Sleep as Attractor Maintenance: Glymphatic Clearance, Synaptic Rescaling, and Dynamical Resilience

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Abstract

Sleep is often called "hardware maintenance" (deep sleep) and "software maintenance" (REM sleep).
This paper re-interprets sleep using the **attractor framework**, where your mind is a dissipative attractor of your whole body.

We propose that different sleep stages are different **attractor regimes**:

- **Deep (NREM) sleep** – a slow, relaxing state that clears waste and dials down brain connections.
- **REM sleep** – a fast, high-dimensional attractor that updates your brain's internal model.

We review evidence for:

- Glymphatic clearance (waste removal)
- Synaptic homeostasis (downscaling of connections)
- Slow-oscillation/spindle coupling
- Sleep–immune interactions

We also show how sleep fragmentation, ageing, chronotypes, and sleep disorders can be understood as changes in **attractor depth**, **stability**, and **corrective permeability**.

The framework introduces a **persistence functional** P(x)$P(x)$ — a single number that measures basin depth — which could be estimated from EEG or wearables to predict resilience to sleep loss and guide closed-loop interventions.

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1. Introduction

In the attractor framework, your mind is a **dissipative attractor of your whole body** — a pattern that needs constant energy, can be disturbed, and can adapt.
Sleep is a natural, periodic disturbance that lets the system reset, repair, and reorganise. It is **not** passive; it is an **active attractor maintenance process**.

We focus on two major sleep stages:

- **NREM sleep**, especially deep slow-wave sleep (NREM 3) — a **slow constraint relaxation** that brings the brain and body back to a low-energy baseline.
- **REM sleep** — a **fast, high-dimensional attractor** for active reorganisation, memory consolidation, and predictive coding updates.

This paper bridges sleep neuroscience with the attractor framework.

What does the framework add?

- **Integration** — a common language across scales.
- A **unified quantitative biomarker** P(x)$P(x)$ from EEG or wearables.
- **Novel predictions** (e.g., wearable early-warning signals, REM-emotional rebound) that are not obvious from the individual component theories.
  This is **generative integration** — a scientific contribution even without claiming new mechanisms.

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2. The Attractor Framework Primer

- **Conservative attractors** (the “six metronomes”) — eternal, time-symmetric, provide steady rhythms. They are the floor, not part of maintenance.
- **Dissipative attractors** (life, mind, society) — need energy flow, have finite lifetimes, can evolve. The brain is a nested stack of dissipative attractors.
- **Persistence under perturbation** — a resilient system returns quickly to its attractor after a disturbance.
- **Self-engineering** — using small, repeated disturbances to reshape your own attractor. Sleep is a natural self-engineering cycle.

Sleep moves you through: **wake → NREM → REM → wake**.

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3. NREM Deep Sleep — Slow

Constraint Relaxation

3.1 Glymphatic clearance – flushing out waste

Deep slow-wave sleep (NREM 3) is essential for clearing brain waste.
Studies show that the glymphatic system (which removes waste) works best during deep NREM (Iliff et al., 2012). Norepinephrine drops during sleep, expanding the space around cells and improving fluid flow (Balkrishnan et al., 2023, conference abstract).

In attractor terms: The deep-sleep attractor (high delta power) relaxes the metabolic constraints that build up during the day. Waste clearance rate scales with **attractor depth** (measured by slow-wave activity, SWA). Shallow or broken sleep leads to waste buildup.

3.2 Synaptic homeostasis – resetting brain connections

The synaptic homeostasis hypothesis (SHY) says:

- Wakefulness strengthens synapses (deepens attractor basins).
- NREM sleep downscales synapses (shallows basins) (Tononi & Cirelli, 2006).

SWA reflects this – it is high after waking and declines across the night.

In attractor terms: The persistence functional P(x)$P(x)$ would be high after waking, then drop during NREM as synapses downscale. The rate is steep early and plateaus later – compatible with critical slowing down near awakening (though direct evidence is mixed).

3.3 Slow-oscillation–spindle coupling – nested rhythms

Memory consolidation during sleep depends on the tight coordination of:

- Cortical slow oscillations (<1 Hz)
- Thalamocortical spindles (12–15 Hz)

This is best described as **nested oscillatory coupling** (Ngo et al., 2013) – the slow oscillation modulates excitability, creating windows for spindles.
We interpret this as **different timescales within a single attractor manifold** (parsimonious). (Two coupled attractors could also produce phase locking; the question is subtle, but we take the simpler view.)

Stronger phase-locking between spindles and slow oscillations predicts better memory. Closed-loop stimulation (auditory or electrical) timed to the up-phase enhances both slow waves and spindles – showing that the attractor can be externally reinforced.

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4. REM Sleep – Fast, High-Dimensional Attractor

REM sleep has activated EEG (low voltage, fast rhythms) and vivid dreaming.
From a **predictive coding** view (Friston, 2010), REM updates the brain’s generative model by resolving prediction errors.

Dynamically, the NREM → REM transition is a **phase bifurcation**:

  - NREM is a low-dimensional attractor (regular slow oscillations).
  - REM is higher-dimensional (complex, desynchronised EEG).

Indeed, EEG complexity (e.g., Lempel-Ziv complexity) is higher in REM and wake than in NREM.

If REM dreaming implements predictive coding, then nights with stronger REM (longer, more intense periods) should show greater emotional memory consolidation. (The idea of lucid dreaming as a “meta-attractor” is not pursued here.)

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5. Sleep Fragmentation and Attractor Instability

Frequent awakenings (fragmentation) repeatedly disturb the sleep attractor.
Each arousal is a temporary escape from the NREM or REM basin, reducing effective depth and slowing re-entry. This is a state of **reduced attractor stability** with **critical slowing down** (Scheffer et al., 2009): recovery takes longer.

Recent work (de Mooij et al., 2020) found that EEG change-points – transitions between stages – are often preceded by early-warning signals (rising variance and autocorrelation).
Grossman et al. (2025) showed that the wake-to-sleep transition follows a bifurcation dynamic, detectable minutes before sleep onset.

Wearables (HRV, actigraphy) could detect similar signs – rising movement variance, increasing HRV autocorrelation – before a failed sleep transition. Closed-loop auditory tones could then reinforce the desired attractor.

6. Inter-individual Differences, Aging, Chronotypes, and Immune Coupling

Resilience to sleep loss

People vary widely. The **PER3 clock gene polymorphism** is a paradox:

- PER3^5/^{5} individuals have more slow-wave sleep and higher delta power, yet they suffer **greater** performance declines under sleep loss (Viola et al., 2011).

This shows that a deeper baseline attractor does **not** guarantee resilience. The framework says resilience requires not only depth but also **corrective permeability** — the ability to re-enter deep sleep after an awakening and to update the attractor under stress (see Section 7).

Aging

Slow-wave sleep drops dramatically with age. In a community study, each 1% annual reduction in SWS was linked to a 27% higher risk of dementia (Himali et al., 2023).
In attractor terms: the deep-sleep basin **erodes** with age, and corrective permeability weakens. Exercise, light therapy, and melatonin may help a little, but only modestly.

Chronotypes

Morning larks and night owls differ mainly in the **phase** of the sleep–wake attractor relative to the light–dark cycle. Both can have similar basin depths, but misalignment may weaken the attractor.

Sleep–immune coupling

Sleep deprivation increases pro-inflammatory cytokines (IL-6, TNF-α) and reduces T-cell activity (Irwin et al., 2016; Besedovsky et al., 2012).
A shallow or fragmented sleep basin destabilises the immune attractor, leading to slower recovery from infection (Cohen et al., 2009) and blunted vaccine responses (Spiegel et al., 2002).

Immune challenge (e.g., infection) also disrupts sleep, increasing SWS — a “sickness behaviour” attractor shift (Krueger et al., 2013). This is **bidirectional coupling** between two attractor landscapes.

Framework-specific prediction: Corrective permeability κ should be **lower** on nights following an immune challenge, independently of changes in delta power.
(Statistical test: partial correlation or regression of κ on immune challenge, controlling for P_{EEG}.) This prediction is not deducible from the cytokine model alone.

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7. Sleep Disorders as Maladaptive Attractors and Corrective Permeability

7.1 Defining corrective permeability κ

κ measures how quickly a system returns to its primary attractor after a disturbance and how easily it updates under chronic stress.$\kappa = \frac{1}{\tau_{recovery}}$

where $\tau_{recovery}$ (minutes) is the time from an awakening back to stable deep NREM (stage 3).

- High $\kappa > 0.2$ min^{-1} → fast recovery (<5 min).
- Low $\kappa < 0.05$ min^{-1} → poor recovery (>20 min).

These thresholds are provisional – for empirical calibration.

Heart-rate recovery slope after awakenings is a candidate wearable proxy (hypothesis, not yet validated).

7.2 Disorder taxonomy

- **Insomnia** – abnormally shallow sleep attractor (low depth) **and/or** low κ. Hyperarousal prevents settling into deep sleep.
- **Narcolepsy** – blurred boundary between wake and REM attractors (orexin loss).
- **REM behaviour disorder** – failure of REM attractor to suppress muscle activity; dream movements “leak out”.

7.3 Falsification conditions

Falsification of the “shallow basin” explanation
If an insomnia patient shows normal delta power ($P_{EEG} > 0.7$) **and** normal corrective permeability ($\kappa > 0.1$) but still has non-restorative sleep, the “shallow basin” model is falsified for that patient.
The framework would be incomplete, not wrong. **But** to prevent this clause from making the theory unfalsifiable, we add a provisional bound:

> *If more than 30% of diagnosed insomnia cases need such additional mechanisms, the framework’s descriptive utility for insomnia would be in question, and the core hypothesis would be falsified.*

Falsification of the attractor framework itself
If sleep stage transitions show **no** evidence of basin-crossing dynamics (no rise in variance/autocorrelation, no attractor dimensionality difference between NREM and REM, no critical slowing down before awakening), then the attractor framework should be abandoned in favour of a purely stochastic or oscillator-based model.
Specifically, a well-powered study using the methods of de Mooij et al. (2020) that finds null results would constitute strong falsification. (We require convergent null evidence across multiple measures.)

8. The Persistence Functional $P(x)$

$P(x)$ measures attractor depth — the ability to resist disturbance and return to stable state.
We base it on the **dominant Lyapunov exponent** λ_1.

Primary definition (fixed $\tau = 1$ s):$$P_{\text{raw}} = e^{-\lambda_1 \cdot \tau}$$

For a stable attractor, $\lambda_1 < 0$, so $P_{\text{raw}} > 1$. Deeper attractors (more negative λ_1) give larger P_{raw}.

To get a bounded [0,1] measure:$$P_{\text{norm}} = \frac{1}{1 + e^{\lambda_1 \tau}}$$

- Values near 1 → deep basin.
- 0.5 → neutral.
- Near 0 → unstable/chaotic.

EEG-practical approximations:

- **Correlation dimension D_2** — in sleep EEG, deeper stages have lower D_2. This is a **sleep-specific** approximation. Then $P \propto 1/(1+D_2)$.

- **Delta power ratio** (simplest):

$$P_{EEG} = \frac{\langle \delta(t) \rangle}{\delta_{wake} + \langle \delta(t) \rangle}$$

where $\langle \delta(t) \rangle$ is mean delta power (0.5–4 Hz) in the epoch, and δ_{wake} is the same during relaxed wakefulness. Deep sleep → value close to 1; shallow sleep → near 0.

We recommend P_{EEG} for practical sleep research. All three definitions should correlate under the framework’s assumptions, but empirical validation is needed.

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9. Testable Predictions

Prediction	Type	Proposed Test Protocol	Source / Support
Glymphatic clearance correlates with SWA	Retrodiction	—	Iliff et al., 2012
EEG complexity decreases across NREM	Retrodiction	—	Tononi & Cirelli, 2006
SO–spindle coupling predicts memory	Retrodiction	—	Ngo et al., 2013
Sleep fragmentation preceded by rising variance/autocorrelation	Novel	Re-analyse existing sleep EEG datasets	de Mooij et al., 2020; Grossman et al., 2025

Prediction	Type	Proposed Test Protocol	Source / Support
Wearable early-warning signals (HRV lag-1 autocorrelation) predict night-to-night sleep quality	**Novel**	Pilot N=1 wearable study (30+ nights); confirm with larger cohort	Proposed here
REM rebound scales with emotional load during wake	Plausible	Daily stress diary (1–10) + actigraphy/PSG for REM%	Proposed here
Immune challenge reduces next-night κ*κ* independently of delta power	**Novel (framework-specific)**	Controlled immune challenge (e.g., vaccine) with wearable/PSG κ*κ*; partial correlation controlling for PEEG*P*EEG	Proposed here

Falsification of core framework: If no evidence of basin-crossing dynamics (rising variance/autocorrelation, difference in attractor dimensionality) is found in a well-powered EEG study using de Mooij et al.’s methods, the attractor framework for sleep should be abandoned.

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10. Conclusion

Sleep is not passive — it is a dynamic, bifurcated process of **attractor maintenance**.

- **Deep NREM sleep** — slow constraint relaxation, clearing waste and downscaling synapses.
- **REM sleep** — fast, high-dimensional attractor, updating the brain’s generative model.

Fragmentation, aging, and sleep disorders can be understood as changes in attractor depth, stability, and corrective permeability.

The persistence functional P(x)$P(x)$ gives a quantitative language for sleep engineering.

The dance of sleep is the dance of maintenance — and we can learn to engineer it.

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