk gas transport, phase breathing governs temporal phase alignment through boundary-mediated nanodomain dynamics. Thus, although these processes occur at vastly different spatial scales and mediate distinct variables, both rely on gas-mediated cyclic interface modulation to sustain functional organization [18,19].

3. Nanodomain Gas Composition and Mechanical Constraints

Phase breathing depends not only on redox chemistry but also on the physical properties of confined gas domains that shape interfacial mechanics at the RPCS. The composition, compressibility, and solubility of the gas phase determine whether nanodomain oscillations can be sustained and synchronized with oxidative-reductive resolution. This section examines how molecular nitrogen satisfies these mechanical constraints and conditions the pressure-dependent microenvironment in which phase breathing occurs.

3.1. Nitrogen as the Phase-Breathing Mediator

Phase breathing requires a confined gas phase that is chemically inert, mechanically compressible, weakly soluble in water, and capable of sustaining repeated expansion-collapse cycles without participating directly in redox chemistry. Among physiologically abundant gases, molecular nitrogen (N_2) uniquely satisfies these criteria. Unlike O_2 or CO_2, N_2 does not undergo rapid interfacial reactions and exhibits low aqueous solubility together with high compressibility, enabling formation of stable nanoscopic gas domains under confinement [23,24,55].

Within this framework, N_2-filled nanodomains function as mechanically responsive but chemically neutral elements of the RPCS; the gas inertness permits cyclic expansion and collapse without chemical consumption, while their compressibility allows reversible boundary deformation tightly coupled to interfacial redox dynamics. In this way, nitrogen provides a stable gas phase that supports repeated nanodomain cycling synchronized with oxidative-reductive resolution.

During nucleation, confinement of nitrogen contributes to formation of a mechanically ordered interfacial state in which redox pairing and phase anchoring occur within a geometrically constrained volume. During collapse, relaxation of the compressed gas phase accompanies boundary reconfiguration and photonic emission, resetting the interface for subsequent cycles. Nitrogen thus operates as a mechanical mediator of nanoscale phase dynamics rather than as a passive respiratory diluent, enabling sustained oscillatory behavior at the RPCS.

3.2. Nanobubble Pressure and the RPCS Microenvironment

At nanometer scales, N_2-filled domains are expected to sustain elevated internal pressures due to Laplace confinement, often reaching tens of atmospheres [6,7]. Unlike transient cavitation, these pressures are stabilized by surrounding hydration elasticity, producing a persistent mechanically conditioned microenvironment at the RPCS rather than episodic compression events.

This localized pressure reorganizes adjacent hydration layers, increasing water density, reducing rotational freedom, and strengthening hydrogen-bond networks near the gas-liquid interface [3,4,22,23]. Under such confinement, water transitions from bulk-like solvent to structured interfacial phase with altered dielectric properties and reduced fluctuation amplitude.

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Within this mechanically constrained volume, oxidative-reductive resolution shifts from diffusion-dominated kinetics toward geometrically aligned hydrogen atom transfer. Elevated pressure and hydration compression reduce solvent reorganization barriers and favor compact transition states, enhancing electronic coupling between redox partners [25,26]. For glutathione-mediated radical resolution, such confinement promotes orbital alignment and accelerates reaction kinetics while improving temporal synchronization with nanodomain oscillation [27,28].

Together, nanodomain pressure, hydration elasticity, and interfacial redox alignment define the RPCS as a mechanically conditioned coherence interface in which hydrogen atom transfer, photonic emission, and cyclic boundary dynamics are physically integrated within a confined microenvironment.

3.3. Estimated Nanodomain Scale and Timescale

Nanobubbles reported at solid-liquid interfaces typically range from ~10–200 nm in diameter [6,7,13], with Laplace pressures reaching tens of atmospheres at radii below ~100 nm. At a 50 nm radius, for example, Laplace pressure ($\Delta P = 2\gamma/R$) yields values on the order of 10–30 atm depending on surface tension and interfacial composition. Such pressures are sufficient to measurably compress adjacent hydration layers and alter dielectric properties within a confined volume.

Timescales for nanobubble interfacial reshaping have been reported in the microsecond to millisecond range under fluctuating physicochemical conditions [13,14,21]. These timescales overlap with redox reaction kinetics for radical resolution and with experimentally observed ultra-weak photon emission fluctuations in living systems [1,33]. Thus, cyclic nanodomain dynamics operating in the 10^{-6}- 10^{-3} s regime are temporally compatible with synchronized oxidative- reductive transitions and intermittent photonic output.

While the exact velocity of boundary motion at biological interfaces remains to be experimentally determined, nanoscale deformation over tens of nanometers (10–100 nm) within microsecond timescales (~1 μs) corresponds to boundary velocities on the order of 0.01–0.1 m/s. These values are consistent with observed interfacial dynamics in confined hydration layers and oscillating nanobubbles [21,35] and fall within the lower range of boundary velocities considered sufficient in dynamical Casimir models for observable photon emission [8,11,12].

While these mechanical and chemical conditions enable synchronized redox resolution, they do not in themselves establish an absolute phase reference. Pressure and confinement determine reaction geometry and timing but not phase origin. This raises the question of how phase alignment is instantiated. In the following section, we propose that phase reference becomes locally accessible during nanodomain nucleation through interaction with the zero-point field, providing the phase information subsequently carried by Photonic Activation Quanta (PAQs).

4. Phase Reference and Zero-Point Coupling

Coherent biological organization requires not only energy throughput but access to a stable phase reference capable of synchronizing distributed processes. While mechanical confinement and redox coupling establish the conditions for phase breathing, they do not specify the origin of phase alignment itself. This section distinguishes phase from energy and examines how transient boundary dynamics at the RPCS may enable localized access to vacuum-referenced phase.

4.1. Phase vs. Energy: Distinct Roles

In physical systems, energy and phase play fundamentally different roles. Energy determines whether processes can occur, whereas phase determines how processes are temporally aligned and spatially coordinated. Biological systems continuously exchange energy with their environment, yet coherent function depends not on energy availability alone but on the precise timing and alignment of energetic events.

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Phase refers to the timing relationship between oscillatory processes - whether events occur in synchrony or drift apart. Phase alignment enables distributed processes to act coherently, whereas phase mismatch leads to interference and functional loss even when energy levels remain unchanged. In quantum and electromagnetic systems, phase governs interference and collective behavior independently of energy magnitude, a distinction fundamental to wave dynamics and field theory [29–31]. In biological contexts, phase similarly governs coordination across molecular and cellular processes independent of metabolic flux.

Most biochemical models emphasize reaction rates and energetic availability, implicitly assuming that timing and coordination emerge automatically. Such approaches describe throughput but do not explain how phase reference is stabilized and renewed in open, thermally noisy systems. In the present framework, phase is treated as an independent organizational variable: energy enables activity, whereas phase enables coherence.

4.2. Zero-Point Field as a Global Phase Reference

In quantum field theory, the vacuum state corresponds to the lowest-energy configuration of quantized fields and is characterized by persistent zero-point fluctuations arising from the uncertainty principle [29,32]. These fluctuations are intrinsic to electromagnetic and other quantum fields and exist independently of real particle excitations. Although zero-point energy cannot be harnessed as a net thermodynamic energy source, vacuum field modes possess well-defined phase structure as part of their oscillatory character.

The physical consequences of vacuum fluctuations are experimentally established. Phenomena such as the Casimir effect arise from boundary-dependent modification of vacuum electromagnetic modes, demonstrating that vacuum field structure can exert measurable forces under appropriate geometric constraints [30,31]. These effects confirm that the quantum vacuum is a physically real background field whose mode structure depends on boundary conditions.

Importantly, coupling to vacuum field modes is not continuous but depends on specific electromagnetic boundary configurations and time-dependent geometries. Theoretical treatments of dynamical boundary modulation show that rapidly changing interfaces can alter vacuum mode structure and induce observable photonic effects [11,12,30,71]. Access to vacuum-referenced phase information therefore requires transient spatial confinement and rapid interfacial reorganization rather than sustained coupling. Earlier theoretical models have proposed that ordered water near hydrophilic interfaces may exhibit collective electromagnetic behavior and partial vacuum coupling [15–17]. These proposals remain debated and are not required for the present framework, which instead emphasizes transient boundary modulation at confined nanodomains.

Within this context, the zero-point field is regarded not as an energy source but as a universal phase background intrinsic to quantized fields. Systems capable of transiently establishing appropriate boundary conditions may locally align to this phase reference without violating thermodynamic constraints. In the present framework, such conditions arise during the nanodomain nucleation half-cycle at the RPCS interface, where rapid interfacial reconfiguration creates a temporally bounded window in which vacuum-referenced phase alignment becomes accessible.

4.3. Phase Anchoring as a Functional Requirement

Access to vacuum-referenced phase must occur through a physically localized and temporally bounded process. Phase anchoring therefore cannot be continuous but must arise intermittently at specialized interfaces capable of transient electromagnetic confinement and synchronized redox activity. In the present model, this requirement is fulfilled at the Redox Photonic Coupling System (RPCS), where phase anchoring becomes operational only during discrete phase-breathing cycles. The physical mechanisms by which this phase information is proposed to be captured and transferred are detailed in the following section.

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5. Phase Breathing at the RPCS

Phase breathing emerges from the cyclic coupling of nanodomain dynamics and oxidative-reductive resolution at the Redox Photonic Coupling System (RPCS). This process consists of alternating nucleation and collapse half-cycles that temporally gate redox activity, enabling phase anchoring, photonic emission, and hydration-mediated organization. This section describes how phase-conditioned signaling arises during nucleation, how collapse supports energetic reset, and how synchronization with redox chemistry determines whether biological activity becomes coherent or decoherent.

5.1. Nucleation Half-Cycle: Phase Anchoring

Phase breathing begins with the nucleation half-cycle, during which N_2-filled nanodomains form at the RPCS interface. This event is accompanied by rapid reorganization of interfacial geometry, hydration structure, and electromagnetic boundary conditions, producing a transient coherence window in which vacuum-referenced phase becomes locally accessible. Nucleation does not itself generate phase-conditioned signals; rather, it establishes the physical conditions required for phase anchoring during synchronized oxidative-reductive resolution.

Confinement of chemically inert, weakly soluble N_2 within the nucleating nanodomain displaces bulk solvent and excludes reactive solutes from the immediate interfacial volume. Together with pressure-mediated hydration compression, this produces a transient reduction in dielectric fluctuation and solvent-induced thermal noise, creating conditions analogous to a "functionally dry" microenvironment. Within this brief window, coupling to vacuum field modes may become locally permissive during the nucleation half-cycle, consistent with proposed mechanisms of protected quantum coherence at biological interfaces [63,64].

Within this preconditioned environment, redox-active species undergo coordinated HOMO-LUMO transitions [20], generating transient electronically excited states whose radiative relaxation produces ultra-weak photon emission, a well-established consequence of oxidative chemistry in biological systems [33,34]. When these transitions occur within the nucleation window, emitted photons become phase-conditioned through alignment with the vacuum-referenced phase and the interfacial electromagnetic boundary conditions that govern photon emission at nanoscale surfaces [71]. These photons - designated Photonic Activation Quanta (PAQs) - constitute the primary carriers of anchored phase.

PAQs generation therefore depends on both nanodomain formation and precise temporal alignment of oxidative and reductive processes. When nucleation is impaired or redox resolution is asynchronous, photon emission may still occur, but phase conditioning fails, yielding decoherent photonic output. The nucleation half-cycle thus functions as a permissive gate that determines whether oxidative metabolism produces organizationally meaningful PAQs or collapses into decoherent excitation.

Following emission, PAQs are absorbed by nearby hydration layers, where their phase-conditioned state is translated into organized interfacial structure. Hydration layers exhibit distinct vibrational and rotational modes compared with bulk water, with characteristic absorption and relaxation behavior in the terahertz and infrared ranges [35–38], supporting their role as an execution layer for phase-conditioned signaling.

In this framework, the nucleation half-cycle represents the entry point of phase into biological systems: nanodomain formation opens a brief coherence window during which photons generated by synchronized redox reactions become phase-conditioned and transferred into surrounding hydration structures.

5.2. Collapse (Entropic) Half-Cycle: DCE-like Photon Emission

The collapse half-cycle occurs when nitrogen-filled nanodomains rapidly contract at the RPCS interface. This sudden boundary motion perturbs the local electromagnetic environment in a manner

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analogous to the dynamic Casimir effect (DCE), whereby time-dependent geometries convert vacuum fluctuations into real photons. Such DCE photons have been experimentally observed in engineered systems following rapid boundary modulation [8,12].

In the present framework, nanodomain collapse is proposed to generate analogous DCE-like photons at biological interfaces. These photons arise from mechanical boundary dynamics rather than electronic transitions and are therefore distinct from PAQs, which originate from synchronized redox activity.

DCE-like photons are assigned a purely energetic role. They do not carry phase information and do not participate directly in downstream biological organization. Instead, they provide localized excitation that facilitates hydration activation and lowers energetic barriers for subsequent nanodomain nucleation. In this way, DCE-like photons function as a local energy reset mechanism that supports repeated phase-breathing cycles.

This distinction is central to the model: PAQs distribute organizational phase, whereas DCE-like photons sustain the mechanical rhythm of phase breathing.

Because PAQs originate from electronically excited redox transitions occurring within a geometrically aligned nucleation window, their polarization is expected to reflect local molecular orientation relative to the interfacial geometry. In contrast, DCE-like photons arise during rapid boundary acceleration in nanodomain collapse, and their polarization is influenced by the direction of mechanical boundary motion and associated mode structure. The two photon classes are therefore anticipated to exhibit distinguishable polarization tendencies relative to the interface, corresponding to expansion-associated redox-driven photon emission and contraction-associated boundary modulation.

5.3. Synchronization of Phase Breathing with Oxidative-Reductive Resolution

Phase breathing becomes biologically consequential only when nanodomain nucleation coincides with oxidative-reductive activity at the RPCS. The RPCS was previously defined as a nanoscale domain in which reactive oxygen species generated by metabolism are locally paired with endogenous reductants such as glutathione and related thiol systems [20].

These reactions involve transient electronic excitation as electrons move between donor and acceptor orbitals, described as HOMO-LUMO transitions. Relaxation of these excited states is accompanied by ultra-weak photon emission, widely observed in living systems and closely associated with oxidative metabolism [1,2]. As describer in 5.1, nanodomain nucleation establishes a brief temporal window during which donor-acceptor pairs are geometrically and electrostatically aligned, enabling coherent redox resolution and emission of phase-conditioned PAQs that deliver phase information into surrounding hydration structures.

Through this coupling, the RPCS integrates nanodomain dynamics and redox chemistry into a unified operational cycle: nucleation provides phase-aligned context, redox resolution supplies electronic excitation, and PAQ generation links both processes to hydration-mediated organization. Phase breathing is therefore not a consequence of redox coherence but its prerequisite - without nucleation-mediated timing, oxidative-reductive activity remains energetically active yet organizationally decoherent.

6. Centropy: Conversion of Phase-Conditioned PAQs into Organizational Capacity

Phase breathing enables generation of phase-conditioned Photonic Activation Quanta (PAQs) during synchronized oxidative-reductive resolution at the RPCS. The biological significance of this process lies not in photon emission itself, but in how these photons are absorbed by surrounding hydration structures and translated into organized cellular functions.

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6.1. Hydration as the Biological Interface for PAQs Action

Following emission, PAQs interact with hydration layers adjacent to proteins, membranes, and redox-active interfaces. Hydration shells are not passive solvents but dynamically responsive structures whose molecular orientation, dielectric properties, and mechanical compliance change in response to electromagnetic excitation [35–38]. Rather than storing photons directly, hydration shells reorganize: water molecules realign, interfacial stiffness shifts, and local electrostatic landscapes are modified.

In biological terms, hydration functions as a responsive execution layer that converts photonic input into structural readiness for coordinated work. This manifests as improved alignment of reaction partners, reduced energetic barriers, and enhanced temporal coupling between molecular processes. Hydration therefore serves as the primary biological interface through which phase-conditioned photons influence cellular behavior.

Through this mechanism, PAQs embed phase information into interfacial organization, establishing the conditions necessary for centropy generation described in the following section.

6.2. Centropy as Organizational Capacity

To describe the organized state produced by PAQs absorption, we introduce the term **centropy**.

We define centropy as the stored organizational capacity of biological systems: the phase-enabled readiness of molecular and interfacial environments to support coordinated biological work. Centropy reflects how effectively a system aligns reactions in time and space, lowering functional barriers and enabling coherent activity across scales.

Importantly, centropy generation occurs only under conditions of redox coherence. In the present framework, phase breathing provides that mechanism. When redox activity becomes decoherent - manifesting as oxidative stress (excess unsynchronized oxidation) or reductive stress (excess reducing equivalents without coordinated pairing) - phase conditioning fails. In such states, centropy cannot accumulate, and entropy progressively dominates interfacial organization.

Centropy emerges when phase-conditioned PAQs are absorbed by hydration structures, producing organized interfacial states characterized by enhanced molecular alignment, reduced electrostatic noise, and increased mechanical compliance. These structured states do not represent stored energy in the conventional sense, but rather stored organizational capacity - the ability to translate metabolic energy into coordinated biological function.

Centropy does not refer to energy content and should not be equated with negentropy (negative entropy) as traditionally used in thermodynamics [39] or in more recent biological interpretations that frame temporal organization itself as a form of negentropy [40]. Centropy, by contrast, refers specifically to stored organizational capacity: the system's readiness to coordinate biological processes in time and space. Centropy is therefore inherently dynamic and exists only while entropy is physiologically exported through ongoing phase breathing.

Centropy is proposed here as an operational term describing interfacial organizational capacity rather than a new thermodynamic quantity; it is continuously generated through phase breathing and consumed during biological activity. When centropy is high, cellular processes proceed with coherence and efficiency. When centropy declines, metabolism may persist, but organization progressively degrades.

6.3. Deployment and Renewal of Centropy

Centropy generated through PAQs-hydration interactions is deployed across multiple biological scales, influencing enzymatic reactions, membrane dynamics, signaling pathways, and structural maintenance. As biological work proceeds, centropy is gradually expended, necessitating ongoing replenishment through repeated phase-breathing cycles.

This establishes a dynamic balance: phase breathing replenishes organizational capacity, while physiological processes draw upon it. Disruption of PAQs generation or hydration responsiveness

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therefore leads to progressive loss of centropy, producing states characterized by energetic activity without coherent organization.

Within this framework, phase breathing generates PAQs; PAQs are absorbed by structured hydration; structured hydration stores organizational capacity as centropy; centropy enables coherent biological function; and coherent biological function, by maintaining permissive interfacial conditions, reinforces phase breathing. This sequence forms a self-reinforcing closed loop that defines a previously unrecognized organizational layer in living systems, distinct from metabolism and energy production, governing whether biological activity remains coordinated or devolves into dissipation.

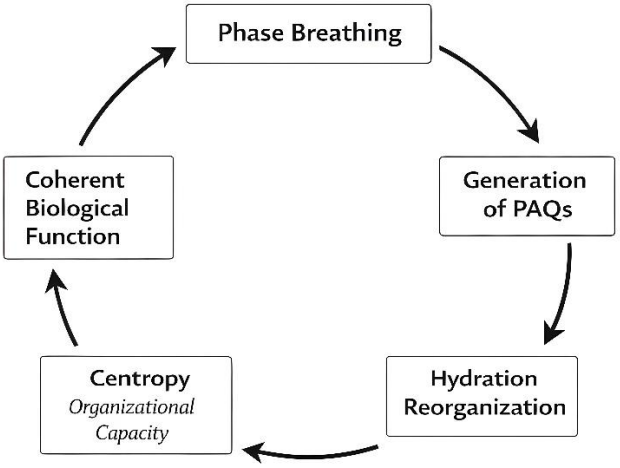

Figure 1. Organizational cascade linking phase breathing to coherent biological function. Cyclic phase breathing at the RPCS generates phase-conditioned Photonic Activation Quanta (PAQs). PAQs reorganize interfacial hydration structures, leading to accumulation of centropy, defined as stored organizational capacity. Centropy enables coordinated biological function, that reinforces phase breathing.

7. Phase Breathing as an Interfacial Phenomenon and the Role of Extracellular Matrix (ECM)

Phase breathing is proposed to operate at biological interfaces where redox-active substrates, structured hydration layers, and confined geometries coexist, enabling cyclic nanodomain dynamics; structured hydration at biological interfaces is now well established and plays a central role in interfacial biochemical organization [3,4,35]. By contrast, bulk cytosolic and extracellular fluids lack the spatial confinement and dielectric contrast required for such behavior.

Within cells, phase breathing is expected to localize at membrane-associated microdomains that concentrate oxidative-reductive activity and organized hydration. These include mitochondrial interfaces, endoplasmic reticulum membranes, and related intracellular membrane assemblies. These sites share common physical features: high protein density, membrane curvature, structured hydration layers, and continuous redox flux [41,42]. Together, they form an integrated intracellular interfacial network supporting phase breathing.

The plasma membrane represents the principal boundary between intracellular and extracellular environments and serves as a coordinating interface between internal and external phase dynamics. Its lipid-protein architecture and associated hydration layers provide a continuous interfacial platform through which intracellular and extracellular processes are coupled [43].

Beyond the plasma membrane, the glycocalyx forms a highly structured extracellular interface enriched in charged polysaccharides and organized water. This region plays a central role in mechanotransduction, redox signaling, and hydration organization, linking membrane-associated

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dynamics with the surrounding tissue environment. Recent work by Ninham and colleagues has emphasized how ion-specific (Hofmeister) effects and nanobubble-related interfacial physics operate at the endothelial surface layer, demonstrating that structured carbohydrate-rich interfaces such as the glycocalyx are governed by competitive ion interactions and hydration forces that influence interfacial organization and gas-liquid behavior [44].

Closely linked to the glycocalyx is the extracellular matrix (ECM), which we propose here to function as a centropy storage and entropy dissipation field. Organizational capacity generated at cellular interfaces is distributed into the ECM, where its large, hydrated volume and mechanical compliance allow coordinated biological signals to be buffered, propagated, and integrated at the tissue scale. The ECM is widely recognized as a mechanically active, signaling-rich environment that regulates cell behavior, buffers stress, and coordinates tissue-level organization [46,47].

Within this framework, the ECM does not serve as a site of phase breathing. Rather, phase breathing is localized to confined interfacial microdomains at intracellular membranes, the plasma membrane, and the glycocalyx, while the ECM provides the surrounding field that stores centropic organization and supports entropy export.

Phase-conditioned hydration states generated by PAQs are proposed to propagate via calcium signaling. Local changes in Ca^{2+} handling - including altered channel gating or modifications in release and reuptake kinetics - can reshape the timing, amplitude, and spread of calcium oscillations and intercellular waves [48–51].These waves may synchronize subcellular redox states and extend to tissue scales, embedding centropy into ECM hydration and matrix organization [3,4,22,44,46,47].

Thus, phase breathing emerges as a distributed interfacial process spanning intracellular membranes, the plasma membrane, and the glycocalyx, forming a coherent multi-compartment system that anchors phase and generates organizational capacity. This interfacial network couples intracellular coherence with extracellular propagation, while the surrounding extracellular matrix functions as a centropy storage and entropy dissipation field that integrates, buffers, and distributes organizational signals at the tissue scale. Together, these compartments constitute a unified biological architecture in which phase anchoring, centropic organization, and entropy export are spatially coordinated to support coherent biological function across scales.

8. Physiological Requirements of Phase Breathing

Phase breathing depends on a specific physiological context in which interfacial hydration, electrolyte composition, gas saturation, boundary compliance, and redox organization converge to support cyclic nanodomain dynamics. Disruption of any of these parameters is predicted to impair phase anchoring and reduce centropic capacity, even when energetic metabolism remains active.

8.1. Electrolyte Balance and Hydration Elasticity

The electrolyte environment plays a central role in conditioning interfacial hydration structure and nanodomain stability. Phase breathing requires hydration layers that retain sufficient elasticity to permit cyclic nucleation and collapse while maintaining electrostatic organization at redox-active interfaces.

Intracellular electrolytes are characterized by high potassium and low sodium concentrations, whereas extracellular compartments exhibit the opposite pattern. Potassium ions possess lower charge density and weaker hydration compared with sodium, promoting flexible hydration shells and reduced interfacial stiffness. Sodium ions, by contrast, impose stronger water structuring and increase electrostatic rigidity [52]. These differences imply that intracellular interfaces exhibit greater hydration elasticity and lower nucleation thresholds than extracellular interfaces.

Extensive work by Ninham and collaborators on Hofmeister phenomena demonstrates that specific ions differentially modulate interfacial hydration, surface tension, and colloidal stability through ion-dependent organization of water [45]. These ion-specific effects extend to gas-water interfaces, where competitive adsorption alters surface charge and nanodomain persistence. Thus,

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physiological electrolyte composition actively conditions nanodomain nucleation and stability rather than serving as a passive background variable.

Calcium ions provide temporally structured electrostatic modulation at both intracellular and extracellular interfaces. Cellular calcium signaling is inherently oscillatory, with localized release and reuptake occurring at membrane-associated microdomains on timescales ranging from milliseconds to seconds [48,49]. Prominent examples include ER–mitochondria contact sites and plasma membrane junctions, where calcium microdomains regulate metabolic activity, membrane dynamics, and redox signaling [42,50,51]. These calcium oscillations transiently reorganize hydration layers and modulate membrane surface charge, creating rhythmic perturbations of interfacial geometry that can gate nanodomain nucleation events. In this framework, calcium acts as a dynamic trigger that may synchronize the cyclicity of phase breathing with cellular signaling rhythms.

In contrast, potassium and sodium function primarily as stabilizers of the electrolyte environment. Potassium-rich intracellular conditions promote hydration elasticity and low interfacial stiffness, while sodium-dominant extracellular environments impose greater structural constraint. Together, these complementary electrolyte gradients establish a steady electrostatic landscape that supports sustained rhythmic cycling. Calcium superimposes transient oscillatory modulation upon this stabilized background, enabling controlled nucleation-collapse dynamics.

8.2. Nitrogen Gas Saturation and Confined Pressure Dynamics

Phase breathing requires sufficient local N_2 saturation to permit recurrent formation of confined gas nanodomains at interfacial RPCS microdomains. Without adequate dissolved N_2 availability, nanoscale gas nucleation cannot be sustained, and cyclic expansion-collapse dynamics required for phase anchoring fail to occur.

Nitrogen is present in biological fluids at concentrations determined by ambient partial pressure and solubility constraints [23,24]. At interfacial sites where hydration structure, surface charge organization, and geometric confinement converge, dissolved N_2 can partition into nanoscale domains consistent with experimentally observed nanobubble formation at biological interfaces [6,7,55]. The persistence of such domains depends not only on interfacial mechanics but on gas saturation to permit repeated nucleation following collapse.

At nanometer scale, confined gas domains sustain elevated internal pressures due to Laplace effects [6,7]. When nitrogen saturation supports stable re-nucleation, these localized pressure states become recurrent rather than transient, establishing mechanically conditioned microenvironments within RPCS interfaces. Such pressure confinement reshapes adjacent hydration structure and alters intermolecular spacing, thereby influencing oxidative-reductive reaction geometry under confined conditions [25,26].

Thus, N_2 saturation functions as a prerequisite boundary condition for phase breathing. When local N_2 availability falls below nucleation threshold, nanodomain cycling cannot be maintained, even if membrane compliance and hydration elasticity remain permissive.

8.3. Interfacial Boundary Compliance

Phase breathing further requires compliant interfacial boundaries capable of undergoing repeated nanoscale deformation without structural failure. The RPCS operates within membrane-associated and glycocalyx-associated microdomains where lipid-protein assemblies, hydration layers, and extracellular polymers together form mechanically flexible interfaces.

Biological membranes are not rigid barriers but dynamically deformable structures whose elasticity is regulated by lipid composition, protein crowding, and hydration state [43,54]. This compliance allows localized curvature, thickness fluctuations, and transient compartmentalization, all of which are necessary for nanodomain nucleation and collapse. Similarly, the glycocalyx provides a hydrated, mechanically responsive extracellular interface that supports deformation while maintaining structural continuity with the plasma membrane [53].

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Beyond mechanical deformation, compliant boundaries also support photonic signaling. Photonic Activation Quanta (PAQs) generated during redox resolution must propagate across hydrated interfaces into surrounding structures. Excessive boundary stiffness or dehydration impairs both nanodomain formation and PAQs transmission, reducing organizational capacity even when metabolic activity persists.

Thus, interfacial boundary compliance constitutes a fundamental physiological requirement for phase breathing, allowing nanodomain cycling, coherent redox resolution, and photonic phase transfer to proceed in a coordinated manner.

9. Boundary Impedance as the Control Variable of Phase Breathing

Phase breathing requires cyclic nanodomain nucleation and collapse. While preceding sections established the molecular and interfacial components of this process, they do not yet identify the variable that determines whether phase breathing actually occurs.

We propose that this control variable is **boundary impedance**.

Boundary impedance (Z_β) is defined here as the effective resistance of an interfacial domain to rapid mechanical and electromagnetic reconfiguration. It represents the combined opposition of membranes, hydration shells, and dielectric boundaries to time-dependent deformation.

Boundary impedance is an emergent physical property integrating:

- interfacial mechanical stiffness
- hydration shell elasticity
- dielectric contrast
- surface charge organization
- geometric confinement

Together, these determine whether an interface can undergo the rapid boundary motion required for nanodomain cycling.

9.1. Boundary Impedance as the Gate for Nanodomain Dynamics and Phase Capture

Nanodomain nucleation and collapse arise from the coordinated interaction of multiple interfacial variables; none of these factors alone determines whether phase breathing occurs. Their combined influence is expressed through boundary impedance (Z_β), which functions as the integrated control variable governing access to cyclic nanodomain dynamics.

In the present work, Z_β is conceptually defined but not yet quantitatively determined. It represents the integrated effect of interfacial mechanical, dielectric, hydration, and geometric properties governing boundary reconfiguration.

We propose that phase breathing becomes dynamically sustainable only when the effective interfacial resistance to boundary motion falls below a critical functional threshold. In such low-impedance regimes, nanodomains can undergo oscillatory expansion and collapse, creating transient temporal windows during which oxidative-reductive resolution may become synchronized.

Conversely, when interfacial stiffness, dielectric rigidity, or hydration immobility increase, boundary motion becomes mechanically constrained. Under these higher-impedance conditions, nanodomain cycling becomes irregular or arrested, coherence windows fail to stabilize, and phase capture does not occur.

Because phase capture generates PAQs, which in turn drive centropy generation through hydration restructuring, the boundary impedance that governs nanodomain cycling also constrains the system's capacity to accumulate centropy.

9.2. Boundary Impedance as a Dynamic Variable

Boundary impedance is not a fixed property of biological interfaces. It is continuously modulated by hydration structure, electrolyte organization, membrane compliance, and oxidative modification.

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Since hydration organization influences interfacial elasticity and dielectric responsiveness, successful phase breathing can indirectly reduce effective boundary impedance, whereas sustained loss of phase breathing promotes interfacial stiffening and impedance elevation.

This dynamic coupling implies that boundary impedance is both a determinant and a consequence of interfacial organization. As a result, biological systems may exhibit threshold behavior: gradual changes in interfacial conditions can produce abrupt transitions between phase-permissive and phase-restricted regimes.

10. Boundary Impedance as the Central Control Axis of Phase-State Biology

The preceding sections established boundary impedance as the integrated physical variable governing access to phase breathing. This permits a higher-order organizational classification at the local level: biological interfaces operate within discrete Phase-States defined by whether phase breathing is accessible, restricted, or arrested.

We define three primary Phase-States - Phase Access, Phase Restricted, and Phase Arrest - representing progressively increasing impedance to nanodomain cycling and phase capture. These states describe local interfacial organizational regimes rather than uniform organism-wide conditions. Because phase breathing is a spatially distributed phenomenon operating across intracellular membranes, plasma membrane microdomains, and glycocalyx-associated interfaces, distinct tissues or subcellular domains may simultaneously occupy different Phase-States depending on their boundary conditions.

Boundary impedance (Z_β) functions as the control variable for local phase accessibility. However, local impedance alone does not define global biological phase organization. Organism-level coherence additionally depends on higher-order coordination variables, including cross-scale phase entrainment between physiological domains (subcellular, cellular, tissue-level) and stable phase alignment among spatially distributed RPCS networks. Even when local impedance permits phase breathing within isolated interfaces, failure of inter-domain phase synchronization may produce system-level decoherence. Thus, boundary impedance governs local phase accessibility, whereas global biological coherence requires multiscale temporal alignment and coordinated phase propagation. Formal characterization of these coordination variables lies beyond the scope of the present work and will be addressed in future investigations.

10.1. Phase Access State

The Phase Access State corresponds to conditions in which phase breathing proceeds efficiently. Nanodomains undergo sustained oscillatory nucleation and collapse, coherence windows form reliably, and oxidative-reductive resolution at the RPCS generates phase-conditioned Photonic Activation Quanta (PAQs). PAQs are absorbed by hydration structures, enabling continuous centropy accumulation. In this regime, metabolic energy is effectively translated into coordinated biological work.

Functionally, the Phase Access State is characterized by:

- robust nanodomain dynamics
- reliable PAQs generation
- high centropy
- elastic hydration interfaces
- coherent biological signaling and coordination

When Phase Access is generalized across distributed biological interfaces, it defines a state of physiological coherence: biological processes remain synchronized across scales, organizational capacity is continuously renewed, and metabolic activity is coupled to coherent function while entropy is physiologically exported.

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10.2. Phase Restricted State

The Phase Restricted State arises when access to phase breathing becomes intermittent or incomplete. Nanodomain cycling persists but loses oscillatory stability. Coherence windows form inconsistently, and PAQs generation becomes sporadic. Redox reactions continue, but temporal alignment with nanodomain nucleation is unreliable. As a result, photon emission increasingly lacks phase conditioning, leading to reduced centropy accumulation.

Functionally, this state is characterized by:

- unstable or subthreshold nanodomain oscillations
- inconsistent PAQs production
- declining centropy reserves
- partial loss of hydration organization
- increasing reliance on compensatory metabolic activity

Biologically, the Phase Restricted State corresponds to adaptive stress physiology. Energetic throughput may increase, but organizational efficiency declines. Systems remain viable yet progressively lose coherence.

10.3. Phase Arrest State

The Phase Arrest State represents functional loss of phase breathing. Nanodomain dynamics fall below the threshold required for coherence window formation, and phase capture ceases. Oxidative-reductive reactions may persist, but photon emissions are no longer phase-conditioned. PAQs are not generated, hydration organization collapses, and centropy cannot be replenished.

Functionally, this state is characterized by:

- arrested or non-oscillatory nanodomain behavior
- absence of PAQs-mediated organization
- centropy depletion
- rigidified interfacial boundaries
- dominance of entropic dissipation

This regime corresponds to degenerative and terminal pathophysiology, where metabolic activity may persist, but without coherent phase coordination.

11. Pathogenesis of Phase Breathing Impairment

Pathology arises when phase breathing becomes impaired due to locally elevated boundary impedance, preventing sustained nanodomain cycling and phase capture. Reduced generation of phase-conditioned Photonic Activation Quanta (PAQs) diminishes centropy renewal within hydration interfaces, progressively destabilizing interfacial organization. Metabolic reactions may continue, but their energetic output becomes organizationally incoherent.

Phase breathing becomes impaired when interfacial conditions change in ways that reduce hydration-layer elasticity or mechanically constrain the cyclic nucleation and collapse of nanodomains. A range of physicochemical factors have the potential to alter these boundary properties. For example, alterations in hydrogen-bond dynamics - such as those associated with increased deuterium content - can modify hydration structure and influence local stiffness [56–58]. Similarly, environmental contaminants including microplastics and per- and polyfluoroalkyl substances (PFAS) may embed within lipid membranes or adsorb at hydration interfaces, perturbing dielectric organization and interfacial compliance [59,60]. Heavy metals can bind interfacial proteins and redox-active sites, altering both mechanical flexibility and electronic coupling within confined domains [61,62].

Such boundary-modifying influences are proposed to elevate local boundary impedance. As impedance rises, temporal alignment between nanodomain nucleation and oxidative-reductive resolution may become unstable, favoring transition toward a Phase Restricted regime characterized

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by diminished phase capture and progressive redox decoherence. Decoherent redox activity manifests as oxidative stress and promotes accumulation at boundaries of lipid peroxidation products, protein cross-linking, and other oxidative byproducts. These oxidative modifications stiffen biological interfaces, further increasing boundary impedance and reinforcing phase restriction. This positive feedback loop progressively suppresses nanodomain oscillation, ultimately driving transition toward Phase Arrest.

Neural tissue may be particularly sensitive to boundary impedance-mediated disruption of phase breathing due to its dependence on sustained temporal coordination across spatial scales. Neurons rely on precise synaptic timing, cytoskeletal integrity, and long-range network synchronization -processes that require stable interfacial organization within membrane-associated and cytoskeletal microdomains. Localized elevation of boundary impedance within such domains may impair nanodomain cycling and reduce centropy renewal, promoting interfacial stiffening and redox decoherence. In this framework, region-specific degenerative or inflammatory phenotypes can be interpreted as spatially confined transitions toward Phase Restricted or Phase Arrest regimes, wherein hydration-level disruption and boundary stiffening progressively diminish coherent biological organization despite preserved systemic metabolic activity.

12. Translational Implications of the Phase-State Framework

The phase breathing mechanism and Phase-State hierarchy described above suggest a potential translational interpretation of biological organization. From this perspective, health and disease may be examined not only in terms of molecular pathways or metabolic throughput, but also in terms of phase accessibility. Disease processes may correspond to localized elevations of boundary impedance that impair phase breathing and reduce centropy, even when metabolic activity persists.

Because phase breathing is spatially distributed, different regions of the same organism may occupy distinct Phase-States simultaneously. Pathology may therefore reflect region-specific disruption of interfacial coherence rather than uniform systemic failure.

Novel diagnostic strategies would aim to identify measurable proxies of phase accessibility and organizational capacity at both local and systemic levels. Therapeutic approaches would focus on restoring the interfacial conditions required for phase breathing - for example by supporting hydration elasticity, limiting oxidative boundary stiffening, optimizing electrolyte composition, and mitigating environmental factors that elevate boundary impedance.

13. Testable Predictions and Experimental Approaches

Although the phase breathing framework is primarily theoretical, it is formulated to be empirically falsifiable through measurable proxies of nanodomain dynamics, interfacial hydration organization and photonic output.

13.1. Prediction Related to Phase Breathing and Nanodomain Dynamics

Hypothesis 1: *Phase Access states will exhibit cyclic nanodomain expansion-collapse dynamics temporally coupled to oxidative-reductive resolution, whereas Phase Restricted and Phase Arrest states will show subcritical or arrested boundary motion.*

Rationale: Phase breathing requires sustained oscillatory nanodomain behavior gated by boundary impedance. Loss of oscillatory dynamics reflects elevated impedance and impaired phase capture.

Testable prediction: In Phase Access regimes, nanoscale interfacial domains will display rhythmic deformation synchronized with structured UPE signatures. In Phase Restricted or Phase Arrest regimes, comparable metabolic excitation will produce diminished or incoherent photonic output alongside suppressed nanodomain motion.

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Methods: Ultra-weak photon emission (UPE) detection [33,34] may be combined with high-resolution interfacial imaging - such as high-speed atomic force microscopy [65] or super-resolution fluorescence methods [66] - together with localized reactive oxygen species (ROS) probes [67], to examine time-resolved correlations between nanodomain dynamics and redox activity.

13.2. Predictions Related to Photonic Polarization and Half-Cycle Origin

Hypothesis 2: *Photonic Activation Quanta (PAQs) generated during the expansion (nucleation) half-cycle exhibit a polarization signature distinct from photons emitted during the collapse half-cycle.*

Rationale: Photons are emitted during both nucleation and collapse half-cycles of phase breathing. Because expansion and collapse differ in both direction and phase conditions, the two photon classes are expected to exhibit distinct polarization patterns relative to the interface.

Testable prediction: Nucleation-associated photons exhibit a polarization tendency consistent with molecular alignment at the interface, whereas collapse-associated photons display a different polarization pattern consistent with rapid boundary motion.

Methods: Time-tagged, polarization-resolved ultra-weak photon emission (UPE) may be recorded using a rotating linear polarizer or a polarization-sensitive single-photon counting camera [69,70]. Nanodomain boundary motion may be monitored concurrently using high-speed atomic force microscopy [65] or super-resolution interfacial reporters [66]. Photon arrival times can then be aligned with inferred expansion and contraction phases using the boundary-motion trace. For each half-cycle, polarization metrics, including degree of linear polarization and preferred orientation relative to the interface, may be calculated to compare polarization characteristics between nucleation- and collapse-associated emission components.

13.3. Prediction Related to Nitrogen Specificity in Phase Breathing

Hypothesis 3: *Phase breathing depends specifically on molecular nitrogen (N_2) as the mechanically permissive gas mediating nanodomain nucleation and collapse at RPCS interfaces.*

Rationale: The phase-breathing framework proposes that confined N_2 nanodomains provide the inert, compressible gas phase required for cyclic expansion-collapse dynamics. If N_2 functions as the mediator of nanodomain cycling, altering the dissolved gas composition should affect the stability and periodicity of nanodomain oscillations and their coupling to photonic emission.

Testable prediction: Biological or biomimetic systems exhibiting Phase Access will show enhanced oscillatory nanodomain dynamics and structured ultra-weak photon emission when equilibrated with N_2-rich environments. Substitution of N_2 with alternative dissolved gases (e.g. O_2, CO_2) is predicted to reduce the stability of nanodomain cycling and weaken temporal coupling between boundary motion and photonic emission.

Methods: Nanodomain dynamics may be monitored using high-speed atomic force microscopy [65] or super-resolution interfacial imaging [66], while ultra-weak photon emission can be measured using single-photon counting detectors [33,34]. Gas composition of the surrounding medium can be systematically varied while maintaining constant metabolic or redox conditions to examine the effect of N_2 availability on nanodomain oscillation and photonic output.

Conclusions

This work introduces phase breathing as an interfacial mechanism by which living systems anchor phase, generate organizational capacity termed centropy, and sustain coherent biological function. By integrating nitrogen-mediated nanodomain dynamics, synchronized oxidative-reductive resolution, and photonic phase conditioning within the Redox Photonic Coupling System, the framework defines a physical pathway through which metabolic activity is translated into structured biological organization.

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From this mechanism emerges a Phase-State hierarchy -Phase Access, Phase Restricted, and Phase Arrest - representing distinct organizational regimes defined by the integrity of phase breathing. These states provide an additional layer for interpreting biological function as phase-dependent rather than solely metabolically driven.

Within this hierarchy, coherent physiological function is associated with sustained Phase Access at critical interfaces, where organizational capacity is continuously renewed and entropy is effectively dissipated. Conversely, progressive elevation of boundary impedance and reduced centropy generation are expected to shift affected regions toward Phase Restricted or Phase Arrest regimes. In this view, degenerative or chronic inflammatory phenotypes can be interpreted as localized transitions in phase accessibility rather than uniform loss of metabolic activity.

These considerations outline a potential diagnostic and therapeutic perspective grounded in phase biology. Rather than focusing exclusively on isolated molecular targets or metabolic throughput, this framework expands biological assessment to include boundary impedance as the controlling interfacial parameter and phase-state as the emergent expression of interfacial organization. By emphasizing restoration of interfacial permissivity to enable recovery of phase breathing, it provides a translational bridge between nanoscale biophysical dynamics and clinical strategies aimed at restoring physiological coherence.

Glossary of Terms

The following terms are introduced and defined in this work to describe the conceptual framework developed by the author.

Phase Breathing: The cyclic nucleation and collapse of confined molecular nitrogen (N_2) nanodomains at the Redox Photonic Coupling System (RPCS), coupled to synchronized oxidative-reductive resolution. Phase breathing provides the physical mechanism by which biological systems intermittently anchor phase, generate phase-conditioned photonic output, and renew organizational capacity.

Centropy: The stored organizational capacity of biological systems arising from phase-conditioned hydration organization. Centropy reflects readiness for coordinated biological work and represents phase-enabled structural permissivity.

Boundary Impedance: An integrated interfacial control variable describing resistance to nanoscale mechanical and electromagnetic reconfiguration. Boundary impedance aggregates the effects of hydration elasticity, interfacial compliance, dielectric structure, surface charge organization, and geometric confinement, determining whether phase breathing can occur.

Phase Access State: The organizational regime in which phase breathing proceeds efficiently, Photonic Activation Quanta (PAQs) are generated reliably and centropy accumulates. In this state, metabolic activity remains coherently organized and entropy is effectively exported through ongoing phase breathing.

Phase Restricted State: A spatially distributed intermediate organizational regime characterized by impaired or intermittent phase breathing, inconsistent Photonic Activation Quanta (PAQs) generation, declining centropy, and partial loss of hydration organization. In this state, entropy progressively accumulates within the system.

Phase Arrest State: A spatially distributed terminal organizational regime in which phase breathing fails, Photonic Activation Quanta (PAQs) generation ceases, centropy cannot be replenished, and interfacial boundaries become rigidified. In this state, entropy dominates as internal entropy production exceeds the system's capacity to export it.

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