What Makes Sleep a Distinct Biological Mode?

Substitution Limits, Organismal Integration, and the Component Evolution of Sleep

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Preprint notice

This manuscript has not been peer reviewed. It is a structured, curated theory synthesis based on published evidence, explicitly marked inferences, and preregistration-ready experiment proposals. It is not a systematic review or meta-analysis. The aim is to convert a broad literature into a smaller set of claims that can lose.

Abstract

Why sleep takes the form of a recurrent organismal mode remains unresolved despite an extensive literature on processes associated with sleep. A central source of confusion is that four questions are often treated as interchangeable: what counts as sleep, how prior wake history is regulated, which operations depend on a sleep mode, and how sleep evolved. Here I present a structured synthesis spanning sleep homeostasis, local network dynamics, plasticity, interregional timing, body-wide physiology, REM sleep, comparative sleep architectures, evolution, and technical counterexamples to global-offline necessity. I distinguish organismal sleep from sleep-like organismal modes, local sleep-like events, and sleep-associated operations, and introduce a typed causal audit separating functional states, sensors, history integrators, messengers, controllers, modes, events, interventions, functions, coupling profiles, observations, and evidence scope. The evidence rejects the inference that maintenance, learning, self-modification, or replay inherently require simultaneous global sleep. It also establishes cross-state occurrence, alternative natural architectures, and narrow operator transfer for selected processes. These findings do not establish broad substitution. I therefore define relative mode dependence against prespecified comparator classes, endpoints, time horizons, resource constraints, and biological equivalence margins. The residual explanatory space is organized into three levels: local mode dependence and state moderation; neural and neuron-body interaction; and batching or safety advantages. The same component discipline is then applied to evolution. Present-day mode dependence does not identify origin, and present-day substitutability does not erase possible historical selection. Phenotype, history-dependent regulation, controller pathways, operations, functions, spatial organi-

zation, and active modes require separate ancestral reconstruction. The decisive empirical task is to determine which, if any, operations retain a mode-specific advantage after fair biological substitution; the decisive historical task is to establish when and how the relevant components became coupled.

Keywords: sleep function; sleep evolution; sleep homeostasis; local sleep; REM sleep; relative mode dependence; substitution; Process S; homology; evolutionary lock-in

1. Introduction: useful operations do not yet explain a biological mode

Sleep is physiologically active, recurrent, reversible, and costly. It reduces normal environmental engagement and, in many species, exposes the organism to opportunity costs or predation. Yet the biological literature has identified no single process whose presence during sleep is sufficient to explain why sleep is organized as a distinct organismal mode. The problem is not a shortage of candidate functions. Sleep has been linked to synaptic regulation, memory, ion homeostasis, genomic stability, vascular and fluid dynamics, immunity, metabolism, tissue repair, and developmental plasticity. The problem is that occurrence during sleep is often treated as if it established necessity of sleep.

The distinction matters because many operations associated with sleep also occur outside organismal sleep. Hippocampal replay occurs during quiet wake and behavioral pauses [1,2]. Local cortical populations can enter off periods while an animal remains behaviorally awake [3]. In awake mice, experimentally imposed cortical on/off dynamics can modify local sleep pressure, synaptic markers, and a narrow memory output [4]. Birds and marine mammals can spatially divide sleep, while chinstrap penguins can accumulate large amounts of sleep in bouts lasting only seconds [5,6,7]. These findings refute a simple global-offline argument: the existence of maintenance, learning, self-modification, or replay does not logically entail that the entire brain or organism must enter simultaneous global sleep.

They do not prove that sleep is broadly replaceable. Wake replay may perform a different computation from sleep replay. A local operator may rescue one endpoint while leaving other neural, bodily, or regulatory states untouched. Unihemispheric sleep is an evolved architecture, not a within-individual substitute for bilateral sleep. A short-term behavioral rescue does not establish tissue restoration, later homeostatic equivalence, durability, or acceptable biological cost. The scientific target is therefore narrower and more tractable than the question “what is sleep for?”:

> **Which operation retains a measurable advantage in a local or organismal sleep mode after fair biological alternatives are tested?**

A second problem is historical. A current sleep-dependent operation need not be the reason sleep originated. It may have been co-opted after a quiescent mode already existed, become specialized through evolutionary lock-in, or lost an ancestral alternative. Conversely, an operation that can now be reproduced exper-

imentally outside sleep may still have favored the origin or maintenance of sleep because that substitute was unavailable to ancestral lineages. Current mode dependence and evolutionary origin therefore require different evidence.

This paper separates four explananda:

1. **Classification:** what counts as organismal sleep, a sleep-like organismal mode, a local sleep-like event, or a sleep-associated operation?
2. **Regulation:** what stores or represents prior wake history, and how is that history translated into sleep expression?
3. **Current mode dependence:** which operations remain non-equivalent outside sleep within a defined comparator class?
4. **Evolution:** which components are homologous, convergent, co-opted, independently gained, elaborated, locked in, or secondarily lost?

Figure 1. Four explananda and the inference barriers between them

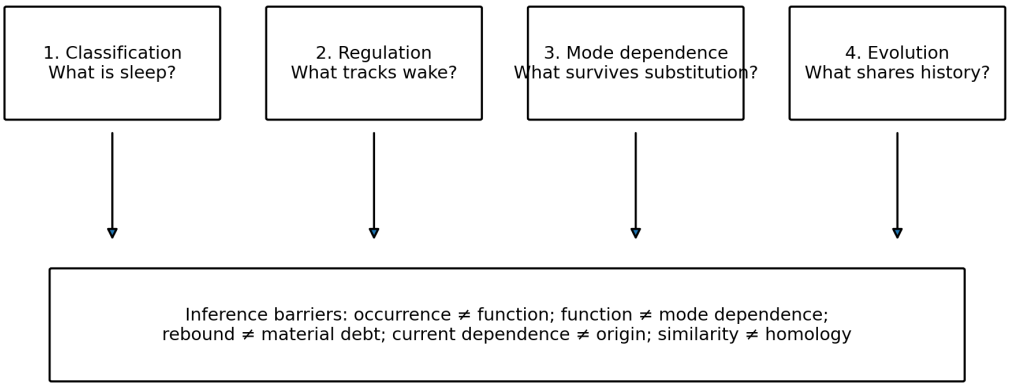

Figure 1: Four explananda and the inference barriers between them.

The paper makes four contributions. First, it proposes an operational taxonomy that does not define sleep by its presumed function. Second, it introduces a typed causal architecture that distinguishes the state requiring restoration from the sensors, messengers, controllers, operations, and readouts used to regulate it. Third, it replaces absolute claims of non-substitutability with bounded comparisons and a multidimensional substitution profile. Fourth, it applies the same component discipline to sleep evolution, where the complete phenomenon should not be treated as one historical unit before its components have been reconstructed.

The central claim is deliberately modest:

> **A useful process occurring during sleep does not establish current sleep-mode dependence. Current dependence must be tested relative to explicit biological comparators, while evolutionary explanation requires a separate reconstruction of component ori-**

gin, elaboration, maintenance, lock-in, and loss.

2. Methods: a structured theory-building synthesis

2.1 Design

This manuscript distills a twelve-module research program developed through broad synthesis, separate adversarial review, revision, and cross-module integration. The active corpus covers: global-offline arguments; Process S and sleep-pressure candidates; control architecture; evolution; plasticity and critical periods; local sleep, replay, and interregional timing; body-wide physiology; anesthesia, torpor, and consciousness boundaries; REM; natural short-sleep and spatially divided architectures; continual-learning agents; and a final master synthesis.

The review is **curated and question-driven**, not systematic. It does not claim exhaustive coverage. Sources were selected because they carried a central claim, provided a counterexample, altered the status of a claim, filled an empty comparison class, or defined a decisive experiment. This method is suitable for theory construction but limits field-wide absence statements. Whenever the manuscript says that no substitute or universal mechanism has been identified, the intended domain is the audited evidence set, not every paper ever published.

2.2 Claim types

The synthesis separates:

- **empirical findings:** what was measured;
- **causal findings:** what an intervention changed;
- **methodological rules:** what evidence is required for a conclusion;
- **epistemic non-implications:** what does not follow from a result;
- **theoretical hypotheses:** proposed present-day mechanisms;
- **historical hypotheses:** proposed evolutionary trajectories;
- **technical counterexamples:** engineered architectures that constrain logical necessity.

A construction is not cited as evidence for itself. A cross-study chain is treated as a hypothesis graph unless all causal links were measured in one system.

2.3 Evidence calibration

Mechanistic depth, taxonomic scope, and independent replication are evaluated separately. A deep mechanism in one cell type can be highly informative while remaining phylogenetically narrow. Conversely, broad comparative occurrence can have little mechanistic resolution. Recent 2025–2026 findings are treated as especially informative but still young.

Equivalence claims follow non-inferiority logic: failure to detect a difference is not evidence of equivalence [8,9]. A substitute must be evaluated against prespecified biological margins, not against a null-hypothesis p value.

2.4 Corrections and contested records

Corrections, errata, formal disputes, and retractions are treated as part of the evidence chain. The awake sharp-wave-ripple intervention by Jadhav and colleagues is therefore read with its 2026 erratum [10,11]. The clearance result of Miao and colleagues is read with its Author Correction and the subsequent formal challenge and reply [12,13,14,15]. A retracted sponge-root phylogenomic article is not used as positive evidence [16].

2.5 AI assistance and responsibility

ChatGPT assisted with literature organization, model comparison, adversarial review, claim governance, experiment architecture, drafting, and document production. Kimi was used in an independent parallel analysis that was examined only after the original ChatGPT synthesis had been frozen; selected ideas were then incorporated in narrower form. AI systems are not authors. All interpretive and editorial decisions remain the responsibility of the human author.

3. What is the object called sleep?

A theory of sleep can become circular if sleep is defined by the mechanism or function the theory is supposed to explain. The present paper therefore uses an operational taxonomy.

3.1 Organismal sleep

Organismal sleep is a recurrent, reversible biological mode or mode complex characterized, where measurable, by a species-specific cluster of:

- altered behavioral organization;
- altered responsiveness or arousal threshold;
- endogenous temporal regulation;
- characteristic neural and bodily dynamics;
- distinction from paralysis, acute pathology, anesthesia, and torpor;
- history-dependent regulation, where demonstrated.

No single criterion is required across all taxa. The definition is a cluster definition, not a claim of one function or one origin.

3.2 Sleep-like organismal mode

A **sleep-like organismal mode** satisfies several sleep criteria but lacks adequate evidence for one or more dimensions, such as homeostatic regulation, state-specific physiology, functional restoration, or distinction from another quiescent condition. Cnidarian work demonstrates that sleep-like, homeostatically regulated modes can exist without a central brain [17,18]. That establishes architectural possibility. It does not date the origin of sleep.

3.3 Local sleep-like event

A **local sleep-like event** is a spatially restricted neural event or regime with partial correspondence to sleep dynamics while the organism remains globally awake or in another mode. Natural local off periods in awake rats are the clearest example [3]. They can predict task errors without converting the whole animal into sleep.

3.4 Sleep-associated operation

A **sleep-associated operation** is a defined process that is enriched, gated, made more effective, or experimentally reconstructable during sleep. Examples include local on/off dynamics, spindle-ripple coupling, selected synaptic changes, REM-theta-dependent operations, dendritic spine selection, and defined immune or vascular effects [19,20,21,22,23]. An operation is not identical to the mode in which it usually occurs.

3.5 Neighboring states

Anesthesia, sedation, torpor, coma, neuromuscular blockade, and pathological unresponsiveness are not simply deeper sleep. They can mimic selected outputs while differing in causal architecture and functional consequences. Behavioral unresponsiveness is also not a sufficient exclusion criterion for external cognitive integration during sleep [24].

3.6 Does sleep form a natural biological kind?

The phrase “natural kind” can mean a shared phenotype, mechanism, regulator, function, or ancestry. Those are different claims. A strong case for a coherent historical sleep character would require converging evidence for:

1. a recurring organismal phenotype;
2. related history-dependent regulation;
3. homologous or transformable components;
4. a coherent ancestral reconstruction;
5. at least partly conserved causal roles.

Current evidence does not establish that all operationally classified sleep modes satisfy all five.

4. Typed causal architecture and relative mode dependence

The term “sleep pressure” often compresses several causal roles into one word. A mechanistic account should distinguish them.

For biological context κ, define:

$$\mathcal{M}_{\kappa} = (I, Z, S, H, B, C, q, e, U, F, G, \mathcal{O}, Y, \mathcal{E}).$$

Here I is an input class; Z a functional tissue, network, or bodily state; S a sensor; H a history integrator or accumulator; B a messenger; C a controller; q a local or organismal mode; e a measured event; U an intervention; F a functional output; G a coupling profile; $\mathcal{O}$ the observation model; Y the measured readout; and $\mathcal{E}$ the evidence scope, depth, and replication status.

Figure 2. Typed causal architecture for sleep regulation and sleep work

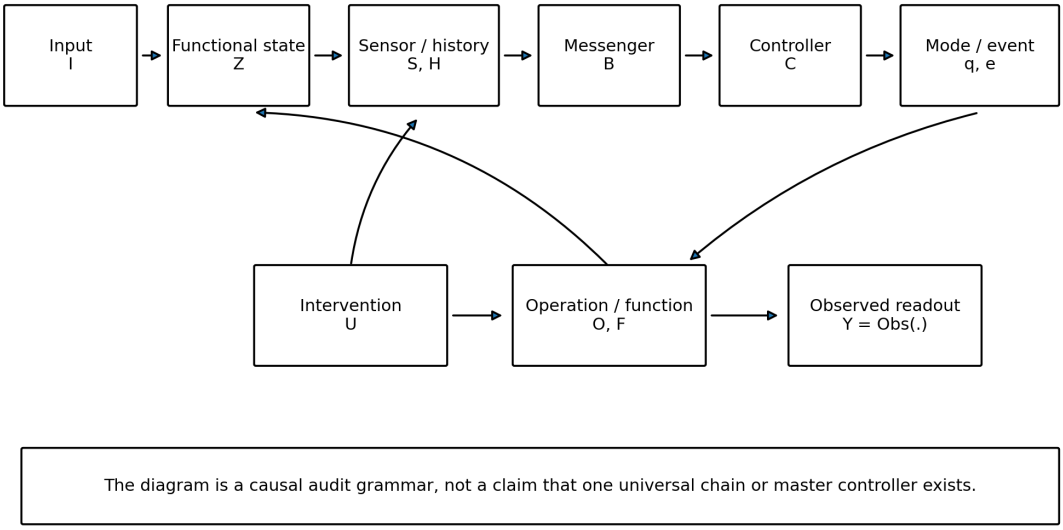

Figure 2: Typed causal architecture for sleep regulation and sleep work.

This grammar prevents three recurrent errors.

First, a messenger or controller state is not necessarily the tissue burden that needs restoration. Adenosine can alter sleep expression without being a universal material store [25]. Tryptamine can carry wake-history information to preoptic control systems without being the complete biological content of Process S [26].

Second, a mode is not an operation. Pharmacologically induced behavioral sleep can fail to reproduce a natural sleep-associated process. In zebrafish, synaptic loss depended on sleep pressure and a particular neuromodulatory configuration, not on behavioral sleep alone [27].

Third, a readout is not restoration. Slow-wave activity, sleep duration, latency, performance, and molecular markers may diverge. Reducing later SWA does not by itself show that all functional states have been restored.

4.1 Process S

The two-process model remains a powerful macroscopic description of sleep regulation [28]. But a predictive scalar does not identify an ontic scalar. Process S may summarize one deep state, a serial cascade, a coupled field, or several partially independent states. Its biological interpretation must remain output- and context-specific.

4.2 Input classes

Four input classes should remain distinct:

- **H:** basal history-dependent regulation during natural wake;
- **A:** allostatic, injury-, infection-, or damage-induced sleep;
- **D:** developmental or remodeling-associated sleep;
- **C:** circadian, motivational, and ecological gating.

A shared sleep output does not prove a shared upstream burden. DNA damage can recruit sleep after UV or mutagenic challenge without being the dominant everyday mammalian Process S [29,30].

4.3 Relative mode dependence

Absolute non-substitutability cannot be demonstrated because no experiment can test every imaginable alternative. The relevant object is **relative mode dependence**.

For operation O, target-state vector $\mathbf{Z}$, functional endpoints $\mathbf{F}$, context κ, comparator class $\mathcal{C}$, resource envelope $\mathcal{R}$, time horizon τ, and biological margins δ:

$$\mathrm{RMD}(O; \mathbf{Z}, \mathbf{F}, \kappa, \mathcal{C}, \mathcal{R}, \tau, \delta).$$

Relative mode dependence is supported when the reference sleep implementation meets prespecified criteria and every admissible tested comparator fails at least one equivalence constraint, after accounting for operator fidelity, timing, off-target effects, and biological cost.

The strongest legitimate conclusion is:

> **Within the tested comparator class, endpoint set, resource envelope, and time horizon, no non-sleep implementation was equivalent.**

It is not:

> the operation requires sleep in principle.

5. Evidence cartography: what occurs outside organismal sleep?

The evidence outside organismal sleep falls into four classes.

Evidence class	Question answered	Example	What it does not establish
E1: Cross-state occurrence	Does the process also occur outside sleep?	Wake replay; continuous molecular turnover	Functional equivalence

Evidence class	Question answered	Example	What it does not establish
E2: Alternative natural architecture	Can sleep be spatially or temporally reorganized?	Unihemispheric sleep; microsleeps	Same-system substitution
E3: Operator transfer	Can a sleep-associated dynamic be induced outside sleep?	Awake cortical on/off induction	Broad restoration
E4: Functional substitution	Does an alternative meet a prespecified endpoint margin?	Narrow behavioral rescue	Multi-domain mode equivalence

5.1 Continuous maintenance

The brain is not maintained only during sleep. Protein turnover, membrane renewal, organelle quality control, ion regulation, and receptor trafficking continue during wake. This alone is enough to reject the literal statement that biological maintenance cannot occur online. It does not show that every rate, coordination pattern, or functional repair can be achieved under normal wake physiology.

5.2 Wake replay

Reverse and remote replay occur during wake [1,2]. Awake sharp-wave ripples can influence spatial memory, although the intervention literature is task-specific and one classic study now carries an erratum affecting the exact stimulation pattern [10,11,31]. Wake replay therefore rejects exclusivity. It does not show that sleep replay is redundant or that identical interregional operations occur under matched conditions.

5.3 Natural local sleep

After extended wake, local cortical populations can enter off periods while the rat remains globally awake, and such events predict task errors [3]. This demonstrates spatially distributed sleep pressure and a natural local alternative to whole-organism sleep. It also shows the cost: an unpredictable local outage can hit a currently required network.

5.4 Awake operator transfer

The strongest narrow transfer result is the induction of cortical on/off periods in awake mice [4]. The intervention reduced later local SWA and synchrony, changed synaptic markers, and rescued one recognition-memory outcome after sleep depri-

vation. Tonic inhibition with a similar reduction in mean firing did not reproduce the effect, implicating temporal organization rather than mere inactivity.

The result is important but bounded. It does not establish long-term safety, REM substitution, broad body restoration, or equivalence across memory systems. It also does not show that reduced coupling itself is necessary; the induced event may be sufficient without identifying why natural sleep normally generates it.

5.5 Spatial division

Fregattbirds sleep in flight and can show unihemispheric or bihemispheric sleep while remaining airborne [5]. Fur seals can suppress REM for long periods at sea without a corresponding conventional rebound [7]. These cases demonstrate that sleep architecture is spatially and mode-specifically plastic. They do not show that all omitted operations were executed elsewhere or that no latent costs remained.

5.6 Temporal fragmentation

Chinstrap penguins can accumulate roughly normal daily sleep through thousands of seconds-long microsleeps [6]. This challenges the assumption that sleep must be consolidated into long bouts. It does not establish equivalence for every species, function, or ecological context.

5.7 Extreme reduction and preserved performance

Pectoral sandpipers can sharply reduce sleep during mating competition, with high performers obtaining more offspring [32]. Dolphins can sustain a narrow echolocation vigilance task for days [33]. Chronic human sleep restriction, however, produces cumulative neurobehavioral deficits even when subjective sleepiness adapts imperfectly [34]. Low observed sleep is therefore not a direct measure of low biological need.

5.8 Technical counterarchitectures

Continual-learning robots and digital systems can learn online through modularity, redundancy, separate evaluation paths, replay buffers, and controlled updates [35]. This constrains any claim that self-modification logically requires global sleep. It does not constitute biological evidence because engineered systems can copy, pause, replace, or externalize state in ways living organisms cannot.

6. What fair substitution has and has not established

Cross-state occurrence is not substitution. A substitute should be described through a multidimensional evidence profile:

$$\mathcal{P}_{sub}^{A,R} = (P_O, P_Z, P_F, P_H, P_T, P_B, P_C),$$

where P_O is operator fidelity, P_Z target-state normalization, P_F functional non-inferiority, P_H homeostatic or controller equivalence, P_T durability, P_B breadth, and P_C cost and side effects.

Figure 3. Evidence classes and the multidimensional substitution profile

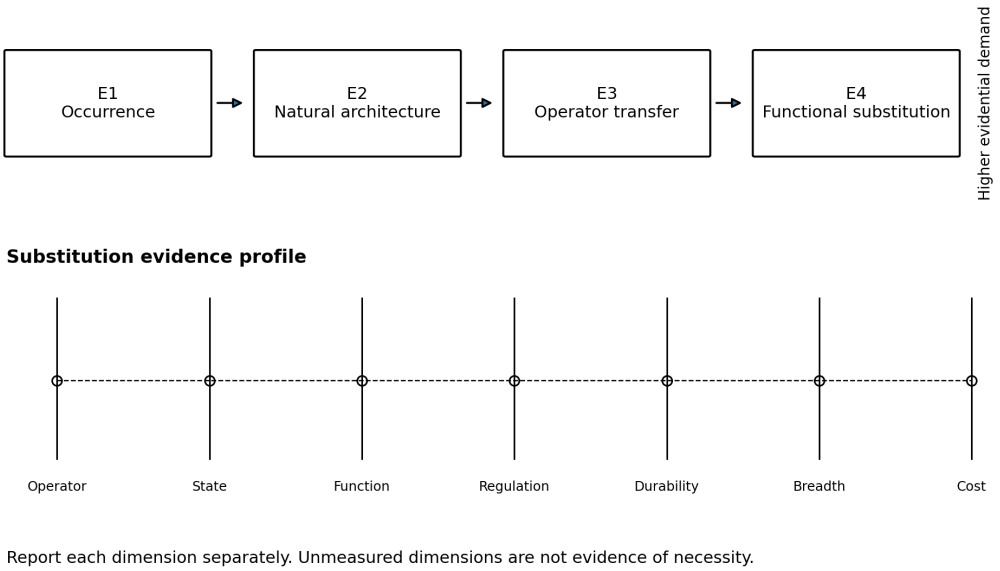

Figure 3: Evidence classes and the multidimensional substitution profile.

6.1 Narrow substitution exists

The Driessen intervention reaches a meaningful level for selected endpoints: the operator was reconstructed outside organismal sleep, local state markers shifted, and one behavior was rescued [4]. That is more than occurrence. It is still narrower than broad mode equivalence.

6.2 Wake replay is not a matched substitute

Wake replay demonstrates that replay is not sleep-exclusive. Existing studies do not generally match sequence content, event number, phase, neuromodulatory context, starting burden, and downstream function across wake and sleep. The equivalence question therefore remains open.

6.3 Natural architectures are not same-system substitutions

Unihemispheric sleep, microsleep, and ecological sleep compression show that evolution can reorganize sleep. They do not compare the same organism, starting state, endpoint set, and resource envelope against ordinary bilateral sleep. They are powerful architecture cases, not simple substitutes.

6.4 Pharmacological state labels are insufficient

A hypnotic may increase behavioral sleep or slow-wave activity while altering natural oscillations, vascular dynamics, or specific operations. Norepinephrine-mediated vasomotion is one example of a process that can be changed by a hypnotic even when sleep-like behavior is present [23]. In human T cells, state-dependent neuroendocrine signaling can be partly reconstructed ex vivo, but the result concerns a specific immune readout rather than a global sleep milieu [22].

6.5 The bounded negative conclusion

Within the audited comparator set, endpoint-specific functional substitution exists only in limited contexts. No evaluated alternative has demonstrated durable, broad neural, bodily, and regulatory equivalence to organismal sleep. This is a corpus-bounded audit result, not proof that such an alternative is impossible.

7. Three-tier hypothesis architecture

The unresolved explanatory space should not be compressed into one vague claim of "coordination." Six separable hypotheses occupy three levels.

Figure 4. Three-tier hypothesis architecture

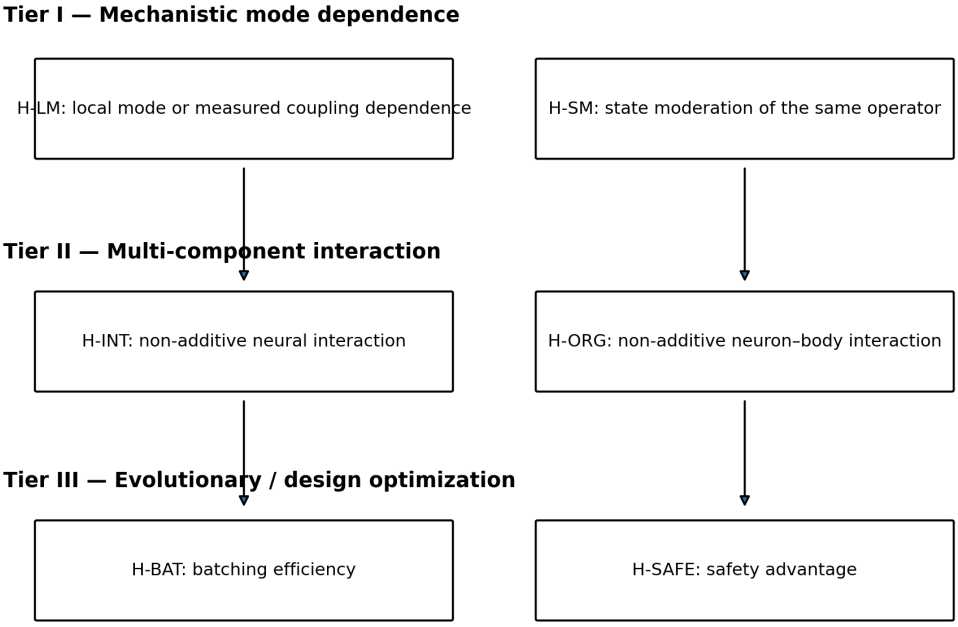

Figure 4: Three-tier hypothesis architecture.

Tier	Hypothesis	Exact question	Decisive evidence
I	**H-LM: local mode dependence**	Does a verified local mode or reduced coupling contribute beyond a matched operator?	Same operator with and without the local mode
I	**H-SM: state moderation**	Does the same operator have different efficacy across modes because of measured moderators?	Operator × mode × moderator design
II	**H-INT: neural interaction**	Do distributed neural operations produce a non-additive gain?	Factorial A, B, A+B design
II	**H-ORG: neuron-body interaction**	Do neural and peripheral operations interact beyond additivity?	Factorial neural × peripheral intervention
III	**H-BAT: batching**	Is consolidated sleep cheaper or faster for equivalent restoration?	Cost-matched distributed alternative
III	**H-SAFE: safety**	Does a declared organismal mode reduce failures relative to local outages?	Error- and fitness-matched comparison

7.1 H-LM: local mode dependence

Local off periods and induced on/off dynamics support the importance of a dynamic regime [3,4]. They do not yet show that reduced task or sensory coupling itself is necessary once the downstream operator is matched. H-LM remains open.

7.2 H-SM: state moderation

Several processes are gated by neuromodulatory or physiological state. Zebrafish synapse loss depended on sleep pressure, adenosine, and low noradrenergic tone [27]. Homer1a-dependent AMPA regulation requires a sleep-associated molecular context [36]. Human T-cell integrin activation is modulated by sleep-associated G-protein-coupled signals [22]. These cases support process-specific gating. The

strongest form of H-SM—an identical downstream operator showing different efficacy across matched modes because a measured moderator mediates the difference—remains incompletely tested.

7.3 H-INT: neural interaction

Sleep oscillations can be precisely coupled. Triple phase-locking among cortical slow oscillations, thalamic spindles, and hippocampal ripples supports memory formation in rodents [19]. Manipulating hippocampo-cortical coupling can affect consolidation [37], and human stimulation that augments hippocampal-prefrontal synchrony can improve memory [38]. Yet timing dependence is not automatically a non-additive interaction. A factorial design must compare the components alone, asynchronous combination, phase-correct combination, and phase-incorrect combination. Null memory effects despite enhanced oscillations also show that rhythm amplification is not sufficient [39].

7.4 H-ORG: neuron-body interaction

Sleep affects hematopoiesis and atherosclerotic inflammation [40], can be recruited after myocardial infarction to limit damage [41], and in Drosophila can gate clearance of brain-derived lipids by peripheral blood cells [42]. These are real pathways. They do not establish a non-additive organismal synergy. H-ORG requires separate manipulation of neural and peripheral arms and a combined condition that exceeds the expected sum.

7.5 H-BAT and H-SAFE

A consolidated mode could be advantageous even if all component operations were technically reproducible elsewhere. Batching may reduce time, energy, switching costs, or ecological exposure. Safety may prevent unpredictable local failures during ongoing behavior. Predation-related asymmetry, in-flight sleep, seasonal sleep reduction, and microsleep motivate these hypotheses [43,5,32,6]. They do not directly test them.

8. Regulation, Process S, and the unresolved burden

8.1 History-dependent regulation is real

Prolonged wake changes later sleep, local dynamics, performance, and molecular states. The two-process model captures a robust macroscopic regularity [28]. The unresolved issue is what Process S compresses biologically.

8.2 Candidate roles

Adenosine

Adenosine links activity and metabolism to sleep expression and mediates part of the effect of prolonged wake [25]. It is a strong regional messenger and gate, not a demonstrated universal store.

Tryptamine-GPR139

Tryptamine from wake-active monoaminergic neurons tracks sleep history and acts through GPR139 in preoptic circuitry [26]. This is a strong mammalian messenger-controller pathway. It may count activity of wake systems rather than a global material burden.

Synaptic strength

Acute prefrontal synaptic potentiation can increase NREM sleep and delta activity [44]. Sleep pressure modulates synapse-number changes in zebrafish [27]. Synaptic strength is therefore a local burden and operation target, but a selective wake rescue that normalizes function and later rebound remains missing.

Intracellular chloride

Wake-history-dependent chloride regulation can affect local cortical sleep pressure, recruitment, and performance [45]. Chloride may be a burden, a coupling variable, or a downstream state in a broader cascade.

Redox and lipid-peroxidation memory

In Drosophila sleep-promoting neurons, mitochondrial electron flow, lipid peroxidation, and a voltage-gated Kvβ-NADP(H) memory form a mechanistically deep controller pathway [46,47]. The mechanism is strong in a narrow cell population and research lineage. It may store controller history rather than the total repair burden of the brain.

DNA stability

Sleep is linked to chromosome dynamics and DNA repair in zebrafish neurons [48,30]. In cnidarians, DNA damage can recruit sleep-like behavior and sleep is associated with reduced damage-response markers [29]. These findings establish challenge and repair pathways; they do not identify DNA damage as the universal basal mammalian Process S. Short-sleeping cavefish can show elevated damage markers without an obvious corresponding aging phenotype, illustrating the difference between marker, mechanism, and fitness cost [49].

8.3 No universal material debt has been identified

No audited candidate closes the full chain required for a universal basal material-debt claim:

natural wake burden → sensor → controller → sleep operation → state and functional rescue

This does not imply that no common burden exists. It means the present evidence does not identify one.

8.4 Drive generation and state competence

A high upstream drive and the capacity to express a stable sleep mode can dissociate. A controller can be masked without repairing the target state; a target state can be repaired while a slow controller continues to report need. This distinction generates a high-value experiment: controller-tissue double dissociation.

9. Stress tests: REM, body coupling, and short-sleep systems

9.1 REM is a mode complex

REM contains tonic and phasic substructure, specific controllers, motor atonia, eye movements, theta dynamics, dendritic events, and narrow causal operations. REM-theta disruption can impair contextual memory [20]. REM can select or prune developing synapses [21], and reactivation of small adult-born ensembles during REM can support memory in mice [50]. These findings support particular REM operations, not one universal REM function.

Long REM suppression in fur seals and paradoxical memory outcomes after pharmacological REM suppression constrain strong universal claims [7,51]. The correct question is not whether REM "has a function," but which operator needs which REM context and remains non-equivalent elsewhere.

9.2 Body-wide physiology

Sleep is embedded in autonomic, thermal, vascular, metabolic, endocrine, and immune physiology. Challenge pathways are especially strong: peripheral injury or inflammation can recruit sleep, and sleep can alter subsequent tissue outcomes [41]. Basal daily regulation is harder. A pathway showing that sleep changes a tissue is not automatically the pathway that causes daily sleep need.

The clearance literature illustrates the danger of overcompression. Hauglund and colleagues identify norepinephrine-mediated vasomotion and fluid dynamics during NREM [23]. Miao and colleagues report reduced clearance under their sleep and anesthesia conditions, but the result has a correction and a formal methodological dispute [12,13,14,15]. "Sleep clears waste" and "sleep reduces clearance" are both too broad without specifying route, tracer, compartment, state, anesthesia, and endpoint.

9.3 Natural short sleep and alternative architectures

Short sleep is not one mechanism. It can reflect:

- ecological suppression;
- controller or gate changes;
- spatial division;
- temporal fragmentation;
- altered burden accumulation;

- improved operation efficiency;
- tolerated residual costs;
- measurement limitations.

Pectoral sandpipers, frigatebirds, penguins, fur seals, dolphins, Drosophila mutants, and cavefish occupy different positions in this design space [32,5,6,7,33,52,49]. Their diversity argues against a fixed dose law. It does not establish absence of sleep need.

10. Does sleep form one coherent historical character complex?

A historical claim needs a defined character. “Sleep” can refer to:

- organismal quiescence;
- increased arousal threshold;
- history-dependent regulation;
- local off dynamics;
- a particular controller;
- slow-wave or active modes;
- one operation or function.

Those components may have different histories.

Cnidarians demonstrate sleep-like modes and homeostatic regulation without central brains [17,18]. Zebrafish possess multiple neural sleep signatures [53]. Drosophila species show divergence in amount, depth, pharmacology, and rebound [54]. Octopus active sleep and jumping-spider retinal movements expand the space of active sleep-like components beyond vertebrates [55,56]. These findings are consistent with a deep precursor, multiple gains, repeated co-option, or a mosaic of homology and convergence.

The animal root itself remains contested. Synteny evidence supports ctenophores as sister to other animals [57], while a corrected partitioned-phylogenomics analysis supports sponges [58,59]. A separate 2025 sponge-root article was retracted and supplies no positive evidence [16]. Root topology alone would not solve sleep history because comparable phenotype and homeostasis data remain sparse in several root taxa.

The strongest current wording is:

> **Current evidence does not establish that all operationally classified sleep modes constitute one homologous, mechanistically coherent historical character complex.**

Figure 5. Component evolution of sleep as a historical model space

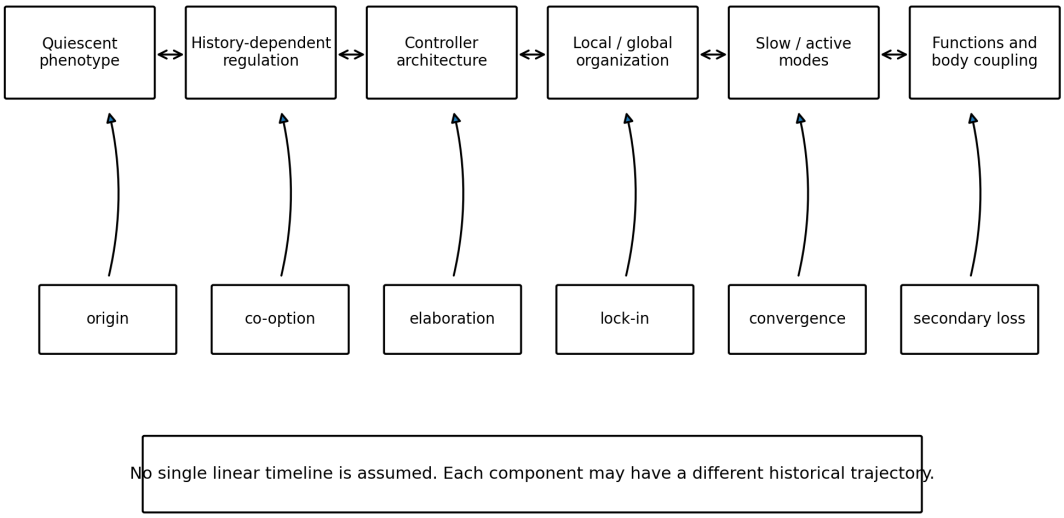

Figure 5: Component evolution of sleep as a historical model space.

11. Component evolution: origin, co-option, lock-in, and loss

11.1 Separate historical processes

A component can undergo:

- **origin:** first appearance;
- **elaboration:** increased complexity or range;
- **co-option:** recruitment for a new role;
- **maintenance:** continued selection because of current benefit;
- **lock-in:** derived dependence after alternatives are lost;
- **convergence:** independent emergence of similar features;
- **secondary loss:** reduction or disappearance.

Current mode dependence may reflect lock-in rather than origin. Current substitutability may rely on a modern intervention unavailable ancestrally.

11.2 Representative component matrix

Component	Present evidence	Historical alternatives	Current verdict
Reversible quiescence with arousal change	Broad across bilaterians and cnidarians	Deep precursor or multiple gains	Deep precursor plausible, not proven

Component	Present evidence	Historical alternatives	Current verdict
History-dependent regulation	Strong in many taxa, variable in form	Ancestral, repeatedly added, or repeatedly lost	Whole-state history unresolved
DNA-damage coupling	Cnidaria and vertebrate mechanisms	Deep co-option, challenge-specific convergence	Causal-role correspondence; homology open
Local off dynamics	Strong in mammalian cortex	Derived cortical specialization or broader network principle	Current mechanism; historical depth unknown
Multiple slow/active modes	Vertebrates, octopus, possible spider component	Homology, convergence, generic network attractor	Component convergence plausible
Unihemispheric organization	Birds and marine mammals	Independent ecological specialization	Strong secondary architecture
REM-associated operations	Multiple narrow vertebrate mechanisms	Early component package, elaboration, or repeated co-option	No universal function identified

11.3 The possible “birth of the counter”

One historical model is that quiescence preceded explicit history-dependent defense, and that an older mode later became coupled to a burden integrator. This model is compatible with existing data but not favored decisively over alternatives. It should be treated as a model to compare, not an answer.

11.4 Current causal evidence constrains history without determining it

If a local operator can be executed during wake, then global sleep is not its only physically possible implementation. That constrains strong necessity stories. It does not show that the operator was historically irrelevant. If a modern REM operation is currently non-equivalent outside REM, that may reflect derived specialization rather than original selection. Functional and evolutionary inference therefore meet at component identity, not at one common proof rule.

12. Decision experiments

The paper’s value depends on experiments that can weaken its branches. Four designs are sufficient for a near-term agenda.

12.1 Process-specific mode × moderator test

Compare natural sleep, EEG-verified quiet wake, and active wake for one defined operation. Measure and selectively manipulate one process-specific moderator rather than invoking a global "sleep milieu." Where relevant, separate damage formation from repair rate.

Interpretation:

- a residual sleep-mode effect after operator and moderator matching strengthens H-LM;
- complete mediation by the moderator strengthens process-specific H-SM;
- no advantage weakens mode dependence for that operation.

12.2 Matched cross-state operator reconstruction

Reconstruct the same downstream operator across quiet wake and sleep, matching target population, event number, phase, amplitude, duration, and starting state. Measure operator fidelity, target-state normalization, function, later regulation, durability, and cost.

This is the direct test of whether a state label contributes beyond the operator itself.

12.3 Factorial interaction test

For two neural operations, or one neural and one peripheral operation, compare:

1. control;
2. A alone;
3. B alone;
4. A+B asynchronous;
5. A+B phase-correct;
6. A+B phase-incorrect.

The interaction scale must be prespecified. A combined effect larger than either component is not sufficient; the relevant quantity is the residual beyond the expected joint effect.

12.4 Controller-tissue double dissociation

Manipulate the controller signal and the candidate target state independently:

- high burden, controller intact;
- high burden, controller expression suppressed;
- burden selectively reduced, controller initially high;
- normal mode expression with the operation blocked;
- burden and controller jointly normalized.

Measure target state, function, mode expression, operation, and delayed rebound. This design distinguishes masking of sleep need from actual restoration.

13. Relationship to existing frameworks

The present architecture is not a universal replacement for established theories. It asks a different question: what evidential step connects a useful process to a distinct mode?

13.1 Synaptic homeostasis

Synaptic homeostasis explains important local and global regularities, and causal evidence links synaptic strength to sleep pressure [44]. It does not by itself establish organismal-mode necessity, because selected synaptic operations can be local, conditional, or transferred across states [27,4].

13.2 Clearance frameworks

Clearance and fluid-dynamics frameworks identify real transport processes [23]. The heterogeneous and contested literature requires route- and assay-specific claims [12,14,15]. A clearance function is not automatically the daily burden that causes sleep.

13.3 Energy restoration and allocation

Energy-based theories can explain why operations are scheduled differently across modes and why sleep quantity is ecologically plastic. They remain compatible with batching. They do not establish a unique material counter or exclude non-energetic mechanisms.

13.4 Adaptive inactivity

Adaptive inactivity explains timing and ecological negotiation. The striking flexibility of sleep supports part of this account [32,5]. History-dependent regulation and local use dependence show that sleep is not merely arbitrary inactivity.

13.5 The window thesis K13

K13 proposes that sleep is a defended physiological window in which selected maintenance chemistry operates faster, with drive coupled to material burden. The present paper retains narrower elements: process-specific state moderation, drive versus state competence, vulnerability surfaces, and a direct sleep-versus-quiet-wake rate comparison. It does not adopt a universal material counter, a single global milieu, or a confirmed linear origin sequence.

14. Limitations and conclusion

14.1 Limitations

This is a curated theory-building synthesis, not a systematic review. The source base is heterogeneous across species, developmental stages, assays, and causal

depth. Some of the most informative mechanistic studies are recent and not independently replicated. Several literatures are research-house concentrated. Negative conclusions are bounded to the audited corpus. The paper proposes an audit architecture rather than one compact universal biological mechanism, and that methodological breadth risks insufficient explanatory compression.

AI assistance enabled a wide synthesis but does not substitute for independent scientific review. The manuscript should therefore be read as a preprint-level argument and experiment agenda, not as a final adjudication of the field.

14.2 Conclusions

Many processes associated with sleep also occur during wakefulness, local sleep-like dynamics, or spatially divided modes. This rejects the inference that maintenance, replay, or self-modification inherently require simultaneous global sleep. It does not establish broad replacement of organismal sleep.

The next empirical task is to determine which, if any, operations retain a mode-specific advantage after matched biological alternatives are tested across target states, functions, regulatory residuals, durability, breadth, and cost. The residual hypotheses fall into three levels: local mode dependence and state moderation; neural and neuron-body interaction; and batching or safety advantages. None has yet won as a universal explanation.

The historical task is different. Current dependence cannot identify origin unless organismal mode, history-dependent regulation, controllers, operations, functions, and spatial organization are reconstructed separately and compared with alternatives available in ancestral lineages. Sleep may ultimately prove to be a coherent mode complex assembled from components with different histories and different degrees of present-day mode dependence. Current evidence establishes neither one universal function nor one whole-state origin.

Declarations

Competing interests

The author declares no competing interests.

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Data and materials availability

The manuscript is based on published literature and a versioned internal claim-and-source register. A compact technical supplement accompanies this preprint.

AI assistance

ChatGPT assisted with research organization, synthesis, adversarial testing, claim governance, experiment design, drafting, and document production. Kimi was used in a separate parallel analysis and selected ideas were incorporated only after independent comparison. The author remains responsible for all claims, interpretations, and final wording.

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