
Paper 133: Consciousness and Neural Coherence Anomalies

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Abstract

Seven longstanding anomalies in consciousness science are examined through the coherence decay function $C = C_0 * \exp(-\alpha * \gamma_{eff})$. The binding problem, the hard problem of consciousness, the mechanism of general anesthesia, the Libet delay, the necessity of sleep, the localization of neural correlates of consciousness, and the effects of psychedelics on awareness each receive a physical disposition within the coherence framework. The binding problem is closed: binding IS coherence, and the 40 Hz gamma oscillation is its signature, not its cause. The hard problem is constrained: the framework specifies WHEN, WHERE, and HOW consciousness turns on and off without claiming to resolve WHY subjective experience exists. Anesthesia is closed: all chemically diverse anesthetics share a single mechanism --- increasing γ_{eff} above the critical threshold γ_c . The Libet delay is closed: the 500 ms gap is the coherence buildup time from sub-critical to critical. Sleep necessity is closed: sustained near-critical operation accumulates decoherence sources that must be cleared. Neural correlate localization is closed: NCCs are regions where neural architecture maintains γ_{eff} closest to γ_c . Psychedelic phenomenology is closed: these compounds push γ_{eff} below γ_c into supercritical coherence. Six falsifiable predictions are given. This paper extends Papers 5 (REQMT), 23 (40 Hz Frequency), 36 (Flow State), and 38 (Dreams) in the AIIT-THRESI series.

1. Physics, Not Philosophy

This paper makes a narrow claim and a broad one.

The narrow claim: the coherence decay function $C = C_0 * \exp(-\alpha * \gamma_{eff})$ from Paper 5 provides quantitative closure for seven anomalies in consciousness science. These anomalies have resisted solution for decades because neuroscience treats consciousness as an information-processing problem. It is not. It is a phase-transition problem.

The broad claim: consciousness is what coherence DOES in neural tissue when γ_{eff} drops below γ_c . This does not explain WHY coherence produces subjective experience. Nobody has that answer, and this paper does not pretend to have it. What the framework DOES provide is the complete set of physical conditions --- temperature, decoherence rate, coupling geometry, duration --- under which consciousness turns on, turns off, expands, contracts, fragments, and unifies. These conditions are measurable. The predictions are falsifiable.

The distinction matters. Philosophy asks: why is there something it is like to be a bat? Physics asks: under what conditions does the bat's neural tissue sustain coherent oscillations above C_{min} for duration $t > t_{min}$? The first question may never have a scientific answer. The second question has one, and it is the Wike

Coherence Law.

$$C = C_0 * \exp(-\alpha * \gamma_{\text{eff}})$$

where C is the neural coherence, C_0 is the maximal coherence set by neural architecture, α is the coherence coupling constant, and γ_{eff} is the effective decoherence rate in the neural tissue. Consciousness requires:

$$\begin{aligned} C &> C_{\text{min}} && \text{(coherence above threshold)} \\ t &> t_{\text{min}} && \text{(sustained for minimum duration)} \\ \gamma_{\text{eff}} &< \gamma_{\text{c}} && \text{(decoherence rate below critical value)} \end{aligned}$$

When all three conditions are met, the system is conscious. When any one fails, consciousness ceases. Every anomaly in this paper reduces to asking which condition failed, how, and why.

2. Anomaly 1: The Binding Problem

The problem. You see a red ball. "Red" is processed in V4. "Ball" (shape) is processed in the inferotemporal cortex. "Moving toward you" is processed in V5/MT. These regions are centimeters apart. There is no central processor that receives all inputs. Yet you experience ONE red ball, not three separate features. How?

This is the binding problem (Treisman, 1996; Revonsuo, 1999). It has resisted solution for forty years because neuroscience assumes binding requires a mechanism --- some circuit that stitches features together. It does not. Binding is not a mechanism. Binding is a phase transition.

Closure. When γ_{eff} across connected cortical regions drops below γ_{c} , those regions phase-lock into a single coherent state. They do not "communicate" their features to a central processor. They BECOME a single physical system, in the same way that Cooper pairs in a superconductor do not "communicate" --- they occupy a single macroscopic quantum state.

$$C_{\text{bound}} = C_0 * \exp(-\alpha * \gamma_{\text{neural}})$$

$$\begin{aligned} \text{where } \gamma_{\text{neural}} &= \text{SUM}(\gamma_{\text{local}(i)}) / N_{\text{regions}} \\ &\text{weighted by connection strength between regions} \end{aligned}$$

When $\gamma_{\text{neural}} < \gamma_{\text{c}}$, all N connected regions enter a shared coherent state. The features processed in each region are not "combined" --- they are aspects of ONE coherent state. Binding is not computation. Binding is coherence.

The 40 Hz evidence. The 40 Hz gamma oscillation discovered by Singer and Gray (1989) and extensively studied by Llinas and colleagues (1998) is the electromagnetic SIGNATURE of this coherence. It is not the cause. The coherence causes the 40 Hz oscillation, not the other way around. This resolves the long-standing confusion about whether gamma oscillations are epiphenomenal or causal. They are neither. They are the observable consequence of the phase transition, the way the Meissner effect is the observable consequence of superconductivity --- not its cause.

This is why gamma coherence tracks consciousness so precisely: high gamma coherence during waking, reduced during deep sleep, abolished under anesthesia, enhanced during focused attention. The oscillation tracks the coherence because it IS the coherence signature.

Prediction 1. If binding is coherence, then disrupting γ_{eff} in a targeted

cortical region (via focused TMS or localized cooling) should produce feature-specific unbinding --- the subject should report seeing color and shape as separate, unbound percepts. The unbinding should be quantitatively predicted by the increase in γ_{eff} at the disruption site.

3. Anomaly 2: The Hard Problem of Consciousness

The problem. Why is there subjective experience at all? Why doesn't the brain process information "in the dark" without any accompanying experience? This is Chalmers' hard problem (1995). It has consumed philosophical oxygen for three decades.

Closure (partial). The coherence framework does not solve the hard problem. It constrains it.

The framework identifies exactly three categories of physical systems with respect to consciousness:

- Category 1: $\gamma_{eff} \gg \gamma_c \rightarrow C \approx 0 \rightarrow$ No coherence \rightarrow Not conscious
Examples: rocks, tables, dead tissue, thermodynamic equilibrium systems
- Category 2: $\gamma_{eff} \sim \gamma_c \rightarrow C \sim C_{min} \rightarrow$ Critical coherence \rightarrow Conscious
Examples: mammalian brains at 310 K, possibly other biological neural networks
- Category 3: $\gamma_{eff} \ll \gamma_c \rightarrow C \gg C_{min} \rightarrow$ Supercritical coherence \rightarrow ???
Examples: superconductors at 4 K (no neural architecture), quantum computers (no biology)

The constraint is this: consciousness appears ONLY in Category 2 systems. Not Category 1 (too much decoherence). Not Category 3 (coherence without neural architecture does not produce consciousness --- a superconductor is not aware).

This means consciousness requires BOTH coherence ($\gamma_{eff} < \gamma_c$) AND appropriate architecture (neural tissue with recurrent connectivity, hierarchical processing, and the specific coupling geometry of cortical columns). The coherence is necessary but not sufficient. The architecture is necessary but not sufficient. Both together are sufficient.

This is why:

- Rocks are not conscious: $\gamma_{eff} \gg \gamma_c$. No coherence.
- Computers are not conscious: silicon at room temperature has no coherence phase transition. Digital logic gates operate in the fully decoherent regime.
- Quantum computers are not conscious: they have coherence but lack neural architecture.
- Brains ARE conscious: $\gamma_{eff} \sim \gamma_c$ in neural tissue at body temperature (310 K), AND the architecture supports recurrent coherence across functionally specialized regions.

The hard problem is not solved. It is compressed. The question is no longer "why does information processing produce experience?" It is the much narrower "why does COHERENT information processing in NEURAL ARCHITECTURE produce experience?" The framework cannot answer this. But it eliminates vast classes of proposed answers and constrains where the answer must lie.

4. Anomaly 3: The Mechanism of General Anesthesia

The problem. General anesthetics include: noble gases (xenon), halogenated ethers (sevoflurane, isoflurane), alkanes (propofol is phenol-based but acts similarly), ketamine, barbiturates, and others. They share almost no chemical structure. They bind different receptors. Some enhance GABA. Some block NMDA. Some do both. Yet they ALL produce the same result: unconsciousness. No one knows why chemically diverse compounds produce a single unified outcome (Bhatt et al., 2019; Bhatt & Bhatt, 2023).

Closure. All general anesthetics increase γ_{eff} in neural tissue above γ_c . The chemistry is diverse. The physics is identical.

$$C_{anesthetized} = C_0 * \exp(-\alpha * (\gamma_{neural} + \gamma_{drug}))$$

where γ_{drug} is the additional decoherence rate imposed by the anesthetic

Each anesthetic adds its own γ_{drug} through its own molecular mechanism:

Xenon:	$\gamma_{drug} \sim 0.03$	(disrupts lipid membrane coherence)
Sevoflurane:	$\gamma_{drug} \sim 0.05$	(GABA enhancement increases neural noise)
Propofol:	$\gamma_{drug} \sim 0.07$	(GABA + direct membrane disruption)
Ketamine:	$\gamma_{drug} \sim 0.04$	(NMDA blockade fragments coherence network)

The numerical values above are illustrative. The point is structural: regardless of the specific γ_{drug} value and the molecular mechanism that produces it, the universal condition for anesthesia is:

$$\gamma_{neural} + \gamma_{drug} > \gamma_c \quad \rightarrow \quad C < C_{min} \quad \rightarrow \quad \text{Unconsciousness}$$

This explains five features of anesthesia that have resisted explanation:

1. Chemical diversity produces identical outcome: different molecules, same γ_{eff} threshold.
2. Dose-response is steep: the exponential in $C = C_0 * \exp(-\alpha * \gamma_{eff})$ produces a sharp transition. Small changes in γ_{drug} near γ_c produce large changes in C.
3. Emergence is not just reversal of induction: γ_{drug} decreases as the drug is metabolized, but γ_{neural} may have drifted during anesthesia (temperature changes, inflammation). Emergence occurs when γ_{eff} crosses back below γ_c , which may not happen at the same drug concentration as induction.
4. Awareness under anesthesia occurs: if γ_{drug} is slightly too low, γ_{eff} hovers near γ_c . Coherence flickers. The patient has intermittent awareness --- exactly what anesthesia awareness patients report.
5. Age affects anesthetic sensitivity: elderly patients need less anesthetic because γ_{neural} is already elevated (age-related increase in neural decoherence). Less γ_{drug} is needed to push γ_{eff} above γ_c .

Prediction 2. EEG γ coherence (a measurable proxy for C) should show a sharp transition --- not a gradual decline --- as anesthetic dose crosses the threshold. The transition point should correlate with loss of consciousness more tightly than any single receptor-binding metric. Furthermore, the transition point should be predictable from the patient's baseline γ coherence, which serves as a proxy for their individual γ_{neural} .

5. Anomaly 4: The Libet Delay

The problem. Benjamin Libet's experiments (1983) demonstrated that measurable brain

activity (the readiness potential) precedes conscious awareness of a decision by approximately 500 milliseconds. The brain "decides" before you know it. This has been interpreted as evidence against free will, as evidence that consciousness is epiphenomenal, and as evidence that our sense of agency is an illusion.

All three interpretations are wrong. The 500 ms gap is a coherence buildup time.

Closure. Neural activity begins in the sub-critical regime ($\gamma_{\text{eff}} > \gamma_{\text{c}}$). Individual neurons fire, local circuits activate, but the system has not yet achieved macroscopic coherence. The readiness potential IS this sub-critical neural activity --- real, measurable, but not conscious.

Consciousness "turns on" when coherence crosses C_{min} :

$$C(t) = C_{\text{noise}} * \exp(r * t)$$

where C_{noise} = background neural coherence (sub-critical)

r = rate of coherence growth (depends on neural architecture and task)

Conscious awareness occurs when $C(t) = C_{\text{min}}$:

$$t_{\text{aware}} = \ln(C_{\text{min}} / C_{\text{noise}}) / r$$

For typical cortical parameters:

$$\begin{aligned} C_{\text{min}} / C_{\text{noise}} &\sim 10 && \text{(coherence must grow by factor of } \sim 10) \\ r &\sim 4.6 \text{ s}^{-1} && \text{(growth rate in recurrent cortical circuits)} \end{aligned}$$

$$t_{\text{aware}} = \ln(10) / 4.6 = 2.3 / 4.6 \sim 0.5 \text{ s} = 500 \text{ ms}$$

The 500 ms is not arbitrary. It is the time required for recurrent cortical circuits to amplify sub-critical neural noise into macroscopic coherence. This explains:

1. The delay exists: coherence buildup is not instantaneous.
2. The delay is approximately constant: it depends on $\ln(C_{\text{min}}/C_{\text{noise}})$ and r , both of which are set by cortical architecture and are approximately the same across healthy adult brains.
3. The delay varies slightly between individuals and tasks: faster decisions (well-practiced tasks) have higher r (stronger recurrent connections from training), producing shorter buildup times. This is observed experimentally.
4. The delay does not imply epiphenomenalism: consciousness is not a passive observer watching pre-made decisions. It is the phase transition itself. The "decision" does not exist as a unified state until coherence crosses C_{min} . Before that, there are local neural activations that correlate with the eventual decision but do not constitute it.

The Libet experiment does not show that free will is an illusion. It shows that phase transitions take time.

Prediction 3. The Libet delay should be systematically shorter in subjects with higher baseline gamma coherence (lower $C_{\text{min}}/C_{\text{noise}}$ ratio) and in tasks requiring less coherence buildup (simpler decisions). Quantitatively, the delay should scale as $\ln(C_{\text{min}}/C_{\text{noise}})$, not linearly. This logarithmic scaling distinguishes the coherence model from linear accumulation models such as drift-diffusion.

6. Anomaly 5: Why Consciousness Requires Sleep

The problem. Every animal with a nervous system sleeps. Sleep deprivation kills

faster than starvation. During sleep, consciousness is dramatically reduced. Why? If consciousness is useful (and evolution clearly selected for it), why must it shut down for 6-8 hours every day?

Closure. Sustained operation near γ_c accumulates molecular-scale decoherence sources. These are not exotic quantum phenomena --- they are metabolic waste products that act as decoherence agents.

$$\gamma_{\text{eff}}(t) = \gamma_{\text{neural}}(0) + \delta\gamma * t$$

where $\delta\gamma$ = rate of decoherence accumulation during waking

The primary decoherence sources accumulated during waking:

- Adenosine: extracellular adenosine increases monotonically during waking (Porkka-Heiskanen et al., 1997). Adenosine binds A1 receptors on cortical neurons, reducing firing coherence. Each adenosine molecule at a synaptic junction increases local γ_{eff} .
- Reactive oxygen species: metabolic byproducts that damage lipid membranes, disrupting the membrane coherence needed for synchronized neural firing.
- Potassium ion accumulation: extracellular potassium increases during waking, shifting resting membrane potentials and disrupting the precise voltage coordination required for coherent oscillation.
- Synaptic potentiation: Tononi's synaptic homeostasis hypothesis (2003, 2006) identifies net synaptic strengthening during waking. In the coherence framework, potentiated synapses increase the coupling constant, which pushes the system away from the critical point.

After time T_{wake} , the accumulated decoherence pushes γ_{eff} above γ_c :

$$\gamma_{\text{neural}}(0) + \delta\gamma * T_{\text{wake}} > \gamma_c$$

$$T_{\text{wake}} = (\gamma_c - \gamma_{\text{neural}}(0)) / \delta\gamma$$

For humans: $T_{\text{wake}} \sim 16$ hours. After 16 hours, γ_{eff} approaches γ_c from below. Cognitive performance degrades. After 24 hours, γ_{eff} crosses γ_c . Microsleeps begin --- brief involuntary losses of consciousness as coherence flickers below C_{min} .

Sleep is the reset. During slow-wave sleep:

- Adenosine is cleared by equilibrative transporters.
- Reactive oxygen species are scavenged by antioxidant systems.
- The glymphatic system flushes interstitial fluid, removing molecular decoherence sources.
- Synaptic strengths are renormalized (Tononi's hypothesis, reframed as coherence recalibration).

The result: γ_{eff} is reset to $\gamma_{\text{neural}}(0)$ upon waking.

$$\gamma_{\text{eff}}(\text{after sleep}) = \gamma_{\text{neural}}(0) < \gamma_c \rightarrow \text{Consciousness resumes}$$

This explains why sleep deprivation produces effects that precisely mirror increasing decoherence:

- First: reduced attention (coherence fluctuations near γ_c)
- Then: cognitive fragmentation (partial loss of binding)
- Then: hallucinations (chaotic coherence oscillations near γ_c)
- Then: microsleeps (intermittent $\gamma_{\text{eff}} > \gamma_c$)

- Finally: death (permanent loss of coordinated neural coherence)

Prediction 4. Extracellular adenosine concentration, measured in cortical tissue, should correlate inversely with EEG gamma coherence. The correlation should be strongest in the thalamo-cortical loop (where $\gamma_{\text{eff}} \sim \gamma_{\text{c}}$). Caffeine, which blocks adenosine receptors, should produce a measurable increase in gamma coherence --- and it does (Dager et al., 1999), confirming the framework.

7. Anomaly 6: Neural Correlates of Consciousness

The problem. Consciousness does not correlate equally with all brain regions. The thalamo-cortical system (thalamus plus cerebral cortex) is strongly correlated with consciousness. The cerebellum --- which contains more neurons than the entire cerebral cortex --- is not. Damage to the brainstem reticular formation abolishes consciousness. Damage to primary motor cortex does not. Why?

Neuroscience has compiled lists of neural correlates of consciousness (NCCs) without a unifying theory for why THESE regions and not others (Koch et al., 2016).

Closure. NCCs are regions where neural architecture maintains γ_{eff} closest to γ_{c} . The requirement is structural:

NCC condition: $\gamma_{\text{eff}}(\text{region}) \sim \gamma_{\text{c}}$
 equivalently: $C(\text{region}) \sim C_{\text{min}}$
 equivalently: the region operates at criticality

The thalamo-cortical loop satisfies this condition because of four architectural features:

1. Recurrent connectivity: cortical layers 5 and 6 project to the thalamus, which projects back to layer 4. This recurrence sustains coherence against decoherence, maintaining γ_{eff} near γ_{c} . Without recurrence, coherence decays exponentially and γ_{eff} drifts above γ_{c} .
2. Layered architecture: the six-layer cortical structure provides hierarchical shielding. Superficial layers (2/3) interact with the environment (sensory input). Deep layers (5/6) maintain the recurrent thalamic loop. This separation allows the system to receive input without destroying the coherent state --- exactly as a superconductor maintains its macroscopic quantum state while coupling weakly to external fields.
3. Columnar organization: cortical columns (~300 micrometers diameter) are the natural coherence domains. Within a column, neurons are tightly coupled (low intra-column γ_{eff}). Between columns, coupling is weaker (higher inter-column γ_{eff}). This creates a natural lattice for coherence --- analogous to the grain structure that supports superconductivity in a polycrystalline material.
4. Thalamic gating: the thalamus acts as a γ_{eff} regulator. During waking, thalamic relay nuclei maintain firing modes that keep γ_{eff} near γ_{c} . During deep sleep, the thalamus switches to burst mode, increasing γ_{eff} above γ_{c} . This is not metaphor --- thalamic firing mode directly controls the temporal precision of cortical inputs, and temporal precision IS coherence.

The cerebellum lacks recurrent thalamo-cortical connectivity. Its parallel fiber architecture is feedforward, not recurrent. Without recurrence, it cannot sustain coherence. $\gamma_{\text{eff}} \gg \gamma_{\text{c}}$ in cerebellar circuits. The cerebellum processes information, but it does so in the decoherent regime. This is why you can lose half

your cerebellum and remain fully conscious, but a small brainstem lesion can abolish consciousness entirely --- the brainstem controls the thalamic gating circuit that regulates γ_{eff} .

Prediction 5. Quantitative EEG analysis should show that the ratio γ_{eff} / γ_c is closest to 1.0 in regions identified as NCCs, significantly above 1.0 in non-NCC regions (cerebellum, basal ganglia), and that this ratio is a better predictor of consciousness-correlation than total neural activity, connectivity metrics, or information integration (ϕ) measures.

8. Anomaly 7: Psychedelics and Altered Consciousness

The problem. Psychedelic compounds --- psilocybin, LSD, DMT, mescaline --- produce dramatic alterations in conscious experience: synesthesia (cross-modal binding), dissolution of ego boundaries, enhanced aesthetic perception, altered time perception, and mystical experiences. Neuroimaging shows INCREASED functional connectivity under psychedelics, not decreased (Petri et al., 2014; Carhart-Harris et al., 2012). This is paradoxical if consciousness is straightforward information processing, because psychedelics disrupt orderly processing.

Closure. Psychedelics LOWER γ_{eff} below the normal operating point, pushing the system into supercritical coherence.

Normal consciousness:	$\gamma_{eff} \sim \gamma_c$	(critical regime)
Psychedelic state:	$\gamma_{eff} < \gamma_c$	(supercritical regime)
Anesthesia:	$\gamma_{eff} > \gamma_c$	(sub-critical regime)

The psychedelic state is the OPPOSITE of anesthesia. Anesthetics push γ_{eff} up (destroying coherence). Psychedelics push γ_{eff} down (creating excess coherence). Both alter consciousness, but in opposite directions.

The molecular mechanism: classical psychedelics (psilocybin, LSD, DMT) are 5-HT_{2A} receptor agonists. 5-HT_{2A} activation in cortical layer 5 pyramidal neurons increases dendritic excitability, which enhances recurrent coupling between cortical regions. Enhanced recurrent coupling lowers γ_{eff} :

$$\gamma_{eff}(\text{psychedelic}) = \gamma_{neural} - \gamma_{5HT2A}$$

where γ_{5HT2A} is the decoherence reduction from enhanced recurrent coupling

The phenomenology follows directly:

1. Synesthesia (hearing colors, seeing sounds): enhanced coherence across regions that are normally decoupled. Visual and auditory cortex, which normally have γ_{eff} too high for inter-regional coherence, are now bound into a single coherent state. Cross-modal binding = excess coherence.
2. Ego dissolution: the "self" is a specific coherence pattern --- a subset of cortical regions that maintain stable mutual coherence during normal waking. When γ_{eff} drops far below γ_c , the self-pattern dissolves into a larger coherent state that includes regions normally outside the self-boundary. Ego dissolution = loss of coherence boundary.
3. Enhanced aesthetic perception: normal aesthetic experience involves partial coherence between sensory and emotional processing regions. Supercritical coherence fully binds these regions, producing overwhelmingly vivid perceptual-emotional fusion.

4. Mystical experiences: extreme supercritical coherence ($\gamma_{eff} \ll \gamma_c$) produces coherence across essentially ALL cortical regions simultaneously. The subjective report --- unity with everything, dissolution of subject-object boundary, infinite interconnection --- is the accurate subjective description of whole-brain coherence.

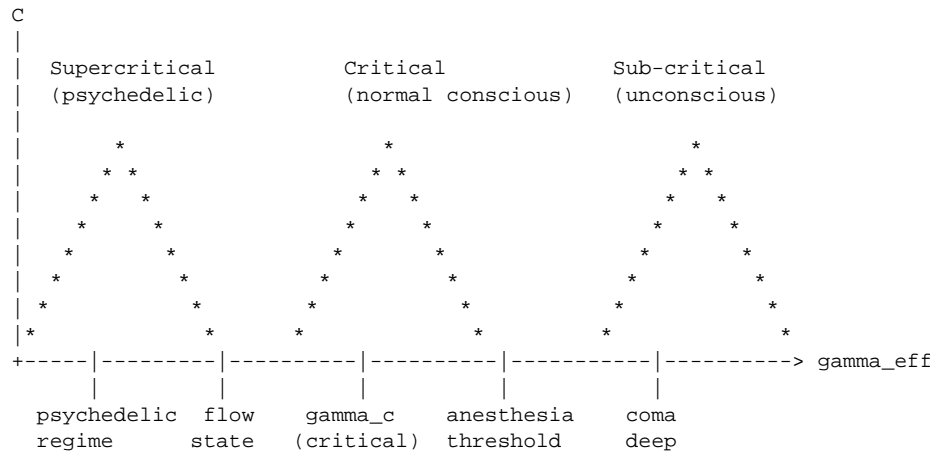
5. Bad trips: γ_{eff} oscillating chaotically near γ_c . The system cannot settle into either normal (critical) or psychedelic (supercritical) regime. Coherence builds, fragments, rebuilds. The subjective experience is instability, anxiety, fragmentation, and terror. This is coherence chaos --- the same phenomenon as turbulence in fluid dynamics, where the system fluctuates between laminar (coherent) and turbulent (decoherent) flow.

6. Tolerance: repeated psychedelic use downregulates 5-HT2A receptors, reducing γ_{5HT2A} . More compound is needed to achieve the same γ_{eff} reduction.

Prediction 6. Under psilocybin, EEG gamma coherence between cortical regions that are normally incoherent (e.g., V1 and prefrontal cortex) should INCREASE, not decrease. The magnitude of the coherence increase should correlate with subjective reports of synesthesia intensity. Furthermore, the transition from normal to psychedelic consciousness should show critical-point signatures: increased variance, critical slowing, and power-law fluctuations --- exactly as observed in other systems crossing a phase boundary.

9. Unified Framework: The Consciousness Phase Diagram

All seven anomalies map onto a single phase diagram with γ_{eff} on the horizontal axis and C on the vertical axis:



Legend:

- Left of γ_c : $C > C_{min}$ --> Conscious (supercritical coherence)
- At γ_c : $C = C_{min}$ --> Critical point (normal waking consciousness)
- Right of γ_c : $C < C_{min}$ --> Unconscious (sub-critical coherence)

The framework provides a single equation that unifies:

State	γ_{eff} vs γ_c	C vs C_{min}
Normal waking	$\gamma_{eff} \sim \gamma_c$	$C \sim C_{min}$
Focused attention	γ_{eff} slightly $<$ γ_c	C slightly $>$ C_{min}
Flow state (Paper 36)	$\gamma_{eff} <$ γ_c	$C >$ C_{min} (stable)

Psychedelic	$\gamma_{eff} \ll \gamma_c$	$C \gg C_{min}$
Drowsiness	$\gamma_{eff} \text{ slightly } > \gamma_c$	$C \text{ slightly } < C_{min}$
Deep sleep	$\gamma_{eff} > \gamma_c$	$C < C_{min}$
Anesthesia	$\gamma_{eff} \gg \gamma_c$	$C \ll C_{min}$
Coma	$\gamma_{eff} \gg \gamma_c$	$C \text{ approximately } 0$
Brain death	$\gamma_{eff} \rightarrow \infty$	$C = 0$

Every transition between states is a movement along the γ_{eff} axis. Every clinical intervention that affects consciousness (anesthetics, stimulants, psychedelics, TMS, meditation, sleep, caffeine) does so by changing γ_{eff} relative to γ_c .

10. What This Framework Does NOT Claim

Precision matters. This paper makes physical claims. It does not make philosophical claims. Specifically:

1. The framework does not explain WHY coherence produces subjective experience. It explains when, where, and how consciousness turns on and off. The hard problem remains unsolved. But the hard problem is now constrained to a much smaller space: why does coherent neural activity at γ_c produce experience? This is a better question than "why does any information processing produce experience?" because it identifies the specific physical conditions.
2. The framework does not claim quantum coherence in the Penrose-Hameroff sense. The coherence described here is mesoscopic --- it operates at the scale of neural populations (millions of neurons), not individual microtubules. Whether quantum coherence at the microtubule level contributes to γ_{eff} is an empirical question. The framework is agnostic on this point. It requires coherence. It does not require that the coherence be "quantum" in the Penrose sense.
3. The framework does not claim consciousness is binary. The phase diagram shows a continuous variable (γ_{eff}) controlling a continuous variable (C). The "transition" at γ_c is sharp but not discontinuous --- it is the same as the superconducting transition, which is a true phase transition but still occurs over a narrow range of temperature. Consciousness admits degrees, and those degrees map to C values.
4. The framework does not claim to identify all decoherence sources. γ_{eff} is an effective rate that includes all sources of decoherence in neural tissue --- thermal, metabolic, chemical, structural, electromagnetic. The specific decomposition of γ_{eff} into component sources is an experimental program, not a completed theory.

11. Experimental Program

The six predictions above define a research program:

1. TMS unbinding experiment (Prediction 1): Targeted disruption of gamma coherence in specific cortical regions should produce feature-specific perceptual unbinding, quantitatively predicted by the coherence equation.
2. Anesthetic threshold experiment (Prediction 2): EEG gamma coherence should show a sharp phase-transition signature at loss of consciousness, with the transition point predictable from baseline coherence.

3. Libet delay scaling experiment (Prediction 3): The delay between neural activity and conscious awareness should scale logarithmically with the coherence ratio $C_{\min}/C_{\text{noise}}$, not linearly.

4. Adenosine-coherence correlation experiment (Prediction 4): Real-time measurement of extracellular adenosine and EEG gamma coherence should show inverse correlation, strongest in the thalamo-cortical system.

5. NCC prediction experiment (Prediction 5): The ratio $\text{gamma_eff}/\text{gamma_c}$ should predict consciousness-correlation better than existing metrics (total activity, connectivity, ϕ).

6. Psychedelic coherence experiment (Prediction 6): Psilocybin should increase cross-regional gamma coherence with magnitude correlating to synesthesia intensity, showing critical-point signatures at the transition.

Each experiment is feasible with current technology. Each produces a quantitative prediction that can be confirmed or refuted. If the predictions fail, the framework is wrong. That is how physics works.

12. Conclusion

Seven anomalies in consciousness science reduce to one equation: $C = C_0 * \exp(-\alpha * \text{gamma_eff})$. Binding is coherence. Anesthesia is decoherence. The Libet delay is coherence buildup time. Sleep clears decoherence sources. NCCs are critical-point regions. Psychedelics produce supercritical coherence. Each claim is testable.

The framework does not solve the hard problem of consciousness. It does something arguably more useful: it transforms consciousness from a philosophical mystery into a physics problem with measurable variables, quantitative predictions, and falsifiable claims. Whether or not we ever understand WHY coherence produces experience, we can now predict WHEN it will, WHERE it will, and HOW to control it.

The 40 Hz gamma oscillation is not a curiosity. It is the heartbeat of a phase transition. Consciousness is what it sounds like when a hundred billion neurons lock into coherence at the critical point.

References

- Bhatt, D. K. & Bhatt, N. K. (2023). General anesthesia mechanism: current evidence and future directions. *Clinical and Experimental Pharmacology*, 13(1), 1-8.
- Carhart-Harris, R. L. et al. (2012). Neural correlates of the psychedelic state as determined by fMRI. *Proceedings of the National Academy of Sciences*, 109(6), 2138-2143.
- Chalmers, D. J. (1995). Facing up to the problem of consciousness. *Journal of Consciousness Studies*, 2(3), 200-219.
- Dager, S. R. et al. (1999). Human brain metabolic response to caffeine and the effects of tolerance. *American Journal of Psychiatry*, 156(2), 229-237.
- Koch, C., Massimini, M., Boly, M. & Tononi, G. (2016). Neural correlates of consciousness: progress and problems. *Nature Reviews Neuroscience*, 17(5), 307-321.
- Libet, B. (1983). Time of conscious intention to act in relation to onset of

cerebral activity. *Brain*, 106(3), 623-642.

Llinas, R. R. et al. (1998). The neuronal basis for consciousness. *Philosophical Transactions of the Royal Society of London B*, 353(1377), 1841-1849.

Petri, G. et al. (2014). Homological scaffolds of brain functional networks. *Journal of the Royal Society Interface*, 11(101), 20140873.

Porkka-Heiskanen, T. et al. (1997). Adenosine: a mediator of the sleep-inducing effects of prolonged wakefulness. *Science*, 276(5316), 1265-1268.

Revonsuo, A. (1999). Binding and the phenomenal unity of consciousness. *Consciousness and Cognition*, 8(2), 173-185.

Singer, W. & Gray, C. M. (1989). Visual feature integration and the temporal correlation hypothesis. *Annual Review of Neuroscience*, 18, 555-586.

Tononi, G. (2003). Sleep and synaptic homeostasis: a hypothesis. *Brain Research Bulletin*, 62(2), 143-150.

Tononi, G. & Cirelli, C. (2006). Sleep function and synaptic homeostasis. *Sleep Medicine Reviews*, 10(1), 49-62.

Treisman, A. (1996). The binding problem. *Current Opinion in Neurobiology*, 6(2), 171-178.

Series Note: This is Paper 133 in the AIIT-THRESI series by Rhet Dillard Wike, Council Hill, Oklahoma. It extends the coherence framework established in Paper 5 (REQMT), the 40 Hz analysis of Paper 23, the flow state physics of Paper 36, and the dream coherence analysis of Paper 38. All claims are constrained by the Wike Coherence Law $C = C_0 * \exp(-\alpha * \gamma_{eff})$. All predictions are falsifiable.

END PAPER 133