

## The dynamical models of sleep: A brief review

Adriano Scibilia<sup>1\*</sup>  and Luigi Fortuna<sup>2</sup> 

<sup>1</sup> *Institute of Intelligent Industrial Systems and Technologies for Advanced Manufacturing, National Research Council, Milan, Lombardy, Italy*

<sup>2</sup> *Department of Engineering, University of Palermo, Palermo, Palermo, Italy*  
[adriano.scibilia@cnr.it](mailto:adriano.scibilia@cnr.it)

### History:

Received: December 23, 2025

Revised: March 2, 2026

Accepted: March 10, 2026

Published online: April 24, 2026

### ABSTRACT

The field of sleep dynamics is a fascinating and complex area of research. The topic of sleep has attracted widespread interest over the last century. The aim of this concise review is to provide some essential items for researchers interested in both nonlinear science and sleep engineering. In particular, the paper focuses on the dynamical modeling of sleep, highlighting the fundamental mechanisms underlying sleep–wake regulation. A brief literature review is presented, and some fundamental mathematical models of sleep are reported with numerical simulations. This review introduces the reader to the fascinating topic of sleep dynamics, underlining its universality and complexity, and stimulates further research in the field.

**Keywords:** Sleep dynamics; Nonlinear systems; Circadian rhythms; Homeostatic regulation; Chaos theory; Complex systems



## 1. Introduction

Sleep is one of the most fundamental and significant biological phenomena in the world.<sup>1</sup> Here, we briefly outline why this topic is so compelling and what the scientific community has learned about the incredible diversity and universality of sleep.

### 1.1. The diversity of sleep across the animal kingdom

Sleep is a behavior observed in nearly every animal group studied, from mammals and birds to insects, fish, and even jellyfish.<sup>2</sup> However, the nature of the sleep of a hummingbird is wildly different from that of a human, yet it serves a similar biological purpose.

Dolphins and whales practice unihemispheric sleep, in which one hemisphere of the brain rests while the other remains awake and alert, allowing them to surface for air while remaining vigilant for predators. Birds also exhibit unihemispheric sleep, enabling flocks to maintain vigilance while resting. Interestingly, they can alternate between non-rapid eye movement (NREM) and REM sleep, but their REM periods are incredibly short, lasting only a few seconds.<sup>3</sup>

Insects such as fruit flies and honeybees enter a state of metabolic rest and reduced responsiveness that meets the criteria for sleep. During this time, they become immobile and require stronger stimuli to awaken them. Sleep-deprived bees are less effective at foraging and communicating with their hive, highlighting sleep's crucial role even in simpler nervous systems.<sup>4</sup> Among large prey

\*Corresponding author:

Adriano Scibilia ([adriano.scibilia@cnr.it](mailto:adriano.scibilia@cnr.it)).

### Citation:

Adriano Scibilia and Luigi Fortuna. The dynamical models of sleep: A brief review. *Nonlinear Sci Cont Eng.* 2026;2(1):025520019. doi: 10.36922/NSCE025520019

**Copyright:** © 2026 The Author(s). This is an Open Access article distributed under the terms of the Creative Commons Attribution License, permitting distribution, and reproduction in any medium, provided the original work is properly cited.

animals, giraffes and elephants sleep for incredibly short periods, often just 2-4 hours a day, and may not lie down to achieve deep sleep. This limited sleep is an evolutionary trade-off to minimize vulnerability to predators.<sup>5</sup> Bats and opossums, in stark contrast with animals such as the little brown bat, can sleep for nearly 20 hours a day, taking advantage of their protected roosting sites to rest extensively.

## 1.2. The phenomenon of sleep is a universal biological imperative

Despite these vast differences in how sleep is manifested across species, several core, universal properties define it as a fundamental biological process:

- Behavioral quiescence and posture: An animal in a sleep-like state is immobile and often adopts a specific resting posture.
- Increased arousal threshold: A sleeping organism requires a stronger stimulus to be awakened than when it is merely resting quietly.
- Homeostatic regulation<sup>6</sup>: Sleep deprivation leads to a compensatory increase in sleep duration and/or depth later, demonstrating a biological need that builds up over time and must be “repaid.”
- Reversibility: Sleep is easily reversible; the animal can quickly return to full alertness and function, unlike hibernation or torpor.

The fact that sleep evolved independently in many different animal lineages, from invertebrates with simple neural networks to highly complex mammals, suggests that it serves a critically important function. Leading theories for its universality include:

- Brain rest and restoration: Sleep allows the brain to consolidate memories, repair cellular damage, and remove waste products and neurotoxins that accumulate during wakefulness.
- Synaptic homeostasis: During wakefulness, synapses are constantly strengthened. Sleep acts as a “reset” mechanism, downscaling and pruning connections to maintain a stable, efficient neural network for future learning.<sup>7</sup>
- Energy conservation: By reducing activity, sleep lowers metabolic rate and conserves energy, especially for animals that forage during limited hours.

The complexity of sleep provides a starting point for investigating its evolutionary<sup>8</sup> and functional significance. The diversity of sleep across organisms does not undermine its importance; rather, it highlights sleep’s flexibility and adaptability as a core biological process that all life has had to optimize in its own unique way. This emphasizes that sleep is far more than a human experience—it is a cornerstone of the biological world.<sup>9</sup>

Dynamical models are the capstone of understanding complex phenomena like sleep, and a substantial body of literature has developed on this topic.<sup>10</sup> The aim of this review is to provide a concise overview of the state of the art in sleep dynamical models. The paper is primarily intended for people working in nonlinear science and complex engineering, introducing them to the problem and encouraging new contributions in this fascinating

field. **Section 2** presents a brief review of the literature, **Section 3** discusses selected mathematical models of sleep dynamics, and **Section 4** provides numerical results. The conclusions emphasize the universality of sleep and explore the potential for linking it to chaos dynamics, inspiring ideas in the emerging field of nonlinear sleep technology.

## 2. A concise state of the art on sleep dynamics

The field of sleep dynamics is a fascinating and complex area of research that studies the temporal patterns and transitions of sleep, moving beyond simple metrics such as total sleep duration. It views sleep not as a static state, but as a dynamic system with various interacting components. Research in this area often uses mathematical models, network analysis, and advanced signal processing techniques to understand the mechanisms underlying sleep and elucidate its relationship with health and disease. Here is a list of influential papers and key research areas regarding sleep dynamics, categorized for clarity.

The following three contributions address foundational and theoretical problems and have a key role in shaping the field. These works laid the groundwork for conceptualizing sleep as a dynamic system, often through the application of physical and mathematical models.

The work by Borbély<sup>11</sup> is a landmark contribution that, while not explicitly about sleep dynamics as a complex system, remains the single most important conceptual framework for the field. It proposed that sleep is regulated by two primary processes: Process S (sleep homeostasis, which increases during wakefulness and decreases during sleep) and Process C (the circadian process, which governs the 24-hour cycle of wakefulness and sleep). This model establishes the fundamental drives that underpin sleep dynamics. In this contribution, an oscillatory trend in sleep characteristics is noted and linked to a single oscillatory device that drives the interaction between these processes.

The work by Phillips and Robinson<sup>12</sup> presents a physiologically based model of the sleep–wake cycle using a simplified “flip-flop” switch mechanism. It models the mutual inhibition between wake-promoting and sleep-promoting brain regions and demonstrates how the circadian and homeostatic drives (derived from the Borbély model) interact to produce realistic sleep–wake dynamics. This work is a key example of how a dynamical systems approach can be used to understand the brain’s sleep–wake regulation.

The work by Comte et al.<sup>13</sup> proposes that sleep dynamics can be understood as a self-organized critical system, akin to a sandpile. The authors argued that sleep is a complex system with alternating slow-wave sleep and paradoxical sleep (REM) episodes, and that the transitions between these states exhibit properties of criticality. This perspective offers a new theoretical lens for analyzing the variability and structure of sleep patterns.

The following three papers contribute to the understanding of sleep architecture and stage transitions. They focus on the micro- and macro-structure of sleep, analyzing the patterns of transitions between sleep stages through patient-based and clinical studies. This allows us to focus on sleep dynamics in real cases.

The study by Sorensen et al.<sup>14</sup> investigated how sleep stage transitions differ in patients with narcolepsy. It found that hypocretin-1 deficiency is associated with increased transitions between REM and NREM sleep, while cataplexy

is linked to more frequent transitions between sleep and wakefulness. This work highlights how analyzing sleep dynamics can provide insights into the mechanisms of specific sleep disorders.

An empirical model of behavioral and physiological dynamics was reported by Prerau et al.<sup>15</sup> This study moved beyond traditional, discrete sleep-stage scoring by proposing a continuous, dynamic model of the transition from wakefulness to sleep. It used a combination of physiological and behavioral data to show that the sleep-onset process is a continuum rather than a single, instantaneous event, a finding particularly relevant to understanding sleep disorders like insomnia.

The study by Yetton et al.<sup>16</sup> examined sleep architecture dynamics and individual differences using a large-scale dataset and a Bayesian network approach. It highlights that traditional measures of sleep architecture (e.g., stage proportions) fail to capture the full picture of sleep dynamics. The dynamic, network-based approach reveals individual differences and patterns related to health and behavioral variables.

In sleep studies, it is also important to examine the link between sleep dynamics and cognition/health. The following contributions explore how the dynamic properties of sleep affect cognitive function and overall health.

The review article by Basner et al.<sup>17</sup> discussed the dynamic neurobehavioral changes induced by both acute and chronic sleep restriction. It argued that a dynamic perspective is necessary to understand how the brain's response to sleep loss changes over time and highlights the long-term neuromodulatory changes that can occur.

The study by Chen et al.<sup>18</sup> investigated the competitive dynamics underlying cognitive improvements during sleep, providing evidence that human sleep functions as a competitive arena for cognitive resources. It used pharmacological methods and connectivity analysis to show that different offline neural mechanisms for long-term and working memories are mutually antagonistic during sleep, and that a "sleep switch" mechanism may toggle between them. This work exemplifies a functional, dynamic perspective on sleep.

A scientific statement from the American Heart Association<sup>19</sup> addressed the impact of sleep disorders and disturbed sleep on brain health. It emphasized how conditions such as obstructive sleep apnea, insomnia, and sleep fragmentation can disrupt the dynamic regulation of synaptic homeostasis, inflammation, and autonomic nervous system function, ultimately contributing to cognitive decline and other health issues.

The following studies focused on developing mathematical models of sleep and outlining future directions for advanced theoretical research. This field is continuously evolving, with new papers leveraging big data, machine learning, and computational modeling.

Rempe et al.<sup>20</sup> reported the basic concepts to build wake, REM, and NREM models. This contribution is highly relevant because it provides a framework for constructing models of varying complexity, offering readers a clear view of the sleep model paradigm. Various sleep modeling concepts were also discussed by Kharchenko and Zhdanova.<sup>21</sup> They outlined robust paradigms for quantitative modeling of sleep dynamics. The study by Viswanath et al.<sup>22</sup> exemplifies modern research on sleep dynamics. By analyzing an extensive dataset from wearable devices, the authors identified distinct sleep phenotypes

and showed that these phenotypes are dynamic rather than static. The findings highlight that temporal sleep dynamics contain significant, nonrandom information about a range of health conditions. Robinson et al.<sup>23</sup> proposed a single-wave model to explain the complex structure of sleep, including the distinct dynamics of NREM and REM episodes. Their work provides a highly quantitative and parsimonious explanation of sleep dynamics.

Next, we highlight recent and relevant contributions that explore sleep as a dynamical system, with particular focus on chaos, complexity, and mathematical modeling. Regarding chaos in electroencephalography (EEG) and sleep stages, the following five studies were considered.

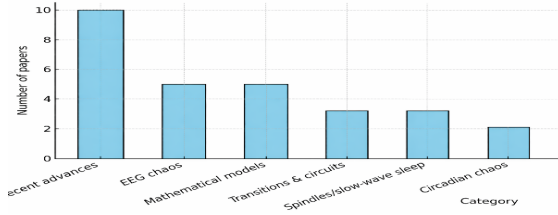
One of the first studies to apply nonlinear dynamics to EEG signals, Babloyantz et al.<sup>24</sup> reported evidence of low-dimensional chaotic attractors across sleep stages. Rösche and Başar<sup>25</sup> demonstrated that EEG signals display deterministic rather than purely stochastic structure, supporting the idea of chaos in brain activity. Fell et al.<sup>26</sup> identified positive Lyapunov exponents in human sleep EEG, further confirming the presence of chaotic signatures. Additionally, Fell et al.<sup>27</sup> compared spectral versus nonlinear measures for sleep stage classification, revealing that nonlinear metrics better capture transitions and subtle dynamics. Kruglikov and Schiff<sup>29</sup> detected unstable periodic orbits in the EEG during sleep, strengthening the view that chaotic dynamics underlie sleep microstructure.

Mathematical models of sleep with emerging complex dynamics have expanded our theoretical understanding. Behn and Booth<sup>7</sup> applied fast-slow analysis to REM sleep dynamics, revealing complex oscillatory structures in NREM-REM cycles. Booth et al.<sup>30</sup> presented a reduced sleep-wake regulatory model as a one-dimensional map, enabling bifurcation analysis of circadian modulation and explaining irregular sleep patterns under circadian/homeostatic shifts. The review by Postnova et al.<sup>31</sup> highlighted nonlinear sleep-wake models, spanning circadian oscillators to chaos, and emphasized the connections between physiological processes and mathematical frameworks.

The topic of chaos at sleep-wake transitions and neural circuits has also received significant attention. Hutt and Longtin<sup>32</sup> used neural field modeling to show how anesthesia and neural inhibition can generate oscillations and chaotic transitions. Paul et al.<sup>33</sup> showed that a thalamocortical circuit model exhibits chaos near sleep-wake transitions, suggesting that chaos can mediate instability. Rasmussen et al.<sup>34</sup> highlighted that chaotic dynamics facilitate flexible transitions between sleep and wake states.

Similarly, studies of spindles and slow-wave sleep have revealed the importance of chaos. Achermann and Borbély<sup>35</sup> explored the interplay of noise and nonlinearities in EEG alpha rhythms, illustrating chaotic features in spindles. Massimini et al.<sup>36</sup> showed that slow oscillations in NREM behave as traveling waves, suggesting that the cortex explores a high-dimensional state space. Foroutannia et al.<sup>37</sup> modeled thalamocortical up-down states and spindles, demonstrating transitions into chaos and linking them to sleep-dependent memory processes.

The roles of the circadian clock and chaos have been widely studied. Granada and Herzel<sup>38</sup> reviewed chaos and bifurcations in circadian clock models and emphasized the role of nonlinear feedback. Yang et al.<sup>39</sup> surveyed nonlinear phenomena in circadian oscillators, including routes to



**Figure 1.** A histogram showing the number of papers across sleep modeling and chaos categories. Image created by the authors with MATLAB.

Abbreviation: EEG: Electroencephalography

chaos and multistability, with relevance to sleep timing.

More recent studies in the last two years continue to push the field forward by integrating theoretical and mathematical approaches with advanced technology for sleep modeling. For example, Duarte et al.<sup>40</sup> employed complexity measures that, although not explicitly focused on chaos, highlight irregular dynamics and provide a foundation for future research. Skeldon and Dijk<sup>41</sup> modernized the two-process model from an oscillator perspective, incorporating thresholds, hysteresis, and links to neuronal mutual inhibition, offering a robust basis for dynamical analysis. Ji et al.<sup>42</sup> presented a mechanistic model showing saddle node on an invariant circle bifurcation for NREM-REM transitions and Arnold tongue entrainment to the approximately 90-minute ultradian rhythm. Yao and Yang<sup>43</sup> extended the Phillips-Robinson framework with time-varying thresholds for bifurcation analysis of sleep-wake transitions—a useful template for piecewise/threshold dynamics.

A stochastic-thermodynamics perspective on REM-NREM ultradian cycling was presented by Sun et al.,<sup>44</sup> providing a framework for noise-driven dynamics. Data-driven mathematical modeling of sleep consolidation in early childhood integrated physiologically based networks with bifurcation sequences to explain nap-to-no-nap transitions,<sup>45</sup> with emphasis on theoretical and clinical results. Additionally, a predictive REM-propensity functional derived from NREM time was introduced, particularly handy for a tractable NREM-to-REM trigger law.<sup>46</sup> Phillips and Robinson<sup>47</sup> introduced biomathematical modeling of fatigue due to sleep inertia, integrating adenosinergic dynamics to capture post-awakening transients. This model is useful for modeling short-time dynamics around state switches.

The dynamics of neurobehavioral impairment and recovery after sleep loss were reported by McCauley et al.,<sup>48</sup> who introduced additional feedback into a model to better match recovery dynamics—an excellent example of identifiability and parameter refits. Kinoshita et al.<sup>49</sup> reported a unified framework for synaptic homeodynamics during the sleep-wake cycle—a useful framework for establishing mechanistic links between network plasticity and macro-states. Finally, Putilov<sup>50</sup> posed the following question of whether the brain's thermostatic mechanism can generate sleep-wake and NREM-REM cycles, offering a multi-process dynamical scaffold. **Figure 1** summarizes these studies across six key topics, illustrating the intersection of advances in mathematical modeling and the detection of chaos in sleep EEG

### 3. Mathematical models of sleep

The development of dynamic models of sleep follows the population dynamics of wake (W), NREM (N), and REM (R) states. More complex dynamics may include subpopulations within these states. Moreover, a key variable that must be taken into account is the so-called *homeostatic pressure* ( $h$ ), which accounts for adenosine accumulation during wakefulness. A circadian oscillator of the Van der Pol family is generally incorporated.

Sleep is a nonlinear dynamical process: the brain's oscillations shift between distinct attractors. These dynamics emerge from the coupling of the previously indicated variables, making interacting actions, nonlinearity, and oscillations the main components that define the sleep model and trends.

In particular, an interaction mechanism  $N$  must be identified. In a simplified model that considers only the previous mechanism, six state variables are used. The circadian oscillator is defined by two state variables and a classical variable  $C$ . A straightforward way to represent the state-space equations is provided. For the population dynamics, three similar equations are written:

$$\tau_W \dot{W} = -W + \sigma(F_1) \quad (1)$$

$$\tau_N \dot{N} = -N + \sigma(F_2) \quad (2)$$

$$\tau_R \dot{R} = -R + \sigma(F_3) \quad (3)$$

where  $\tau_W$ ,  $\tau_N$ , and  $\tau_R$  are the time constants of the respective populations. The function  $\sigma(\cdot)$  represents a sigmoid nonlinear function defining both the nonlinearity of the process and the interaction among variables. The following functions are defined:

$$F_1 = A_1 W + A_2 N + A_3 R + A_4 C + A_5 h \quad (4)$$

$$F_2 = B_1 W + B_2 N + B_3 R + B_4 C + B_5 h \quad (5)$$

$$F_3 = C_1 W + C_2 N + C_3 R + C_4 C + C_5 h \quad (6)$$

In general, the generic function is identified as  $F_i$ , and the interaction term among the variables as  $f_{ij}$ .

The homeostatic pressure evolves as:

$$\dot{h} = \frac{(1-h)W}{\tau_{h,wake}} - \frac{h(N+R)}{\tau_{h,sleep}}, \quad h \in [0,1] \quad (7)$$

where  $\tau_h$  denotes the characteristic time constant of the homeostatic process that regulates the accumulation and dissipation rates of sleep pressure. Larger values of  $\tau_h$  correspond to slower homeostatic adaptation, while smaller values imply faster buildup and decay.

The circadian drive is simplified with a second-order oscillator:

$$\dot{x}_c = \omega \left( y_c + \mu \left( x_c - \frac{x_c^3}{3} \right) \right) \quad (8)$$

$$\dot{y}_c = -\omega x_c + I_{light} \quad (9)$$

$$c = \tanh(x_c) \quad (10)$$

The homeostatic pressure builds during the wake phase and is dissipated during sleep. The presented model is a six-state-space equation system, where  $\sigma(x)$  is given as:

$$\sigma(x) = \frac{1}{1 + e^{\alpha x}} \quad (11)$$

With:

$$\alpha = -k \text{sig}(x - \theta) \quad (12)$$

In **Equations 1–3**,  $x = F_1$  in **Equation 1**,  $x = F_2$  in **Equation 2**, and  $x = F_3$  in **Equation 3**, while  $k \text{sig}$  and  $\theta$  are scalar constants. Moreover, in **Equations 1–3**, a noise signal can be added, while in the expressions of  $F_1$ ,  $F_2$ , and  $F_3$ , a constant value, such as baseline drive, can be added.

*Remark 1.* Oscillation dynamics can be a priori fixed as a sinusoidal function.

*Remark 2.* NREM and REM activities can be estimated from the EEG as:

$$\text{NREM} \approx \frac{\delta \text{ power at } t}{\max \delta \text{ power}}, \text{ with } \delta \in [0.5, 4] \text{ Hz} \quad (13)$$

$$\text{REM} \approx \frac{\theta \text{ power at } t}{\max \theta \text{ power}}, \text{ with } \theta \in [4, 8] \text{ Hz} \quad (14)$$

More complex models include the so-called neuromodulator effects. In particular, we have:

$$\dot{F} = \frac{\alpha_F W - B_F F}{\tau_F} \quad (15)$$

The models could be improved to provide a higher-state-variable representation if, in the sleep population, two NREM and two REM components are introduced. Therefore, the sleep components are now:  $W$ ,  $N_1$ ,  $N_2$ ,  $R_1$ , and  $R_2$ .  $N_1$  indicates the light sleep;  $N_2$  indicates the intermediate stage, sleep spindles, and K complexes;  $R_1$  indicates the REM precursor or tonic REM; and  $R_2$  indicates the full phasic REM characterized by bursts of REM, vivid dreams, and autonomous irregularities.

Subsequently, the numerical results in the next section are derived from the following three models:

- Model 1: Three state variables ( $W$ ,  $R$ , and  $N$ ).
- Model 2: Six state variables ( $W$ ,  $R$ ,  $N$ , homeostatic pressure, and two circadian oscillators).
- Model 3: Nine state variables ( $W$ ,  $N_1$ ,  $N_2$ ,  $R_1$ ,  $R_2$ , homeostatic pressure, neuromodulator equation, and two circadian oscillators).

The models discussed in this section are, in a certain sense, qualitative, and their task is to allow the most specialized reader to understand the sleep dynamical process. Each variable must be considered in the context of a macro system, not as a specific variable in a biochemical process involving highly complex mechanisms.

Sleep is a complex phenomenon, and the interesting aspect lies in its unusualness. Each living organism has its own sleep. Throughout history, the characteristics of sleep have remained the same. Each organism on a given day has a different sleep pattern from another organism. We are in the presence of a phenomenon that is sensitive to both parameters and initial conditions. Moreover, sleep is a cooperative action phenomenon that exhibits spatial diversity; its signature feature is universality. The universality of sleep can lead to evaluating it as a strong dynamical process with chaotic, strange attractors. To verify this conjecture, modeling and simulation are the core of the next section. The more complex the system, the more likely it is to discover chaos.

## 4. Numerical simulations

The results were derived from Models 1–3 (**Figures 2–5**). The parameters are included in the Matlab code reported in **Section A1–A3**. The numerical integration method adopted was the Euler method. The simulations were performed to demonstrate the immediate numerical implementation of the mathematical model and to encourage readers to conduct further experiments. Indeed, the chosen parameters, and in particular the  $K \text{sig}$  values, were selected to discover chaos in the dynamical behavior. The complexity of the model has a fundamental role in discovering strange attractors.

### 4.1. Model 1

**Figure 2A** presents the trends of wake, NREM, and REM. In the state-space representation, the attractor is shown in **Figure 2B**. The cyclic behavior of the process was remarked. Moreover, the recurrence plot of variable  $N$  is shown in **Figure 5A**. Cyclic behavior was also observed, and the presence of chaos was not evident.

### 4.2. Model 2

The trends of the state variables are shown in **Figure 3A**. **Figure 3B**, shown in 3D, exhibits an emerging chaotic attractor. In **Figure 5B**, the recurrence plot of the  $R$  trend is presented.

### 4.3. Model 3

The trend of the state variables is presented in **Figure 4A**, while the emerging chaotic attractor is shown in **Figure 4B**. In **Figure 5C**, the recurrence plot of the  $R_1$  trend is presented.

## 5. Discussion

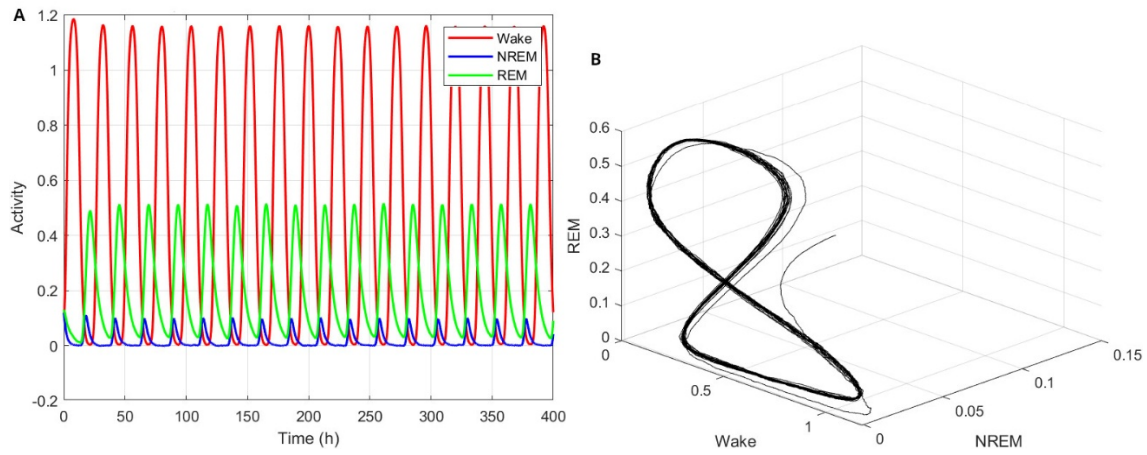
The increasing complexity of the mathematical model is directly reflected in qualitative changes of the attractor geometry. In Model 1 (three state variables), the phase profile showed predominantly cyclic and quasi-periodic behavior, and the recurrence plot featured long, continuous diagonal lines, indicating strong periodicity and temporal regularity.

In Model 2, the introduction of homeostatic and circadian dynamics increased dimensionality and introduced slow-fast interactions. The recurrence plot shows fragmentation of diagonal structures, shorter line segments, and a more irregular recurrence point distribution. This indicates a partial loss of strict periodicity and the emergence of sensitive dependence on internal interactions.

In Model 3, the addition of neuromodulatory feedback and subdivision of REM/NREM populations further increased nonlinear coupling. The recurrence plot shows a highly fragmented texture, with broken diagonals and complex recurrence clustering, consistent with the presence of chaotic attractors. The attractor projections in state-space representation display folding and stretching structures typical of nonlinear chaotic systems.

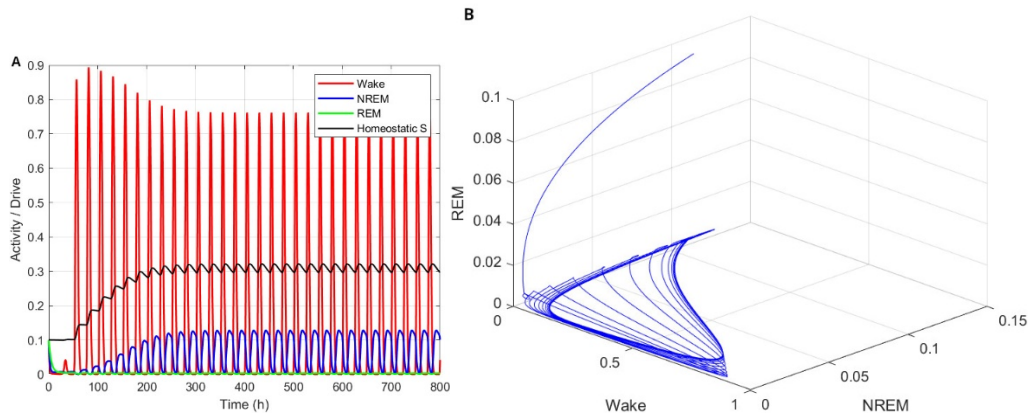
Therefore, the comparative analysis confirms that increasing model complexity enhances dynamical richness and promotes the emergence of chaotic regimes. The progressive fragmentation observed in recurrence structures provides qualitative evidence for the study's central





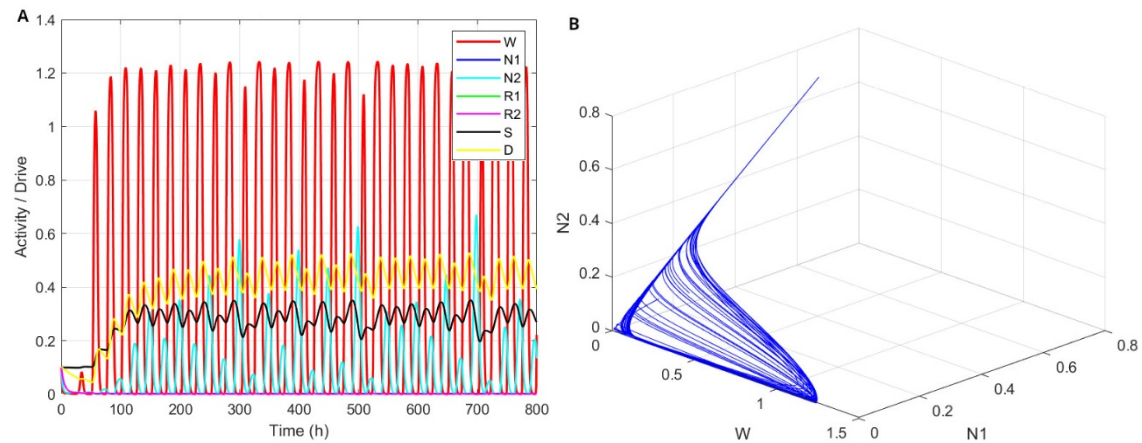
**Figure 2.** Sleep-wake dynamics with circadian drive from Model 1 in the (A) time domain and (B) state-space representation. Image created by the authors using MATLAB.

Abbreviations: NREM: Non-rapid eye movement; REM: Rapid eye movement



**Figure 3.** 5D sleep-wake dynamics from Model 2 in the (A) time domain and (B) state-space representation (W-N-R). Image created by the authors using MATLAB.

Abbreviations: NREM: Non-rapid eye movement; REM: Rapid eye movement



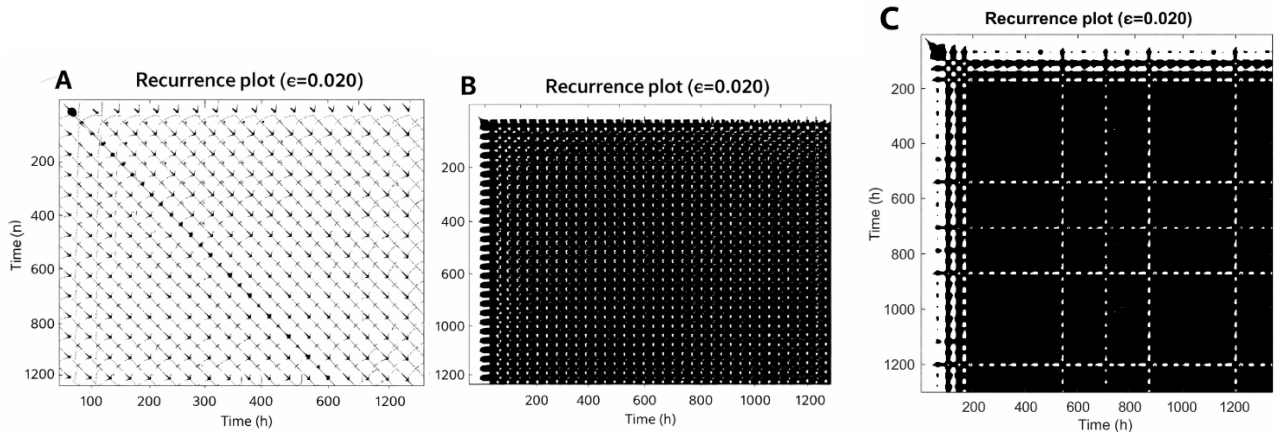
**Figure 4.** 8D Sleep-wake dynamics from Model 3 in the (A) time domain and (B) state-space representation (sample projection). Image created by the authors using MATLAB

concept.

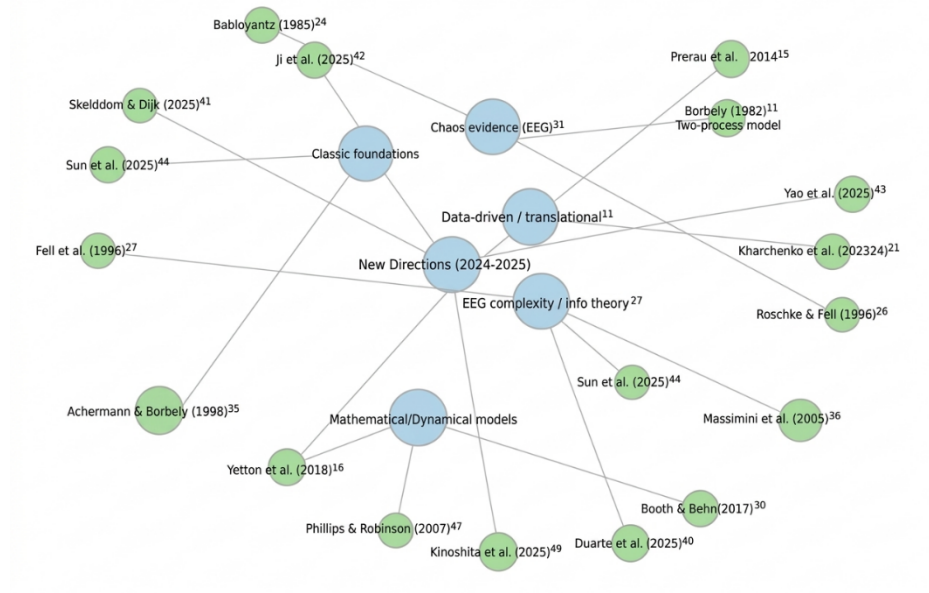
The simulation results showed the emergence of chaos. Moreover, the Lyapunov experiments provide greater certainty about the existence of chaos. The bifurcation parameter used was  $K_{sig}$ , even though chaos could be explored by adding noise. Suggestions for further work

include deriving the bifurcational diagram.

Although the present analysis remains qualitative, future work will include quantitative recurrence quantification analysis metrics and Lyapunov exponent estimation to rigorously characterize chaotic regimes.



**Figure 5.** Recurrence plots of non-rapid eye movement activity for (A) Model 1, (B) Model 2, and (C) Model 3. Image created by the authors using MATLAB.



**Figure 6.** Map of sleep dynamics references. Image created by the authors using Draw.io. Abbreviation: EEG: Electroencephalography

## 6. Conclusion

This review provides guidelines for studying the dynamical models of the sleep process. In **Figure 6**, a map of sleep dynamics reference is shown. Essentially, it emerges that the cluster shown in **Figure 1**. **Figure 7** presents the Gantt-style timeline for the sleep dynamics research theme. From the authors' point of view, given the previous graphs, the issue of chaos in sleep studies will be addressed in the near future. The contribution of this work is to the field of sleep technology. This means modeling sleep using a system-based control. Moreover, people interested in the implementation of chaotic electronic circuits could derive new devices from sleep dynamics. This means that sleep dynamics could help both neuroscience-based technologies and electronic technologies based on chaos.

## Acknowledgments

None.

## Funding

None.

## Conflict of interest

Luigi Fortuna is the Honorary Editor-in-Chief of this journal, but was not in any way involved in the editorial and peer-review process conducted for this paper, directly or indirectly. Separately, other authors declared that they have no known competing financial interests or personal relationships that could have influenced the work reported in this paper.

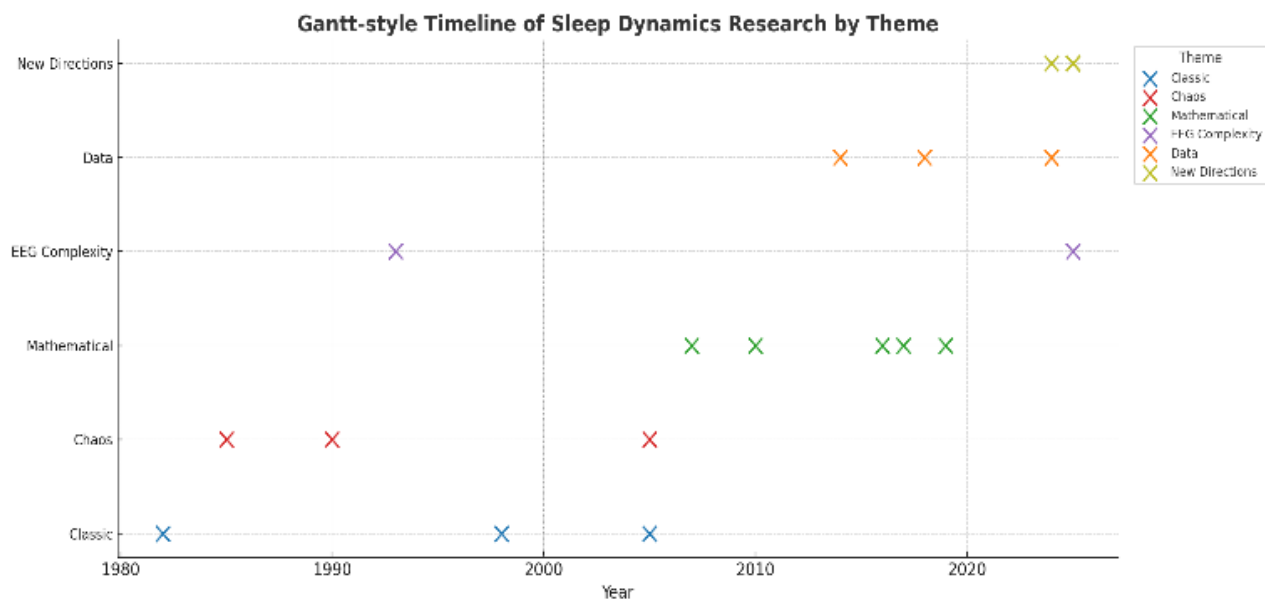
## Author contributions

*Conceptualization:* All authors

*Visualization:* All authors

*Writing—original draft:* All authors

*Writing—review & editing:* All authors



**Figure 7.** A Gantt chart showing the time distribution of the development of topics related to sleep dynamics in research efforts over the last few decades. Image created by the authors using MATLAB.  
Abbreviation: EEG: Electroencephalography.

## Availability of data

Not applicable.

## AI Tools Statement

All authors confirm that no AI tools were used in the preparation of this manuscript.

## References

- Walker M. *Why We Sleep: Unlocking the Power of Sleep and Dreams*. Scribner. 2017.
- Rattenborg NC, Ungurean G. The evolution and diversification of sleep. *Trends in Ecology & Evolution*. 2023;38(2):156-170.  
<https://www.doi.org/10.1016/j.tree.2022.10.004>
- Roth TC, Lesku JA, Amlaner CJ, Lima SL. A phylogenetic analysis of the correlates of sleep in birds. *J Sleep Res*. 2006;15(4):395-402.  
<https://www.doi.org/10.1111/j.1365-2869.2006.00559.x>
- Sauer S, Herrmann E, Kaiser W. Sleep deprivation in honey bees. *J Sleep Res*. 2004;13(2):145-152.  
<https://www.doi.org/10.1111/j.1365-2869.2004.00393.x>
- Lima SL, Rattenborg NC, Lesku JA, Amlaner CJ. Sleeping under the risk of predation. *Anim Behav*. 2005;70(4):723-736.  
<https://www.doi.org/10.1016/j.anbehav.2005.01.008>
- Vyazovskiy VV, Tobler I, Gilestro GF. The temporal structure of behaviour and sleep homeostasis. *PLoS One*. 2012;7(12):e50677.  
<https://www.doi.org/10.1371/journal.pone.0050677>
- Rich MM, Wenner P. Sensing and expressing homeostatic synaptic plasticity. *Trends in Neurosci*. 2007;30(3):119-125.  
<https://www.doi.org/10.1016/j.tins.2007.01.004>
- Siegel JM. Sleep function: an evolutionary perspective. *Lancet Neurol*. 2022;21(10):937-946.  
[https://www.doi.org/10.1016/S1474-4422\(22\)00210-1](https://www.doi.org/10.1016/S1474-4422(22)00210-1)
- Dudley CA, Erbel-Sieler C, Estill SJ, et al. Altered patterns of sleep and behavioral adaptability in NPAS2-deficient mice. *Sci*. 2003;301(5631):379-383.  
<https://www.doi.org/10.1126/science.1082795>
- Olbrich E, Claussen JC, Achermann P. The multiple time scales of sleep dynamics as a challenge for modelling the sleeping brain. *Philos Trans A Math Phys Eng Sci*. 2011;369(1952):3884-3901.  
<https://www.doi.org/10.1098/rsta.2011.0082>
- Borbély AA. A two process model of sleep regulation. *Hum Neurobiol*. 1982;1(3):195-204.
- Phillips AJK, Robinson PA. A quantitative model of sleep-wake dynamics based on the physiology of the brainstem ascending arousal system. *J Biol Rhythms*. 2007;22(2):167-179.  
<https://www.doi.org/10.1177/0748730406297512>
- Comte JC, Ravassard P, Salin PA. Sleep dynamics: a self-organized critical system. *Phys Rev E Stat Nonlin Soft Matter Phys*. 2006;73(5):056127.  
<https://www.doi.org/10.1103/PhysRevE.73.056127>
- Sorensen GL, Knudsen S, Jennum P. Sleep transitions in hypocretin-deficient narcolepsy. *Sleep*. 2013;36(8):1173-1177.  
<https://www.doi.org/10.5665/sleep.2880>
- Prerau MJ, Brown RE, Bianchi MT, Ellenbogen JM, Purdon PL. Tracking the sleep onset process: an empirical model of behavioral and physiological dynamics. *PLoS Comput Biol*. 2014;10(10):e1003866.  
<https://www.doi.org/10.1371/journal.pcbi.1003866>
- Yetton BD, McDevitt EA, Cellini N, Shelton C, Mednick SC. Quantifying sleep architecture dynamics and individual differences using Bayesian networks. *PLoS One*. 2018;13(4):e0194604.  
<https://www.doi.org/10.1371/journal.pone.0194604>
- Basner M, Rao H, Goel N, Dinges DF. Sleep deprivation and neurobehavioral dynamics. *Curr Opin Neurobiol*. 2013;23(5):854-863.  
<https://www.doi.org/10.1016/j.conb.2013.02.008>
- Chen PC, Niknazar H, Alaynick WA, Whitehurst LN, Mednick SC. Competitive dynamics underlie cognitive improvements during sleep. *Proc Natl Acad Sci*. 2021;118(51):e2109339118.  
<https://www.doi.org/10.1073/pnas.2109339118>
- Gottesman RE, Lutsey PL, Benveniste H, et al. Impact of Sleep Disorders and Disturbed Sleep on Brain Health: A Scientific Statement From the American Heart Association. *Stroke*. 2024;55(3).  
<https://www.doi.org/10.1161/str.0000000000000453>



20. Rempe MJ, Best J, Terman D. A mathematical model of the sleep/wake cycle. *J Math Biol.* 2010;60(5):615-644. <https://www.doi.org/10.1007/s00285-009-0276-5>
21. Kharchenko V, Zhdanova IV. The wave model of sleep dynamics and an invariant relationship between non-REM and REM sleep. *Clocks & Sleep.* 2023;5(4):686-716. <https://www.doi.org/10.3390/clockssleep5040046>
22. Viswanath VK, Hartogenesis W, Dilchert S, et al. Five million nights: temporal dynamics in human sleep phenotypes. *npj Digit Med.* 2024;7(1). <https://www.doi.org/10.1038/s41746-024-01125-5>
23. Robinson PA, Phillips AJK, Fulcher BD, Puckeridge M, Roberts JA. Quantitative modelling of sleep dynamics. *Philos Trans A Math Phys Eng Sci.* 2011;369(1952):3840-3854. <https://www.doi.org/10.1098/rsta.2011.0120>
24. Babloyantz A, Salazar JM, Nicolis C. Evidence of chaotic dynamics of brain activity during the sleep cycle. *Phys Lett A.* 1985;111(3):152-156. [https://www.doi.org/10.1016/0375-9601\(85\)90444-X](https://www.doi.org/10.1016/0375-9601(85)90444-X)
25. Röscke J, Başar E. The EEG is Not a Simple Noise: Strange Attractors in Intracranial Structures. In: *Springer Series in Brain Dynamics.* Springer Berlin Heidelberg; 1988:203-216. [https://www.doi.org/10.1007/978-3-642-71531-0\\_13](https://www.doi.org/10.1007/978-3-642-71531-0_13)
26. Fell J, Röscke J, Beckmann P. Deterministic chaos and the first positive Lyapunov exponent: a nonlinear analysis of the human electroencephalogram during sleep. *Biol Cybern.* 1993;69(2):139-146. <https://www.doi.org/10.1007/BF00226197>
27. Fell J, Röscke J, Mann K. Discrimination of sleep stages: a comparison between spectral and nonlinear EEG measures. *Electroencephalogr Clin Neurophysiol.* 1996;98(5):401-410. [https://www.doi.org/10.1016/0013-4694\(96\)95636-9](https://www.doi.org/10.1016/0013-4694(96)95636-9)
28. Kruglikov SY, Schiff SJ. Interplay of electroencephalogram phase and auditory-evoked neural activity. *J Neurosci.* 2003;23(31):10122-10127. <https://www.doi.org/10.1523/JNEUROSCI.23-31-10122.2003>
29. Behn CGD, Booth V. A fast-slow analysis of the dynamics of REM sleep. *SIAM J Appl Dyn Syst.* 2012;11(1):212-242. <https://www.doi.org/10.1137/110832823>
30. Booth V, Xique I, Diniz Behn, CG. One-dimensional map for the circadian modulation of sleep in a sleep-wake regulatory network model for human sleep. *SIAM J Appl Dyn Syst.* 2017;16(2):1089-1112. <https://www.doi.org/10.1137/16M1071328>
31. Postnova S, Voigt K, Braun HA. A Mathematical Model of Homeostatic Regulation of Sleep-Wake Cycles by Hypocretin/Orexin. *J Biol Rhythms.* 2009;24(6):523-535. <https://www.doi.org/10.1177/0748730409346655>
32. Hutt A, Longtin A. Effects of the anesthetic agent propofol on neural populations. *Cogn Neurodyn.* 2009;4(1):37-59. <https://www.doi.org/10.1007/s11571-009-9092-2>
33. Paul K, Caulier LJ, Llano DA. Presence of a chaotic region at the sleep-wake transition in a simplified thalamocortical circuit model. *Front Comput Neurosci.* 2016;10:91. <https://www.doi.org/10.3389/fncom.2016.00091>
34. Rasmussen R, Jensen MH, Heltberg ML. Chaotic Dynamics Mediate Brain State Transitions, Driven by Changes in Extracellular Ion Concentrations. *Cell Systems.* 2017;5(6):591-603.e4. <https://www.doi.org/10.1016/j.cels.2017.11.011>
35. Achermann P, Borbély AA. Temporal evolution of coherence and power in the human sleep electroencephalogram. *J Sleep Res.* 1998;7(S1):36-41. <https://www.doi.org/10.1046/j.1365-2869.7.s1.6.x>
36. Massimini M, Huber R, Ferrarelli F, Hill S, Tononi G. The Sleep Slow Oscillation as a Traveling Wave. *J Neurosci.* 2004;24(31):6862-6870. <https://www.doi.org/10.1523/jneurosci.1318-04.2004>
37. Foroutannia A, Nazarimehr F, Ghasemi M, Jafari S. Chaos in memory function of sleep: A nonlinear dynamical analysis in thalamocortical study. *J Theor Biol.* 2021;528:110837. <https://www.doi.org/10.1016/j.jtbi.2021.110837>
38. Granada AE, Herzel H. How to Achieve Fast Entrainment? The Timescale to Synchronization. Khanin R, ed. *PLoS ONE.* 2009;4(9):e7057. <https://www.doi.org/10.1371/journal.pone.0007057>
39. Yang DP, McKenzie-Sell L, Karanjai A, Robinson PA. Wake-sleep transition as a noisy bifurcation. *Phys Rev E.* 2016;94(2). <https://www.doi.org/10.1103/physreve.94.022412>
40. Duarte CD, Pacheco M, Iaconis FR, et al. Statistical complexity analysis of sleep stages. *Entropy.* 2025;27(1):76. <https://www.doi.org/10.3390/e27010076>
41. Skeldon AC, Dijk DJ. The complexity and commonness of the two-process model of sleep regulation from a mathematical perspective. *npj Biol Timing Sleep.* 2025;2(1). <https://www.doi.org/10.1038/s44323-025-00039-z>
42. Ji Y, Xu F, Shuai J, Yang D, Yao C. Dynamical mechanism for the interplay of circadian, homeostatic, and ultradian rhythm in normal human sleep. *Phys Rev E.* 2025;111(4). <https://www.doi.org/10.1103/physreve.111.044215>
43. Yao C, Yang D. Mechanistic modeling of sleep-wake transitions via circadian-modulated threshold dynamics. *bioRxiv.* Preprint online July 15, 2025. <https://www.doi.org/10.1101/2025.07.10.664059>
44. Sun H, Ishbulatov Y, Karavaev A., Zakharov D., Zaikin A. A thermodynamic model of the REM-NREM sleep cycle. *Chaos, Solitons & Fractals.* 2025;190:115732. <https://www.doi.org/10.1016/j.chaos.2024.115732>
45. Athanasouli C, Stowe SR, LeBourgeois MK, Booth V, Diniz Behn CG. Data-driven mathematical modeling of sleep consolidation in early childhood. *J Theor Biol.* 2024;593:111892. <https://www.doi.org/10.1016/j.jtbi.2024.111892>
46. Ginsberg AG, Cruz MEC, Weber F, Booth V, Diniz Behn CG. A predictive propensity measure to enter REM sleep. *Front Neurosci.* 2024;18. <https://www.doi.org/10.3389/fnins.2024.1431407>
47. Phillips AJK, Robinson PA. A Quantitative Model of Sleep-Wake Dynamics Based on the Physiology of the Brainstem Ascending Arousal System. *J Biol Rhythms.* 2007;22(2):167-179. <https://www.doi.org/10.1177/0748730406297512>
48. McCauley ME, Van Dongen HPA, Belenky G, et al. The dynamics of neurobehavioral impairment and recovery after sleep loss. *Front Environ Health.* 2024;3:1362755. <https://www.doi.org/10.3389/fenvh.2024.1362755>
49. Kinoshita T, Tatsuki F, Yoshida Y, et al. A unified framework to model synaptic dynamics during the sleep-wake cycle. *PLoS Biol.* 2025;23(6):e3003198. <https://www.doi.org/10.1371/journal.pbio.3003198>
50. Putilov AA. Can the Brain's Thermostatic Mechanism Generate Sleep-Wake and NREM-REM Sleep Cycles? A Nested Doll Model of Sleep-Regulating Processes. *Clocks & Sleep.* 2024;6(1):97-113. <https://www.doi.org/10.3390/clockssleep6010008>

## Appendix

### A1. Matlab code for Model 1

```

%% Parameters
aW = 0.8; aN = 0.5; aR = 0.2; % decay rates
bWN = 1.0; bNW = 1.0; % mutual inhibition W-N
bWR = 0.5; bRW = 0.5; % inhibition W-R
bNR = 0.8; bRN = 0.8; % inhibition N-R
cR = 0.6; % NREM drives REM
kC = 0.3; kCN = 0.2; kCR = 0.2; % circadian modulation strengths

I_W = 0.5; I_N = 0.3; I_R = 0.2; % baseline drives
kSig = 12; theta = 0.5; % sigmoid parameters

%% Simulation settings
tspan = [0 400]; % simulation time (hours)
dt = 0.01; % integration step
time = tspan(1): dt:tspan(2);

%% Initial conditions
W0 = 0.11; N0 = 0.12; R0 = 0.13;
X = [W0; N0; R0];
%% Preallocate

W = zeros(size(time));
N = zeros(size(time));
R = zeros(size(time));
%% Sigmoid function
sigmoid = @(x) 1./(1 + exp(-kSig*(x - theta)));
%% Circadian signal (approx 24h sine wave)
C = @(t) sin(2*pi*t/24);
%% Euler integration
for i = 1:length(time)
    t = time(i);

% Current state
    W_i = X(1); N_i = X(2); R_i = X(3);
    % Nonlinear interactions + circadian modulation
    dW = -aW*W_i + sigmoid(I_W - bWN*N_i - bWR*R_i + kC*C(t));
    dN = -aN*N_i + sigmoid(I_N - bNW*W_i - bNR*R_i - kCN*C(t));
    dR = -aR*R_i + sigmoid(I_R - bRW*W_i - bRN*N_i + cR*N_i - kCR*C(t));

% Optional: small noise to induce transient chaos
    noiseLevel = 0.01;
    dW = dW + noiseLevel*randn;
    dN = dN + noiseLevel*randn;
    dR = dR + noiseLevel*randn;

% Euler update
    X(1) = W_i + dt*dW;
    X(2) = N_i + dt*dN;
    X(3) = R_i + dt*dR;

% Save
    W(i) = X(1); N(i) = X(2); R(i) = X(3);
end

```

## A2. Matlab code for Model 2

```

%% Parameters
aW = 0.8; aN = 0.5; aR = 0.2; % decay rates
bWN = 1.2; bNW = 1.2; % mutual inhibition W-N
bWR = 0.5; bRW = 0.5; % Wake-REM inhibition
bNR = 0.8; bRN = 0.8; % NREM-REM inhibition
cR = 0.6; % NREM promotes REM
kSig = 14; theta = 0.5; % sigmoid
alphaS = 1.0; gammaW = 0.01; gammaN = 0.05;
% homeostatic dynamics
kC = 0.3; % circadian coupling

noiseLevel = 0.; % optional noise

%% Circadian oscillator parameters (van der Pol type)
muC = 0.2; omegaC = 2*pi/24; % period ~24h

%% Simulation settings
tspan = [0 800]; dt = 0.01;
time = tspan(1):dt:tspan(2);

%% Initial conditions
W0 = 0.1; N0 = 0.1; R0 = 0.1; S0 = 0.1; C0 = 0.0; dC0 = 0.01;
X = [W0; N0; R0; S0; C0; dC0];

%% Preallocate
W = zeros(size(time)); N = zeros(size(time)); R = zeros(size(time));
S = zeros(size(time)); C = zeros(size(time));

%% Sigmoid function
sigmoid = @(x) 1./(1 + exp(-kSig*(x - theta)));

%% Euler integration
for i = 1:length(time)
    t = time(i);

    % Current states
    W_i = X(1); N_i = X(2); R_i = X(3); S_i = X(4); C_i = X(5); dC_i = X(6);

    % Circadian input
    circ = C_i;

    % Population dynamics
    dW = -aW*W_i + sigmoid(-bWN*N_i - bWR*R_i + kC*circ) + noiseLevel*randn;
    dN = -aN*N_i + sigmoid(alphaS*S_i - bNW*W_i - bNR*R_i) + noiseLevel*randn;
    dR = -aR*R_i + sigmoid(cR*N_i - bRW*W_i - bRN*N_i) + noiseLevel*randn;

    % Homeostatic sleep pressure
    dS = gammaW*(1-S_i)*W_i - gammaN*S_i*N_i;

    % Circadian oscillator (van der Pol)
    ddC = muC*(1 - C_i^2)*dC_i - omegaC^2*C_i;

    % Euler update
    X(1) = W_i + dt*dW;
    X(2) = N_i + dt*dN;
    X(3) = R_i + dt*dR;
    X(4) = S_i + dt*dS;
    X(5) = C_i + dt*dC_i;
    X(6) = dC_i + dt*ddC;

    % Save for plotting
    W(i) = X(1); N(i) = X(2); R(i) = X(3); S(i) = X(4); C(i) = X(5);
End

```

### A3. Matlab code for Model 3

```

%% Parameters
% Decay rates
aW = 0.8; aN1 = 0.5; aN2 = 0.5; aR1 = 0.2; aR2 = 0.2;
% Mutual inhibition / promotion
bWN = 1.2; bNW = 1.2; bWR = 0.5; bRW = 0.5;
bNR = 0.8; bRN = 0.8; cR = 0.6; cN = 0.3; % promotion factors
% Sigmoid parameters
kSig = 14; theta = 0.5;
% Homeostatic
alphaS = 1.0; gammaW = 0.01; gammaN = 0.05;
% Circadian oscillator
muC = 0.2; omegaC = 2*pi/24;
kC = 0.3;
% Neuromodulator
tauD = 50; % slow dynamics
% Noise
noiseLevel = 0.;

%% Simulation
tspan = [0 800]; dt = 0.01; time = tspan(1):dt:tspan(2);

%% Initial conditions
W0 = 0.1; N10 = 0.1; N20 = 0.1; R10 = 0.1; R20 = 0.1; S0 = 0.1; C0 = 0.0; dC0 = 0.01; D0 = 0.1;
X = [W0; N10; N20; R10; R20; S0; C0; dC0; D0];

%% Preallocate
W=zeros(size(time)); N1=zeros(size(time)); N2=zeros(size(time));
R1=zeros(size(time)); R2=zeros(size(time)); S=zeros(size(time));
C=zeros(size(time)); D=zeros(size(time));

%% Sigmoid
sigmoid = @(x) 1./(1 + exp(-kSig*(x - theta)));

%% Euler integration
for i=1:length(time)
    t = time(i);

    % Current states
    W_i = X(1); N1_i = X(2); N2_i = X(3); R1_i = X(4); R2_i = X(5);
    S_i = X(6); C_i = X(7); dC_i = X(8); D_i = X(9);

    % Circadian input
    circ = C_i;

    % Population dynamics with nonlinear interactions
    dW = -aW*W_i + sigmoid(-bWN*(N1_i+N2_i) - bWR*(R1_i+R2_i) + kC*circ + D_i) + noiseLevel*randn;
    dN1 = -aN1*N1_i + sigmoid(alphaS*S_i + cN*N2_i - bNW*W_i - bNR*(R1_i+R2_i)) + noiseLevel*randn;
    dN2 = -aN2*N2_i + sigmoid(alphaS*S_i + cN*N1_i - bNW*W_i - bNR*(R1_i+R2_i)) + noiseLevel*randn;
    dR1 = -aR1*R1_i + sigmoid(cR*(N1_i+N2_i) - bRW*W_i - bRN*(N1_i+N2_i)) + noiseLevel*randn;
    dR2 = -aR2*R2_i + sigmoid(cR*(N1_i+N2_i) - bRW*W_i - bRN*(N1_i+N2_i)) + noiseLevel*randn;

    % Homeostatic sleep drive
    dS = gammaW*(1-S_i)*W_i - gammaN*S_i*(N1_i+N2_i);

    % Circadian oscillator (van der Pol)
    ddC = muC*(1 - C_i^2)*dC_i - omegaC^2*C_i;

    % Neuromodulator / fatigue
    dD = (W_i - D_i)/tauD;

    % Euler update
    X(1) = W_i + dt*dW; X(2) = N1_i + dt*dN1; X(3) = N2_i + dt*dN2;
    X(4) = R1_i + dt*dR1; X(5) = R2_i + dt*dR2; X(6) = S_i + dt*dS;
    X(7) = C_i + dt*dC_i; X(8) = dC_i + dt*ddC; X(9) = D_i + dt*dD;

    % Save
    W(i)=X(1); N1(i)=X(2); N2(i)=X(3); R1(i)=X(4); R2(i)=X(5);
    S(i)=X(6); C(i)=X(7); D(i)=X(9);
End

```