Sleep as an Evolutionary Solution to the PlasticityStability DilemmaA Unified Hypothesis of Bidirectional Memory Adjudication and Instinctual Maintenance

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Abstract

A fundamental theoretical schism persists in sleep research: the **Synaptic Homeostasis Hypothesis (SHY)** emphasizes sleep's role in restoring metabolic balance through global synaptic downscaling, while the **Active Systems Consolidation Theory** focuses on the selective transfer and reorganization of memory traces during sleep. While each explains some phenomena, neither fully addresses (1) the **high selectivity of memory consolidation** or (2) why individuals lacking real-world experience exhibit complex **instinctual dreams**. More critically, both overlook a core evolutionary question: **how does the nervous system maintain innate instinctual circuits that are crucial for species survival but may lie dormant for long periods in modern environments?**

This paper proposes the **Offline Neural Plasticity Homeostasis Hypothesis (ONPH)**, redefining sleep as an **active, algorithmic offline maintenance program** executed by the nervous system in a sensory-isolated state. ONPH rests on two core mechanisms:

Pillar I: The Bidirectional Adjudication Pathway for Ontogenetic Memory.
We propose that lasting memory formation is not unilaterally completed during sleep but follows a closed loop of "**sleep-phase synaptic tagging - wake-phase behavioral confirmation**." Sleep (particularly slow-wave sleep) performs an initial screening based on **emotional salience**, establishing molecular tags on memory traces. Wakeful behavioral reuse or cognitive recall then provides the necessary **confirmation signal**, triggering synaptic capture and stabilization. Unconfirmed tags are pruned by the system, implementing **active forgetting**. This mechanism naturally explains the spacing effect and resolves the logical paradox of how "selective retention" emerges from "global downscaling."

Pillar II: The Anti-Degradation Maintenance of Phylogenetic Memory.
To counter the evolutionary risk posed by Hebbian "use it or lose it" principles—namely, the potential degradation of key survival instinct circuits due to prolonged disuse—**REM sleep acts as an endogenous instinct generator**. By periodically and forcibly activating genetically encoded Fixed Action Patterns (e.g., predation, escape, mating), sleep ensures these "hardware drivers" remain functionally ready, even in stimulus-impoverished modern

environments. This explains a series of seemingly disparate phenomena, from Jouvet's cat experiments to human adolescent sexual dreams.

Within this framework, **dreaming** is reinterpreted as **spurious signal propagation** arising during high-load neural plasticity regulation—a byproduct of the maintenance process, not its primary function. The bizarreness of dream content and its high rate of forgetting both stem from the lack of wake-phase behavioral confirmation.

ONPH not only bridges the gap between SHY and active consolidation theories but also provides novel mechanistic explanations for sleep disorders like insomnia and PTSD, deriving non-pharmacological intervention strategies such as "**signal masquerading**" and "**wake closure therapy**." The hypothesis proposes a series of testable experimental predictions, aiming to advance sleep research from phenomenological description to algorithmic analysis.

Keywords: Sleep Function; Memory Consolidation; Synaptic Plasticity; Instinct; Evolutionary Neurobiology; Computational Modeling; Sleep Disorders

Chapter 1: The Nervous System's Core Dilemma: From the Plasticity-Stability Paradox to the Evolutionary Origin of Sleep

1.1 Revisiting the Plasticity-Stability Paradox

The remarkable adaptability of the vertebrate nervous system stems from its **plasticity**—the ability to encode new experiences by adjusting synaptic strength. However, unconstrained plasticity leads to two critical issues: (1) **Catastrophic Forgetting**: new information continually overwriting old, disrupting system continuity; (2) **Instinctual Circuit Interference**: excessive experience-driven plasticity could disrupt genetically blueprinted, survival-critical fixed action patterns (e.g., breathing, heart rate regulation, escape responses). Conversely, an overly stable system cannot adapt to environmental changes. This "**Plasticity-Stability Paradox**" is a central challenge in nervous system design.

Sleep, a conserved behavior occupying nearly one-third of life in all vertebrates, likely represents an evolutionary solution. The ONPH posits that sleep's essence is the **temporal separation of "online learning" and "offline sorting,"** allowing for deep network optimization without interrupting real-time survival behaviors.

1.2 Contributions, Contradictions, and Blind Spots of Prevailing Theories

Current dominant theories are one-sided and have unavoidable limitations:

Theory	Core Proposition	Contribution	Limitations (ONPH Perspective)
Synaptic Homeostasis Hypothesis (SHY)	Sleep restores metabolic and energy homeostasis by globally downscaling total synaptic strength.	Explains sleep's restorative function; links synaptic strength to system energy budget.	1. **Selectivity Paradox**: Cannot explain why some low-strength memories are retained. 2. **Dream Vacuum**: Cannot explain instinct-themed dreams (e.g., chasing, falling).
Active Systems Consolidation Theory	During slow-wave sleep, memory traces are replayed and transferred along the hippocampal-neocortical axis for long-term storage.	Establishes sleep's causal role in memory consolidation; reveals replay mechanisms.	1. **Confirmation Gap**: Implies sleep is sufficient for consolidation, neglecting the decisive role of wakeful feedback. 2. **Instinct Neglect**: Focuses solely on acquired experience, unable to explain sleep-related activity of innate behavioral patterns.
Threat Simulation Theory	The function of dreaming is to simulate potential threats to train and enhance survival responses.	Provides a functionalist explanation for dream content.	1. **Overly Specific**: Cannot explain numerous non-threatening, even pleasant dreams. 2. **Mechanistic Vagueness**: Does not clarify the neural implementation of this "simulation" or its relation to sleep physiology.

More fundamentally, none of the existing theories directly addresses: **How does the nervous system prevent the "disuse" degradation of innate instinctual circuits that were crucial in evolutionary history but may be rarely triggered in a modern individual's lifetime?**

1.3 ONPH: Sleep as an Offline Maintenance Algorithm

The ONPH advocates abandoning the outdated view of sleep as passive "rest," instead analogizing it to the **offline maintenance window of a complex computing system**. During this window, the brain severs most external input/output, utilizing internally generated data (e.g., sharp-wave ripples, PGO waves) to execute key tasks:

1. **Data Sorting & Persistence**: Screening and tagging valuable short-term memories, integrating them into long-term knowledge networks upon confirmation.
2. **System Self-Test & Calibration**: Periodically activating and testing key innate functional modules (instinctual circuits) to prevent failure from prolonged silence.
3. **Garbage Collection & Resource Reset**: Clearing invalid temporary connections, globally adjusting synaptic excitability, and restoring network dynamic range.

This perspective elevates sleep from a passive physiological process to an **active, purposive information-processing phase**.

Chapter 2: The Bidirectional Adjudication Mechanism for Ontogenetic Memory

2.1 From Microscopic Tagging to Macroscopic Cycle: Extending the STC Theory

At the cellular level, the "**Synaptic Tagging and Capture (STC)**" theory by Frey and Morris demonstrates that early-phase LTP establishes a transient "tag" at the postsynaptic membrane. Only when subsequently synthesized plasticity-related proteins (PRPs) are "captured" by this tag does E-LTP transform into stable late-phase LTP.

ONPH extends this microscopic mechanism to the macroscopic **sleep-wake cycle**: the **sleep phase provides the optimized environment for establishing "tags"** (low interference, specific neuromodulator background), while the **wake phase provides the decisive signal triggering "capture"** (neuromodulator release driven by attention, behavior, or emotion).

2.2 Phase One: Sleep-Phase Synaptic Tagging Based on Emotional Salience

During non-rapid eye movement (NREM) sleep, particularly slow-wave sleep (SWS), hippocampal sharp-wave ripples (SWRs) couple with neocortical slow oscillations and spindles, forming the physiological basis for memory replay.

Crucially, this replay is not an indiscriminate playback of daily experiences but is strictly regulated by the **emotional salience network**:

- The **amygdala** (fear/threat) and **ventral tegmental area/nucleus accumbens** (reward/motivation) act as "**affective gates**," modulating the intensity and priority of hippocampal replay.

- Replay events amplified by this gating system induce specific **molecular tags** (e.g., persistent autophosphorylation of CaMKII, modification of specific cytoskeletal proteins) at involved synapses. These tags are inherently unstable, with a half-life of approximately 24-48 hours.

The tag's significance: It represents the system's preliminary assessment of a memory trace's "potential importance" and sets a temporary "pending" status for its subsequent fate (consolidation or clearance).

2.3 Phase Two: Wake-Phase Behavioral Confirmation-Driven Synaptic Stabilization

This is ONPH's core innovation, distinguishing it from traditional consolidation theories. The sleep-phase tag only grants a "**ticket to compete**" for consolidation, not consolidation itself.

- **Confirmation Signal**: When an individual, after waking, reactivates neural circuits associated with a tagged memory through **behavioral practice, environmental re-exposure, or active recall**, a confirmation signal is generated.
- **Neuromodulators as Capture Messengers**: Confirmation behavior typically accompanies heightened attention and motivation, triggering pulsed release of acetylcholine, norepinephrine, and dopamine in relevant brain regions.
- **Synaptic Capture & Structural Stabilization**: These neuromodulators act as "**capture instructions**," guiding newly synthesized PRPs to be precisely transported to tagged synaptic sites, completing the transformation from E-LTP to L-LTP and achieving lasting synaptic changes (e.g., spine enlargement, AMPA receptor clustering).

Explanatory Power: This mechanism perfectly explains the "**spacing effect**"—why distributed learning is superior to massed learning—as it allows for multiple "sleep-tag-wake-confirm" cycles, reinforcing the same memory trace layer by layer.

2.4 Active Forgetting as Functional Clearance

Within the ONPH framework, forgetting is an **active, functional network optimization process**.

- **Decay of Unconfirmed Tags**: If a synaptic tag receives no confirmation signal within 24-48 hours of formation, it degrades naturally.
- **Clearance Under Homeostatic Pressure**: During subsequent sleep cycles, the global synaptic downscaling process driven by SHY mechanisms preferentially weakens and eliminates these relatively weak connections that have lost tag protection.
- **Functional Significance**: This ensures the brain's memory resources are not occupied by vast amounts of trivial daily minutiae (e.g., a passerby's face, irrelevant conversation fragments), thereby maintaining cognitive system efficiency and focus. **Memory selectivity is, in essence, the selection of behavioral relevance.**

Chapter 3: The Homeostatic Maintenance Mechanism for Phylogenetic Memory

3.1 The Evolutionary Crisis Under the "Use It or Lose It" Rule

The core principle of Hebbian plasticity is "neurons that fire together, wire together." A necessary corollary is: "neurons that **long-term do not fire together will weaken their connection**." This poses a severe evolutionary challenge for genetically encoded **Fixed Action Patterns (FAPs)**—such as complex predation sequences, specific courtship displays, stereotyped escape responses to predators.

In modern or protected environments, many such survival-critical circuits may go untriggered by natural contexts for years. According to plasticity rules, these "**silent hardware**" face the risk of **disuse atrophy**. Should a real survival crisis arise, a degraded predation or escape circuit could be fatal. Therefore, evolution must have developed a mechanism to maintain these circuits' integrity **in the absence of external triggers**.

3.2 REM Sleep as the Endogenous Instinct Generator

The ONPH proposes that **Rapid Eye Movement (REM) sleep** is the evolved solution, with one of its core functions being to act as an **endogenous instinctual circuit activator**.

- **PGO Waves: Endogenous "Test Pulses"**: Ponto-Geniculo-Occipital (PGO) waves, originating in the pons and propagating to the thalamus and cortex, are considered REM-specific endogenous excitatory signals. ONPH reinterprets their function as **system self-test triggers**, used to sequentially "light up" different instinctual circuits.
- **Offline Running of Motor Programs**: Driven by PGO waves and other mechanisms, pattern generators in the brainstem and basal ganglia activate, sending detailed, sequential motor commands (e.g., stalking, pouncing, biting) to the motor cortex.
- **Muscle Atonia: A Safe "Sandbox"**: To prevent these intense virtual actions from causing actual harm, REM sleep induces strong inhibition of spinal motor neurons via brainstem pathways (e.g., from the locus coeruleus), causing near-complete skeletal muscle paralysis. This creates a safe "**neural sandbox**" allowing the brain to run complete motor programs at zero risk.

Key Evidence: Jouvet's classic experiment—lesioning brainstem areas responsible for muscle atonia in cats resulted in animals standing up and performing complete, coordinated hunting or attack behavior sequences during REM sleep. This proves these complex motor programs are **continuously generated** during REM, merely blocked from output under normal conditions.

3.3 Developmental Evidence Chain: From Fetal Movements to Adolescent Sexual Dreams

1. **Fetal and Neonatal Period**: Human fetuses in utero and newborns spend a very high proportion of sleep time (up to 50%+) in REM sleep ("active sleep"). Sensory input is extremely limited at this stage. ONPH posits this is the brain utilizing endogenous activity to "**burn-in**" and "**stress-test**" developing basic motor, sensory integration, and even life-sustaining circuits like breathing and sucking.
2. **Adolescent Sexual Dreams**: With rising gonadal hormone levels, sexual dreams and nocturnal emissions in adolescence often precede actual sexual experience. This is not merely a physiological reflex but represents the brain, during REM sleep, beginning to **integrate and calibrate** the newly matured neuro-endocrine-motivational-motor circuits related to reproduction, preparing for potential reproductive behavior.

3.4 Cross-Species Comparison and Evolutionary Gradient

ONPH predicts that the proportion and complexity of REM sleep should positively correlate with a species' need for plasticity in instinctual behavior.

- **Reptiles**: Instincts largely controlled by rigid brainstem/hypothalamic circuits; low plasticity需求; REM sleep is minimal or absent.
- **Birds & Mammals**: Possess many complex instincts requiring postnatal refinement (e.g., song learning in songbirds, food caching in rodents); REM sleep proportion increases significantly.
- **Higher Primates & Humans**: Survival heavily depends on complex social cognition, emotion regulation, and tool use—skills combining innate templates with acquired learning. Consequently, human REM sleep may not only maintain classical instincts but also involve **social scenario simulation** and **emotional circuit calibration**, leading to unprecedentedly complex dream content.

Chapter 4: The Nature of Dreams: A Cognitive Byproduct of High-Load Plasticity Regulation

4.1 The Spurious Signal Propagation Model

Under the ONPH framework, dreams are not designed to be "experienced"; they are the inevitable "**sidecar effect**" of high-intensity neural regulation during sleep.

- **Signal Sources**:
  - **NREM Phase**: High-intensity memory replay activity in the hippocampus.
  - **REM Phase**: Brainstem-driven high-intensity activation of instinctual circuits.
- **Signal Diffusion**: These intense, localized neural impulses inevitably **diffuse (spill over)** into adjacent associative cortices responsible for sensation and high-level cognition.
- **Narrative Synthesis**: Although the prefrontal cortex's logic and reality-testing functions are suppressed during sleep, the brain's posterior "**interpreter**" system spontaneously

attempts to weave these **random, fragmented internal signals** into a coherent narrative with spatiotemporal and causal relationships. This is the **dream** we perceive.
- **Conclusion**: Dream content **reflects** the neural circuits being maintained (e.g., daytime worries corresponding to emotional memory replay, falling sensations possibly corresponding to vestibular system calibration), but the **specific plot** of a dream may have no deep symbolic meaning; it is a **cognitive hallucination** generated from forced narration of neural noise.

4.2 Mechanism Explaining Rapid Dream Forgetting

Over 95% of dreams are quickly forgotten upon waking. ONPH offers a straightforward explanation:

- **The Core Paradox of Lack of Behavioral Confirmation**: Dreams are purely endogenous experiences. Upon waking, we **almost never act out dream behaviors**, and rarely find scenes perfectly corresponding to bizarre dreams in reality.
- **System Judged as Noise**: According to ONPH's bidirectional adjudication mechanism, neural activity from dreams **completely lacks wake-phase confirmation signals (** $C_{ij} \approx 0$**)**. Therefore, it is judged by the sleep homeostatic maintenance system as invalid "**system noise**."
- **Prioritized Clearance**: During subsequent sleep cycles or upon waking, these unconfirmed, dream-related synaptic changes are **actively and rapidly pruned and cleared** to prevent false memories from contaminating the real-world knowledge base. **Forgetting dreams is an adaptive mechanism to protect the integrity of reality cognition.**

4.3 Lucid Dreaming and the Border with Pathological States

- **Lucid Dreaming**: Occurs during REM sleep when the individual becomes aware they are dreaming and may exert some control. This corresponds to **abnormal partial activation of the prefrontal cortex during REM**, allowing "online" self-monitoring functions to intrude into the "offline" maintenance state—a **brief hybrid of offline and online states**.
- **Hallucinations in Schizophrenia**: ONPH provides a heuristic perspective: positive symptoms of schizophrenia (e.g., auditory/visual hallucinations) may resemble "**dreaming while awake**." Hypotheses suggest patients might have aberrant PGO-wave-like activity or sensory gating deficits, causing endogenously generated signals to be misattributed as external sensory input. This hints that neural mechanisms maintaining the sleep-wake state boundary may be involved in the pathology of certain mental illnesses.

Chapter 5: Computational Modeling and Testable Predictions

5.1 Mathematical Model Based on Plasticity Budget Allocation

We formalize ONPH's core ideas into a mathematical model centered on the concept of **plasticity budget allocation**. Assume the nervous system has limited metabolic and molecular resources for synaptic modification per unit time (e.g., a sleep-wake cycle), denoted as total budget B_{total}.

Synaptic Weight Change Equation:

$$\Delta W_{ij} = \underbrace{\alpha \cdot R_{ij} \cdot S_{ij}}_{\text{Replay Gain}} - \underbrace{\beta \cdot D \cdot (1 - I_{ij})}_{\text{Global Downscaling}} + \underbrace{\gamma \cdot C_{ij} \cdot S_{ij}}_{\text{Confirmation Gain}} + \underbrace{\delta \cdot I_{ij} \cdot A_{rem}}_{\text{Instinct Maintenance}}$$

Constraint (Plasticity Budget):

$$\sum |\Delta W| \leq B_{total}$$

Variable Definitions:

- R_{ij}: Replay strength of the circuit during sleep (normalized 0-1).
- S_{ij}: Synaptic tag state established during sleep (1=tagged, 0=not tagged).
- D: Global downscaling pressure, positively correlated with SWS duration.
- C_{ij}: Confirmation strength during wakefulness (frequency/intensity of behavioral reactivation).
- I_{ij}: Genetically determined instinct weight (0-1, e.g., respiratory center ≈1, complex predation sequence ≈0.8, a specific skill ≈0.1).
- A_{rem}: Endogenous activation strength of the circuit during REM sleep.
- $\alpha, \beta, \gamma, \delta$: Gain coefficients modulated by neural state.

5.2 Key Inferences and Simulation Scenarios

1. **Optimal Memory Consolidation Strategy**: The model predicts maximizing ΔW requires combining **"high-intensity learning (high R_{ij}) + sleep (establishing S_{ij}) + timely wake-phase review (high C_{ij})"**.
2. **Active Forgetting Scenario**: When $S_{ij} = 1$ but $C_{ij} \to 0$ and $I_{ij} \approx 0$, ΔW is negative, achieving active clearance of that memory.
3. **Instinct Maintenance Scenario**: Even with $C_{ij} = 0$ (no real-world experience), if I_{ij} and A_{rem} are sufficiently high, ΔW can remain zero or positive, preventing weight decay.
4. **Budget Competition Effect**: If daytime learning leads to many tagged memories (high $\sum S_{ij}$), it can crowd out the budget for instinct maintenance ($\sum I_{ij} \cdot A_{rem}$), potentially reducing instinctual/emotional dreams or causing short-term fluctuations in emotion regulation.

5.3 List of Falsifiable Experimental Predictions

ONPH's value lies in its testability. Key predictions include:

1. **Prediction I (Bidirectional Adjudication)**: After a learning task, if **artificially preventing** any task-related behavioral or cognitive rehearsal during wakefulness (depriving C_{ij}), the memory consolidation gain from subsequent sleep will be **significantly diminished or absent** (achievable via behavioral restriction or specific brain region inhibition).
2. **Prediction II (Instinct Anti-Degradation)**: During a critical developmental period in animals, **selectively depriving REM sleep** (e.g., using the small platform over water method while preserving SWS as much as possible) will, in adulthood, lead to **significant deficits** in performing complex species-typical instinctive behaviors (e.g., nest building, courtship sequences), while simple learned skills may be less affected.
3. **Prediction III (Dream Forgetting)**: Using lucid dream marking techniques, awaken subjects during REM and record the dream. Then, **force one group to vividly "re-enact" or describe the dream in writing upon waking (providing C_{ij})**, while the other group does nothing. Predict the former group's memory retention for that dream will be **significantly longer** than the latter's.
4. **Prediction IV (Plasticity Competition)**: Following days of intensive skill training, monitoring subjects' dream reports and REM sleep physiological metrics will predict a **reduction in instinctual/emotional themed dreams** and possible changes in REM microstructure (e.g., PGO wave density).

Chapter 6: Clinical Pathological Reconstruction and Mechanistic Intervention

6.1 Insomnia: Safety Protocol Lockdown and Environmental Mismatch

- **ONPH Pathological Model**: Entering deep sleep (especially the offline maintenance state) minimizes the ability to perceive external threats. Thus, the brain evolved a strict "**safety check protocol**" continuously assessing environmental safety. Chronic modern stress (work, social) causes sustained low-grade activation of the amygdala-hypothalamic-pituitary-adrenal axis, sending false "**environment unsafe**" alarms to wake-maintaining systems (e.g., brainstem reticular formation, hypothalamic orexin neurons). The system thus judges "conditions unsuitable for offline maintenance," refusing to initiate or maintain sleep. **Insomnia is not a "malfunction" but an adaptive "vigilance" state in a misjudged dangerous environment.**
- **Intervention New Paradigm — Safety Signal Masquerading**:
  - **Principle**: Bypass high-level cognition to send "safe" somatic sensory signals directly to primitive brain safety monitors.
  - **Methods**: Use weighted blankets (deep pressure touch), rhythmic white noise mimicking heartbeat or maternal blood flow, specific temperature regulation, etc., to simulate sensory input in a safe nest.
  - **Cognitive Offloading Technique**: Perform a "brain dump" before bed, externalizing worries and plans onto paper or digital devices, aiming to reduce

cognitive load entering the sleep processing queue and lower the system's alert level for "unresolved problems."

6.2 PTSD: The Non-Convergent Threat Simulation Loop

- **ONPH Pathological Model**: Traumatic events impart extremely high emotional weight (very high S_{ij}) to related memory networks. Sleep (especially REM) attempts to integrate and digest this high-load information through **repetitive simulation (nightmares)**, ideally leading to "habituation" or "cognitive restructuring." However, PTSD nightmares are often **extremely vivid and end in awakening**, meaning the simulation process is **forcibly interrupted** before reaching a "safe resolution" convergence point. The brain's maintenance system thus judges the "threat simulation task as unfinished," causing the program to be **repeatedly and preferentially restarted** in subsequent sleep cycles, forming a vicious loop of frequent nightmares.
- **Intervention New Strategy — Wake Closure Therapy**:
  - **Principle**: Leverage ONPH's "wake confirmation" mechanism to provide trauma memory with a **new, benign outcome** in an absolutely safe waking state and "write" it into the system through repeated confirmation.
  - **Method**: An enhanced version of **Imagery Rehearsal Therapy (IRT)**. Under therapist guidance, patients not only rewrite nightmare endings but also, while awake, repeatedly "experience" this new ending through **role-playing, physical action, or intense positive imagination**, sending strong C_{ij} confirmation signals to the brain.
  - **Goal**: Overwrite the old, unfinished threat simulation with this new, safe "program closure," thereby terminating the automatic replay of nightmares at night.

6.3 The Bidirectional Link Between Sleep Disorders and Neurodegenerative Diseases

- **ONPH Perspective**: Sleep's synaptic downscaling (SHY mechanism) is tightly coupled with the waste clearance function of the glymphatic system. ONPH predicts that chronic sleep disorders (especially reduced SWS) may lead to:
  1. **Synaptic Homeostasis Dysregulation**: Redundant weak connections cannot be effectively cleared, reducing neural network efficiency and increasing energy consumption.
  2. **Metabolite Accumulation**: Sleep-dependent glymphatic flushing weakens, lowering clearance rates of neurotoxic substances like β-amyloid and tau proteins.
  3. **Increased Oxidative Stress**: Persistent synaptic activity in inefficient networks generates more reactive oxygen species.
- **Significance**: This provides a **mechanistic explanation** for "sleep disorders being a significant risk factor and prodromal marker for neurodegenerative diseases like Alzheimer's." Improving sleep, especially deep sleep, may not only alleviate symptoms but act on the core pathophysiology of the disease.

Chapter 7: Conclusion and Future Roadmap

7.1 ONPH's Theoretical Status and Integrative Value

The Offline Neural Plasticity Homeostasis Hypothesis attempts to provide a **unifying framework** addressing several core splits in sleep research:

- **It reconciles SHY and Active Consolidation Theories**: Treating global downscaling as a background "cleanup" process, while bidirectional adjudication based on tagging-capture serves as the foreground mechanism for "selective consolidation."
- **It fills the theoretical vacuum in instinct research**: Providing a clear, testable functional explanation for REM sleep and instinctual dreaming.
- **It connects normal function with clinical pathology**: Reinterpreting disorders like insomnia and PTSD from a mechanistic level and deriving novel intervention ideas.

ONPH does not claim to be the sole explanation for sleep function but proposes that sleep is a **multi-objective optimized offline maintenance system**, whose algorithm simultaneously serves the integration of individual experience and the preservation of species' genetic heritage.

7.2 Experimental Verification Roadmap

Verifying ONPH requires multi-level research:

1. **Molecular & Cellular Level**: Identify sleep-phase-specific "synaptic tag" molecules and prove their causal relationship with PRP capture triggered by subsequent wake-phase behavioral confirmation.
2. **Circuit & Systems Level**:
   - Use optogenetics/chemogenetics to selectively inhibit reactivation of specific memory engram cells during wakefulness, verifying the blocking effect on sleep-dependent consolidation (Prediction I).
   - Precisely deprive REM (not NREM) in developing animals, tracking long-term deficits in instinctive behavior and corresponding anatomical/functional changes in neural circuits (Prediction II).
3. **Behavioral & Clinical Level**:
   - Conduct prospective studies testing the efficacy of interventions based on "safety signal masquerading" and "wake closure" for chronic insomnia and PTSD compared to traditional methods.
   - In large cohorts, longitudinally study the relationship between mid-life SWS quality and late-life cognitive decline/neuropathological burden.

7.3 Clinical Translation and Artificial Intelligence Inspiration

- **Clinical Translation**: ONPH pushes sleep medicine from "sedative-hypnotic" treatment towards "**plasticity environmental regulation**." Future therapies may involve "creating"

a safe offline maintenance environment for the brain or helping it complete non-convergent information processing loops.

- **AI Inspiration**: Artificial neural networks have long struggled with **catastrophic forgetting** and **continual learning**. ONPH suggests a viable direction: introducing an "**offline phase**" where the network pauses new input and, based on internally generated data or preset "instinct" priorities, performs **selective replay, consolidation, and pruning of weights**. This may be a bio-inspired path towards more robust and efficient AI learning algorithms.

Concluding Remarks

Sleep is far from a blank in life. ONPH depicts a vision: beneath the nightly quiet, our brains engage in a grand, dual-track project—meticulously sorting the individual's yesterday while faithfully guarding the species' yesteryear. The spark of dreams is the glow from this precise machine operating at full capacity. Understanding this offline survival algorithm may not only revolutionize how we treat diseases but perhaps also illuminate our path to building truly intelligent machines.

(Detailed mathematical formulas and parameter calibration procedures are provided in the supplementary material: Computer Simulation Model Description.)

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We particularly thank the **Zenodo** research data sharing platform. Operated by CERN, Zenodo practices the spirit of open science, providing an equal, permanent, and citable dissemination channel for researchers worldwide—especially independent scholars and small teams—truly lowering the threshold for academic communication and serving as key infrastructure for the open science movement.

Accordingly, we declare: **The final version of this article will have its global first release on Zenodo and will be fully published exclusively through this platform and related open-access channels**, ensuring its immediate and permanent openness to the entire human scientific community.

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References (Selected Key Literature)

1. Tononi, G., & Cirelli, C. (2014). Sleep and the price of plasticity. *Neuron*.
2. Diekelmann, S., & Born, J. (2010). The memory function of sleep. *Nature Reviews Neuroscience*.
3. Frey, U., & Morris, R. G. (1997). Synaptic tagging and capture. *Nature*.
4. Jouvet, M. (1979). What does a cat dream about? *Trends in Neurosciences*.
5. Stickgold, R., & Walker, M. P. (2013). Sleep-dependent memory triage. *Nature Neuroscience*.
6. Liu, Y., et al. (2023). Targeted memory reactivation during sleep improves consolidation. *Science*.
7. Rasch, B., & Born, J. (2013). About sleep’s role in memory. *Physiological Reviews*.
8. Hobson, J. A., & Friston, K. J. (2012). Waking and dreaming consciousness. *Progress in Neurobiology*.

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