Specific Regulation of Cognitive Functions by Circadian Rhythms

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Abstract

Circadian rhythm is an endogenous near-24-hour timing system in organisms that regulates various cognitive functions. Cognitive performance typically declines during circadian troughs and recovers during peaks, with different functions exhibiting distinct temporal variations. Existing studies often fail to distinguish between the underlying driving mechanisms, making it difficult to systematically elucidate the specific regulatory mechanisms of different cognitive functions; furthermore, there is a lack of relevant neural evidence. This paper aims to distinguish between circadian rhythm effects driven by different mechanisms, explore the differentiated impacts of each mechanism on cognitive functions, and explain this specificity at the level of neural mechanisms. Future research needs to integrate multimodal neural data with computational models, combine targeted neuromodulation techniques to establish causal relationships, and explore precision light intervention strategies based on specific time windows, thereby providing precise cognitive protection schemes for populations with circadian rhythm disorders.

Full Text

Preamble

Specific Regulation of Cognitive Functions by Circadian Rhythms Kaikai Yan [1], Bowen Guo [2], Xing Chen [1], Tianxin Mao [1*], Hengyi Rao [1,2*] ([1] School of Intelligent Business and Management, Shanghai Key Laboratory of Brain-Machine Synergistic Information Behavior, MRI Research Center, Shanghai International Studies University, Shanghai 201600; [2] School of Humanities and Social Science, University of Science and Technology of China, Hefei 230026)

The circadian rhythm is an endogenous biological timing system with a period of approximately 24 hours that regulates a wide range of cognitive functions.

Cognitive performance typically declines during circadian troughs and recovers during peak periods; however, different cognitive functions exhibit distinct temporal patterns.

Existing research often fails to distinguish between the underlying driving mechanisms of these rhythms, making it difficult to systematically explain the specific regulatory processes governing different cognitive functions. Furthermore, there is a notable lack of corresponding neuroscientific evidence to support these observations.

This paper aims to differentiate the circadian rhythm effects driven by various mechanisms and explore their differential impacts on cognitive functions. Additionally, it seeks to explain this specificity from the perspective of underlying neural mechanisms.

Future research should integrate multimodal neural data with computational models and utilize targeted neuromodulation techniques to establish causal relationships. Furthermore, exploring precision light intervention strategies based on specific time windows will be essential for providing targeted cognitive protection for populations suffering from circadian rhythm disruptions.

关键词

Circadian Rhythm, the Two-Process Model of Sleep-Wake Regulation, and Cognitive Function

Introduction

The regulation of human physiological and behavioral processes is governed by a complex interplay between internal biological clocks and external environmental cues. Central to this regulation is the circadian rhythm, an endogenous oscillation with a period of approximately 24 hours that coordinates various metabolic, hormonal, and behavioral functions. In the context of sleep and alertness, the most widely accepted theoretical framework is the "Two-Process Model of Sleep-Wake Regulation." This model provides a robust foundation for understanding how sleep pressure and circadian timing interact to determine our state of arousal and subsequent cognitive performance.

The Two-Process Model of Sleep-Wake Regulation

The Two-Process Model, originally proposed by Borbély [?], posits that sleep regulation is governed by the interaction of two independent yet interacting processes: Process S and Process C.

Process S (The Homeostatic Process): This represents the sleep debt or sleep pressure that accumulates during wakefulness and dissipates during sleep. The longer an individual remains awake, the higher the levels of Process S, leading to an increased propensity for sleep. Mathematically, this is often modeled as a function that increases exponentially during wakefulness and decreases exponentially during sleep.

Process C (The Circadian Process): This is the endogenous rhythm generated by the suprachiasmatic nucleus (SCN) of the hypothalamus. Unlike Process S, Process C is independent of the duration of prior wakefulness or sleep. It oscillates with a near-24-hour period, facilitating alertness during the day and promoting sleep during the biological night.

The interaction between these two processes determines the timing, duration, and structure of sleep. Under normal conditions, Process C opposes the rising sleep pressure of Process S during the late afternoon and evening, allowing humans to maintain stable levels of alertness throughout the day. However, when these processes are misaligned—such as during shift work or jet lag—cognitive function can be significantly impaired.

Impact on Cognitive Function

Cognitive function is not a static trait but fluctuates significantly based on the state of the sleep-wake regulatory system. Research has consistently shown that both the homeostatic sleep drive and the circadian phase exert profound influences on various cognitive domains, including attention, executive function, memory, and processing speed.

1. **Attention and Vigilance:** Sustained attention is perhaps the most sensitive cognitive domain to sleep loss. As Process S increases

1. 引言

Circadian rhythms are endogenous timing systems evolved by organisms to adapt to the Earth’ s rotation. These rhythms regulate a wide range of physiological and behavioral processes—including sleep-wake cycles, hormone secretion, core body temperature, metabolism, and cognitive functions—with a period of approximately 24 hours [?, ?, ?, ?, ?]. The stable operation of this system is a fundamental requirement for maintaining individual health and cognitive performance. However, factors prevalent in modern society, such as high-intensity work schedules, trans-meridian travel, and exposure to blue light from electronic devices, frequently lead to sleep deprivation and circadian misalignment [?, ?, ?].

Extensive research has demonstrated that circadian disruption significantly impairs cognitive function, and prolonged wakefulness further exacerbates this impairment [?, ?, ?, ?, ?]. For instance, sleep deprivation has been shown to weaken vigilant attention, working memory, and executive functions while increasing subjective sleepiness [?, ?, ?, ?, ?]. Notably, these cognitive impairments are not uniformly distributed throughout the day but exhibit specific diurnal fluctuations: performance typically reaches a nadir from early morning to late morning (04:00-12:00) and shows recovery from late afternoon to night (16:00-00:00). This phenomenon is known as the "circadian rhythm effect" [?, ?, ?, ?, ?]. Further research has found that these circadian effects are not perfectly synchronized across different cognitive domains. For example, the peak phases of cognitive functions such as subjective sleepiness, vigilant attention, and working memory may differ [?, ?, ?, ?].

In contrast, some studies have found that risky decision-making and moral behavior do not exhibit significant rhythmic fluctuations [?, ?, ?, ?]. These observations suggest that the circadian regulation of cognitive function may be domain-specific, manifesting both as phase differences between functions and as varying levels of sensitivity to circadian signals. Preliminary neuroimaging studies support this view, showing that cortical and subcortical structures exhibit distinct patterns of activity fluctuation throughout the day [?, ?, ?]. Consequently, systematically elucidating how circadian rhythms differentially regulate cognitive functions and their neural substrates through various internal mechanisms has become a critical scientific question in current research.

However, existing research on circadian rhythm effects still faces several limitations. First, the conceptual definition and underlying physiological mechanisms of these effects remain unclear. On one hand, interpretations of the internal driving mechanisms vary across studies, often failing to explicitly distinguish the independent contributions of different processes. This conceptual inconsistency makes it difficult to accurately assess the specific impact of each mechanism on cognitive function, thereby limiting the precision and effectiveness of intervention strategies. On the other hand, while physiological mechanisms provide the theoretical foundation for distinguishing these internal drivers, current research is insufficiently systematic. Most studies focus narrowly on molecular clocks or the central pacemaker, lacking an integrated framework that encompasses multi-dimensional interactions across molecular, central nervous, and endocrine systems.

Second, the specificity of circadian rhythm effects has not been fully revealed, and the cognitive domains covered by existing research remain relatively limited. Early theories posited that circadian effects were global, suggesting that fluctuations in physiological indicators like core body temperature influenced all types of cognitive functions identically [?, ?, ?, ?, ?]. However, recent studies indicate phase differences in the circadian peaks of different cognitive functions [?, ?, ?, ?]. These differences may reflect the selective involvement of different neural oscillators or the dynamic regulation of cortical arousal systems [?, ?, ?, ?]. Furthermore, empirical research has primarily concentrated on a narrow range of domains—such as vigilant attention, working memory, and executive function —lacking systematic comparisons across broader cognitive dimensions and failing to explore specific regulatory patterns while distinguishing between internal driving mechanisms. Finally, evidence regarding neural mechanisms remains fragmented. Although some studies have begun to uncover the neural basis of circadian effects—identifying diurnal fluctuations in arousal-related regions such as the thalamus and hypothalamus [?, ?, ?] and noting that EEG θ power, which is closely linked to cortical arousal, also exhibits significant rhythmicity [?, ?, ?]—these findings are mostly limited to single cognitive functions and lack cross-functional integration or comparative analysis.

To address these gaps, the present study aims to systematically review the impact of circadian rhythms on cognitive function and their underlying neural mechanisms. This work focuses on three main areas: (1) defining and distinguishing circadian rhythm effects driven by different internal mechanisms (providing a theoretical basis for this distinction by integrating physiological evidence from molecular clocks, the central pacemaker, and the endocrine system); (2) comprehensively summarizing the specific manifestation patterns of circadian effects across cognitive domains, including subjective sleepiness, vigilant attention, working memory, complex executive function, risky decision-making, and moral behavior; and (3) integrating existing neuroscientific evidence to systematically outline the regulatory mechanisms of circadian rhythms across different cognitive functions and propose directions for future research. By distinguishing circadian effects under different internal drivers and combining behavioral and neural evidence, this study aims to construct a clear framework for how circadian rhythms influence cognitive function. This will provide a mechanistic explanation for understanding their role in human cognition and offer practical guidance for fatigue management and intervention in high-risk scenarios where sleep deprivation is unavoidable.

2.1 昼夜节律的概念与研究范式

The classic two-process model of sleep-wake regulation (Borbély, 1982; Borbély et al., 2016; Daan et al., 1984; Dijk et al., 1992) posits that cognitive performance during wakefulness is regulated by two relatively independent internal driving mechanisms. The middle section of [Figure 1: see original paper]D illustrates the schematic of this two-process model (adapted from Borbély et al., 2016). (1) The homeostatic process (Process S) reflects an individual’ s sleep pressure, which accumulates exponentially with the duration of wakefulness, thereby inhibiting cognitive performance. (2) The circadian process (Process C) is driven by the suprachiasmatic nucleus (SCN) and generates rhythmic oscillations of approximately 24 hours, exerting periodic modulation on cognitive functions. Research has demonstrated that subjective sleepiness, vigilant attention, and complex executive functions all follow the patterns of the two-process model. During the first approximately 16 hours of daytime wakefulness, Process C

can partially counteract the sleep homeostatic pressure that gradually accumulates with time spent awake, thereby maintaining cognitive performance at a relatively high level. As wakefulness extends further into the biological night, the circadian arousal-promoting effect diminishes while the homeostatic sleep pressure remains unmitigated, typically causing cognitive function to drop to its lowest point at the circadian trough. When wakefulness continues from the night into the following day, cognitive function recovers to a relative peak during the circadian crest as the circadian arousal-promoting signals strengthen (Mai et al., 2019; Goel et al., 2013; Valdez et al., 2012, 2019).

Neural projection pathways of the clock: the SCN coordinates downstream nuclei to regulate sleep-wake transitions; (C) Endocrine system: the rhythmic secretion of melatonin and cortisol regulated by the SCN; (D) System integration: the overall framework of environmental input, central coordination, and physiological/behavioral output, adapted from Borbély et al. (2016) (middle section).

While this model provides a theoretical framework for defining circadian effects, the terminology used in existing literature remains inconsistent. Although common terms such as "time of day" (Li et al., 2020), "diurnal rhythm" (Peng et al., 2023), and "circadian rhythms" (Carrier & Monk, 2000) all describe intraday fluctuations in cognitive performance, their underlying physiological driving mechanisms may differ fundamentally and require further clarification. Based on this, the present paper proposes two operational criteria to distinguish between circadian effects in the broad and narrow senses. First, whether critical masking factors such as light and activity are strictly controlled in the experimental design; second, whether mathematical models are employed in data analysis to separate sleep homeostasis (Process S) from the circadian rhythm (Process C). When a study fails to meet either of these criteria, the reported intra-day fluctuations in cognition are regarded as broad circadian effects (Process S + C), reflecting the combined influence of endogenous rhythms and sleep homeostasis. When both criteria are met, the isolated

cognitive components driven purely by the endogenous biological clock are defined as narrow circadian effects (Process C). Clarifying these conceptual differences depends largely on the choice of research paradigm. Different paradigms vary significantly in their degree of control over external masking factors, leading to inconsistent results across studies and influencing whether the revealed effects are broad or narrow circadian effects.

Common research paradigms include: (1) Time of Day (TOD) protocol: cognitive measurements are taken at least twice within a single day under natural sleep-wake conditions without interfering with the subjects' schedules (Blake, 1967). This method is easy to implement but fails to strictly control for confounding factors such as light, activity, and food intake, making it difficult to isolate endogenous rhythmic effects; thus, it typically reflects broad effects (S + C) (Munnilari et al., 2024). (2) Constant Routine (CR) protocol: subjects are required to remain awake for more than 24 hours in a semi-recumbent position, with strict control over light, temperature, posture, and food intake. Cognitive performance is monitored every 1–2 hours (Duffy & Dijk, 2002), which effectively mitigates external interference. (3) Forced Desynchrony (FD) protocol: by artificially setting non-24-hour (e.g., 20 or 28 hours) sleep-wake cycles, Process S and Process C are decoupled, allowing for the precise assessment of endogenous Process C effects (Meyer et al., 2024). In comparison, both CR and FD paradigms enable continuous recording for over 24 hours and effectively control for confounding variables. They offer superior advantages in separating Process S from Process C and are therefore widely used to investigate narrow circadian effects (Blatter & Cajochen, 2007; Borbély, 2022).

2.2 昼夜节律的生理机制

Distinguishing between circadian rhythm effects driven by different intrinsic mechanisms relies on a systematic understanding of their underlying physiological processes. Early research focused primarily on the isolated roles of molecular clocks or the central clock (Chen et al., 2022; Patke et al., 2020), often overlooking the fact that the formation and maintenance of circadian rhythms depend on a multi-level clock network spanning from the cellular to the systemic level. At the core of this system lies the functional coupling between the molecular clock, the central clock, and the endocrine system. These components work in synergy to maintain endogenous rhythms and achieve synchronization between the organism and its external environment. Understanding the hierarchical regulation and hormonal output functions of the Suprachiasmatic Nucleus (SCN) as the central pacemaker is a prerequisite for analyzing the differences between generalized and narrow-sense rhythmic effects, as well as for elucidating the neural basis of specific fluctuations in cognitive function.

2.2.1 分子时钟

The molecular clock serves as the cell-autonomous foundation of circadian rhythms. Its core mechanism consists of a transcriptional-translational feedback loop (TTFL), which is present in nearly all cell types (Chen Jianghui et al., 2022; Patke et al., 2020). The core of this loop is composed of essential clock genes and their protein products: the CLOCK and BMAL1 proteins form

heterodimers that bind to the promoter regions of the *Period* (*Per1/2*) and *Cryptochrome* (*Cry1/2*) genes, thereby driving their transcription.

Subsequently, PER and CRY proteins accumulate in the cytoplasm, undergo phosphorylation, and translocate into the nucleus. Once inside the nucleus, they inhibit the transcriptional activity of the CLOCK-BMAL1 complex, effectively suppressing their own expression. As PER and CRY proteins gradually degrade, this inhibition is lifted, allowing a new round of the transcriptional cycle to restart. This process generates an endogenous oscillation of approximately 24 hours (Albrecht, 2023; Honma, 2018). The molecular clock provides the fundamental cellular-level mechanism for various physiological and behavioral rhythms in the organism, as illustrated in [Figure 1A: see original paper].

2.2.2 中央时钟与外周时钟系统

The mammalian circadian rhythm system is organized in a hierarchical structure. The suprachiasmatic nucleus (SCN) serves as the central pacemaker, composed of a population of highly synchronized neurons that maintain rhythmic firing and gene expression with a period of approximately 24 hours, even in the absence of external inputs (Hastings et al., 2023; Patton et al., 2020). The SCN primarily receives environmental temporal information through three pathways: (1) the retinohypothalamic tract (RHT), which directly transmits light intensity signals; (2) the intergeniculate leaflet (IGL), which conveys non-visual light signals; and

(3) the raphe nuclei-SCN pathway, which transmits non-photic signals such as sleep/wake states (Berson et al., 2002; Starnes & Jones, 2023). These inputs drive the synchronization of the SCN with the environment. Once synchronized, the SCN coordinates systemic rhythmic activities through neural projections and endocrine signaling (Jones et al., 2021; Xie & Chen, 2024). Its neural outputs primarily project to key nuclei and regions within the hypothalamus, including the paraventricular nucleus (PVN), the dorsomedial nucleus (DMH), and the subparaventricular zone (sPVZ). These projections, in turn, regulate sleep-wake transition nodes, such as the sleep-promoting ventrolateral preoptic area (VLPO), as well as the arousal-maintaining lateral hypothalamus (LH) and locus coeruleus (LC) (Cajochen & Schmidt, 2025; Starnes & Jones, 2023; see [Figure 1B: see original paper]). This neural modulation of the arousal/sleep switch forms the basis for circadian influence on global brain excitability and likely represents the neurophysiological mechanism underlying the ubiquitous circadian effects observed across most cognitive functions.

Simultaneously, the SCN is responsible for coordinating peripheral clocks located throughout various tissues and organs (such as those in the liver, kidneys, and heart) to maintain overall physiological rhythm synchronization (Xu et al., 2023; Bautista et al., 2025). Peripheral clocks are not only regulated by the SCN but can also be reset by local factors such as feeding schedules. When phase misalignment occurs between peripheral clocks and the SCN rhythm, it can lead to metabolic disorders and cognitive impairment.

2.2.3 内分泌系统

The suprachiasmatic nucleus (SCN) transmits temporal signals throughout the body by regulating the rhythmic release of endocrine hormones. Melatonin and cortisol serve as two critical rhythmic output signals in this process. On one hand, the SCN projects to the paraventricular nucleus (PVN) via polysynaptic pathways, thereby inhibiting the activity of the pineal gland (PIN) [?, ?, ?]. During the night, as the inhibitory effect of the SCN is lifted, the pineal gland begins to synthesize and secrete large quantities of melatonin. Melatonin levels typically begin to rise in the early evening (18:00–20:00), reach their peak in the early morning hours (02:00–04:00), and subsequently decline, forming a characteristic "low by day, high by night" pattern [?, ?, ?]. The timing of the initiation of melatonin secretion, known as dim light melatonin onset (DLMO), is considered the "gold standard" for determining the phase of the endogenous biological clock [?, ?]. Furthermore, melatonin can provide feedback by acting on receptors within the SCN, participating in the regulation of endogenous rhythmic phases.

On the other hand, the SCN also drives the rhythmic secretion of cortisol \cite{Dong et al., 2014; O' Byrne et al., 2021}. Approximately 1-2 hours before awakening in the morning, the SCN sends signals to activate the hypothalamic-pituitary-adrenal (HPA) axis. This stimulation causes cortisol levels to rise sharply upon awakening, reaching a peak 30–45 minutes after waking—a phenomenon known as the cortisol awakening response (CAR).

response, CAR) [**?**, **?**]. At night, the SCN inhibits HPA axis activity, causing cortisol levels to reach their nadir before bedtime \cite{O' Byrne et al., 2021}, as shown in [Figure 1C: see original paper]. These hormonal rhythms serve not only as biomarkers for the phase of endogenous rhythms but may also exert differential effects on various neural circuits by acting on receptors widely distributed throughout the brain. Consequently, they constitute a potential hormonal mechanism underlying the specific rhythms observed in cognitive functions.

In summary, the circadian rhythm system is a multi-level regulatory network encompassing molecular clocks, a central clock, peripheral clocks, and the endocrine system. As the core pacemaker, the suprachiasmatic nucleus (SCN) integrates environmental zeitgebers—primarily light—and coordinates peripheral clocks and systemic physiological functions through two primary pathways: neural projections and hormone secretion. On one hand, the SCN directly regulates subcortical arousal systems to influence global brain excitability. On the other hand, the SCN modulates the rhythmic secretion of hormones such as melatonin and cortisol, transmitting temporal signals via humoral pathways to diverse brain regions [Figure 1D: see original paper]. This mechanism suggests that the impact of circadian rhythms on cognitive function likely involves both generalized arousal regulation and function-specific modulation dependent on particular neural circuits. The following section will discuss these effects from a behavioral perspective.

To examine whether different cognitive functions exhibit consistent or differentiated rhythmic patterns at various levels, and to further explore their underlying neural foundations.

3．昼夜节律对认知功能的影响

Based on the aforementioned multi-level regulatory mechanisms of circadian rhythms, a core question arises: do different behavioral and cognitive functions exhibit consistent circadian patterns, or do they manifest function-specific differences? To address this question, this paper systematically reviews the impact of circadian rhythms on the behavioral performance of various cognitive functions from the perspectives of both broad and narrow circadian rhythms.

To systematically investigate the influence of circadian rhythms on cognitive functions and the temporal distribution of their peaks and troughs throughout the day, this study conducted a systematic literature search. The search for Chinese literature covered the China National Knowledge Infrastructure (CNKI), VIP, and Wanfang databases, using keywords or

摘要

The search terms included “sleep deprivation,” “sleep restriction,” “circadian rhythm,” “time-of-day effects,” or “diurnal rhythm,” combined with “cognition.” English literature databases, including Web of Science, PubMed, and PsycINFO, were searched using the following Boolean strategy: (sleep restriction OR sleep manipulation OR sleep deprivation OR circadian OR “time of day” OR “diurnal rhythm”) AND (cognitive OR sleepiness OR fatigue OR attention OR memory OR executive OR “decision making” OR risky OR morality OR ethical). Inclusion criteria were defined as follows: (1) cognitive performance must have been measured at least twice within a single day (at minimum once in the morning and once in the afternoon), with the clock time corresponding to the peak or trough of cognitive performance either explicitly provided or inferable from charts; (2) the study must cover at least one of the following cognitive functions: subjective sleepiness (assessed via the Karolinska Sleepiness Scale, KSS), vigilant attention (measured by the Psychomotor Vigilance Task, PVT), complex executive function (evaluated by the Digit Symbol Substitution Test, DSST), working memory, risky decision-making, or moral behavior (note: due to the limited number of studies for the latter three categories, task paradigms were not restricted).

A total of 73 studies were ultimately included, yielding 109 effect sizes. Figure 2 [Figure 2: see original paper] utilizes violin plots to illustrate the distribution of peak and trough times across various cognitive functions, along with the number of effect sizes (K). To ensure task consistency, working memory results were restricted to those based on the N-Back task. To maintain consistency regarding participant chronotypes, effect sizes from subjects with extreme chronotypes are not displayed in the figure, although they have been incorporated into the overall analysis.

3.1.1 主观困倦

Extensive research consistently demonstrates that the generalized circadian rhythm exerts a significant regulatory effect on subjective sleepiness. During the first approximately 16 hours after awakening, the arousal-promoting effect of Process C partially offsets the gradually accumulating sleep pressure, maintaining subjective sleepiness at a relatively low level [?, ?, ?]. Following a night of total sleep deprivation, subjective sleepiness typically peaks in the early morning between 06:00 and 08:00 due to the weakening of the Process C arousal signal and the continuous accumulation of Process S. Subsequently, as the circadian arousal signal reactivates, sleepiness levels tend to alleviate around 18:00 to 20:00 in the evening [?, ?, ?, ?, ?, ?, ?]. For instance, using a Constant Routine (CR) paradigm with measurements every four hours, Lo et al. [?] found that in both sleep-restricted and control groups, subjective sleepiness peaked around 08:00 after a night of sleep deprivation and reached a trough around 20:00. Santhi et al. [?]

further investigated gender differences using a Forced Desynchrony (FD) paradigm. Their results showed that both men and women experienced peak sleepiness around 06:00 and recovery around 18:00, with no gender-specific effects observed.

This recovery phenomenon typically occurs during the “wake maintenance zone” (WMZ), which is the 2–3 hour period prior to the onset of evening melatonin secretion [?, ?, ?, ?]. At this time, despite high sleep pressure, the circadian system maximally promotes arousal [?, ?, ?], which manifests behaviorally as a significant decrease in subjective sleepiness. Monitoring 23 participants during 40 hours of sleep deprivation under a CR paradigm, McMahon et al. [?, ?] found that sleepiness increased significantly in the early morning but was markedly relieved during the WMZ. This pattern has been replicated in larger samples [?, ?] and specifically within male populations [?, ?].

The K values labeled below represent the number of independent effect sizes included within each cognitive function category. Peak times correspond to the optimal cognitive state: lowest sleepiness, highest attention and executive function, lowest risk preference, and highest moral standards; conversely, troughs correspond to the poorest states. Panel A: Results from all studies after excluding data from extreme chronotypes, reflecting both generalized and narrow circadian rhythm effects. Panel B: Studies meeting the criteria for Panel A with a measurement duration of ≥ 24 hours covering the WMZ, reflecting generalized circadian rhythm effects with complete temporal coverage. Panel C:

Narrow circadian rhythm effects that meet the screening criteria for Panel A.

3.1.2 警觉性注意

Existing results consistently demonstrate that generalized circadian rhythms exert a significant influence on vigilant attention. Following a full night of sleep deprivation, an individual’ s level of vigilant attention exhibits regular diurnal fluctuations, typically dropping to its lowest point in the early morning and recovering significantly around the evening (Graw et al., 2004; McMahon et al., 2021). For instance, using a Constant Routine (CR) paradigm, Graw et al. (2004) measured performance on a vigilant attention task every 225 minutes during 40 hours of sustained wakefulness; their results showed that alertness was lowest at 08:00 and recovered significantly by 22:00. Similarly, Wright et al. (2002), employing a Forced Desynchrony (FD) paradigm with measurements every 4 hours, found that vigilant attention reached a trough at 06:00 and rose again around 18:00.

Subsequent studies have further examined factors such as gender, age, and measurement methods, yielding consistently similar results. Using an FD paradigm, Santhi et al. (2016) found that for both men and women, vigilant attention was lowest around 06:00 and recovered by approximately 18:00. Adam et al. (2006) compared changes in alertness between younger men (mean age 25.2 years) and older men (mean age 66.4 years) during 40 hours of sleep deprivation, finding that both groups reached their lowest point at 08:00 and recovered around 23:00. Blatter et al. (2006) confirmed this same pattern when comparing a younger group (20–31 years) with an older group (57–74 years) within the same timeframe.

In a 62-hour sustained wakefulness experiment, Reifman et al. (2019) conducted assessments using both computer-based and mobile devices; they found that regardless of the measurement tool, alertness was lowest around 08:00 and recovered by approximately 20:00. These studies indicate that vigilant attention undergoes a recovery phenomenon during the Wake Maintenance Zone (WMZ) (McMahon et al., 2018, 2021; Zeeuw et al., 2018). Furthermore, the influence of generalized circadian rhythms on vigilant attention is highly stable and consistent, remaining unaffected by age, gender, or the specific measurement instruments employed.

3.1.3 工作记忆

Most studies have identified significant circadian rhythm effects on working memory performance, typically characterized by poorer performance in the early morning followed by a gradual improvement from the afternoon to the evening [?, ?, ?]. For instance, Lo et al. [?] utilized an N-back task within a Constant Routine (CR) paradigm, assessing working memory every four hours. Their results demonstrated that, regardless of whether participants were in the sleep-restricted or control group, working memory levels reached a trough around 08:00 and recovered to a peak around 20:00. Similarly, Santhi et al. [?] employed the same task within a Forced Desynchrony (FD) paradigm and found that working memory performance across genders was poorest around 06:00 and optimal around 22:00.

In a 40-hour sleep deprivation study using the CR paradigm, McMahon et al. [?] and Zeeuw et al. [?] also employed the N-back task; their results consistently showed that working memory performance was at its lowest between 05:00 and 08:00, with significant improvements observed between 15:00 and 22:00. Beyond the N-back task, other measures of working memory support a similar pattern of circadian variation. For example, phonological and visuospatial working memory tasks [?, ?], verbal and logical reasoning tasks [?, ?, ?], memory search tasks [?, ?], dual-task performance [?, ?], and the four-box task [?, ?] have all found that working memory is poorer from morning to noon (05:00–11:00) and superior from afternoon to evening (14:00–23:00). However, some studies have failed to observe significant circadian effects. For instance, Dourte et al. [?] found no regular fluctuations in an object-location binding task.

Similarly, Rey-Mermet and Rothen [?] observed no circadian rhythm effects in numerical/spatial complex span and updating tasks. Some studies have even reported opposing fluctuation patterns. For example, research using the N-back task found that working memory was at its worst between 19:00 and 21:00, while performance peaked between 09:00 and 11:00 [?, ?, ?]. These inconsistent findings may stem from methodological differences. Specifically, some studies may have failed to effectively isolate endogenous circadian components due to an insufficient number of daily measurements, incomplete coverage of time periods, or the exclusion of critical windows such as the Wake Maintenance Zone (WMZ). Consequently, their results may primarily reflect the cumulative effects of homeostatic sleep pressure rather than circadian rhythms.

3.1.4 复杂执行功能

Existing research has demonstrated that generalized circadian rhythms exert a significant regulatory effect on complex executive functions. Specifically, after undergoing a full night of sleep deprivation, an individual' s complex executive performance typically reaches its lowest point in the early morning, followed by a marked recovery during the evening hours [?, ?].

This rhythmic pattern has been validated across a variety of experimental paradigms. For example, Goel et al. (2013) and Tucker et al. (2018) observed this phenomenon within the constant routine (CR) paradigm.

Continuous wakefulness experiments lasting 51 hours and 40 hours, respectively, were conducted under these conditions. The results revealed that complex executive functions reached their lowest point between 08:00 and 11:00, followed by a recovery to peak levels between 22:00 and 23:00. Similarly, a study by Wright et al. (2002) utilizing the Forced Desynchrony (FD) paradigm reported comparable findings: performance was at its worst around 10:00 and showed significant recovery around 22:00. These findings suggest that the period of performance recovery may be concentrated near the Wake Maintenance Zone (WMZ).

To directly investigate the recovery effect of the Wake Maintenance Zone (WMZ), Shekleton et al. (2013) employed a Constant Routine (CR) paradigm under conditions of 50-hour sleep deprivation. By measuring individual melatonin levels to precisely identify the WMZ period, they found that performance on complex executive functions was at its lowest at 08:00, yet showed significant recovery during the 20:30 period corresponding to the WMZ. Similarly, Spaeth et al. (2015) conducted an 88-hour continuous wakefulness experiment where participants were randomly assigned to either total sleep deprivation (with placebo or caffeine intervention) or partial sleep deprivation (regular naps combined with placebo or caffeine). Their results indicated that, regardless of the intervention method, complex executive functions followed a stable pattern of morning decline and evening recovery, demonstrating the robust nature of this rhythmic effect. Furthermore, Honn et al. (2020) utilized 12 variants of the Digit Symbol Substitution Test (DSST) and found a consistent pattern among 37 participants: performance reached its nadir at 11:00 and peaked at 19:30 across all task variations. Taken together, these studies suggest that the regulation of complex executive functions by generalized circadian rhythms follows a robust "morning low, evening high" pattern. The evening recovery effect aligns with the WMZ period, and this rhythmic characteristic exhibits high consistency across different experimental paradigms, intervention conditions, and task variants.

3.1.5 风险决策

Although it has been established that most cognitive functions are regulated by circadian rhythms, the specific impact of circadian rhythms and sleep deprivation on risky decision-making remains unclear [?]. In recent years, researchers have increasingly focused on this area and accumulated preliminary evidence. Most existing studies suggest that generalized circadian rhythms significantly influence an individual's risk preference, typically manifesting in a pattern where risk-taking is lower in the morning and gradually increases during the afternoon and evening [?, ?, ?, ?]. For instance, [?] utilized the Balloon Analogue Risk Task (BART) to compare decision-making behavior at 09:00 and 15:00, finding that individuals exhibited a significant increase in risk-seeking behavior in the afternoon; notably, they maintained a high risk preference even after receiving negative feedback (such as a balloon explosion). Similarly, [?] tested the BART performance of 50 men at three time points—10:00, 14:00, and 19:00—and observed a pattern where risk preference peaked in the afternoon and was lowest in the morning. Using a Constant Routine (CR) paradigm involving 38 hours of continuous wakefulness, [?] conducted BART assessments every two hours and found that risk preference peaked at 13:30 and reached its nadir at 03:45. Furthermore, [?] compared risky decision-making performance at 07:30 and 22:00, also finding higher risk preference in the evening.

However, some studies have failed to observe significant rhythmic effects. For example, [?] randomly assigned participants to complete the BART task in either the morning (07:30-09:00) or the evening (16:30-22:00) while simultaneously measuring chronotypes, but found no significant "Time × Chronotype" interaction effect. This discrepancy with the majority of research findings may stem from methodological limitations: the broad measurement windows (particularly the evening window, which spanned over five hours) and the between-subjects design make it difficult to effectively capture continuous changes in endogenous rhythms. Instead, such designs may introduce confounding variables related to situational factors or individual differences, ultimately weakening the observable circadian rhythm effects.

3.1.6 道德行为

Regarding the influence of generalized circadian rhythms on moral behavior, existing research findings exhibit significant divergence. Some strictly controlled laboratory studies have found that moral behavior may follow a "morning high, evening low" pattern, suggesting that individuals are more inclined toward moral actions in the morning, while unethical behavior increases in the afternoon or evening. For instance, Kouchaki and Smith (2014) conducted four experiments and found that

unethical behavior was significantly more prevalent in the afternoon than in the morning. Similarly, Ingram et al. (2016) observed less dishonest behavior during morning sessions of a matrix reasoning task. Gunia et al. (2014) also found that morning-type individuals were more likely to exhibit honest behavior in the morning, while Mozgai et al. (2017) reported a higher incidence of moral behavior during morning human-computer interactions. Furthermore, Cornwell et al. (2021) randomly assigned participants to complete a general knowledge test in either the morning or the evening and found that cheating rates increased significantly at night.

However, evidence from large-sample studies and research with higher ecological validity has failed to observe a stable circadian effect on moral behavior. For example, Arechar et al. (2017) conducted an online random number guessing task with 2,336 participants and found no differences in self-reported accuracy between daytime and nighttime. Several real-world studies—investigating whether grocery stores overcharge customers (Vranka et al., 2019), whether restaurant customers return excess change (Azar et al., 2013), whether people evade fares (Bucciol et al., 2013), or whether doctors escort hospital transfers (Brøchner et al., 2020)—all reported no significant diurnal differences. Crucially, a recent meta-analysis (Jiang et al., 2023, $N = 7,161$) and a large-sample study (Ingram et al., 2016; Jiang et al., 2023; Zickfeld et al., 2025, $N = 1,006$) further support the conclusion that moral behavior lacks a significant circadian rhythm effect. These inconsistent results stem primarily from methodological differences: varying sensitivity of measurement tools to dishonest behavior (where high-sensitivity tasks detect dishonesty directly while low-sensitivity tasks are prone to random interference); differences in causal inference power between experimental and correlational designs (experimental studies verify causality but lack ecological validity, while correlational studies offer high ecological validity but weaker inference); and inconsistent time windows across studies, where precise intervals capture rhythms while broad intervals introduce situational confounding.

The aforementioned studies indicate that subjective sleepiness, vigilant attention, and complex executive functions all exhibit a robust "morning low, evening high" pattern [Figure 2A: see original paper], suggesting that these functions may be influenced by general regulatory mechanisms related to overall arousal levels. In contrast, research findings for working memory and risky decision-making show high heterogeneity across studies, while moral behavior fails to exhibit a stable rhythmic pattern [Figure 2B: see original paper]. These discrepancies may stem from methodological factors, such as insufficient measurement duration or incomplete coverage of time points, but they also suggest that the neural circuits underlying different cognitive functions may possess functional specificity in their response to rhythmic regulation.

3.2 狭义昼夜节律对认知表现的影响

When investigating narrow circadian rhythm effects, it is necessary to isolate the influence of Process C (the circadian process) from Process S (the homeostatic sleep pressure process). Because “time of day” and “duration of wakefulness” are highly coupled under natural conditions, researchers typically employ the dual-process model and its derivatives to achieve post-hoc separation during analysis.

This methodological approach allows for the isolation of cognitive fluctuations driven purely by Process C, thereby revealing the intrinsic rhythmic patterns of human performance independent of accumulated sleep debt.

3.2.1 主观困倦

Existing evidence consistently demonstrates that the circadian rhythm, in its narrow sense, exerts a significant regulatory effect on subjective sleepiness. By modeling data from 8 male participants and 1,114 shift workers, Reifman and Gander (2004) found that subjective sleepiness driven by Process C reached its nadir during the later periods of the day (specifically at 20:48 and 16:48, respectively).

Åkerstedt and Folkard (1995) conducted an analysis of

data from 15 truck drivers during a 500-kilometer journey, revealing that subjective sleepiness peaked in the early morning at approximately 06:00 and reached its lowest point around 17:00 in the afternoon. Muck et al. (2022) measured subjective sleepiness using a 24-hour constant routine (CR) paradigm after participants underwent simulated day or night shifts. Their results indicated that, regardless of the shift condition, subjective sleepiness peaked between 07:00 and 08:00 and reached its nadir between 18:00 and 19:00.

3.2.2 警觉性注意

As the two-process model continues to evolve and receive validation across various application domains, researchers—having clarified the rhythmic characteristics of subjective sleepiness—have further investigated the impact of the circadian rhythm (in its narrow sense) on vigilant attention. Empirical studies demonstrate that Process C exhibits endogenous oscillations of approximately 24 hours and significantly influences an individual’ s vigilant attention.

For example, Van Dongen et al. [?] measured vigilant attention using a Constant Routine (CR) paradigm involving 36 hours of continuous wakefulness; they found that Process C-driven alertness reached its nadir around 06:00 and peaked around 00:00. Similarly, research by Muck et al. [?] revealed that for participants in both simulated day-shift and night-shift conditions, Process C-driven vigilant attention performed worst between 07:00 and 08:00 and reached its optimal level between 18:00 and 19:00.

3.2.3 复杂执行功能

Compared to subjective sleepiness and vigilant attention, evidence regarding the impact of the circadian rhythm in its narrow sense on complex executive functions remains relatively limited. However, existing findings indicate that these functions are also subject to significant circadian regulation. Muck et al. (2022) found that, regardless of whether participants were in simulated day-shift or night-shift conditions, the circadian process (Process C) drove complex executive functions to a trough between 05:00 and 06:00, while peak performance was reached between 13:00 and 15:00.

The aforementioned studies demonstrate that even after controlling for homeostatic sleep pressure, subjective sleepiness, vigilant attention, and complex executive functions consistently exhibit a "morning low, evening high" rhythmic pattern [Figure 2C: see original paper]. This suggests that the circadian rhythm, in its narrow sense, may exert a general regulatory influence on cognitive functions by modulating shared arousal mechanisms. Simultaneously, the peak phases of different cognitive functions under this rhythmic pattern show significant differences. This indicates that the endogenous biological clock may provide specific temporal regulation of particular brain regions or networks through differentiated neural or endocrine pathways.

3.3 小结

Comprehensive behavioral evidence indicates that both broad and narrow circadian rhythm studies demonstrate a consistent "low in the morning, high in the evening" pattern across subjective sleepiness, vigilant attention, and complex executive functions. This suggests that the circadian system may exert a generalized regulatory influence over these functions. At the same time, the phase differences in peak performance across various cognitive functions, as well as the heterogeneity observed between different cognitive domains, reflect the functional specificity of circadian regulation. Together, these patterns suggest that circadian rhythms influence cognitive function through both a universal regulatory mechanism based on global arousal and differentiated regulatory mechanisms that depend on distinct neural circuits. The following sections will integrate neural evidence within this framework.

4. 昼夜节律影响认知功能的神经机制

To understand the phenomenon observed at the behavioral level—where different cognitive functions exhibit both rhythmic consistency and distinct pattern variations—an integration of existing neuroimaging and physiological evidence suggests that circadian rhythms may influence cognitive function through two primary mechanisms. First, they exert general regulation by modulating overall arousal levels; second, they provide task-specific regulation by modulating the activity of particular task-related brain regions or functional networks.

4.1.1 广义昼夜节律的影响

Studies utilizing functional magnetic resonance imaging (fMRI) suggest that activity in brain regions associated with arousal may exhibit rhythmic fluctuations [?, ?, ?]. The hypothalamus, thala-

mus, and locus coeruleus (LC) are critical components of the arousal system and play key roles in circadian regulation. The hypothalamus coordinates the transition between wakefulness and sleep through various neurotransmitter systems [?, ?]; the thalamus acts as an information relay station, regulating cortical synchrony and information flow [?, ?]; and the LC modulates cortical arousal and attentional states through the release of norepinephrine [?, ?]. The generalized circadian system regulates overall brain excitability through these regions, providing the necessary physiological foundation for various cognitive functions. Several empirical studies provide evidence for this mechanism. Schmidt et al. [?] used fMRI to scan 16 extreme morning types and 15 extreme evening types at 1.5 hours and 10.5 hours after waking. The results showed that evening types exhibited stronger activity in the anterior hypothalamic suprachiasmatic area (SCA) and the LC 10.5 hours after waking, which was accompanied by reduced subjective sleepiness. In a 40-hour constant wakefulness experiment, Maire et al. [?] further discovered that dynamic changes in thalamic activation were highly synchronized with fluctuations in subjective sleepiness. The maintenance of nocturnal alertness also depends on high levels of activity in the SCA and LC [?, ?, ?, ?]. Furthermore, Muto et al. [?] found that thalamic and hypothalamic activation exhibited circadian fluctuations during the performance of N-back tasks. Maire et al. [?] also observed that thalamic activation during the Psychomotor Vigilance Test (PVT) showed rhythmic fluctuations throughout the day, peaking between 23:00 and 07:00 the following day, and reaching a trough between 18:00 and 23:00 the next day. Using resting-state fMRI, Xing et al. [?] found that the fractional amplitude of low-frequency fluctuation (fALFF) and regional homogeneity (ReHo) in the thalamus and parts of the sensory cortex showed an upward trend during the 09:00–13:00 and 21:00–05:00 periods, which correlated positively with PVT performance. Regarding working memory, Muto et al. [?] found that both thalamic and hypothalamic activation exhibited circadian fluctuations during N-back tasks. Reichert et al. [?] further discovered that during the Wake Maintenance Zone (WMZ), hypothalamic activation was positively correlated with N-back performance and could predict performance levels following sleep deprivation.

Electroencephalogram (EEG) studies have shown that power in the θ band (4-8 Hz) exhibits circadian fluctuations, and its enhancement serves as a reliable physiological marker of decreased cortical arousal. Research has found that the increase in θ power during wakefulness is related not only to Process S but also closely linked to Process C, reaching its peak during the biological night [?, ?, ?, ?]. The enhancement of θ oscillations reflects strengthened synaptic connectivity resulting from prolonged wakefulness, making the thalamocortical network more prone to entering a highly synchronized state. The inefficient information processing in this state forms the electrophysiological basis for the decline in cognitive function during circadian troughs [?, ?]. Studies have also identified a transient recovery phenomenon during the WMZ: although θ oscillations generally

increase after prolonged wakefulness, EEG activity across different brain regions is no longer fully synchronized near the WMZ, and is accompanied by pupil dilation. This suggests that the brain can modulate its activity patterns within this time window to partially offset the fatigue and cognitive decline caused by continuous wakefulness [?, ?].

4.1.2 狭义昼夜节律的影响

After controlling for Process S, the circadian rhythm in its narrow sense also demonstrates a regulatory effect on the arousal system. For example, Muto et al. [?] utilized the two-process model to isolate circadian effects and found that activity in the thalamus and hypothalamus still exhibited significant circadian fluctuations during the performance of N-back tasks. This suggests that even when the influence of sleep pressure is excluded, the endogenous biological clock independently regulates the activity levels of these core arousal-related brain regions.

Synthesizing the available neural evidence, research into both broad and narrow circadian rhythms indicates that circadian processes regulate arousal-related structures—including the hypothalamus, thalamus, and locus coeruleus (LC)—as well as their ascending projections that drive cortical states. This regulation dynamically shapes the characteristic “circadian trough decline”and the transient fluctuations observed during the Wake Maintenance Zone (WMZ).

The recovery time window of these processes may exert a relatively non-specific and universal influence across multiple categories of cognition.

4.2.1 广义昼夜节律的影响

Studies using fMRI have demonstrated that, in addition to the global regulation of the arousal system, generalized circadian rhythms exert local and task-specific influences on brain regions associated with specific cognitive functions. Specifically, Maire et al. (2018) found that during the performance of the Psychomotor

Vigilance Task (PVT), activation in regions such as the postcentral gyrus, visual area V2, and the putamen exhibited circadian fluctuations, indicating that the regulation of alertness possesses significant regional specificity. Muto et al. (2016) observed that during the PVT, BOLD responses across nearly the entire brain—with the exception of the dorsolateral prefrontal cortex (dlPFC)—showed circadian variations. Furthermore, the peak phases differed across brain regions: the occipital lobe and limbic system reached their peaks earlier, while the temporal lobe and prefrontal cortex (PFC) exhibited later peaks.

Notably, the peak of neural activity in the prefrontal cortex occurs relatively late, whereas behavioral improvements in complex executive functions appear earlier, suggesting that the two are not perfectly aligned in their temporal sequence. This temporal discrepancy suggests that complex executive functions may rely more heavily on the specific regulation of particular brain regions like the PFC, while subjective sleepiness and basic alertness depend more on the general regulation of the arousal system. Because the activity of the arousal system peaks later, the behavioral improvement of functions dependent on this system lags behind; conversely, brain regions subject to specific regulation may enter a high-efficiency state earlier, allowing executive functions to show earlier behavioral improvement. This may result in a lack of perfect synchrony between the peak neural activity of relevant brain regions and behavioral performance. Xing et al. (2023) found that the fALFF/ReHo of key nodes in the default mode network (DMN) during the resting state showed a downward trend between 21:00 and 05:00 the following day, which was negatively correlated with PVT performance. Regarding working memory, Muto et al. (2016) found that during the N-back task, activity in the bilateral anterior insula fluctuated in sync with melatonin secretion, characterized by higher activation levels in the evening. Sherman et al. (2015), after monitoring sleep-wake patterns via actigraphy for 10 consecutive days, performed fMRI scans on healthy older adults during an associative memory task; the results showed that circadian stability was positively correlated with associative memory performance and hippocampal activation levels.

ERP studies indicate that the electrophysiological representations of high-level cognitive processing also exhibit rhythmic fluctuations. The P300, a classic ERP component reflecting attentional resource allocation and cognitive updating (Polich, 2007), shows significant circadian variation in its amplitude.

For example, An et al. (2009) found that applying short-wavelength blue light during the biological night significantly enhanced P300 amplitude, suggesting that exogenous rhythmic signals can modulate neural processes related to attention and cognitive updating. Huang et al. (2006) observed significant diurnal fluctuations in P300 amplitude during an oddball task. Notably, no stable linear relationship has been found between P300 amplitude and melatonin concentration, suggesting that its variations may more directly reflect the rhythmic changes in cognitive functions regulated by the suprachiasmatic nucleus (SCN) and may possess independent phase characteristics.

4.2.2 狭义昼夜节律的影响

Muto et al. (2016) found that neural activity in several subcortical regions—such as the midbrain, cerebellum, basal ganglia, and thalamus—as well as certain cortical areas, including the primary sensorimotor cortex, occipital pole, and intraparietal sulcus, is exclusively influenced by Process C. In contrast, regions such as the occipital pole and thalamus are simultaneously affected by both Process S and Process C. This suggests that the effects of these two processes are not merely additive, but rather exhibit a dynamic synergy within specific brain regions.

Synthesizing the available neurological evidence, generalized circadian rhythms exert differential temporal regulation on specific cortical networks and their information processing procedures, thereby selectively influencing the efficiency and peak phases of various cognitive functions. However, neural research focusing specifically on narrow-sense circadian rhythms remains limited. Only Muto et al. (2016) have attempted to use the dual-process model to explore the susceptibility of different brain regions to Process C, yet a systematic analysis of the rhythmic phase characteristics of their neural activity has not been conducted. Consequently, future research needs to further investigate the specific regulatory patterns and temporal characteristics of Process C across different brain networks under conditions where sleep homeostasis is strictly controlled.

4.3 小结

Evidence synthesized from both behavioral and neural levels reveals that arousal-related systems—including the thalamus, hypothalamus, and locus coeruleus—exhibit stable rhythmic fluctuations in activity across both broad and narrow circadian rhythm research paradigms. This suggests that circadian rhythms may exert a generalized, synchronous influence on a wide range of cognitive functions by modulating overall arousal levels. Such findings provide a neural-level explanation for the “morning low, evening high” behavioral patterns commonly observed in cognitive domains such as subjective sleepiness and vigilant attention.

Simultaneously, specific brain regions and functional networks responsible for distinct cognitive functions display differentiated rhythmic activity profiles. This indicates that circadian rhythms may also exert specific modulation through functionally distinct neural circuits. These mechanisms help explain the phase differences in peak behavioral performance across various cognitive functions, as well as the dissociation in their sensitivity to circadian signaling.

5. 未来研究方向

This paper systematically reviews the concepts, research paradigms, and physiological mechanisms of circadian rhythms. It compares the effects of generalized and specialized circadian rhythms on various cognitive functions and summarizes their general and specific neural mechanisms. This study highlights the multi-level specificity of circadian rhythm influences on cognitive function, providing theoretical support for time management and interventions in environments characterized by sleep deprivation, such as shift work and transmeridian occupations. However, several key questions remain regarding the neural implementation, causal mechanisms, and intervention feasibility of this dual-regulatory model. Future research should focus on three dimensions: mechanistic validation, causal establishment, and translational application.

5.1 采用多模态神经成像验证双重调控机制

Although behavioral research evidence supports the possibility that circadian rhythms regulate cognitive functions through both general and specific mechanisms, the corresponding neural mechanisms remain unclear.

Current research is primarily limited by two factors: first, there is a lack of direct neural evidence mapping these two regulatory modes to distinct patterns of brain activity; second, existing studies are mostly based on the linear assumptions of the dual-process model, failing to fully characterize the potential nonlinear interactions between Process S and Process C. Future research should utilize paradigms that strictly control for sleep homeostasis, integrating multimodal neuroimaging data with nonlinear computational models. Such efforts should aim to isolate neural signals associated with Process C and systematically examine their spatiotemporal synchrony and heterogeneity across different brain regions or functional networks. This approach will facilitate a neural-level verification of the co-existence of general and specific regulatory mechanisms.

5.2 发展基于认知节律特异性的光照干预

Existing research indicates that different cognitive functions exhibit specific periods of vulnerability and recovery throughout the circadian cycle (see [Figure 2: see original paper]), providing a theoretical foundation for implementing precision-timed light interventions targeting specific cognitive domains. Current light intervention studies have largely focused on adjusting overall rhythmic phases or improving broad cognitive functions [**?**, **?**, **?**].

For example, Rüger et al. (2006) found that exposure to bright light during the early morning trough following sleep deprivation significantly improved individual alert attention. Chellappa et al. (2011) further demonstrated that a single exposure to blue light during the biological night not only reduced subjective sleepiness but also enhanced the accuracy of working memory. However, existing research has not yet systematically examined whether implementing interventions during the rhythmic trough of a specific cognitive function can produce relatively specific behavioral improvements. Future research could utilize the rhythmic characteristics of different cognitive functions to apply standardized, parameter-adjustable light stimuli during theoretically predicted rhythmic troughs. By simultaneously evaluating behavioral performance and associated changes in neural activity, researchers can test whether cognitive rhythm specificity is plastic and determine its potential intervention value. This would represent not only a theoretical breakthrough but also provide a basis for individualized cognitive regulation in real-world scenarios such as shift work and trans-meridian travel.

5.3 利用靶向神经调控确立因果机制

Existing research has primarily utilized correlation analysis to identify several key brain regions associated with specific cognitive rhythmic fluctuations, such as the thalamus and the anterior insula [?, ?, ?]. However, it remains unclear whether the rhythmic activity in these regions is the cause, a concomitant phenomenon, or a consequence of cognitive rhythmic fluctuations. Traditional neuromodulation techniques struggle to effectively target these deep subcortical structures [?, ?, ?], making it difficult to establish a definitive causal link between circadian rhythms and cognitive functions. Consequently, future research must leverage novel non-invasive neuromodulation methods capable of overcoming traditional technical bottlenecks to target deep brain regions.

Emerging temporal interference (TI) electrical stimulation provides a potential solution to this challenge [?, ?]. TI achieves non-invasive and precise stimulation of deep brain regions through the difference-frequency interference of two high-frequency electrical fields. This method offers both high safety and superior penetration, making it suitable for modulating key circadian rhythm nuclei such as the suprachiasmatic nucleus (SCN) and the thalamus [?, ?]. Future studies on causal mechanisms could build upon the cognitive rhythm-related brain regions identified in previous reviews, implementing neural interventions at specific targets corresponding to different cognitive functions. By testing whether these interventions can specifically alter expected cognitive rhythm patterns, researchers can establish causal associations between specific brain region activities and particular cognitive rhythms at a mechanistic level.

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The Circadian Rhythm and Its Specific Regulation of Cognitive Function YAN Kaikai1, GUO Bowen2, CHEN Xing1, MAO Tianxin1*, RAO Hengyi1,2* (1 School of Business and Management, Shanghai International Studies University; Shanghai Key Laboratory of Brain-Machine Intelligence for Information Behavior; Shanghai International Studies University Magnetic Resonance Imaging Research Center, Shanghai, 201600; 2 School of Humanities and Social Sciences, University of Science and Technology of China, Hefei, 230026)

Abstract

The circadian rhythm is an endogenous timing system with an approximately 24-hour period that regulates various cognitive functions. During the trough of the rhythm, cognitive performance typically declines, while it recovers during the peak, with distinct temporal patterns observed across different functions. Most existing studies have not distinguished the underlying driving mechanisms, making it difficult to systematically explain the specific regulatory mechanisms of different cognitive functions, and there is a lack of relevant neural evidence. This review aims to disentangle circadian effects driven by distinct mechanisms, explore their differential impacts on cognitive functions, and interpret this specificity from a neural mechanism perspective. Future research should integrate multimodal neural data with computational models, combine targeted neuromodulation techniques to establish causal relationships, and explore precise light-based intervention strategies based on specific time windows, thereby providing precise cognitive protection for individuals with circadian rhythm disorders.

Keywords

circadian rhythm, two-process model of sleep-wake regulation, cognitive function

Note: Figure translations are in progress. See original paper for figures.