Sleep Health 7 (2021) 293–302

Contents lists available at ScienceDirect

Sleep Health

Journal of the National Sleep Foundation

journal homepage: sleephealthjournal.org

Adverse impact of polyphasic sleep patterns in humans: Report of the National Sleep Foundation sleep timing and variability consensus panel

Matthew D. Weaver, PhD[a,b,#], Tracey L. Sletten, PhD[c,#], Russell G. Foster, PhD, FRS[d], David Gozal, MD, MBA, PhD (Hon)[e], Elizabeth B. Klerman, MD, PhD[a,b,f], Shantha M.W. Rajaratnam, PhD[a,b,c], Till Roenneberg, PhD[g], Joseph S. Takahashi, PhD[h,i], Fred W. Turek, PhD[j], Michael V. Vitiello, PhD[k], Michael W. Young, PhD[l], Charles A. Czeisler, PhD, MD, FRCP[a,b,*]

[a] Division of Sleep and Circadian Disorders, Departments of Medicine and Neurology, Brigham and Women's Hospital, Boston, Massachusetts, USA
[b] Division of Sleep Medicine, Harvard Medical School, Boston, Massachusetts, USA
[c] Turner Institute for Brain and Mental Health and School of Psychological Sciences, Monash University, Clayton, Australia
[d] Sleep & Circadian Neuroscience Institute, Nuffield Department of Clinical Neurosciences, University of Oxford, Oxford, United Kingdom
[e] Department of Child Health, University of Missouri, Columbia, Missouri, USA
[f] Department of Neurology, Massachusetts General Hospital, Boston, Massachusetts, USA
[g] Institute for Medical Psychology, Ludwig Maximilian University of Munich, Munich, Germany
[h] Department of Neuroscience, The University of Texas Southwestern Medical Center, Dallas, Texas, USA
[i] Howard Hughes Medical Institute, The University of Texas Southwestern Medical Center, Dallas, Texas, USA
[j] Center for Sleep and Circadian Biology, Department of Neurobiology, Northwestern University, Evanston, Illinois, USA
[k] Department of Psychiatry and Behavioral Sciences, University of Washington, Seattle, Washington, USA
[l] Laboratory of Genetics, The Rockefeller University, New York, New York, USA

ARTICLE INFO

Keywords:
Polyphasic sleep
Sleep patterns
Circadian misalignment
Health
Performance

ABSTRACT

Polyphasic sleep is the practice of distributing multiple short sleep episodes across the 24-hour day rather than having one major and possibly a minor ("nap") sleep episode each day. While the prevalence of polyphasic sleep is unknown, anecdotal reports suggest attempts to follow this practice are common, particularly among young adults. Polyphasic-sleep advocates claim to thrive on as little as 2 hours of total sleep per day. However, significant concerns have been raised that polyphasic sleep schedules can result in health and safety consequences. We reviewed the literature to identify the impact of polyphasic sleep schedules (excluding nap or siesta schedules) on health, safety, and performance outcomes. Of 40,672 potentially relevant publications, with 2,023 selected for full-text review, 22 relevant papers were retained. We found no evidence supporting benefits from following polyphasic sleep schedules. Based on the current evidence, the consensus opinion is that polyphasic sleep schedules, and the sleep deficiency inherent in those schedules, are associated with a variety of adverse physical health, mental health, and performance outcomes. Striving to adopt a schedule that significantly reduces the amount of sleep per 24 hours and/or fragments sleep into multiple episodes throughout the 24-hour day is not recommended.

© 2021 The Authors. Published by Elsevier Inc. on behalf of National Sleep Foundation. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/)

Introduction

Individuals have long sought to optimize their sleep—minimizing sleep duration while preserving daytime alertness. Many wish to slip rapidly into a restful sleep that is free of nighttime awakenings. Since the role of sleep in memory consolidation and performance was discovered,[1,2] some have sought to use sleep to improve performance.[3] Others view sleep as more of a nuisance than a necessity, and want to minimize the time spent sleeping in order to increase the time available for productive wakefulness. Systematic efforts to do so date back more than a century, when it was recognized that the deepest (and presumably the most restorative) sleep occurs in the first hours of sleep, it was suggested that sleep be broken up into multiple, short sleep episodes, in an attempt to enhance the

*Corresponding author: Charles A. Czeisler, PhD, MD, FRCP, Division of Sleep and Circadian Disorders, Departments of Medicine and Neurology, Brigham and Women's Hospital, 221 Longwood Avenue, Suite 438A, Boston, MA 02115, USA. Tel.: 617-732-4013

E-mail address: charles_czeisler@hms.harvard.edu (C.A. Czeisler).

# These authors contributed equally.

https://doi.org/10.1016/j.sleh.2021.02.009
2352-7218/© 2021 The Authors. Published by Elsevier Inc. on behalf of National Sleep Foundation. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/)

"efficiency" of sleep.[4] Over the past 5 years, interest in polyphasic sleep has increased markedly with the formation of the Polyphasic Sleep Society and the popularization of the claim by polyphasic sleep advocates to have the ability to thrive on as little as 2 hours of sleep per day.[5] The current concept of polyphasic sleep is based loosely on the discovery that sleep is not a unitary process, but rather cycles between REM sleep and non-REM sleep on average every 80-120 minutes.[6] This has led some to strive to have their sleep episodes include an integer number of such cycles, and for reasons that are unstated and unsubstantiated, seek to avoid partial cycles. Such individuals try to schedule their sleep in 90-minute increments, even though REM/non-REM sleep cycles differ in period length between people,[7] vary in period length within individuals during the night and between nights,[8] and start at different phases of the REM/non-REM sleep cycle within the same person across nights,[9] which is compounded by variable latency to the onset of sleep, ie, to when these cycles start.[6] Given Nathaniel Kleitman's hypothesis that a Basic Rest-Activity Cycle (BRAC) with a 80-120-minute period persists throughout day and night,[10] polyphasic sleep advocates recommend dividing sleep into multiple, brief episodes distributed across the 24-hour day, with the aim of minimizing time spent asleep by restricting sleep duration, since deep slow wave sleep occurs in the first hours of each sleep episode.[11] Such a practice usually requires ending the sleep episode by an alarm or other stimulus, rather than spontaneously. While polyphasic sleep is widespread in many mammals, birds, and in human infants, in all such cases these short sleep episodes end spontaneously and are not purposefully curtailed. Significant concerns have been raised about the potential health and safety consequences of such fragmented and shortened sleep schedules.[12]

Biology of human sleep

Adequate sleep is necessary for effective daytime functioning and optimal health, whereas insufficient sleep results in impaired performance and adverse health consequences.[13-17] Adequate sleep can be obtained by various strategies. Historical evidence suggests that prior to the advent of artificial lighting, human sleep may have been split into two or more nighttime sleep episodes, particularly during seasons with a short photoperiod (ie, when the duration of nighttime darkness far exceeds the time people are able to sleep each night).[18] Actigraphic studies of pre-industrial societies living in regions without large seasonal changes in night length find that sleep is consolidated at night, even when it is dark for 11-12 hours.[19-21] Farmers in hotter climates worked late at night and started early in the morning, thereby shortening their sleep at night, for which they compensated with an extended afternoon siesta.[22] Although the siesta culture has receded in recent decades associated with industrial and agricultural changes, one-third of United States adults still report taking a nap during the daytime.[23] A single nighttime episode of 6-9 hours of relatively consolidated sleep has become the normative pattern in the industrialized world.[24] Sleep behavior may be highly diverse among individuals, cultures and different stages of industrialization; all of these patterns increase average sleep duration rather than reducing it, which is a central aim of modern polyphasic sleep strategies discussed below.

The daily cycle of sleep and wakefulness is characterized by a robust circadian variation in both sleep and wake propensity (ie, the probability of falling asleep or awakening, respectively) that is driven by a circadian pacemaker located in the suprachiasmatic nucleus (SCN) of the hypothalamus.[25,26] Synchronization of that pacemaker to the 24-hour day is mediated by retinal light exposure sensed by intrinsically-photosensitive retinal ganglion cells (ipRGCs) that contain the photopigment melanopsin,[27] and then conveyed from the ipRGCs to the SCN via the monosynaptic retinohypothalamic tract.[28] When synchronized to the 24-hour solar day-night cycle, the central circadian pacemaker in humans promotes the strongest circadian drive for sleep to occur just before dawn, near the end of the habitual nocturnal sleep episode, and the strongest circadian drive for wakefulness to occur just before dusk.[29] The circadian variation in sleep-wake propensity interacts non-linearly with the homeostatic sleep-wake regulatory process, which is characterized by a buildup in sleep propensity during wakefulness and a non-linear dissipation of sleep propensity during sleep, with greater deep, slow-wave sleep in the beginning of the sleep episode.[11,30,31] The biological drive for sleep is consequently greatest at night and lowest during the daytime in humans, with a secondary peak of sleep propensity in the mid-afternoon.[29,32] Scheduling sleep during the daytime, therefore, may result in less actual sleep (eg, from increased time to fall asleep and more time spent awake) during the sleep episode (ie, lower sleep efficiency), although sleep deficiency may raise sleep efficiency even during times of low circadian drive for sleep.[11,33] Furthermore, an endogenous circadian rhythm of REM sleep propensity promotes REM sleep in the early morning hours.[8] Therefore, allocating an insufficient amount of time for sleep and then attempting to distribute that limited amount of time available for sleep across the day and night, both of which are features of the most popular polyphasic sleep schedules (as described below), will result in less sleep obtained because of the influence of circadian rhythmicity on sleep, and may disproportionately deprive individuals of REM sleep.

Description of polyphasic sleep

In polyphasic or segmented sleep schedules, there are multiple sleep episodes across the 24-hour day, typically with a core or anchor sleep during the night and shorter episodes during the day.[5] Multiple different polyphasic sleep schedules have been advocated, encompassing a variety of sleep patterns with variation in length of core sleep and the number of daytime sleep episodes. Polyphasic sleep schedules are fundamentally different from the habitual night plus daytime nap or siesta practiced in many countries around the world, which are taken to increase the total amount of sleep obtained. Proponents claim that for the most optimal ("*Uberman*"), polyphasic sleep schedule, adherants should strive to achieve six 20-minute sleep episodes spaced evenly across the 24-hour day for a total targeted sleep duration of 2 hours per 24 hours.[5] Those who are unable to attain this goal are urged to adopt the *Everyman* polyphasic sleep schedule, which incorporates a 3-hour nighttime core sleep with three 20-minute daytime sleep episodes, designed to achieve a total targeted sleep duration of 4 hours per 24 hours. Another example is the *Triphasic* sleep schedule: sleep episodes after dusk, before dawn, and in the afternoon for a total of 4-5 h per 24 hours.

Claimed benefits of polyphasic sleep

Polyphasic sleep schedules, advocated by groups like the Polyphasic Sleep Society (PSS), are claimed to improve sleep density and stability (though the definitions of these terms are unclear) and to decrease the overall time required to be spent asleep for optimal performance.[5] The PSS and other proponents of polyphasic sleep claim that the increased discretionary wake time available as a result of reduced sleep will result in improved productivity.[5] These groups further claim that such schedules will help you live longer, improve your memory and mood, and dream more frequently.[5]

Purpose

As part of a larger systematic review on the impact of sleep timing on health and safety, we specifically reviewed the literature to

identify the impact of polyphasic sleep on sleep, health, safety, or performance outcomes.

Methods

We searched the PubMed database for original research and systematic literature reviews in the English language. We included MeSH terms and free text terms found in the Title or Abstract related to sleep timing (eg, sleep timing variability, napping, sleep patterns) and at least one sleep, health, safety, or performance outcome (eg, metabolism, cardiovascular, mental health). We excluded studies of Obstructive Sleep Apnea, Insomnia, Restless Legs Syndrome, and Narcolepsy (Appendix A). The search was executed on May 20, 2020. There were no publication date restrictions. The systematic review protocol for this question was not registered in advance.

The search returned 40,672 potentially relevant publications. A team of postdoctoral fellows and junior faculty members reviewed titles and abstracts for inclusion or exclusion. Due to the volume of potentially relevant publications, we did not require multiple raters to evaluate each potential paper. After review of titles and abstracts, 2,023 articles remained for full-text review in the larger systematic review. We defined polyphasic sleep as the practice of deliberately distributing restricted sleep opportunities across multiple episodes over the 24-hour day, including episodes with similar durations of sleep opportunity, or including a core or anchor sleep opportunities during the night and shorter episodes during the day. We did not include studies of afternoon naps or siestas, since they do not resemble the common schedules advocated by polyphasic sleep enthusiasts, and represent supplemental sleep rather than restricted sleep that is deliberately fragmented, and rationed into strictly limited bouts. We did include studies of biphasic sleep where sleep was divided during the night into two distinct sleep episodes. This intentional division of sleep during the night more closely approximates the prescribed polyphasic sleep schedules and yielded relevant evidence that could generalize to similarly fragmented sleep schedules. We searched the titles, abstracts, and keywords of the 2,023 articles eligible for full-text review to isolate the subset of articles that evaluated polyphasic or biphasic sleep, identifying 22 full-length manuscripts that specifically evaluated polyphasic or biphasic sleep and at least one relevant outcome in a population that was not constrained to a specific diagnosis (eg diabetic patients). Here we summarize the evidence produced by these studies. Full-text review was performed and relevant study characteristics were extracted collaboratively by two raters (MDW and TLS). Summary information is presented in Table 1. The risk of bias for each study was assessed using the Newcastle-Ottawa Quality Assessment scale.[34] Two reviewers (MDW and TLS) independently evaluated each paper for the quality assessment. Disagreements on quality assessments were to be resolved by a third reviewer (CAC). The risk of bias was considered by the consensus panel in the development of their recommendations. Consensus recommendations were developed using a modified Delphi process that required unanimous agreement. This work does not constitute human subject research and was not subject to Institutional Review Board review.

Findings

Lack of evidence of the claimed benefits of polyphasic sleep on sleep

By design, polyphasic sleep schedules decrease total sleep time, resulting in acute and chronic sleep deficiency. Though the definitions used by polyphasic sleep advocates of "sleep density" and "sleep stability" are unclear,[5] the evidence does not support any improvement in sleep architecture, duration, or quality from the polyphasic sleep schedule. We identified six studies that specifically compared a polyphasic or biphasic sleep schedule to a consolidated (normal) sleep schedule.[35-40] Roach *et al.* compared participants following a consolidated sleep schedule with one sleep-wake cycle every 28 hours to a biphasic sleep schedule with a sleep-wake cycle every 14 hours.[37] Those following the biphasic sleep schedule reported lower sleep quality, had longer sleep onset latencies, more arousals, and spent more time in lighter stages of sleep. A prospective crossover study by Weitzman *et al.* compared a 3-hour polyphasic schedule (opportunity to sleep for 1 hour every 3 hours) to an 8-hour consolidated sleep schedule.[39] Participants on the 3-hour polyphasic schedule achieved sleep for 56% of the sleep opportunities, compared to 90% during the consolidated schedule and had reductions in deep Slow Wave Sleep and REM sleep. Other studies assigning participants to polyphasic or biphasic sleep schedules, ranging from 30 minutes of sleep opportunity every 90 minutes, or 60 minutes of sleep every 180 minutes, etc. (ie, an ultrashort forced desynchrony protocol) to split nighttime sleep, have repeatedly shown extended sleep latencies, prominent sleep fragmentation, and marked reductions in REM sleep compared to consolidated sleep.[36,41-45]

Lack of evidence of the claimed benefits of polyphasic sleep on memory, mood, and performance

No studies demonstrated an improvement in memory retention on a polyphasic sleep schedule. Monks following a split-sleep schedule with a nocturnal awakening for prayer during the night reported more complaints of memory lapses than controls who followed a consolidated sleep schedule.[36] Sailors assigned to a schedule of 60 minutes sleep and 160 minutes wake for 40 hours performed worse on word recall tests than sailors who were completely sleep deprived over the 40-hour study interval.[46]

Similarly, we identified no peer-reviewed published evidence that supported improvements in mood while subjects were on polyphasic sleep schedules. Rather, polyphasic sleep schedules were associated with significant deterioration in mood in multiple studies, with depression ratings increasing with duration of exposure to the polyphasic sleep schedules.[43,47] Irritability and emotional discomfort are also associated with polyphasic sleep schedules.[39]

Evidence supporting improved productivity with polyphasic sleep schedules is scant. Stampi reported on the outcomes from three extended yacht races, including one transatlantic race.[48] Sailors with the shortest mean sleep episodes (20-60 minutes) placed highest in the race. This evaluation, however, did not differentiate between wakefulness and performance improvement; increased wakefulness by necessity increased the opportunity to perform a task requiring wakefulness (navigation), while actual performance of the navigation task was not assessed. Other studies that incorporated more intensive monitoring report worse performance on biphasic or polyphasic sleep schedules in core domains of cognitive function, including visual vigilance,[38] steadiness of hand,[49] tapping speed,[43] psychomotor performance,[50] and memory recall,[46] as well as steeper time-on-task performance deterioration.[38] Increase in sleepiness is also commonly reported on these schedules,[40,51] even when total sleep time is greater on the polyphasic sleep regimen,[52] possibly as a consequence of circadian disruption.

Lack of evidence of the claimed benefits of polyphasic sleep on health

There are no controlled studies that support the claimed health benefits of polyphasic sleep. Rather, the limited existing literature currently testing these types of polyphasic sleep schedules demonstrates their adverse health effects. Polyphasic sleep schedules deliberately promote sleep restriction, resulting in total sleep times that are typically far less than required for optimal health. Polyphasic schedules cause sleep deficiency, sleep at adverse circadian phases,

Table 1
Summary of relevant literature

Reference	Study design	Sample size	Age (y)	Exposure	Outcome(s)	Major findings	Bias rating
1935 Husband[49]	Prospective crossover study	n = 1	-	One month of 8 h continuous sleep followed by one month of 6 h segmented sleep (2300-0200 h, 0500-0800 h).	Eleven mental and motor tasks related to speed, accuracy, bodily sway, and intelligence.	3 of the 11 tests showed deterioration; diminished steadiness of hand, tapping speed, and bodily sway.	5
1974 Hartley[38,#]	Prospective laboratory study	n = 36	-	5-day experiment with 3 groups: a) 8 h continuous sleep b) 4 h continuous sleep c) 4 h sleep in 80 min episodes distributed across 24 h.	70 min visual vigilance test (Correct choices and false alarms).	80-min interval group was less impaired compared to 4-h continuous group. 80-min group had worst time-on-task deterioration. 80-min false alarm rate was comparable to 8-h group (more cautious). Both 4-h groups were impaired relative to 8-h group.	6
1974 Weitzman *et al.*[39,#]	Prospective crossover study	n = 7	23-40	a) 1 week baseline b) Up to 10 day exposure: 3 h sleep-wake schedule (8 × 1 h sleep times across 24 h day) c) 7 days of nocturnal 8-h sleep time	Characterization of sleep stages. Sequential plasma. Rectal temperature.	Participants slept for 56% of sleep opportunities, compared to 90% of baseline intervals. REM sleep was significantly diminished. Sleep onset REM sleep occurred more often in the last 5 days of ultradian protocol. NREM sleep Stages 3 and 4 reached 75% of baseline. During the last 3 days, the % of total sleep time for Stages 3 and 4 was same as baseline. Irritability and emotional discomfort were common. Circadian patterns of total sleep time, cortisol and body temperature were persistent. 3 h cortisol cycle entrained to sleep-wake cycle. Mean 24 h output of Growth Hormone was not changed but there was no evening peak. Recovery sleep patterns suggested that sleep deprivation had occurred.	8
1975 Carskadon & Dement [40,#]	Prospective laboratory study	n = 5	-	One-week protocol of 90-min schedule comprised of 30 min darkness for sleep and 60 min wakefulness following one week of regular 8 h sleep each night. *One additional subject completed with 15 min sleep and 75 min wakefulness.	Characterization of sleep stages.	Total sleep time decreased throughout the experiment, followed by a doubling of total sleep time on recovery day 1 compared to baseline. Sleep-onset REM episodes were common (79 of 110 instances of REM sleep). REM sleep rarely occurred during two consecutive sleep episodes (6 of 110 intervals).	5
1975 Moses *et al.*[51]	Prospective laboratory between-subjects study	n = 8 nap n = 10 exercise	18-22	2 groups: nap vs exercise. After baseline sleep (2200-0600 h), 40 h period of 10 epochs of 220 min with 1 h nap vs stationary bicycle exercise.	Oral temperature, total sleep time, sleep stage characterization.	Total sleep time and amount of REM sleep negatively correlated with circadian temperature pattern. Compared to baseline, the nap group had a reduction in the percentage of time asleep, higher percentages of Stage 1 and Stage 4 sleep, and lower percentages of Stage 2 and REM sleep.	5

(*continued on next page*)

Table 1 (*Continued*)

Reference	Study design	Sample size	Age (y)	Exposure	Outcome(s)	Major findings	Bias rating
1976 Lubin *et al.* [46]	Prospective laboratory study	n = 40	18-22	60 min sleep, 160 min wake for 40 h (n = 10). Compared with sleep deprivation groups completing 60 min exercise or non-sleep bedrest.	Wilkinson Auditory Vigilance Test, Wilkinson Addition Test, Williams Word Memory Test, Stanford Sleepiness Scale	Participants in the 60 min sleep arm performed worse on the word memory test and the Stanford Sleepiness Scale than participants who were completely sleep deprived. The 60-min sleep arm performed better than the complete sleep deprivation arm on the addition test. Participants were able to sleep for 61% of the allotted sleep time.	5
1977 Carskadon & Dement [92]	Prospective laboratory study	n = 10	17-21	90 min sleep-wake schedule (30 min sleep, 60 min play) beginning at midnight, vs. the same schedule beginning at 0900 h.	Sleep stage characterization, Stanford Sleepiness Scale.	Higher sleepiness pre-sleep predicted REM sleep in subsequent sleep interval. Greater REM sleep predicted greater sleep reductions. SWS did not diminish subjective sleepiness.	5
1978 Moses *et al.* [93]	Prospective laboratory study	n = 38	18-22	60-min sleep, 160 min wake for 40 h (n = 8). Compared with sleep deprivation groups completing 60-min exercise or non-sleep bedrest.	Sleep stage characterization, oral temperature, Stanford Sleepiness Scale, Wilkinson Auditory Vigilance Test, Wilkinson Addition Test	Minimum oral temperature associated with maximum nap sleep time, errors in vigilance and sleepiness. Naps with the most sleep were followed by the highest sleepiness ratings and the worst performance.	4
1981 Lavie & Scherson [44]	Prospective laboratory study	n = 9	20-25	15 min wake, 5 min sleep for 12 h	Sleep stage characterization	Only two participants achieved REM sleep. Stage 1 sleep showed 90-min rhythmicity. Stage 2 was prevalent in the mid-afternoon.	8
1989 Lavie & Segal [94]	Prospective laboratory study	n = 6 morning types, n = 5 evening types	23-26 22-25	Two ultrashort 7/13 min sleep-wake protocols. Attempting sleep: one adaptation night with 8-h sleep, then 7-min sleep/13-min wake for 24 h. Resisting sleep: 24-h sleep deprivation, then 7-min resisting sleep/13-min wake for 24 h.	24 h structure of sleep propensity comparing chronotypes	After night of sleep: Increased nocturnal sleepiness in morning types. Increased nocturnal sleepiness in evening types 2 h later than morning types. Sleep Stage 2 rose earlier and steeper in morning types. Morning types had 45% more sleep per trial and twice as much Stage 2 sleep per trial. One dropout after attempting sleep protocol. After sleep deprivation: Morning types had bimodal distribution of sleepiness. Evening types had no discernible afternoon sleepiness and a less pronounced nocturnal peak. Evening types achieved more sleep in the morning (1100-1200 h). Morning types had distinct sleep gates, while evening types did not.	7
1989 Stampi [48]	Prospective Field Study	n = 99		Three extended yacht races.	Total sleep time, duration of sleep episodes, race performance	Sailors with shortest mean sleep episodes (20 min-1 h) had best performance. Race performance was significantly worse for sleep episodes >2 h.	6
1992 Wehr [95]	Prospective laboratory study	n = 7		4-week exposure to 10 h light, 14 h darkness	Sleep, sleepiness, melatonin secretion	Cumulative sleep increased, sleep divided (sometimes) into 2 episodes with 1-3 h of wake between them. Melatonin secretion expanded. The rising phase of drowsiness expanded.	7

(*continued on next page*)

Table 1 (*Continued*)

Reference	Study design	Sample size	Age (y)	Exposure	Outcome(s)	Major findings	Bias rating
1995 Bonnet & Arand[35]	Prospective laboratory study	n = 12 per group	18-30	4 h continuous sleep before 24 h wake vs. 4 × 1 h naps during 24-h shift	Sleep stages, performance, mood.	Relative to continuous 4-h, the 4 × 1-h group had more Stage 3 and 4 sleep and fewer awakenings. Overall performance was similar, but time of day effects were stronger for the 4 × 1 h group, with better performance in the late afternoon and worse performance in the early morning.	5
1998 Porcu *et al.*[96]	Prospective field study	n = 9	19.7 ± 1.1	2 h activity and 4 h rest repeated over one 24-h interval	Sleep stages, microsleeps	Linear increase of Stage 2 and REM sleep throughout the 4 cycles, Stage 1 sleep was quadratic, with increased Stage 1 in the last cycle. Wake increased from 2nd to 3rd cycle and decreased in the 4th cycle. SWS most prominent in 2nd and 3rd cycle. Sleep latency decreased across cycles. Few microsleeps observed during activity (<1% of epochs).	8
2005 Buysse *et al.*[41]	Prospective laboratory study	n = 17 older n = 19 younger	76.3 ± 5 23.2 ± 3	60 h on a 90-min sleep-wake protocol: 30 min sleep, 60min wakefulness	Mood, Vigor, Psychomotor Vigilance Task	Older adults had smaller circadian variation of sleep propensity compared to younger adults. Wakefulness and performance rhythms showed increased circadian variation amongst older adults. Young adults got more sleep (18 min) compared to older adults (14 min). Sleep latency was lower among younger adults, and they had fewer awakenings. Sleep efficiency was 66% in the younger group and 53% in the older. Young adults reported more vigor, faster response times, and had more correct responses.	5
2007 Kripke *et al.*[47]	Prospective laboratory study	n = 50 n = 56	18-31 mean 23 59-75 mean 67	90-min protocol of 30 min bed, 60 min wake. After 30 h baseline, administered 3 × 3 h bright light at one of 8 times for three consecutive days.	Circadian phase shift (primary aim), depression scores before, during, and after experiment.	Increased depression from home rating to last day in laboratory.	8
2010 Kline *et al.*[43]	Prospective laboratory study	n = 25	19.6 ± 1.0 [SEM]	1 h sleep in dark, 2 h wake in dim light, repeated for 50-55 h	Psychomotor Vigilance Task, mood, sleep, and sleepiness	Average 24-h sleep duration 5.1 h, sleep efficiency 65%, acrophase 0630 h. Performance, mood, sleepiness showed robust circadian variation. Mood deteriorated from day 1 to day 2.	8
2011 Arnulf *et al.*[36,#]	Prospective (observational) field study	n = 10 monks/nuns n = 10 controls	18-67 (53.0 ± 6.7 for monks)	Monks and nuns maintaining split-sleep night vs controls. First rest period 1930 h, wake at 2345 h, second sleep 230 h	Interviews, 48 h core body temperature, 1 week sleep diaries and actigraphy	Core body temperature peak and trough values and clock times not different from controls. Core body temperature rhythm biphasic in monks and nuns. Monks and nuns had earlier sleep onset, shorter sleep time, difficulties with sleep latency, duration and daytime function, more frequent hallucinations.	5

(*continued on next page*)

Table 1 (*Continued*)

Reference	Study design	Sample size	Age (y)	Exposure	Outcome(s)	Major findings	Bias rating
2015 Neu *et al.*[45]	Prospective laboratory study	*n* = 18	26.2 ± 4.7	3 h early sleep (2330-230 h) and 3 h late sleep (430-730 h), spaced by 2 h sustained wakefulness	Sleep EEG power spectra, psychomotor vigilance task, subjective sleepiness	Similar sleep efficiency and total sleep time but more wake after sleep onset in first sleep. Expected phasic NREM and REM sleep predominances. Sleepiness was stable after both sleeps, psychomotor vigilance task decreased after the second sleep.	8
2017 Roach *et al.*[37]	Between-groups	*n* = 29	22.5 ± 2.2 22.6 ± 2.9	Forced desynchrony with activity: rest ratio of 2:1. Consolidated sleep with one sleep-wake cycle every 28-h. Split condition with one sleep-wake cycle every 14-h.	Sleep (polysomnography)	Split sleep condition was associated with lower sleep quality, longer sleep onset latency, more arousals, greater Stage 1 sleep, lower % of wake after sleep onset, and higher % of slow wave sleep.	8
2018 Maire *et al.*[97]	Prospective laboratory crossover study	*n* = 31	24.8 ± 3.3	Naps: 8-h sleep, Cycles of 160-min wake, 80-min sleep, for 56-h. Sleep deprivation: 40-h wakefulness.	Sleep, sleepiness, vigilance, cortical activity.	Compared to total sleep deprivation, sleepiness and reaction time was improved for those who napped.	8
2020 Al-Abri *et al.*[52]	Prospective cross-sectional (observational) study	*n* = 405	32.8 ± 11.5	One week of actigraphic measurement of sleep. Categorized as monophasic, biphasic (post-dawn), biphasic (afternoon siesta), polyphasic (3 sleep periods/24 h).	Epworth Sleepiness Scale (ESS) and Pittsburgh Sleep Quality Index (PSQI)	Polyphasic sleep (3 sleep periods/24 h) was associated with higher ESS compared to other sleep patterns. No difference in sleep quality. No difference in night sleep duration. Polyphasic sleep pattern had higher total 24-h day sleep duration.	6

# Indicates studies with a control group of consolidated sleep.

reduced sleep efficiency, sleep interruption and fragmentation, circadian disruption, REM sleep deficiency, and sleep inertia. An extensive body of scientific literature conclusively demonstrates the adverse consequences of these exposures.[53-61] No studies of polyphasic sleep support the claimed increased in life expectancy.

The negative consequences of sleep deficiency

Sleep deficiency has repeatedly been shown to diminish multiple aspects of health, safety, and performance.[13-16] The National Sleep Foundation recommends that adults seek 7-9 hours of sleep per night for optimal health, though 6-10 hours may be appropriate for some.[17] While dividing sleep into more than one episode is feasible (such as an anchor sleep supplemented by a nap), maintaining adequate total sleep time is necessary to prevent deterioration in neurobehavioral performance and symptoms of sleepiness.[62] Polyphasic sleep schedules do not meet recommendations for sufficient duration of total sleep time, and in fact, lead to Behaviorally Induced Insufficient Sleep Syndrome[63] with chronic sleep deficiency. Over time, the homeostatic sleep pressure rises to levels that can result in involuntary transitions to sleep, which may cause errors and accidents.[64-66] Once homeostatic sleep pressure is high enough, chronic sleep deficiency may facilitate the ability of individuals practicing polyphasic sleep to sleep during the biological daytime, which may be why advocates instruct those attempting to practice polyphasic sleep to endure the adverse consequences for a month or more until they are "trained" to sleep at any time of the day or night. The underlying sleep deficiency, however, impairs cognitive function, productivity, performance, and threatens safety.[30,67] Chronic sleep deficiency is also associated with a vast number of adverse long-term health outcomes including impaired growth, development, and metabolism,[68,69] increased risk of cardiovascular disease and diabetes,[70] accelerated cognitive decline,[71] and premature death.[72-76] In addition, multiple sleep episodes, particularly on a background of chronic sleep deficiency, amplify the intensity and frequency of impairment after each episode due to sleep inertia.[60] The independent decrements produced by sleep deficiency, circadian misalignment, and sleep inertia combine to profoundly diminish performance and health on polyphasic sleep schedules.

Multiple studies have also shown that the *self-reported* impairments of those who have chronic sleep deficiency do not align with objective performance: they fail to accurately assess how impaired they are by their sleep deprivation.[30,77-79] The self-reported experience of polyphasic sleepers regarding their performance, therefore, is likely to overestimate their level of alertness.

No studies of polyphasic sleep have demonstrated an increase in life expectancy. Sleep variability through following a polyphasic sleep schedule is markedly different from variability resulting from compensatory sleep, ie, extending sleep on free days to reduce sleep deficiency that accumulated during work/school days. Adults in the Swedish National March Cohort who reported sleeping ≤5 hours per night had a higher adjusted mortality rate than those consistently sleeping 6-7 hours per night; however, if those short sleepers reported extending sleep on weekends (by sleeping ≥6 hours), their adjusted mortality rate was no different from someone who regularly slept 6-7 hours per night.[80] Many prospective, population-based studies examining sleep duration and mortality suggest a U-shaped relationship, with both short and long durations of sleep associated with all-cause mortality, death from cardiovascular events, and cancer-specific mortality.[72-76] These studies used 7 hours of sleep as the referent category in the primary analyses, and did not always properly account for reasons for long sleep duration (eg, illness, obstructive sleep apnea) that may also affect mortality.[81] Polyphasic sleep schedules (eg, Uberman, Everyman, Triphasic) are designed to target considerably less than 7 hours of sleep per 24 hours (typically 3-5

hours). Therefore, extrapolating the findings relating to sleep duration and mortality from short sleep duration induced by polyphasic sleep schedules would predict an increased risk of premature death rather than any improvement in life expectancy.

The negative consequences of sleep at adverse circadian phases

Sleep on a polyphasic schedule also results in sleep outside of the temporal windows of increased sleep propensity driven by the circadian clock; such sleep is lower quality, fragmented, and more difficult to initiate.[29,33] Thus, the total duration of the actual sleep obtained is often much shorter than the time allotted for sleep.[33] Sleep and wakefulness at adverse circadian phases also results in circadian misalignment, in which the light-dark cycle and endogenous circadian rhythms are out of phase with each other.[82] Circadian misalignment has been associated with adverse effects on metabolism, the cardiovascular system, mood and decision-making, and increased risk of cancer.[83,84] In addition, many hormonal rhythms and other biological processes are altered by sleep. For these biological functions to occur optimally, sleep and circadian rhythms must be aligned. For example, secretion of growth hormone, cortisol, leptin, ghrelin, and other key regulators are adversely affected by misalignment of the circadian system.[85-87] The timing of REM sleep is also modulated by the circadian system.[8] The REM sleep deficiency often observed in studies of polyphasic sleep[39,44,51] is likely attributable to circadian misalignment. REM sleep is important for brain development, memory consolidation, problem solving, creativity, and may also serve to prepare the central nervous system for wakefulness.[88,89] One rationale for some polyphasic sleep schedules assumes that scheduling sleep episodes to occur every 90 minutes will somehow entrain the REM/Non-REM sleep cycle. As detailed above, this assumption is not correct.[6] Finally, interrupted or fragmented sleep in general is also associated with impaired learning and memory.[1,90,91]

In summary, while the direct evidence evaluating polyphasic sleep schedules is limited, an extensive body of literature demonstrates the adverse effects of key features of such schedules. Evaluations of the long-term impact of polyphasic sleep strategies are unlikely to materialize since such experiments would have significant ethical concerns.

Limitations

While the original review criteria for finding relevant studies was not targeted specifically to identify studies of polyphasic sleep, we believe the original review criteria focus on sleep timing variability was sufficiently sensitive to identify relevant literature for this project. Our search terms are included in Appendix A. We focused on evidence supporting or refuting PSS claims, but there may be other individuals or groups with perspectives that are not directly addressed in this argument. We are also limited by our reliance on English language original articles in peer-reviewed outlets. The conclusions drawn are limited to individuals over 17 years, since infants and young children, who do have polyphasic sleep patterns, differ from adults in multiple relevant areas of physiology (eg, development, hormonal secretion, circadian rhythmicity). In addition, we did not identify any studies where polyphasic sleep was implemented with preservation of normal sleep duration. None of the polyphasic sleep schedules of which we are aware maintain the recommended minimum of 7 hours of sleep per 24 hours. In fact, it is the stated goal of the polyphasic sleep advocates to significantly increase the duration of wakefulness by distributing sleep in a polyphasic schedule. Thus, we are unable to explicitly identify the separate impacts of fragmenting sleep and limiting sleep duration in a polyphasic sleep schedule.

Conclusions

The consensus panel unanimously agrees:

1. The claims of benefits from polyphasic sleep schedules are not supported by scientific evidence.
2. Polyphasic sleep schedules and the sleep deficiency inherent in the schedules that are most highly promoted in popular culture have been associated with adverse physical and mental health, as well as with decreased performance.
3. Striving to adopt a schedule that significantly reduces the amount of sleep per 24 hours and/or fragments sleep into multiple episodes throughout the 24-hour day can have significant adverse consequences for daytime performance, mood and health; and is clearly not recommended.

Funding

This project was supported by the National Sleep Foundation. The National Sleep Foundation had no role in the preparation of the manuscript or the decision to publish. Dr. Takahashi is an investigator in the Howard Hughes Medical Institute. Dr. Czeisler reports grants to BWH from NHLBI, NIA, NIOSH, and DOD, and serves as the incumbent of an endowed professorship provided to Harvard University by Cephalon, Inc. Dr. Klerman reports grants from NIH, the FAA, and salary support from Harvard University and the Massachusetts Institute of Technology during the conduct of the study.

Acknowledgments

We are grateful for logistical support and coordination from Katherine Draghi, Meredith Ellison, Anita Roach, and the National Sleep Foundation. We also thank Bryan Baxter, Lauren Booker, Ivy Mason, Megan Mulhall, Rebecca Robbins, and Melissa St. Hilaire for assistance with the literature review.

Conflicts of Interest

Dr. Weaver reports grants from the Brigham Research Institute, NHLBI and NIOSH, and personal fees from University of Pittsburgh, outside the submitted work.

Dr. Weaver and Dr. Sletten report personal fees from the National Sleep Foundation during the conduct of the study.

Dr. Klerman reports personal (consulting) fees from Wellesley College, CQ University (Australia), Puerto Rico Science Technology and Research Trust, the National Sleep FoundationPfizer, and Sanofi-Genzyme; non-financial (ttravel and registration) support from the Sleep Research Society, Santa Fe Institute, UT Austin, World Conference of Chronobiology, Gordon Research Conference, Stanford University, Northwestern University Solvay Institute (Belgium), Catholic University (Portugal);and personal fees and non-financial support from LMU (Germany), and NIH. All these are outside the submitted work. Dr. Klerman reports non-financial (travel) support from the National Sleep Foundation related to the submitted work.

Dr. Rajaratnam reports positions as the Program Leader for the CRC for Alertness, Safety and Productivity, Australia and the Director of the Sleep Health Foundation. He received grants from Vanda Pharmaceuticals, Philips Respironics, Cephalon, Rio Tinto, BHP Billiton and Shell which are not related to this paper. He also received equipment support and consultancy fees through his institution from Optalert, Compumedics, Teva Pharmaceuticals, and Circadian Therapeutics, which are not related to this paper.

Dr. Roenneberg is the founder and Chief Scientific Officer of the company Chronsulting UG and consults several other companies (Vanda Pharmaceuticals, Chiesi GmbH, jetlite GmbH, Condor Instruments, Salzgitter AG; PricewaterhouseCoopers; Weightwatchers; KGK Science Inc.); none of these activities created conflicts with the content of this paper.

Dr. Takahashi's conflicts of interest are all outside the submitted work. He is a co-founder and SAB member of Synchronicity Pharma, Inc.

Dr. Turek reports personal fees from Nature's Bounty Board of Advisers and grants from Vanda Pharmaceuticals, outside the submitted work.

Dr. Czeisler's conflicts of interest are all outside the submitted work. They include institutional gifts from: Mary Ann & Stanley Snider via Combined Jewish Philanthropies, Optum, Philips Respironics Inc, ResMed Foundation, San Francisco Bar Pilots, Sanofi SA , Sysco Corp, and Teva Pharmaceuticals Industries Ltd; and gifts and grants from Jazz Pharmaceuticals Plc Inc; and grants from Regeneron Pharmaceuticals, and Dayzz Ltd. They include personal fees from Bose Corporation, Boston Red Sox, Columbia River Bar Pilots, Institute of Digital Media and Child Development, Klarman Family Foundation, Samsung Electronics, Zurich Insurance Company Ltd, Washington State Board of Pilotage Commissioners, Ganésco Inc, Teva Pharma Australia, AARP, M. Davis and Company, Physician's Seal, United States Aircraft Insurance Group, National Sleep Foundation, Sleep Research Society, and Tencent; and non-financial support from Boston Celtics, CurtCo Media Labs LLC, Schneider Inc; and personal fees and an equity interest in Vanda Pharmaceuticals Inc. Dr. Czeisler is the incumbent of an endowed professorship provided to Harvard University by Cephalon, Inc. In addition, Dr. Czeisler receives royalty income from McGraw Hill, the New England Journal of Medicine, and Philips Respironics for the Actiwatch-2 and Actiwatch-Spectrum devices. Dr. Czeisler has served as an expert witness on a number of civil matters, criminal matters, and arbitration cases, including those involving the following commercial and government entities: Advanced Power Technologies; Alvarado Hospital, LLC; Amtrak; Bombardier, Inc.; C&J Energy Services; Casper Sleep, Inc.; Columbia River Bar Pilots; Complete General Construction Company; Dallas Police Department; Delta Airlines/Comair; Fédération des Médecins Résidents du Québec (FMRQ); FedEx; Greyhound Lines, Inc./Motor Coach Industries/FirstGroup America; H.G. Energy LLC; Maricopa County, Arizona, Sheriff's Office; Murrieta Valley Unified School District; Pomerado Hospital, Palomar Health District; Puckett EMS; Purdue Pharma; South Carolina Central Railroad Company, LLC.; Steel Warehouse, Inc.; Union Pacific Railroad; United Parcel Service. Dr. Czeisler served as a voluntary board member for the Institute for Experimental Psychiatry Research Foundation, Inc. Dr. Czeisler's interests were reviewed and managed by Brigham and Women's Hospital and Partners HealthCare in accordance with their conflict of interest policies. Dr. Foster, Dr. Gozal, Dr. Vitiello, and Dr. Young report no conflicts to declare.

Supplementary materials

Supplementary material associated with this article can be found in the online version at doi:10.1016/j.sleh.2021.02.009.

References

1. Stickgold R. Sleep-dependent memory consolidation. *Nature*. 2005;437(7063):1272–1278.
2. Pilcher JJ, Huffcutt AI. Effects of sleep deprivation on performance: a meta-analysis. *Sleep*. 1996;19(4):318–326.
3. Capellini I, Nunn CL, McNamara P, Preston BT, Barton RA. Energetic constraints, not predation, influence the evolution of sleep patterning in mammals. *Funct Ecol*. 2008;22(5):847–853.
4. Basner M. Arousal threshold determination in 1862: Kohlschütter's measurements on the firmness of sleep. *Sleep Med*. 2010;11(4):417–422.
5. Polyphasic Sleep Mastery. *Boost Energy and Productivity While Sleeping Less*. Polyphasic Society; 2016.
6. Carskadon MA, Dement WC. Normal human sleep: an overview. *Principles Pract Sleep Med*. 2005;4:13–23.
7. Chang AM, Bjonnes AC, Aeschbach D, et al. Circadian gene variants influence sleep and the sleep electroencephalogram in humans. *Chronobiol Int*. 2016;33(5):561–573.
8. Czeisler CA, Zimmerman JC, Ronda JM, Moore-Ede MC, Weitzman ED. Timing of REM sleep is coupled to the circadian rhythm of body temperature in man. *Sleep*. 1980;2(3):329–346.
9. Schulz H, Dirlich G, Zulley J. Phase shift in the REM sleep rhythm. *Pflügers Archiv*. 1975;358:203–212.
10. Kleitman N. Basic rest-activity cycle—22 years later. *Sleep*. 1982;5(4):311–317.
11. Dijk D-J, Czeisler CA. Contribution of the circadian pacemaker and the sleep homeostat to sleep propensity, sleep structure, electroencephalographic slow waves, and sleep spindle activity in humans. *J Neurosci*. 1995;15(5):3526–3538.
12. Wozniak P. Polyphasic sleep: 5 years later! A followup to "polyphasic sleep: facts and myths". Available at: https://www.supermemo.com/en/archives1990-2015/articles/polyphasic2010. 2010. Accessed June 12, 2020.
13. Goel N, Rao H, Durmer JS, Dinges DF. Neurocognitive consequences of sleep deprivation. *Paper presented at: Seminars in Neurology*. 2009.
14. Lockley SW, Barger LK, Ayas NT, Rothschild JM, Czeisler CA, Landrigan CP. Effects of health care provider work hours and sleep deprivation on safety and performance. *Joint Commission J Qual Patient Saf*. 2007;33(11):7–18.
15. Spiegel K, Sheridan JF, Van Cauter E. Effect of sleep deprivation on response to immunizaton. *JAMA*. 2002;288(12):1471–1472.
16. Medic G, Wille M, Hemels ME. Short-and long-term health consequences of sleep disruption. *Nat Sci Sleep*. 2017;9:151.
17. Hirshkowitz M, Whiton K, Albert SM, et al. National Sleep Foundation's sleep time duration recommendations: methodology and results summary. *Sleep Health*. 2015;1(1):40–43.
18. Ekirch AR. Sleep we have lost: pre-industrial slumber in the British Isles. *Am Hist Rev*. 2001;106(2):343–386.
19. Yetish G, Kaplan H, Gurven M, et al. Natural sleep and its seasonal variations in three pre-industrial societies. *Curr Biol*. 2015;25(21):2862–2868.
20. Lee EE, Amritwar A, Hong LE, Mohyuddin I, Brown T, Postolache TT. Daily and seasonal variation in light exposure among the old order Amish. *Int J Environ Res Public Health*. 2020;17(12):4460–4476.
21. de la Iglesia HO, Fernandez-Duque E, Golombek DA, et al. Access to electric light is associated with shorter sleep duration in a traditionally hunter-gatherer community. *J Biol Rhythms*. 2015;30(4):342–350.
22. Webb WB, Dinges DF. Cultural perspectives on napping and the siesta. *Sleep Alertness*. 1989:247–265.
23. Taylor P, Punk C, Craighill P. Pew Research Center. 2009. *Nap time URL:* http://www.pewsocialtrends.org/2009/07/29/nap-time/*[accessed 2017-03-30][WebCite Cache ID 6mrdEseqh]*. 2017.
24. Ekirch AR. Segmented sleep in preindustrial societies. *Sleep*. 2016;39(3):715–716.
25. Moore RY. Suprachiasmatic nucleus in sleep–wake regulation. *Sleep Med*. 2007;8:27–33.
26. Borbély AA, Daan S, Wirz-Justice A, Deboer T. The two-process model of sleep regulation: a reappraisal. *J Sleep Res*. 2016;25(2):131–143.
27. Panda S, Sato TK, Castrucci AM, et al. Melanopsin (Opn4) requirement for normal light-induced circadian phase shifting. *Science*. 2002;298(5601):2213–2216.
28. Fuller PM, Gooley JJ, Saper CB. Neurobiology of the sleep-wake cycle: sleep architecture, circadian regulation, and regulatory feedback. *J Biol Rhythms*. 2006;21(6):482–493.
29. Strogatz SH, Kronauer RE, Czeisler CA. Circadian pacemaker interferes with sleep onset at specific times each day: role in insomnia. *Am J Physiol Regul Integr Compar Physiol*. 1987;253(1):R172–R178.
30. Cohen DA, Wang W, Wyatt JK, et al. Uncovering residual effects of chronic sleep loss on human performance. *Sci Transl Med*. 2010;2(14). 14ra13-14ra13.
31. Dijk DJ, Brunner DP, Borbely AA. Time course of EEG power density during long sleep in humans. *Am J Physiol*. 1990;258(3 Pt 2):R650–R661.
32. Mitler MM, Carskadon MA, Czeisier CA, Dement WC, Dinges DF, Graeber RC. Catastrophes, sleep, and public policy: consensus report. *Sleep*. 1988;11(1):100–109.
33. Czeisler CA, Weitzman E, Moore-Ede MC, Zimmerman JC, Knauer RS. Human sleep: its duration and organization depend on its circadian phase. *Science*. 1980;210(4475):1264–1267.
34. Wells G, Shea B, O'connell D, et al. The Newcastle-Ottawa Scale (NOS) for assessing the quality if nonrandomized studies in meta-analyses. 2009. *Epub Available from: URL:* http://www.ohri.ca/programs/clinical_epidemiology/oxford.htm *[cited 2009 Oct 19]*. 2016.
35. Bonnet MH, Arand DL. Consolidated and distributed nap schedules and performance. *J Sleep Res*. 1995;4(2):71–77.
36. Arnulf I, Brion A, Pottier M, Golmard JL. Ring the bell for Matins: circadian adaptation to split sleep by cloistered monks and nuns. *Chronobiol Int*. 2011;28(10):930-941.
37. Roach GD, Zhou X, Darwent D, Kosmadopoulos A, Dawson D, Sargent C. Are two halves better than one whole? A comparison of the amount and quality of sleep obtained by healthy adult males living on split and consolidated sleep-wake schedules. *Accid Anal Prev*. 2017;99:428-433.
38. Hartley L. A comparison of continuous and distributed reduced sleep schedules. *Q J Exp Psychol*. 1974;26(1):8–14.
39. Weitzman ED, Nogeire C, Perlow M, et al. Effects of a prolonged 3-hour sleep-wake cycle on sleep stages, plasma cortisol, growth hormone and body temperature in man. *J Clin Endocrinol Metab*. 1974;38(6):1018–1030.
40. Carskadon MA, Dement WC. Sleep studies on a 90-minute day. *Electroencephalogr Clin Neurophysiol*. 1975;39(2):145–155.

41. Buysse DJ, Monk TH, Carrier J, Begley A. Circadian patterns of sleep, sleepiness, and performance in older and younger adults. *Sleep*. 2005;28(11):1365–1376.
42. Dement W, Sidoric K, Simmons J, et al. Life on Basic Rest-Activity Cycle (BRAC)—sleep studies of a ninety minute day. *Psychophysiology*. 1972. Paper presented at: APSS 1971.
43. Kline CE, Durstine JL, Davis JM, Moore TA, Devlin TM, Youngstedt SD. Circadian rhythms of psychomotor vigilance, mood, and sleepiness in the ultra-short sleep/wake protocol. *Chronobiol Int*. 2010;27(1):161–180.
44. Lavie P, Scherson A. Ultrashort sleep-waking schedule. I. Evidence of ultradian rhythmicity in 'sleepability. *Electroencephalogr Clin Neurophysiol*. 1981;52(2):163–174.
45. Neu D, Mairesse O, Newell J, Verbanck P, Peigneux P, Deliens G. Does more sleep matter? Differential effects of NREM- and REM-dominant sleep on sleepiness and vigilance. *Neurophysiol Clin*. 2015;45(2):167-175.
46. Lubin A, Hord DJ, Tracy ML, Johnson LC. Effects of exercise, bedrest and napping on performance decrement during 40 hours. *Psychophysiology*. 1976;13(4):334–339.
47. Kripke DF, Elliott JA, Youngstedt SD, Rex KM. Circadian phase response curves to light in older and young women and men. *J Circadian Rhythms*. 2007;5:4.
48. Stampi C. Polyphasic sleep strategies improve prolonged sustained performance: a field study on 99 sailors. *Work Stress*. 1989;3(1):41–55.
49. Husband RW. The comparative value of continuous versus interrupted sleep. *J Exp Psychol*. 1935;18(6):792.
50. Bonnet MH, Arand DL. Consolidated and distributed nap schedules and performance. *J Sleep Res*. 1995;4(2):71-77.
51. Moses J, Hord D, Lubin A, Johnson L, Naitoh P. Dynamics of nap sleep during a 40 hour period. *Electroencephalogr Clin Neurophysiol*. 1975;39(6):627–633.
52. Al-Abri MA, Al Lawati I, Zadjali F, Ganguly S. Sleep patterns and quality in omani adults. *Nat Sci Sleep*. 2020;12:231–237.
53. Zitting K-M, Münch MY, Cain SW, et al. Young adults are more vulnerable to chronic sleep deficiency and recurrent circadian disruption than older adults. *Sci Rep*. 2018;8(1):1–14.
54. Laposky AD, Van Cauter E, Diez-Roux AV. Reducing health disparities: the role of sleep deficiency and sleep disorders. *Sleep Med*. 2016;18:3–6.
55. Scheer FA, Hilton MF, Mantzoros CS, Shea SA. Adverse metabolic and cardiovascular consequences of circadian misalignment. *Proc Natl Acad Sci*. 2009;106(11):4453–4458.
56. Newman AB, Nieto FJ, Guidry U, et al. Relation of sleep-disordered breathing to cardiovascular disease risk factors: the Sleep Heart Health Study. *Am J Epidemiol*. 2001;154(1):50–59.
57. Tsuno N, Besset A, Ritchie K. Sleep and depression. *J Clin Psychiatry*. 2005;66(10):1254–1269.
58. Buxton OM, Broussard JL, Zahl AK, Hall M. Effects of sleep deficiency on hormones, cytokines, and metabolism. *Impact of Sleep and Sleep Disturbances on Obesity and Cancer*. Springer; 2014:25–50.
59. Depner CM, Stothard ER, Wright KP. Metabolic consequences of sleep and circadian disorders. *Curr Diab Rep*. 2014;14(7):507.
60. McHill AW, Hull JT, Cohen DA, Wang W, Czeisler CA, Klerman EB. Chronic sleep restriction greatly magnifies performance decrements immediately after awakening. *Sleep*. 2019;42(5):1–7.
61. McHill AW, Hull JT, Wang W, Czeisler CA, Klerman EB. Chronic sleep curtailment, even without extended (>16-h) wakefulness, degrades human vigilance performance. *Proc Natl Acad Sci U S A*. 2018;115(23):6070–6075.
62. Mollicone DJ, Van Dongen HP, Rogers NL, Dinges DF. Response surface mapping of neurobehavioral performance: testing the feasibility of split sleep schedules for space operations. *Acta Astronaut*. 2008;63(7-10):833–840.
63. Hublin C, Sallinen M. Behaviorally induced insufficient sleep. *Sleep Med Clin*. 2012;7(2):313–323.
64. Goel N. Neurobehavioral effects and biomarkers of sleep loss in healthy adults. *Curr Neurol Neurosci Rep*. 2017;17(11):89.
65. McHill AW, Hull JT, Wang W, Czeisler CA, Klerman EB. Chronic sleep curtailment, even without extended (>16-h) wakefulness, degrades human vigilance performance. *Proc Natl Acad Sci*. 2018;115(23):6070–6075.
66. Goel N, Rao H, Durmer JS, Dinges DF. Neurocognitive consequences of sleep deprivation. *Semin Neurol*. 2009;29(4):320–339.
67. Rosa RR. Sleep loss, sleepiness, performance, and safety. *Sleep: A Comprehensive Handbook*. 2006:203–207.
68. Potter GD, Skene DJ, Arendt J, Cade JE, Grant PJ, Hardie LJ. Circadian rhythm and sleep disruption: causes, metabolic consequences, and countermeasures. *Endocr Rev*. 2016;37(6):584–608.
69. Qian J, Scheer F. Circadian system and glucose metabolism: implications for physiology and disease. *Trends Endocrinol Metab*. 2016;27(5):282–293.
70. Mullington JM, Haack M, Toth M, Serrador JM, Meier-Ewert HK. Cardiovascular, inflammatory, and metabolic consequences of sleep deprivation. *Prog Cardiovasc Dis*. 2009;51(4):294–302.
71. Osorio RS, Gumb T, Pirraglia E, et al. Sleep-disordered breathing advances cognitive decline in the elderly. *Neurology*. 2015;84(19):1964–1971.
72. Yin J, Jin X, Shan Z, et al. Relationship of sleep duration with all-cause mortality and cardiovascular events: a systematic review and dose-response meta-analysis of prospective cohort studies. *J Am Heart Assoc*. 2017;6(9). https://doi.org/10.1161/JAHA.117.005947.
73. Cappuccio FP, D'Elia L, Strazzullo P, Miller MA. Sleep duration and all-cause mortality: a systematic review and meta-analysis of prospective studies. *Sleep*. 2010;33(5):585–592.
74. Gallicchio L, Kalesan B. Sleep duration and mortality: a systematic review and meta-analysis. *J Sleep Res*. 2009;18(2):148–158.
75. Stone CR, Haig TR, Fiest KM, McNeil J, Brenner DR, Friedenreich CM. The association between sleep duration and cancer-specific mortality: a systematic review and meta-analysis. *Cancer Causes Control*. 2019;30(5):501–525.
76. Li W, Wang D, Cao S, et al. Sleep duration and risk of stroke events and stroke mortality: a systematic review and meta-analysis of prospective cohort studies. *Int J Cardiol*. 2016;223:870–876.
77. Bermudez EB, Klerman EB, Czeisler CA, Cohen DA, Wyatt JK, Phillips AJ. Prediction of vigilant attention and cognitive performance using self-reported alertness, circadian phase, hours since awakening, and accumulated sleep loss. *PLoS One*. 2016;11:(3) e0151770.
78. Sletten TL, Segal AY, Flynn-Evans EE, Lockley SW, Rajaratnam SM. Inter-individual differences in neurobehavioural impairment following sleep restriction are associated with circadian rhythm phase. *PLoS One*. 2015;10:(6) e0128273.
79. Van Dongen H, Maislin G, Mullington JM, Dinges DF. The cumulative cost of additional wakefulness: dose-response effects on neurobehavioral functions and sleep physiology from chronic sleep restriction and total sleep deprivation. *Sleep*. 2003;26(2):117–126.
80. Akerstedt T, Ghilotti F, Grotta A, et al. Sleep duration and mortality—does weekend sleep matter? *J Sleep Res*. 2019;28(1):e12712.
81. Bianchi MT, Thomas RJ, Westover MB. An open request to epidemiologists: please stop querying self-reported sleep duration. *Sleep Med*. 2017;35:92–93.
82. Vetter C. Circadian disruption: what do we actually mean? *Eur J Neurosci*. 2020;51(1):531–550.
83. Roenneberg T, Merrow M. The circadian clock and human health. *Curr Biol*. 2016;26(10):R432–R443.
84. Rajaratnam SMW, Licamele L, Birznieks G. Delayed sleep phase disorder risk is associated with absenteeism and impaired functioning. *Sleep Health*. 2015;1(2):121–127.
85. Wright Jr. KP, Drake AL, Frey DJ, et al. Influence of sleep deprivation and circadian misalignment on cortisol, inflammatory markers, and cytokine balance. *Brain Behav Immun*. 2015;47:24–34.
86. Scheer FA, Hilton MF, Mantzoros CS, Shea SA. Adverse metabolic and cardiovascular consequences of circadian misalignment. *Proc Natl Acad Sci U S A*. 2009;106(11):4453–4458.
87. Morris CJ, Aeschbach D, Scheer FA. Circadian system, sleep and endocrinology. *Mol Cell Endocrinol*. 2012;349(1):91–104.
88. Lara-Carrasco J, Nielsen TA, Solomonova E, Levrier K, Popova A. Overnight emotional adaptation to negative stimuli is altered by REM sleep deprivation and is correlated with intervening dream emotions. *J Sleep Res*. 2009;18(2):178–187.
89. Peever J, Fuller PM. The biology of REM sleep. *Curr Biol*. 2017;27(22):R1237–R1248.
90. Djonlagic I, Saboisky J, Carusona A, Stickgold R, Malhotra A. Increased sleep fragmentation leads to impaired off-line consolidation of motor memories in humans. *PLoS One*. 2012;7(3):e34106.
91. Stickgold R, Hobson JA, Fosse R, Fosse M. Sleep, learning, and dreams: off-line memory reprocessing. *Science*. 2001;294(5544):1052–1057.
92. Carskadon MA, Dement WC. Sleepiness and sleep state on a 90-min schedule. *Psychophysiology*. 1977;14(2):127–133.
93. Moses J, Lubin A, Naitoh P, Johnson LC. Circadian variation in performance, subjective sleepiness, sleep, and oral temperature during an altered sleep-wake schedule. *Biol Psychol*. 1978;6(4):301–308.
94. Lavie P, Segal S. Twenty-four-hour structure of sleepiness in morning and evening persons investigated by ultrashort sleep-wake cycle. *Sleep*. 1989;12(6):522–528.
95. Wehr TA. In short photoperiods, human sleep is biphasic. *J Sleep Res*. 1992;1(2):103–107.
96. Porcu S, Casagrande M, Ferrara M, Bellatreccia A. Sleep and alertness during alternating monophasic and polyphasic rest-activity cycles. *Int J Neurosci*. 1998;95(1-2):43–50.
97. Maire M, Reichert CF, Gabel V, et al. Human brain patterns underlying vigilant attention: impact of sleep debt, circadian phase and attentional engagement. *Sci Rep*. 2018;8(1):970.