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Biphasic sleep and human health: A theoretical paradigm for personalized sleep

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ABSTRACT

As our understanding of sleep evolves, a recurring recommendation remains that adults should sleep in a single, uninterrupted nocturnal bout – commonly referred to as monophasic sleep. However, the scientific foundation for this standard may be less canonical than previously assumed, particularly when considering individual variability in sleep needs and organization. Historical texts and recent research suggest an alternative view: that sleep can be naturally segmented into two periods across the 24h cycle – a pattern known as biphasic sleep. This review explores the physiological implications of biphasic sleep, examining its potential benefits and limitations under healthy conditions and in particular contexts, such as shift working. With this available data, an original hypothesis is proposed, consisting of a novel framework of sleep distribution according to sleeping types. This aims to stimulate new research about biphasic schedules and how sleep architecture might adapt to them over time. The strengths and weaknesses of existing research will be critically assessed, culminating in recommendations for future investigations and methodological approaches. These insights may ultimately inform more personalized and flexible sleep guidelines better aligned with individual and societal needs.

1. Introduction

It has been postulated and accepted that 7h–9h of sleep in one single session should be the norm for every healthy young adult [1]. However, even the same individual can see themself challenging this dogmatic monophasic sleep pattern throughout their life, as babies have polyphasic sleep, which develops into a biphasic sleep pattern in children, and later into a monophasic sleep in adults [2]. Interestingly, later in life, sleep patterns often change back into a biphasic schedule in retired people [3]. This begs the question of whether monophasic sleep is the true default mode for humans or simply a product of our current social organization. More importantly, we should question whether these different sleep schedules might benefit or harm our mental and physical health.

Despite the widely accepted view of sleep as a monophasic process, growing evidence from the field of somnology suggests that this model may not represent the full spectrum of human sleep variability. This is the first literature review to integrate historical and sociological perspectives with physiological mechanisms, and health implications of biphasic sleep. By synthesizing current research and proposing an original hypothesis, this work aims to lay the groundwork for future experimental studies and promote a more personalized approach to sleep, offering a critical reevaluation of biphasic sleep's role in human biology.

1.1. Background on sleep propensity and architecture

The currently leading model for why and when we sleep is called the "2-process model", which consists of the C-process and the S-process [4].

1.1.1. C-process (circadian rhythmicity)

One of the most important mechanisms our body uses to control sleep is the modulation of melatonin, which is low during the day and high during the night (Fig. 1), in both diurnal and nocturnal animals, indicating that it has no intrinsically sedative effect [5]. By the end of the day, the decrease in light reaching the eyes stimulates the brain to produce this hormone [5,6], which then regulates multiple systems of metabolism and consciousness [7].

This way, melatonin production marks the physiological response to darkness, also called the "biological night". To determine its beginning, researchers use the initial uptick in the early evening under low light, known as dim-light melatonin onset or DLMO [8,9]. This usually starts on average 2h before habitual sleep onset time in healthy people. The moment when sleepiness sets in, and from when constantly high levels of

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sleep propensity are observed, is called the 'sleep gate' [10]. Opposed to it, there is during the day the 'wake maintenance zone', when sleep propensity is at its lowest, at 1h–3h before habitual sleep time, terminating before the start of DLMO [11].

Conversely, cortisol is a hormone responsible for increasing arousal, energy, immune readiness, and metabolic activity during wake hours [12]. Particularly, upon waking, the Cortisol Awakening Response (CAR) consists of a sharp increase in cortisol levels within the first 30–45 min after waking from sleep. This is a distinct, rapid burst of cortisol secretion that is superimposed on the regular circadian rhythm of cortisol, which generally peaks in the early morning and declines throughout the day [13].

Circadian rhythms are entrained by other environmental or external time cues (called *Zeitgebers*) besides light. For example, meal timings can promote or hinder sleepiness [14] and lower temperatures can facilitate sleep induction [15]. Specific types of neurons are also tuned with the circadian rhythm, for example, orexinergic neurons play a critical role in regulating sleep and wakefulness by releasing the excitatory neuropeptides orexin-A and orexin-B that stimulate other arousal-related neurons to ensure consolidated alertness and arousal during wakefulness [16–18].

Another important influencing factor is the individual's chronotype, a reflection of the individual's phase of entrainment/synchronization to light (Fig. 1). This expression of circadian rhythms is dictated by individual (genetics and age) and environmental (light) factors [19]. There are two extremes in the gradient of chronotypes: late-type (or owls) and early-type (or larks) subjects. Late-type persons were recorded to have a delayed phase of temperature regulation compared to the early-type, subjective sleepiness is also reported later in the day, with a slower increase in sleep pressure – i.e., sleepiness arises later in late-type and takes longer to reach the same level as in the early-type individuals [20,21]. Accordingly, melatonin onset was recorded to increase up to 2.5h later in late-type individuals [22], which was even reported to be aggravated during daylight saving time, explaining the observed inability of patients with delayed sleep-wake phase disorder to adapt to the new advanced schedule [23].

1.1.2. S-process (homeostatic sleep pressure)

Another factor in this sleep induction model is the physiological buildup throughout the day of the somnogenic neurotransmitter adenosine and its dissipation during sleep, being this one of the factors of increased sleep pressure throughout the day (Fig. 1). This process indicates to the body the length of sleep-wake periods [24–26], gauging the amount of previous sleep and thus of current sleep deprivation [27]. This endogenous molecule functions as a hypnogenic neuromodulator when its extracellular concentration is increased in the cerebrospinal fluid [25,28]. Caffeine is able to restrain its effect by blocking adenosine receptors from activating the sleep-inducing cascade. However, these adenosine receptor antagonists do not truly reduce sleep pressure, as adenosine keeps building up, resulting in an intensified effect afterward (e.g. the so-called "caffeine crash") [25,26].

1.1.3. Sleep stages and cycles

Upon sleep initiation, thalamus and cortex neurons synchronize their activity – creating a state of enclosure. This produces long-traveling waves with higher amplitude and lower frequency that can be observed in electroencephalogram (EEG) recordings. The characteristics of these waves change not only with the wakefulness state but also with the specificities of different sleep stages that occur throughout the night [5].

The time we spend asleep can be divided into two broad categories: Rapid Eye Movement sleep (REM sleep), and non-REM sleep [29] (Fig. 2). Non-REM sleep is the first to occur upon sleep initiation, and it is further subdivided into three sections distinguished by specific wave criteria in EEG recordings. These stages are labeled sequentially (N1, N2, and N3) by their decrease in measured wave frequency and manifestation of specific wave events (respectively, 4–12 Hz, 4–12 Hz with spindles and k-complex events, and 0.5–4Hz) [30,31], thus why N3 – with its characteristic Slow Wave Activity (SWA) – is also called Slow Wave Sleep (SWS), or Deep Sleep [32].

The functions attributed to sleep vary with its different stages. For example, the sleep spindles that occur during non-REM sleep have been linked to cognitive performance [33] and memory consolidation [34], specifically for declarative memories in the hippocampus [35]. In contrast to this, non-hippocampal procedural memories (a type of non-declarative memory), are enhanced during REM sleep [35,36]. As for SWA, it increases in nights following sleep deprivation, hinting at its importance for proper recovery and dissipation of sleep pressure [37,38].

Upon waking, a characteristic grogginess follows, called "sleep inertia". For an average of 30 min (although it can range from 15 to 90 min) afterward, performance measurements are affected and there is an increased reporting of sleepiness and disorientation [39]. The mechanisms behind this event are still being investigated, but another process of sleepiness/alertness other than circadian rhythmicity (C-process) or sleep pressure (S-process) must exist to explain its manifestation – since

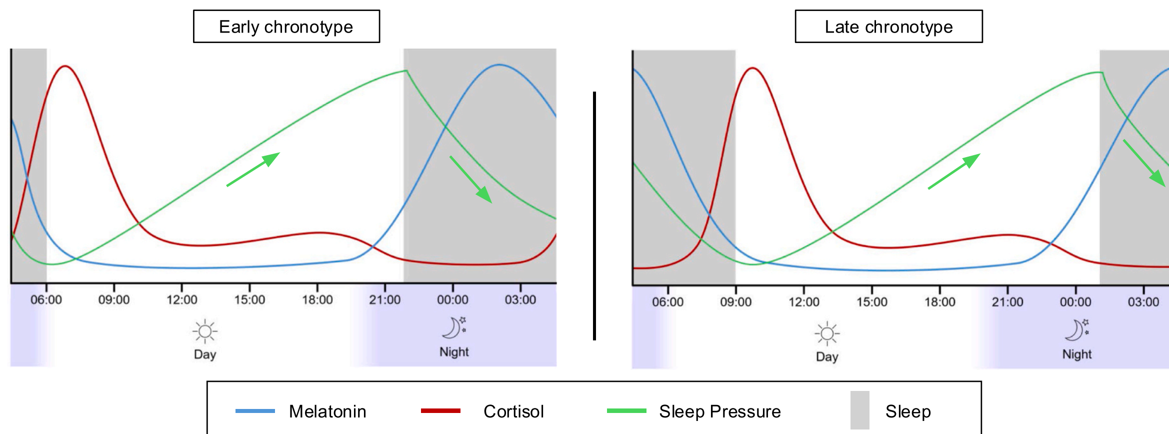

Fig. 1. Circadian and homeostatic regulation of sleep in early and late chronotypes. Schematic representation of the interplay between sleep (grey shadow) and the circadian rhythms of melatonin (blue line), cortisol (red line), and sleep pressure (green line) in early (lark) and late (owl) chronotypes. Green arrows illustrate the progressive buildup of sleep pressure. Chronotype-dependent shifts in circadian phase influence the timing of melatonin secretion and cortisol release, affecting preferred sleep onset and wake times. (For interpretation of the references to colour in this figure legend, the reader is referred to the Web version of this article.)

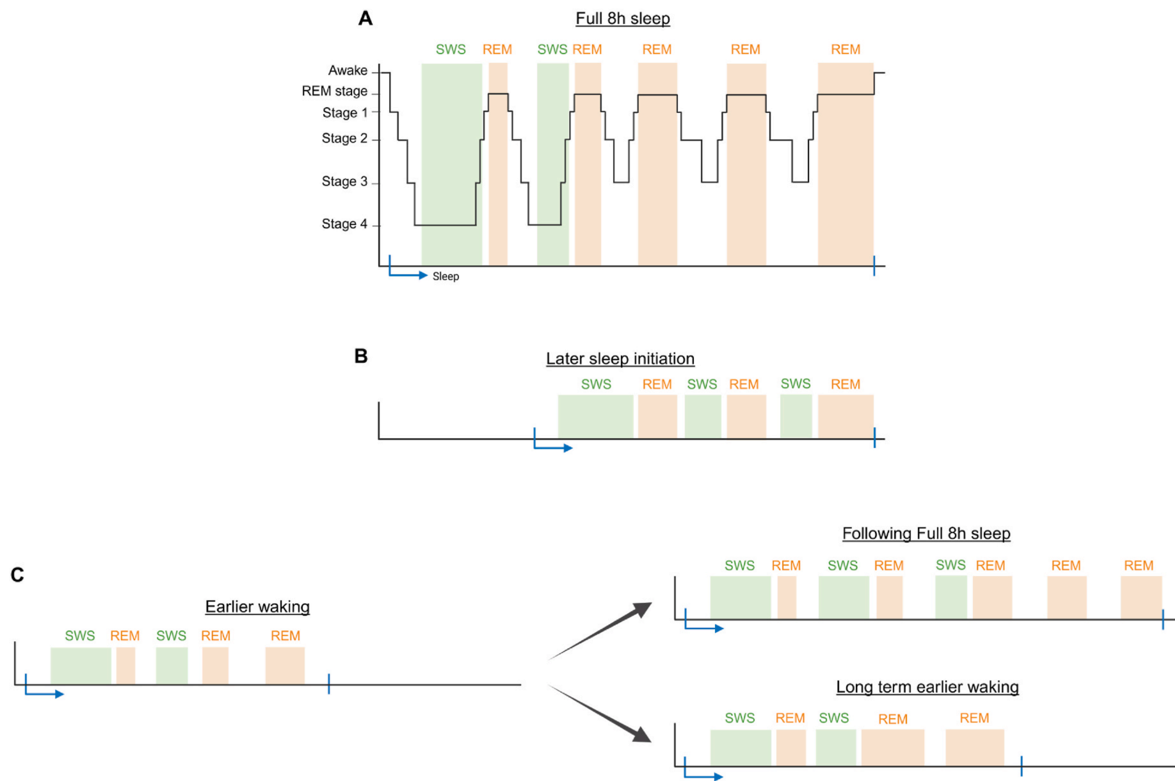

Fig. 2. – **Effects of sleep curtailment on sleep architecture and distribution.** A) Typical hypnogram of an 8-h monophasic sleep period showing the temporal distribution of non-REM and REM sleep stages. B) Delayed sleep onset tends to increase the proportion of slow-wave sleep (SWS) early in the night. C) Early awakening reduces REM sleep duration while preserving SWS. However, over successive nights, REM rebound occurs, with REM stages shifting to earlier in the night.

it happens even when subjects wake up at the correct time and after restoring sleep. However, sleep inertia is still influenced by these known processes, as it is also worsened after prior sleep loss and when the subjects are woken up during the biological night [39]. A recent publication links this event to the dynamics of the waking brain. EEG recordings revealed that awakening begins in frontal and central regions and spreads to the back, and that awakening during non-REM sleep shows slower transitions while REM sleep skips directly to higher frequency waves, characteristic of wakefulness. Furthermore, even among the transitional slow wave activity, certain types of waves improve alertness, while others worsen it, demonstrating the complexity of this process [40].

Sleep becomes even more complex when we realize that we do not go through each stage just once per night. Instead, stages 1–3 and REM repeat in cycles throughout the night, each lasting about 90 min in a healthy young adult [41]. Additionally, the ratio of REM/non-REM in each cycle also changes throughout the night (Fig. 2). REM sleep distribution depends on the phase position relative to the circadian clock (C-process) [42] as it increases across the sleep bout – lasting 1–10 min in the first cycle and up to 50 min in the last [2,43]. SWS is only observed in the first cycles [44], comprising most of each and declining throughout the night – alongside the decreasing sleep pressure (S-process) [37,38].

1.2. Sleep need

Sleep need is here defined as the amount of sleep an individual requires across a 24h period to maintain good vigilance, function optimally and sustain good health. Many factors influence the amount of time we need to sleep, and plenty of them are even variable day by day, such as the amount of previous wakefulness and its arousal intensity, or personal preference and social events.

The median length of the night throughout a year represents around 12h of the total 24h day. So, if circadian rhythms were to strictly induce sleep as darkness settles in, and wakefulness as the day dawns, humans would spend half of their lives sleeping, and although we acknowledge some seasonal variation in sleep – sleeping slightly more in the winter when the nights are longer [45,46] – humans still do not sleep the entire scotoperiod. Furthermore, with the current technology, humans bypass this day/night contrast by turning on the lights. Studies have shown that until the widespread use of artificial light, the elongation of the awake period was not as common, although these people still do not sleep through the entire night [47,48].

A third of the working adults polled in the U.S. reported sleeping on average less than 7h a night [49] – sleeping times that are often overestimated [50]. A study during the COVID-19 social restriction period showed the magnitude of sleep deficit due to pre-pandemic social time pressure [51]. This study showed that late-types are more prone to 'social jetlag' – the difference between social and biological clocks, or the difference between the midpoint of sleep on free days and on workdays [51,52]. Indeed, it has been shown that patients with sleep disorders and people with a late chronotype report better sleep quality on free days than on workdays, mediated by social jetlag [53]. This data fuels an ongoing discussion about whether the current societal organization and technology disallow proper sleep.

Intrinsic individual factors are also important to this discussion, however, although long- and short-sleepers have been identified in the literature [54], it is still unclear if they *need* to sleep more or less, or whether they simply *can*.

It has been proposed that short sleepers may avoid the effects of sleep deprivation due to a reduced need for REM sleep and less stage 2 sleep, thereby allowing them to shorten their total sleep time [55–57], as they tend to exhibit higher sleep efficiency and shorter sleep onset latency [54].

Conversely, while long sleepers do exist, habitual sleep durations exceeding 10 h are neither typical nor feasible for the general population

[58], and may even be associated with adverse outcomes [59]. Sleep extension to approximately 10 h has not been shown to produce significant improvements in alertness or cognitive performance among healthy individuals with normal baseline sleep [60,61]. Furthermore, evidence suggests that the restorative benefits of sleep extension follow an exponential saturation curve, wherein the greatest gains occur in the initial hours of sleep, with diminishing returns observed as sleep duration increases [62].

Extensive research has shown that sleep deprivation induces widespread neurobehavioral and physiological impairments [51,63–65]. Social constraints on sleep have further contributed to chronic sleep restriction in modern society [51–53,63].

Current evidence suggests that the commonly recommended 7–9 h of daily sleep [1] may be sufficient to optimize recovery while minimizing the adverse effects of sleep restriction [56,60,61,66,67]. However, these findings may also reflect factors such as the distribution of sleep phenotypes in the population and long-term social habituation to monophasic sleep patterns.

1.2.1. Shortened sleep and circadian misalignment

Aside from the effects of sleep deprivation, shortened sleep influences other aspects of the brain's circadian and homeostatic mechanisms, resulting in altered sleep stage distribution (Fig. 2).

When shortening sleep by initiating it later than usual (postponing it), we observe an increase in SWA [37,38,68] while REM sleep seems to remain largely unaltered, being the extra time allocated to SWS subtracted from stage 2 sleep [69–73].

Contrastingly, when waking up an individual before the completion of their normal sleep duration the amount of REM sleep is expected to be reduced – with no detriment to either non-REM sleep stage – given that the largest amount of REM sleep is observed in the later cycles of sleep [2,42]. However, after a few nights of sleep curtailment, REM sleep was reported to be redistributed to earlier in the night (still without reaching baseline levels), presumably as a way for the body to compensate for the heightened suppression of this stage [68,73].

Circadian misalignment refers to when the natural sleep-wake cycle (i.e., circadian rhythmicity of core body temperature, melatonin, and cortisol) is out of sync with the external environment, particularly the day-night cycle. In a society structured around early-rise monophasic sleep, individuals with different sleeping types may experience circadian misalignment. This asynchrony can be further aggravated by poorly timed biphasic sleep patterns [23,51,53], and has been linked to negative outcomes such as sleep deprivation and reduced sleep quality on workdays [51–53], disrupted feeding behaviors and metabolism [74], and mood disturbances, including depression [75–77].

1.3. What is biphasic sleep

Biphasic sleep refers to an arrangement that consists of portioning the sleep time into two separate segments called phases or bouts. The term Polyphasic Sleep further extends this idea by dividing sleep into more than two bouts. Both patterns have been reported to occur naturally in normal adults [78]. As we will discuss further, these sleep arrangements can be assembled into varying patterns, with possibly different outcomes for human health and life quality.

The following review examines these physiological principles and implications of biphasic sleep, with the goal of proposing a novel framework for personalized sleep distribution and concrete directions for future research, as well as potential clinical applications.

2. Biphasic sleep

2.1. Historical, cultural and evolutionary perspectives

Historian Roger Ekirch, drawing on ancient and medieval texts, identified a common biphasic sleep pattern in pre-industrial northern hemisphere cultures, where individuals would sleep shortly after sunset, awaken around midnight for various activities (e.g., eat, read, engage in sexual intercourse, sew, pray, or reflect and relax), and then return to sleep until dawn. This pattern, referred to as "*first*" and "*second*" *sleep*, reflects a culturally normative partitioning of rest [79].

Contradictorily, in a study of three pre-modern era hunter-gatherer/horticulturalist societies from Tanzania, Namibia, and Bolivia (all close to the equator), it was observed that they sleep continuously 6–7h per night. They initiate their sleep about 3 h after sunset and awake before sunrise, with the duration of sleep increasing by 1h from the summer to the winter season [46]. A possible explanation for the monophasic sleep observed in this study is, as concluded by the researchers, that this pattern was "probably not present before humans migrated into Western Europe" and higher latitudes.

However, as reported by Ekirch, segmented sleep was observed, not only during winter but throughout the entire year, suggesting that it was not simply a result of longer winter nights. Furthermore, biphasic sleep has been reported in other cultures from more equatorial regions as well – from South America, Africa, and Asia [80,81] and even the study by Yetish and colleagues reported that naps were observed in 22 % of summer days.

Napping represents another form of biphasic sleep observed across many cultures worldwide [82]. While it remains uncertain whether biphasic sleep represents the ancestral human norm, available evidence suggests that such patterns were far from uncommon across diverse historical and cultural contexts.

This variability of sleep patterns matches what we can observe in nature. Most animals – and specifically over 86 % of mammalian species – are biphasic or polyphasic sleepers [83]. The reason why animals today have such distinct sleep patterns is riddled with evolutionary gaps, divergent and convergent evolution, and overall exceptions. Despite these differences, all sleep patterns can be categorized based on three aspects: circadian placement, intermittency, and total sleep duration [84].

Circadian placement ties inactivity to the Earth's day/night cycle. Light, temperature shifts, food availability, and predator activity create exceptionally different environmental conditions to which species must adapt. This leads to non-adaptive selection, where continuous activity, during the day or night, is either inefficient or dangerous [84]. For humans, this inactive period aligns with nighttime, classifying us as diurnal animals.

Sleep intermittency varies widely across species and, according to some studies, may correlate with the predator-prey dynamics. Prey animals, unable to afford prolonged periods of unconsciousness, tend to exhibit polyphasic sleep patterns [83,84]. However, diverging evidence claims that sleeping patterns might be more related to energetic constraints instead, given that smaller mammals need to eat more frequently and thus show polyphasic sleep with shorter sleep cycles and longer total sleep durations [85,86]. Humans have a medium body weight among mammals and occupy a unique position in the predation dynamics, having evolved from prey species to, only recently in their evolutionary history, the planet's apex predator.

Both of these aspects might also influence total sleep duration, as smaller mammals typically have more total sleep [85,86] and preliminary evidence reports that species facing higher predation risk typically show less REM and non-REM sleep, reflecting limitations on total sleep time [86–88]. Similarly, although humans are no longer constrained by the need to forage or hunt, modern societal structures impose new limitations, often restricting sleep opportunities, perhaps even more so that in the past.

From these evolutionary clues, we might hypothesize about the three aspects of human's innate sleep pattern: that we are primitively diurnal, that our sleep pattern is either intermediately segmented (i.e., biphasic) or monophasic, and that we have a total sleep requirement neither excessively long nor short.

Chronotypes may have evolved in response to security needs, as

groups with staggered circadian rhythms would benefit from having individuals awake to stand guard throughout the night [89,90]. In modern societies, where work schedules favor early rising and monophasic sleep, there may be an inadvertent selection for morning-oriented individuals, thought it remains essential to recognize and accommodate those who fall outside this pattern.

2.2. Biphasic sleep in contemporary contexts

2.2.1. Sleep throughout life

Another aspect to consider when discussing sleep segmentation in humans is that the monophasic arrangement is not constant even throughout an individual's lifespan. When starting their lives, infants show a very pronounced polyphasic sleep pattern. Then, through the process of "sleep consolidation", the child experiences a reduction of daytime sleeping and an increase during the night, eventually resulting in a biphasic pattern with a night bout and a midday nap [91,92].

Later in life, as humans age, total sleep time remains approximately the same (albeit a small decrease from adult to elder years, in the nocturnal sleep), and arousal during sleep increases, resulting in more fragmented sleep [1,3,93,94]. After retirement – perhaps without the pressure of work schedules – elderly people often return to biphasic sleep patterns, both at night due to fragmented sleep and during the day in the form of naps [3].

2.2.2. Napping

As we observe in children and elders, many cultures have maintained the habit of afternoon napping through to adulthood [82]. The occurrence of napping periods manifests mainly in the afternoon, being linked to ultradian circadian rhythmicity, such as core body temperature or glycemic variations, as several indicators show a marked drop of measurements in the early to mid-afternoon period: the postprandial somnolence, colloquially referred to as "noon dip" or "afternoon slump" [95,96]. Short naps taken in this mid-afternoon circadian dip (14:00h to 17:00h) have been shown to improve sleep latency and efficiency, and objective performance measures over naps taken either later in the evening [97,98] or earlier in the afternoon [99–101].

These daytime periods of sleep are predominantly comprised of non-REM sleep [102]. Relative to their overall ratios in nocturnal sleep, a 1h nap sees the percentages of stage 1 and SWS increased, while stage 2 and REM sleep percentages are reduced [34,103].

Individual differences in napping habits have been observed, with individuals generally falling into two categories: "nappers" and "non-nappers" [104]. The naturally habitual nappers tend to benefit from naps more strongly, even showing a decline when they are deprived of it – although non-habitual nappers also observe performance benefits [104]. They also show a greater amount of high frequency EEG waves, which might indicate that they maintain a tonic level of alertness that allows them to more quickly disperse sleep inertia following the nap and thus perform better right after it [40,105]. Although it is still unclear, being a napper seems to be a predominantly intrinsic genetic characteristic [106], and it might not be a skill that can be trained or habituated to Ref. [107].

Nonetheless, various studies have reported the benefits of napping in the general population, including enhanced alertness, improved performance on cognitive and motor tasks, reduced sleepiness, and lower subjective fatigue [34,108,109]. Additionally, napping has not been shown to negatively impact nocturnal sleep quality or duration [110], and even brief naps, as short as 15 min, can help sustain alertness and performance following sleep deprivation [111].

2.2.3. Shift working

Shift-type schedules are commonly employed in critical industries, such as for healthcare and emergency workers, military staff, sailors, and truck drivers, where 24h work coverage is necessary, which explains why they are so widely represented in the current studies. Shift workers face increased risks of metabolic, cardiovascular, and mental health problems linked to circadian misalignment, insufficient total sleep time, and sleep fragmentation [112,113]. To maintain alertness throughout the day and mitigate the effects of circadian misalignment – particularly in night work –, split sleep schedules are often adopted, allowing workers at least one sleep opportunity during their biological night, and helping to reduce sleep pressure between shifts (Fig. 3) [114–116]. In this context, napping has been shown to improve psychomotor speed, recovery between shifts, subjective sleepiness, blood pressure regulation, and heart rate variability, as well as reductions in fatigue, anxiety, and lapses in attention [117–119].

However, these biphasic sleep patterns might not be a substitute for consolidated nocturnal sleep. They might not resolve the core issue of circadian misalignment inherent to shift work, as daytime sleep episodes are usually lighter and more fragmented compared to night sleep, often resulting in non-restorative sleep [120].

The restorative benefits of biphasic sleep most likely depend on timing, duration, and environmental conditions – short or poorly timed naps may offer limited recovery. Carefully designed scheduling and individualization of sleep strategies are key to optimizing outcomes in this population.

2.3. Biphasic sleep laboratory studies

An in-lab highly controlled study (i.e., light exposure, social interactions) by Jackson and colleagues looked at nighttime shift workers and asked how split sleep compares to continuous daytime sleep and continuous night sleep [115]. Performance measurements showed no significant differences between sleep conditions, while subjective sleepiness was lowest, and total sleep time and REM sleep were longer in nighttime sleepers, followed by the biphasic sleep, and then the daytime sleepers. An important caveat is that this study examined only one possible biphasic sleep configuration, and although the chosen schedule aimed to align sleep onset with high sleep propensity and wake time with lower propensity for most people, it was not individualized to each participant's chronotype. While they used a questionnaire to screen for extreme early or late types, the delayed schedule considered may have favored only true late-types, and their actual phase of entrainment might even be different from what was reported.

To account for this, in another study looking at shift work, researchers aimed to directly compare nocturnal monophasic to biphasic sleep, but with the equally spaced sessions distributed across a 28h desynchronized schedule, for 13 days [121]. This desynchrony protocol (relative to a normal 24h day) aimed to isolate the homeostatic and circadian components. Both conditions resulted in the same total amount of sleep, with the biphasic schedule showing more SWS, and no differences reported in overall performance. However, performance showed less variability in biphasic sleep when the effects of circadian rhythmicity and prior wake time are accounted for. This can be explained by shorter accumulation of sleep pressure (Fig. 3) and the matching of sleep bouts with circadian oscillation of performance, as all metrics are typically lower near the body temperature nadir and decline proportionally to the amount of time awake [4].

To understand the influence of chronotypes, another experiment by Short and colleagues looked at "6h-on/6h-off" shift schedules and compared a baseline monophasic sleep and two biphasic sleep schedules: "early work" (03:00h to 08:00h and 15:00h to 20:00h) and "late work" (09:00h to 14:00h and 21:00h to 02:00h), both of them with a total 10h in bed per 24h [122]. This study showed that total sleep time was slightly higher in the baseline monophasic sleep schedule – agreeing with another 6h-on/6h-off shift working study in submarines, where the military staff slept less during mission and returned to their higher baseline sleep duration afterward in monophasic sleep [114]. In the Short study, this can be attributed to the increased sleep latency in the biphasic conditions – particularly during the 09:00h sleep initiation – or increased wakefulness after sleep onset (sleep fragmentation) –

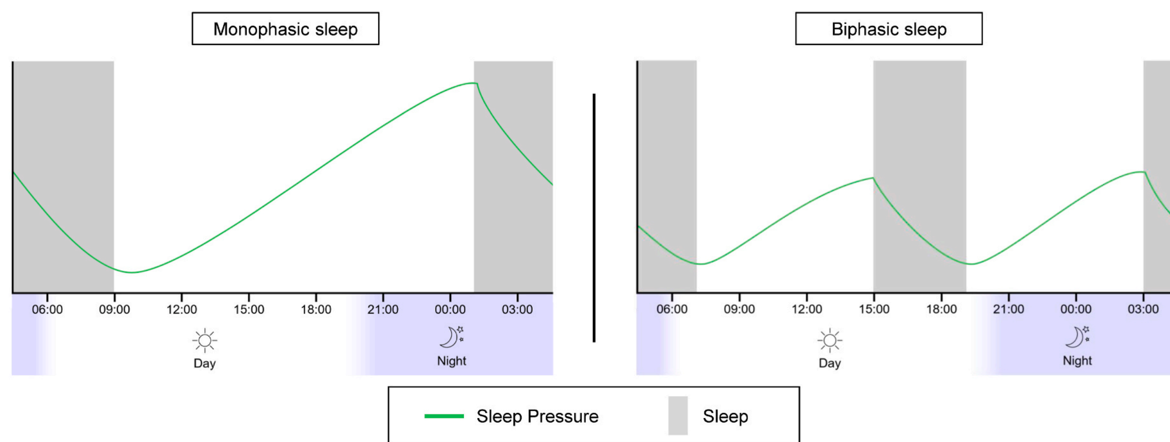

Fig. 3. – **Homeostatic stability in biphasic sleep schedules.** Schematic representation showing the hypothesized effect of regular biphasic sleep (equally split between a daytime nap and a nocturnal sleep) on maintaining a more stable sleep pressure over time. Compared to monophasic sleep, biphasic schedules may allow for less buildup of sleep pressure, promoting sustained alertness. Sleep bouts are represented in grey shadowing and sleep pressure as a green line. (For interpretation of the references to colour in this figure legend, the reader is referred to the Web version of this article.)

especially in the sleep period between 15:00h and 20:00h [122]. However, both effects can be explained by circadian rhythmicity: at 09:00h, the body expects to wake rather than to start sleeping, and while sleep can occur at 15:00h during the noon dip, it is difficult to sustain it for long due to the overlap with the "wake-maintenance zone" closer to 20h [11]. In the study, sleep architecture was conserved across conditions, with all stages returning to baseline after habituation. Performance on the psychomotor vigilance test showed no significant differences between monophasic and biphasic sleep conditions. Subjective sleepiness however was significantly higher in the "late work" condition compared to both the monophasic sleep and "early work" conditions, although it did not accumulate over the experimental period [122].

A research work from Thomas Wehr revealed that when healthy individuals were shifted from a 16h/8h light/dark photoperiod to a 10h/14h shorter day, they exhibited an unconsolidated biphasic sleep pattern [123]. In this short photoperiod condition, the total amount of sleep increased from 7.26 ± 0.21h to 8.36 ± 0.82h. However, they observed that sleep naturally split into two equally sized bouts, with 1–3h of interval between them during which the subjects were awake. Additionally, after the experimental period ended, 24h melatonin measurements and sleepiness tests were performed in a protocol of constant dim light and no sleep allowed. These measurements showed that even in the absence of light cues, the secretion of this hormone and the rating of rising drowsiness during the "night period" both increased in length after the 4 weeks experiment, relative to the baseline week. These findings show that humans have the capability to engage in biphasic sleep (particularly under conditions of lengthier darkness), and they indicate a modification in the circadian rhythm mechanisms of melatonin production and sleepiness accordingly.

2.4. Polyphasic sleep

Polyphasic schedules have also been theorized, as some of them have been popularized in media for allegedly being followed by famous people, such as Leonardo DaVinci or Nikola Tesla. Some of these schedules, like the "Uberman", aim at reducing the total amount of sleep time by engaging in short bouts of 30 min every 3.5h, resulting in a final 3h of sleep per day. Interestingly, it has been reported that test subjects might be capable of reorganizing their sleep cycles in a way that some of these sleep bouts focus on SWS and others on REM sleep, resulting in percentages of each equal to those observed in consolidated sleep [124]. Researchers also concluded that another key feature of this adaptability is the individual's increased ease of falling asleep quickly and throughout the day, given that with such short naps, any uncounted time awake in bed is wasted sleep time [124].

Other polyphasic sleep schedules harness the idea of "core sleep" by employing what is termed an "anchor sleep", a larger session at a fixed daily time [124]. Such is the "Everyman" schedule, according to which sleep is organized in one anchor sleep period lasting 6h and three other bouts of 30 min each, totalizing 7.5h across the 24h. Similarly, researchers have studied a schedule of 4h of sleep, followed by naps of 20, 50, and 80 min, which showed reduced sleep efficiency but identical performance measurements, except during the more frequent instances of sleep inertia [124].

However, a recent meta-analysis from the National Sleep Foundation was published, reviewing the existing literature and concluding that polyphasic sleep patterns are detrimental to physical parameters, mental health, and performance, as they result in sleep deficiency and fragmentation [125].

2.5. Potential drawbacks associated with biphasic sleep

While biphasic sleep has been associated with potential benefits in certain contexts, it is equally important to consider its possible drawbacks.

Sleep fragmentation specifically refers to the interruption and breaking up of sleep into multiple brief episodes, whether voluntarily or pathologically, preventing sustained periods of deep and restorative sleep. It has been linked to subjective fatigue, impaired inhibition, and weaker cognitive performance - particularly in tasks requiring memory or sustained focus [126].

Fragmented and irregular sleep schedules are also closely associated with eating disorders. Disrupted sleep may exacerbate psychological (mood, reward processing), hormonal (ghrelin, leptin), and metabolic (energy expenditure) imbalances, resulting in irregular eating patterns, including skipping meals, nighttime eating, and increased risk for binge episodes [127–129].

Further studies in murine models have shown that sleep fragmentation might induce inflammation and the elevation of stress hormones [130], disrupts gut microbiota homeostasis and host response [131], and leads to white matter abnormalities, reduced myelin integrity, and neuroinflammation [132].

Similarly to monophasic sleep, a poorly managed biphasic schedule may result in sleep curtailment and deprivation, with the corresponding psychological, physiological, and metabolic consequences [51,63–65]. It can also exacerbate circadian misalignment, between sleep and the circadian rhythms of core body temperature, melatonin, and cortisol [74–76] Moreover, it may even contribute to the development of

insomnia due to reduced nocturnal sleep pressure [133] - although this depends on factors such as the length, timing, and frequency of naps, as well as individual sleep needs, patterns, and reason to why they nap [27, 134,135].

3. Discussion

3.1. A theoretical paradigm of biphasic sleep

3.1.1. Biphasic sleep session arrangement

Given the strong influence of the circadian component upon sleep initiation, the success of a biphasic sleep pattern likely depends on how sleep periods are distributed throughout the day (Fig. 4).

Across history and cultures, two main biphasic sleep schemes can be identified: a night-night and a day-night configuration. The foremost consists of two nocturnal sleep bouts separated by a few hours of wakefulness. This can be further categorized (although it is in fact a continuous gradient) into two subtypes: early night-night biphasic sleep and late night-night biphasic sleep, for early and late chronotypes, respectively. The other type of schedule involves complementing an anchoring nocturnal sleep bout with another during the day. Again, this can be subdivided into a morning-night or afternoon-night biphasic sleep (one having the second bout in the morning and the other in the afternoon), for early and late chronotypes, respectively (Fig. 5).

Regarding the schedule with two nocturnal bouts, Wehr has shown that human physiology is naturally prepared to gear into a second sleep after a 1–3h intermission [123]. Furthermore, preliminary studies reported a possible biological predictive process that anticipates the awakening [136,137], and a circadian adaptation, by lengthening the melatonin and sleepiness peaks, as well as temperature fluctuation with wakefulness [123,136]. Just as for monophasic sleep, the variability in circadian rhythmicity proposedly induces the manifestation of chronotypes and thus the individual preference for an early or late-onset night-night biphasic sleep (Fig. 5).

Daytime sleep is most likely to occur during the mid-afternoon, a period consistently identified in the literature as the peak window for napping, during which it tends to be more efficient [95,97]. Even when the interval between sleep bouts is held constant, biphasic sleep schedules with a secondary sleep in the mid-afternoon, as opposed to earlier in the day, have been associated with reduced subjective sleepiness, preserved cognitive performance and shorter sleep latency. In contrast, sleep initiated later in the evening, such as 21:00h, is typically preceded by prolonged sleep latency, aligning with the concept of the “wake-maintenance zone”, a circadian window of reduced sleep propensity occurring approximately between 19:00h to 21:00h [122]. Taken together, these findings suggest that the optimal timing for daytime sleep lies between the end of the morning wakefulness period and the onset of the wake-maintenance zone, typically around 15:00h to 17:00h, coinciding with the circadian dip in alertness (Fig. 4) [122,138].

However, the optimal timing for daytime sleep is influenced by prior wake time duration and also by the individual’s chronotype. For early chronotypes, the typical mid-afternoon sleep window may overlap with the beginning of the wake-maintenance zone (Fig. 4), as they also tend to experience increased sleepiness earlier in the night. This results in higher sleep fragmentation, as the body is less primed for sleep [122].

An alternative option for early-types could be to engage in their daytime sleep in the late morning, rather than in the afternoon (Fig. 4), since a weaker circadian dip has also been reported at around 10:00h [137]. This way, regardless of the subject being an early- or a late-type, the choice of a split day/night daytime sleep can be correlated not only with circadian rhythmicity but also with sleep pressure, as it would effectively allow the body to restore some of the homeostatic buildup of the day while still leaving enough waking time before the second sleep. By truncating the duration of waking between the two sleep periods,

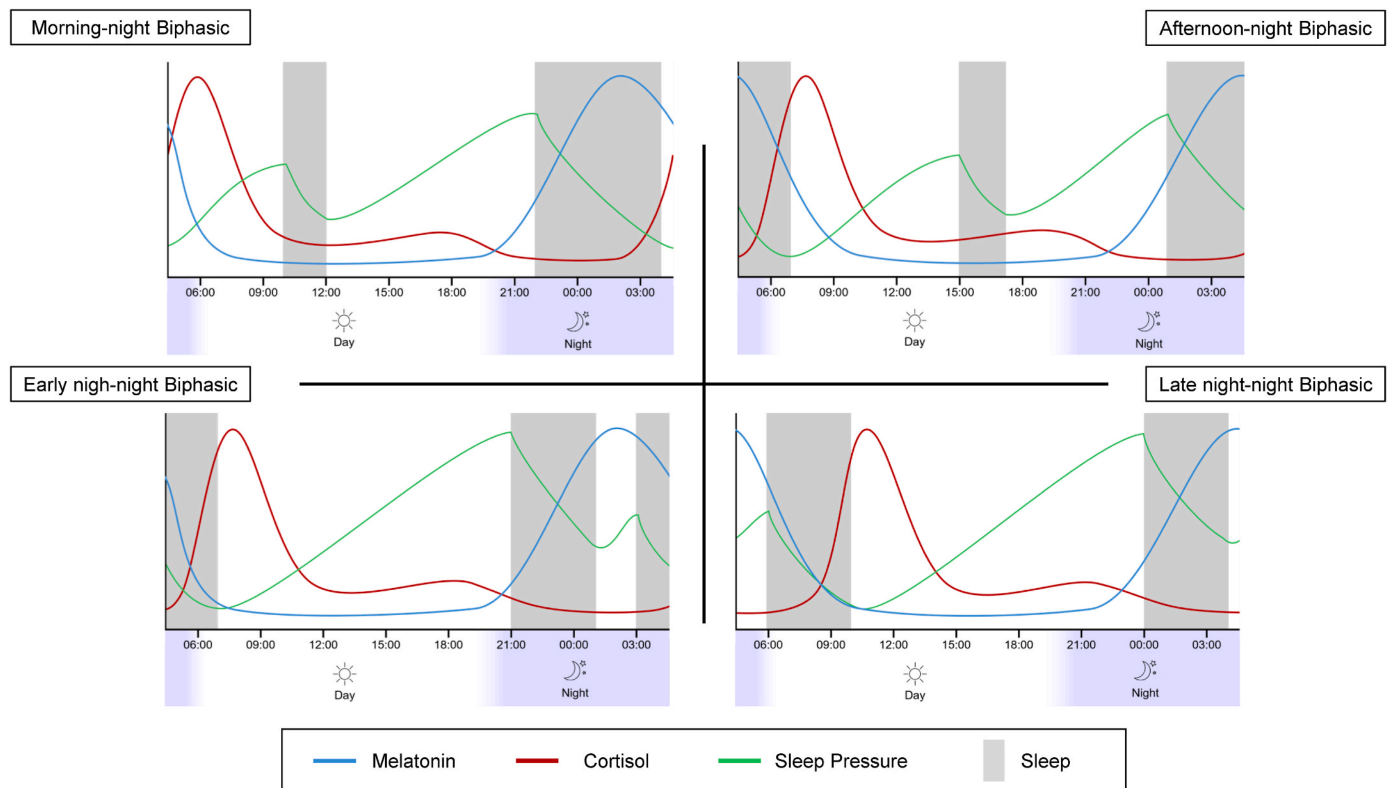

Fig. 4. – **Interaction between circadian rhythmicity and the four biphasic sleep patterns.** Schematic representation of sleep configurations (grey shadow) overlaid on circadian curves for melatonin (blue), cortisol (red), and sleep pressure (green). This figure expands on the model in Fig. 1, exploring how different distributions of biphasic sleep might align with internal biological rhythms. (For interpretation of the references to colour in this figure legend, the reader is referred to the Web version of this article.)

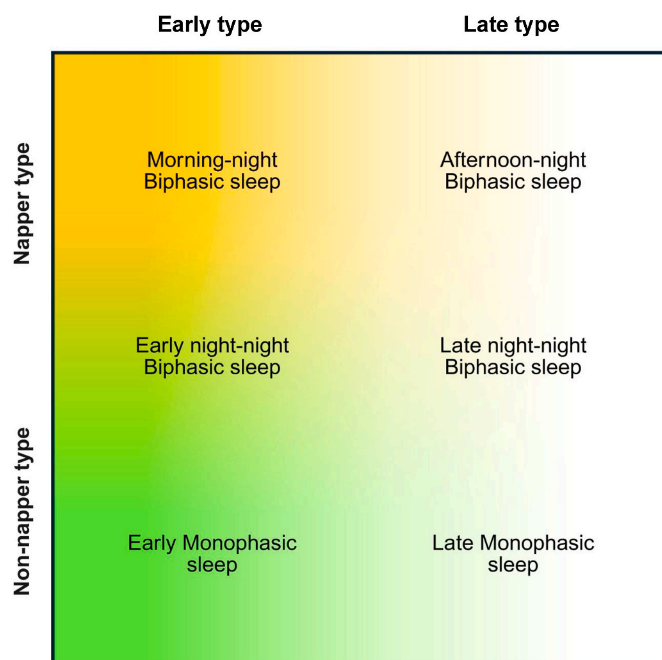

Fig. 5. – **Hypothesized chronotype and napper type interaction and its implications for optimal sleep distribution.** Two-dimensional illustration combining chronotype (lark to owl; dark-to-light gradient) and nap propensity type (habitual napper to non-napper; yellow-to-green gradient). The resulting continuous quadrants represent hypothesized sleep profiles most compatible with each combination, suggesting that personalized biphasic sleep strategies may optimize alertness and health outcomes. (For interpretation of the references to colour in this figure legend, the reader is referred to the Web version of this article.)

positive effects on sleepiness and performance might derive from the reduction of sleep pressure throughout both wake phases [121], possibly eliminating the characteristic alertness dip at the end of the day (Fig. 3).

Accordingly, two uneven day-night schedules might be hypothesized, an afternoon-night biphasic pattern (e.g., sleep from around 15h–17h and 1h–7h) for late-type people and a morning-night schedule (e.g., sleep from 10h to 12h and 22h–4h) for early-types (Figs. 4 and 5) could be further explored.

Another important factor that might influence the choice between schedules is being a habitual napper or a non-napper (Fig. 5). Habitual nappers have shorter sleep latency during the day, they more easily disperse sleep inertia after waking (allowing for an easier sleep-wake transition twice a day), and they see greater improvements in memory after napping, as well as a more efficient sleep [104,105]. Moreover, the performance of habitual nappers even declines when they are deprived of it (with equivalent total time in bed across the 24h), which suggests they may benefit more from one of the daytime sleep schedules than from monophasic sleep [104].

The CAR hormonal surge is governed by underlying circadian rhythmicity and modulated by environmental and neurocognitive factors – most notably, it is specifically triggered by the act of awaking [13,139,140]. This supports its compatibility with a biphasic sleep paradigm, as the typical once-daily occurrence of CAR may simply reflect the convention of a single daily awakening. With consistent adaptation to a biphasic sleep schedule, it is conceivable that the CAR could shift to occur twice daily, aligning with both wake periods. Furthermore, while biphasic sleep may include two episodes of wakefulness, it might also be that only the morning awakening (as coming from an anchor sleep) will interact robustly with the endogenous circadian system that drives cortisol, possibly not compromising the wake period (Fig. 4).

3.1.2. *Biphasic sleep stages and cycles*

Findings from Stampi's research on polyphasic sleep underscore the remarkable adaptability of the human sleep system. The body is not only a well-regulated system but also a highly flexible one, capable of reorganizing its sleep architecture to accommodate non-traditional sleep patterns. These studies suggest that sleep stages can be distributed across multiple bouts in such a way that preserves the overall quantity of REM and SWS, with stage 2 sleep typically serving as a buffering phase [124].

The implications of these findings for biphasic sleep patterns remains somewhat ambiguous. While the evidence is not yet conclusive, it is plausible to hypothesize that, at least in the case of nocturnal biphasic sleep, the progression and distribution of sleep stages across the 24-h cycle may closely resemble that of monophasic sleep. This assumption is based on the observation that neither circadian rhythmicity nor sleep pressure appears to be significantly changed.

In contrast, day-night biphasic sleep presents a more complex scenario. In this pattern, the relationship between sleep stages and circadian rhythms and sleep homeostasis cannot be assumed to remain constant. Given the strong circadian regulation of REM sleep, it is likely to remain concentrated in the nocturnal segment, potentially increasing its relative proportion during nighttime sleep. Should future research demonstrate that the body can adapt to a biphasic sleep schedule while preserving the overall distribution of sleep stages, SWS could either be primarily allocated to the daytime sleep bout or distributed more evenly between both episodes. This distribution would likely depend on factors such as the duration of each sleep bout and the amount of prior wakefulness. In the latter scenario, the reduction in nocturnal sleep duration would be mainly attributed to a decreased in SWS, thereby preserving adequate REM sleep at night. This could also ensure sufficient dissipation of sleep pressure to maintain alertness until the next sleep episode.

3.1.3. *Interbout activities*

In diurnal biphasic sleep patterns, social and occupational activities typically occur between the two sleep bouts. In contrast, nocturnal biphasic sleep involves an interbout wakefulness period during the night, when the majority of the population is inactive. Due to both the temporal isolation of this period and its relatively short duration, certain types of activities may be more suitable than others. Historical records have documented a wide range of interbout activities [79], and modern technologies have further expanded these possibilities. Nevertheless, two critical factors must be considered when evaluating appropriate activities for this interval: the effects of sleep inertia and the maintenance of sleep propensity for the second sleep period.

Sleep inertia, characterized by transient cognitive and motor impairment upon awakening, can significantly influence the quality and safety of activities undertaken during this wakefulness period. Its intensity and duration are affected by several variables, including the circadian phase and the sleep stage at the time of awakening. Specifically, awakening during the biological night and from deeper sleep stage (particularly those dominated by SWA) has been shown to intensify the negative effects of sleep inertia [39,40]. Given that the interbout interval in nocturnal biphasic sleep occurs during the night and typically follows the first cycles of sleep (where SWA is most prominent), careful consideration must be given to the potential cognitive drowsiness during this period. As such, individuals are advised to assess their typical duration of sleep inertia length and avoid engaging in high-risk, cognitively demanding, or operational critical tasks that require rapid decision-making or precise motor coordination.

Moreover, with a second sleep episode impending, it is advisable to refrain from engaging in stimulating activities that could reduce sleep propensity. These include exposure to blue light, which suppresses melatonin secretion [141,142] and vigorous physical exercise, which elevates levels of arousal-related neurotransmitters like adrenaline and acetylcholine [143]. Such factors may interfere with the body's ability to initiate and maintain subsequent sleep. In addition, biphasic sleep patterns themselves may alter sleep architecture, with potential

attenuation of SWS and increase likelihood of disorders of arousal in susceptible persons.

While strenuous exercise should generally be avoided during the interbout period, sexual activity may be an exception. Despite involving physical exertion, it is typically characterized by a brief peak of physiological arousal followed by a rapid decline. This post-coital period is associated with the release of hormones such as prolactin and oxytocin, both of which have been linked to increased sleepiness and rapid sleep onset [144–147]. Accordingly, sexual activity may not be non-disruptive but potentially conductive to initiating the second sleep bout.

3.2. Clinical relevance and applications

Biphasic sleep is a clinically relevant phenomenon, both as a natural sleep preference observed across cultures and as a potential therapeutic strategy for selected populations. Research has consistently demonstrated that individuals with sleep disorders, late chronotype, or irregular work schedules such as shift workers, are especially susceptible to sleep deprivation and 'social jetlag' [51–53,112,113]. In such populations, where sleep opportunities are often constraint by external demands (e.g., work shifts spaced less than 8h apart or early school/-work start times that conflict with individual circadian rhythms), studies indicate that there are no significant differences in sleep quality or cognitive performance between monophasic and biphasic sleep patterns [115,121,122].

More importantly, when biphasic sleep is carefully timed to ensure adequate total sleep within a 24-h period, it appears to be less detrimental than alternative patterns such as consolidated daytime monophasic sleep, partial sleep deprivation or extreme polyphasic schedules [115,117–119,125,148,149]. This suggests that biphasic sleep may be a preferable option in various contexts, including for individuals with extreme chronotypes, shift workers, new parents, and students. It is particularly noteworthy that chronotypes tend to be significantly delayed during adolescence, yet educational institutions often impose early start times and examinations, which may exacerbate sleep deprivation in this group [150].

Biphasic sleep may be therapeutically useful in delayed sleep-wake phase disorder (DSWPD) [77] and sleep maintenance insomnia. When appropriately timed, biphasic sleep can help reduce sleep debt and accommodate social obligations, although it may also exacerbate the delayed sleep-wake schedule. Some evidence suggests that social jet lag in individuals with DSWPD is associated with a lower risk of comorbid depression [77], potentially indicating that increased social interaction and earlier wake times on workdays serve as protective factors. An interesting observation made by Ekirch is that as historical references to the concept of "two sleeps" began to disappear, reports of conditions such as sleep maintenance insomnia started to rise [79]. While this correlation may be coincidental, coinciding with advances in medical diagnostics and a growing awareness of sleep disorders, it may also suggest that some individuals are naturally predisposed to a biphasic sleep pattern. In such cases, the body might attempt to an innate split-sleep schedule [151].

This raises the important question of whether contemporary societal expectations for uninterrupted, monophasic sleep may be inadvertently exacerbating sleep disturbances. By imposing rigid norms around continuous nighttime sleep, individuals who deviate from this model may experience heightened stress and anxiety, ultimately further impairing sleep quality [152]. In response to this issue, recent research has explored Paradoxical Intention therapy, which promotes acceptance of diverse chronotypes and views sleep interruption as a normal feature rather than a pathological one. This approach aims to reduce performance anxiety around sleep and improve overall sleep quality (Fig. 6). According to this emerging perspective, as long as total sleep time is sufficient – accounting for both sleep and wake periods during the night – embracing a biphasic pattern can serve as a therapeutic strategy to ease anxiety and support better sleep health [153].

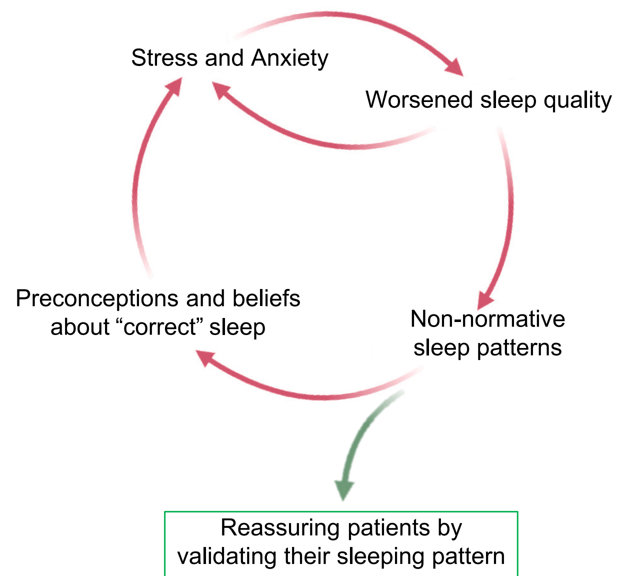

Fig. 6. – Cycle of sleep-related distress and the potential for intervention. Diagram depicting a self-reinforcing loop in which deviation from socially accepted sleep norms induces stress and anxiety, further degrading sleep quality. The figure also illustrates a potential exit point from this cycle through patient education and validation, promoting acceptance of individualized sleep patterns and reducing psychological burden, thus leading to improved sleep quality.

Whether the objective is to mitigate sleep deprivation or to align more closely with an individual's natural sleep schedule, a personalized approach that considers biological, cultural and social factors is essential when considering biphasic sleep as part of a viable health strategy.

3.3. Controversies and gaps in the literature

Despite growing interest in biphasic sleep as an alternative to the conventional monophasic model, there are still plenty of open questions from the scientific community and preconceptions from the broader public. In this section, we aim to point out some of those concerns, identify gaps in the literature, and outline approaches to future studies.

Some scholars argue that biphasic or segmented sleep was the norm in pre-industrial societies, citing historical documents across continents [79,80]. However, interpretations differ, with some researchers questioning the interpretation of these references, describing some of them as possibly ambiguous and selective, suggesting that sleep behaviors might have been more varied and more dependent on specific context than on universally natural sleep patterns [154]. Nonetheless, this analysis agrees even more closely with our proposed model, as it indicates that segmented sleep might not have been the only or even the predominant sleep distribution in the early modern period.

There is also controversy regarding the relevance of historic biphasic sleep patterns to contemporary lifestyles. The industrial revolution and artificial lighting profoundly changed sleep patterns, raising debate over whether a return to biphasic sleep is beneficial or feasible in the context of modern demands. Furthermore, there is an unconscious modern cultural bias in sleep research against biphasic sleep, particularly in response to more mainstream media and digital influencers who often disseminate inaccurate information, recommending a change in sleep patterns without concern for the individual's context and physiological responses.

While some studies have proposed potential cognitive and physiological benefits linked to biphasic sleep, others note limited or inconclusive evidence. The continuous study of fundamental sleep features

will certainly also prompt researchers to analyze additional parameters other than performance and sleepiness – such as network features, like cortical excitability [155] and functional connectivity [156–158], or molecular signals, like pleiotrophin [159], associated with sleep-loss-induced cognitive impairment – or use better-informed EEG sleepiness assessment techniques [160]. Further research is needed to better understand the circadian regulation of various sleep-related processes, including the modulation of orexin neurons and the dynamics of cortisol secretion. For example, it remains unclear whether the Cortisol Awakening Response would adapt to a second awaking in biphasic sleep schedules. This uncertainty also underscores the need for studies on the long-term health outcomes of biphasic sleep, including the potential for physiological habituation. Additionally, the impact of interbout activities on the quality of the second sleep episode, as well as the level of physiological alertness during the intervening wake period, warrants closer examination.

Constant routine protocols, although of difficult implementation, are valuable for assessing health outcomes by allowing for better isolation of the human physiological components. However, such studies often lack the social and psychological complexity inherent of real-world contexts. Additionally, cross-sectional or short-term observational designs may fail to capture the long-term risks or benefits associated with different sleep patterns. This highlights the need to complement existing knowledge with robust longitudinal research. Despite the inherent methodological challenges in human sleep research – such as reliance on self-reports [50], subjective interpretations of sleep quality, and difficulty of translating findings from animal models –, advances in actigraphy techniques [161], along with the development of specialized scales and models for assessing sleep distribution [162], are proving to be increasingly valuable tools in addressing these limitations.

In the discussed studies on shift work, where individuals follow a prescribed sleep pattern, researchers have found no significant differences in performance and sleep quality between the monophasic and biphasic sleep conditions [115,121,122]. However, participants in biphasic sleep reported higher subjective sleepiness and perceived impairment [115,122]. This discrepancy between objective and subjective measures suggests that these perceptions may stem more from societal beliefs – particularly the widespread notion that a single, consolidated night's sleep is healthier – rather than from actual physiological differences. To investigate this further, expectation-controlled studies are needed, where participants are either separated according to their prior beliefs about sleep scheduling, or they receive different contextual information about the effectiveness of their assigned sleep schedule. Crossover study designs could also be employed, wherein participants initially complete a questionnaire assessing their expectations and preferred sleep schedules and then adhere to that preferred schedule during the first experimental phase. In the second phase, participants would receive education on the potential benefits of an alternative sleep schedule, which they would subsequently adopt. This approach would enable researchers to evaluate whether reported increase in sleepiness is rooted in physiological responses or are instead influenced by negative preconceptions and expectations associated with unfamiliar sleep patterns.

Another individual factor is the stringency of the sleep times imposed, although suitable for the questions posed, it might instead add a variable when chronotypes and other sleeping types are not considered. Further studies are needed in which sleep patterns are not imposed but instead allowed to emerge naturally, or in which researchers, informed by prior knowledge of participants' sleep types, can tailor sleep schedules to better align with individual physiological predispositions. In this context, a deeper investigation into chronotypes and other sleep phenotypes is essential, particularly with regard to their physiological and genetic underpinnings. Such an understanding would provide a more objective basis for assigning participants into different groups. Alternatively, the use of forced desynchrony protocols offers a valuable method for disentangling the effects of circadian rhythmicity from homeostatic pressure, thereby enhancing our understanding of the independent contributions of each process.

Few studies have explored the clinical implications of adopting biphasic sleep schedules in individuals with sleep disorders or chronic health conditions. Patients diagnosed with sleep maintenance insomnia may be particularly valuable for such investigations, as their sleep often naturally fragments into a biphasic pattern, suggesting a possibly natural physiological inclination toward a nocturnal split-sleep schedule [151]. When evaluating whether a biphasic approach improves sleep quality in this population, it is important to ensure that participants are not sleep deprived at baseline. Controlling for sleep deprivation is critical to accurately assess the benefits of biphasic sleep, as its effects may otherwise be confounded by underlying sleep deficits.

4. Conclusion

This review has examined biphasic sleep as a biologically grounded and culturally widespread sleep pattern, challenging the prevailing notion that monophasic sleep is the universal ideal. While widely promoted as the normative model, monophasic sleep may not reflect the full range of human sleep flexibility observed across historical periods, developmental stages, and diverse lifestyles. From infancy to old age, sleep naturally evolves – from polyphasic to biphasic, to monophasic, and often back to biphasic in later life – suggesting that consolidated sleep is more a product of industrialized society than a universal biological standard.

Importantly, in many real-world contexts, biphasic sleep does not need to outperform monophasic sleep; rather, it simply needs to offer advantages over chronic sleep restriction or deprivation. This is especially relevant for populations subject to irregular schedules, such as shift workers, new parents, and students. Current evidence suggests that, under certain conditions, biphasic sleep does not impair cognitive performance and may, in fact, sustain more stable alertness throughout the day. It may also provide benefits by enabling sleep to occur at biologically advantageous times, such as during the biological night, and by allowing for more frequent dissipation of homeostatic sleep pressure. However potential trade-offs remain, particularly with regard to circadian rhythm disruption and changes in subjective sleepiness.

In specific occupations, such as the military deployments or maritime work, sleep arrangements are often integrated into the work environment, reducing commute time. Time off is mainly used for rest and sleep, and management of light exposure and social zeitgebers is facilitated and encouraged. For most people, however, this is not their reality. The 9:00h to 17:00h work schedule is the standard in our current society, which, coupled with long commutes and social and domestic obligations, largely disallows mid-day slumber or the extension of nocturnal time in bed. This might, however, be changing. With the advent and recent surge of remote work and flexible schedules, we are possibly being given an opportunity to disrupt the accepted dogmas and explore new (or old) systems for organizing our sleep in a more individualized way.

The literature also highlights that the effectiveness of biphasic or monophasic sleep patterns may depend heavily on individual differences, namely, chronotype and nap propensity. Here, we hypothesize a model that integrates the characteristics of these sleeping types into a matrix of the corresponding sleeping patterns.

Clinically, reevaluating biphasic sleep might help mitigate sleep deprivation in certain populations and prove beneficial for individuals with sleep maintenance insomnia or those experiencing social jetlag. Instead of pathologizing mid-night wakefulness, by imposing on patients a consolidated sleep schedule, we propose that clinicians should consider it a natural reflection of an individual's propensity for biphasic sleep. By accounting for time awake, proper scheduling allows for a higher total sleep amount, and validation potentially reduces sleep-related stress and performance anxiety, thus improving sleep quality outcomes.

Nevertheless, biphasic sleep is not without trade-offs, as it may disrupt circadian rhythmicity or exacerbate social misalignment, especially in environments that restrict mid-day rest. Indeed, most available studies investigate biphasic sleep under artificial circumstances – such as forced sleep restriction or shift work – rather than in naturalistic, health-promoting contexts. As such, the field remains limited by short-term and cross-sectional experiments, lacking long-term, ecologically valid, and chronotype-sensitive experimental designs, as well as clinical trials in populations with sleep disorders or chronic health conditions.

Like most aspects of human behavior, optimal sleep is likely to be a highly individualized process. Humans exhibit extraordinary adaptability, and the exploration of diverse sleep patterns can reveal critical individual differences in sleep need and timing, potentiating the reexamination of society's assumptions about "correct" sleep. Here, we propose to shift the paradigm of healthy sleep from one consolidated nocturnal bout to a range of schedule options, allowing for proper sleep across the 24h day – as some people might benefit from monophasic sleep, and others from biphasic, in different arrangements. Ultimately, we hope this work contributes to a broader, more flexible understanding of healthy sleep and encourages a move toward personalization rather than standardization, so that more people can access the kind of sleep that truly meets their individual needs.

CRediT authorship contribution statement

João Sena-Ribeiros: Writing – review & editing, Writing – original draft, Conceptualization. **Cátia Reis:** Writing – review & editing, Supervision.

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Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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