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Circadian Rhythm

Daytime Sleep Propensity After Moderate Circadian Phase Shifts Induced With Bright Light Exposure

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Summary: Moderate circadian phase shifts were induced by 3 days of bright light exposure, without changing the habitual sleep schedule. Daytime sleep propensity was evaluated with multiple sleep latency tests (MSLT) conducted before and after the light treatment. Phase shifts were estimated using the core body temperature rhythm recorded during constant routines. The subjects were divided into three groups according to the timing of the bright light exposure. Morning bright light exposure (Morning group) advanced the circadian phase by about 1.2 hours, evening bright light (Evening group) delayed the circadian phase by 1.6 hours on average; whereas, bright light administered in the afternoon (Afternoon group) did not change the circadian phase. After the light treatment, daytime sleep latencies decreased in the Evening and Afternoon groups, but did not change in the Morning group. Reduced sleep latencies in the Afternoon group probably reflect an increase in the manifest sleep tendency induced by the protocol itself. It is suggested that, in the presence of a high physiological sleep tendency, a moderate circadian phase delay may increase further daytime sleep propensity, whereas a moderate circadian phase advance may help to maintain daytime sleep propensity at a lower level. **Key Words:** Daytime sleep propensity—MSLT—Circadian rhythms—Phase shifts—Bright light.

Current hypotheses on sleep–wake regulation include at least two interacting components to explain variations in sleep propensity (1–3). A circadian component (process "C"), which depends on the output of the circadian pacemaker, actively or passively promotes wakefulness during the day and increases sleep tendency during the habitual sleep period. A second component, the homeostatic component (process "S"), is responsible for the increase in sleep tendency associated with an increased duration of prior time awake.

The 24-hour component of sleep propensity, defined as the ability to fall asleep at different times of the day, was studied under artificial days of very short duration (4–6). These experiments have shown a pronounced circadian rhythm in sleep propensity, with a peak around 0600 hours and a minimum around 2100 hours. Facilitated transitions from wake to sleep were also observed in late evening and in mid-afternoon, and zones where sleep is rarely initiated were identified around 2000 to 2300 hours in the evening and 1000 to 1100 hours in the morning. The same pattern in sleep propensity was also observed with a normal sleep schedule when, in addition to daytime sleep latencies, nighttime sleep latencies were obtained following 15-minute awakenings every 2 hours during the night (7).

These circadian variations in sleep propensity seem to maintain a stable phase relationship with the endogenous rhythm of body temperature. When the sleep–wake cycle and the endogenous circadian temperature rhythm are desynchronized, either spontaneously in free-running studies (8–10) or by a forced desynchrony protocol (3), subjects fall asleep more easily close to the time of their body temperature minimum, and sleep is rarely initiated (or sleep latencies are longer) when body temperature is at the maximum. The shortest sleep latencies are observed at the minimum of the temperature rhythm. Then, sleep latencies increase steadily over two-thirds of the temperature rhythm and show a steep lengthening before the temperature starts to decline. After this zone of long sleep latencies, sleep propensity increases abruptly on the falling limb of the temperature cycle (over one-third

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of the cycle). Recently, Lack and Lushington (11) combined constant routines and ultra-short sleep–wake schedules to identify "sleep propensity phase times" that appear to be locked to the endogenous temperature rhythm. They were able to confirm the presence of the maximum sleep propensity close to the temperature minimum as well as the opening of the evening "sleep gate" about 5 hours earlier in all the subjects. In addition, they observed a significant 12-hour rhythm in sleep propensity in eight of 14 subjects and identified: a mid-afternoon peak in sleepiness about 14 hours before the temperature minimum, an early evening minimum in sleep propensity preceding the temperature minimum by about 8 hours, and a late morning drop in sleepiness that occurred about 6 hours after the temperature minimum.

These results strongly suggest that sleep propensity and body temperature rhythms are driven by the same circadian oscillator and that a phase shift of the endogenous temperature rhythm will be accompanied with a phase shift of circadian sleep propensity. A major difficulty in the study of circadian regulation of daytime sleep propensity is changing the phase relationship between the sleep–wake cycle and the circadian pacemaker without affecting sleep duration and, thus, the homeostatic process. All the protocols cited above involved at least some sleep deprivation or abnormal sleep schedules to expose the circadian rhythm of sleep propensity. Bright light exposure has been used to change the phase of the circadian oscillator without changing the timing of sleep (12–15), but the resulting effects on daytime sleep propensity have never been reported. In the present study, the effects of phase shifts on daytime sleep propensity were measured by inducing moderate circadian phase shifts with timed bright-light exposure while maintaining normal sleep hours.

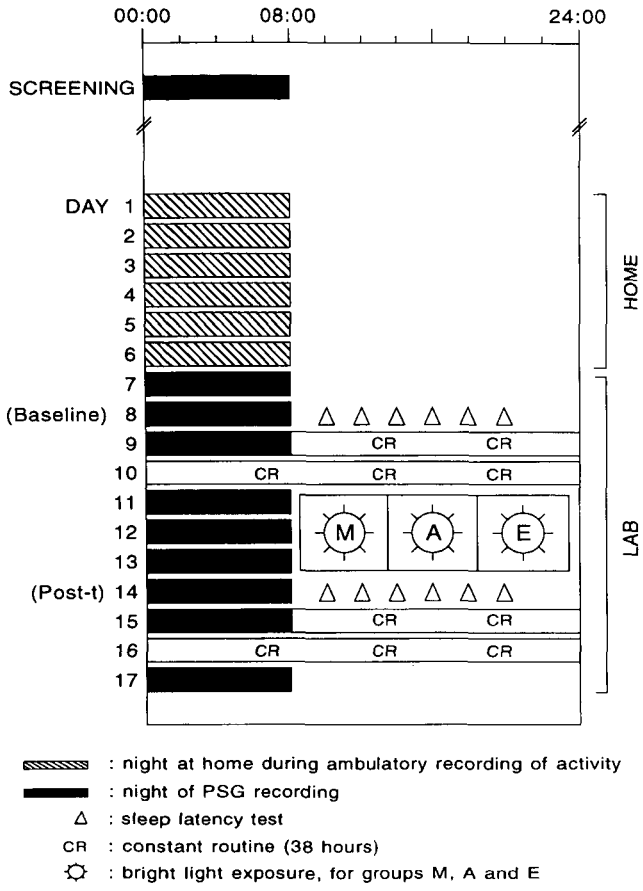

FIG. 1. Research protocol. The horizontal axis identifies hours from midnight to midnight. The vertical axis identifies the 17 days of the protocol. Baseline sleep-latency tests were conducted on Day 8, before the first constant routine, and post-treatment sleep latency tests on Day 14, following the last day of bright-light exposure. Group M received bright light in the morning, Group A in the afternoon, and Group E in the evening.

METHODS

Subjects

Twenty-three subjects (18 men and 5 women, 18–33 years old) completed the protocol. They were free of any drugs or medication, had no sleep or vigilance problem and provided 10-day sleep–wake logs showing a regular sleep–wake schedule. Good physical and psychological health were confirmed by interviews, questionnaires [medical, Minnesota Multiphasic Personality Inventory (MMPI), Beck Depression Scale], blood and urine tests, optometric exam (16), and all-night polysomnography recording. Volunteers with night work or transmeridian travel in the past 3 months, with extreme morning or evening chronotypes (17), or with any history of psychiatric or neurological disorders were excluded. All the subjects were nonsmokers and were required to abstain from alcohol, caffeine, and all medication for the duration of the study. The women were not taking any oral contraceptives and had regular menstrual cycles; they completed the laboratory part of the study during the follicular phase of their cycle, as confirmed by urinary luteinizing hormone (LH) tests administered during the last 3 days in the laboratory. All the subjects signed an informed consent approved by the institutional ethical committee and were paid for their participation.

Procedures

The experimental schedule is illustrated in Fig. 1. For each subject, the experiment lasted 17 consecutive days with 6 days at home and 11 days in the chronobiology laboratory. The research was conducted over a 3-year period, and all the experiments were performed in the summer, between May and September.

Subjects of the three groups were studied in random order over the 3 years of experimentation.

During the 6 days at home, each subject was required to maintain a strict sleep schedule with bedtime at midnight and waketime at 0800 hours (except for one subject: 2300–0700 hours). Naps were forbidden. Compliance was verified by 24-hour activity recording with an ambulatory monitor (Vitalog, Vilalog Monitoring Inc. or Mini-Logger, Mini-Mitter Co.). One subject did not respect his schedule and was excluded from the study. Night 7 was an adaptation night of polysomnography. During the evening of Day 7, the subjects were admitted to the chronobiology laboratory for the next 10 days. Night sleep was recorded by polysomnography and these results are reported elsewhere (18).

Bright light exposure

Timed bright light exposure was used to create moderate phase shifts according to the phase–response curve reported by Czeisler et al. in 1989 (9). Bright light was administered on three consecutive days (Days 11–13, see Fig. 1). The subjects were divided into three groups depending on the timing of the bright light exposure: a Morning group (n = 8, 6 men and 2 women), an Evening group (n = 8, 6 men and 2 women), and an Afternoon group (n = 7, 6 men and 1 woman). The groups were matched for age. Bright light exposure was administered for 5 hours each day: from 0830 to 1330 hours in the Morning group to create a circadian phase advance, from 1830 to 2330 hours in the Evening group to create a phase delay, and from 1330 to 1830 hours in the Afternoon group. This last group was used as a control group since no phase shift was expected after bright light exposure in the afternoon. One subject of the Evening group (woman, 26 years old) was exposed to bright light from 1730 to 2230 hours. This subject was used to a sleep schedule from 2300 to 0700 hours, and the protocol was exactly the same for her as for the other subjects except that all procedures were advanced by 1 hour. In the analyses, her results were averaged with those of the other subjects of her group as if they were obtained at the same clock hour.

Bright light was delivered by three panels 2 × 2 ft (Medic-Light), one facing the subject and one on each side, while the subject was sitting at a white table. Luminance varied from 10.5 to 14.5 kcd/m^2. Illuminance, measured at the subject's eye level every 10 minutes, ranged between 6 and 13 klux. The subjects were allowed to go outside, wearing sunglasses, for a maximum of 45 minutes at 1330 hours for the Morning group, 1725 hours for the Evening group, and 1225 hours for the Afternoon group. At all other times, the subjects remained in indoor lighting (<100 lux).

Core body temperature rhythm

Core body temperature was recorded during two 38-hour constant routines. The first constant routine occurred on Days 9–10, before the first bright light exposure, and the second constant routine on Days 15–16, after the second day of evaluation of daytime sleep propensity (see Fig. 1). Each constant routine started at 0800 hours and terminated at 2200 hours on the next day. During that time, the subjects stayed in a semi-recumbent posture, were not allowed to sleep, and were served 25 small snacks at regular intervals. The subjects stayed in bed all the time and used a urinal or a bedpan when necessary. Ambient lighting was ~40 lux on average, with a maximum of 70 lux at eye level. Body temperature was measured by a disposable rectal thermistor (Yellowsprings Instruments Co.) and recorded every 40 seconds either via a DC amplifier of the polygraph (Grass, 78D) (13 subjects) or via a Mini-Logger monitor (Mini-Mitter Inc.) (10 subjects). Clock time of the minimum of the circadian temperature rhythm was calculated by a two-harmonic regression model (20) and was used to estimate the endogenous circadian phase before and after the bright light treatment.

Multiple sleep latency test (MSLT)

Daytime sleep propensity was measured with the multiple sleep latency test (MSLT) conducted on Day 8, 2 days before the light treatment, and on Day 14, the day after the last bright light exposure (see Fig. 1). Sleep latency tests were administered according to standard procedures (21) and given six times each day at 2-hour intervals from 1000 hours. Subjects were in bed in a darkened and quiet room and were instructed to keep their eyes closed and let themselves fall asleep. Sleep latency was defined as the time from lights off to the beginning of the first minute of stage 1 or as 20 minutes if no sleep occurred. MSLTs were scored in 20-second epochs according to standard criteria (22) by an experienced sleep technician unaware of the experimental conditions.

Data analysis

The results were analyzed with ANOVA Group × Day for temperature and daily means of daytime sleep latencies and by an ANOVA Group × Day × Hour on individual sleep latency tests. The Group factor had three levels, Morning, Afternoon, and Evening groups;

TABLE 1. *Decimal hour (mean ± SEM) of the estimated minimum of the temperature rhythm recorded during a constant routine before and after bright light treatment. ANOVA results show the Group × Day interaction*

Groups	Before	After	Phase shift	ANOVA	Tukey
Morning	5.63 (0.42)	4.40 (0.42)	Advance: 1.23 (0.52)[a]	$F_{2,19} = 7.71$	M vs. E: $p < 0.005$
Evening	5.22 (0.46)	6.85 (0.61)	Delay: 1.62 (0.68)[a]	$p < 0.004$	M vs. A: ns
Afternoon	5.82 (0.39)	5.35 (0.44)	Advance: 0.48 (0.31)		E vs. A: $p < 0.05$

[a] $p < 0.05$, post-hoc paired t tests.

the Day factor had two levels, before and after the light treatment; and the Hour factor had six levels for the six sleep latency tests conducted each day. The Greenhouse-Geisser correction was applied for repeated measures. Since the direction of the expected circadian phase shifts were in the opposite direction for the Morning and the Evening groups, and since no effect was expected for the Afternoon group, a significant Group × Day interaction was predicted. Significant interactions were followed by pairwise between-group comparisons with Tukey tests conducted on difference scores (post- minus pre-treatment). Within-group changes were assessed by two-tailed post-hoc t-tests. For the analyses, daytime sleep latencies were log transformed to normalize the distributions.

RESULTS

Endogenous circadian temperature rhythm

Table 1 shows the circadian phase shifts observed in the temperature minima of the three groups of subjects, estimated during the constant routines before and after bright light treatment. Due to technical difficulties, temperature data from the second constant routine of one subject of the Morning group could not be analyzed, leaving the results of seven subjects for temperature phase shifts in this group. The significant Group × Day interaction shows that the light treatment did not produce the same effect in the three groups: the phase of the temperature minimum advanced in the Morning group and delayed in the Evening group. As expected, no significant phase shift was observed in the Afternoon group.

Daytime sleep latencies

Daily means

Analyses conducted on averaged daytime sleep latencies showed that the bright light treatment had a different effect according to the timing of bright light administration ($F_{GROUP\times DAY} = 12.31$; df = 2,20; $p < 0.001$). Figure 2 illustrates group averages for sleep latencies before and after treatment. Tukey tests showed that the subjects who received bright light in the morning reacted differently from those who received the treatment in the afternoon ($p < 0.05$) or in the evening ($p < 0.01$), whereas the effects in the latter two groups were similar. An ANOVA conducted on pre-treatment scores showed that mean daytime sleep latencies in the three groups were not statistically different ($p = 0.08$) before the administration of bright light treatment.

Diurnal variations

Figure 3 illustrates for the three groups the mean sleep latencies before and after treatment for each set of six tests. The results of the ANOVA Group × Day × Hour (with Greenhouse-Geisser correction) not only confirmed that bright light had a different effect on sleep propensity in the three groups but also showed that this interaction varied according to the time of day ($F_{GROUP\times DAY\times HOUR} = 2.22$, df = 7.02,70.23, $p < 0.05$).

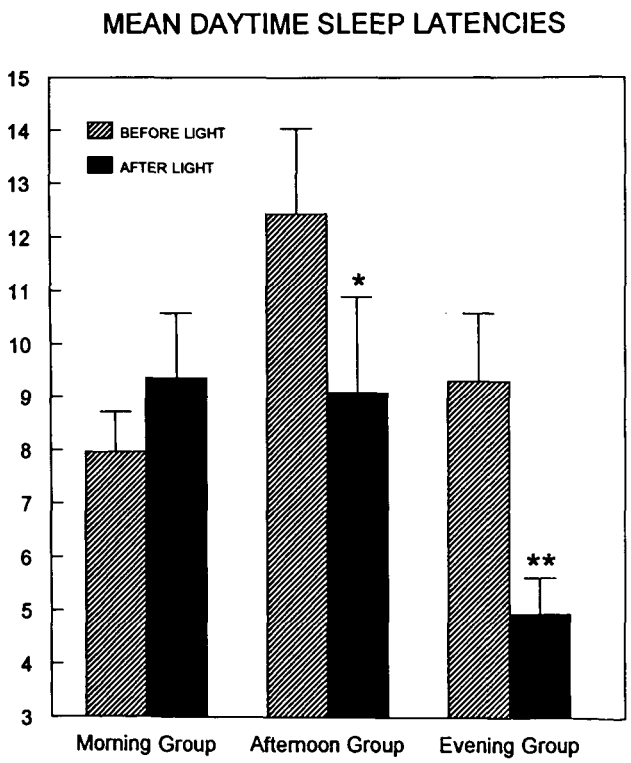

FIG. 2. Mean daytime sleep latencies in minutes (and SEM) averaged over the six tests administered on the day before (Before Light) and on the day after (After Light) bright light treatment (*$p < 0.05$, **$p < 0.01$, post-hoc paired t tests).

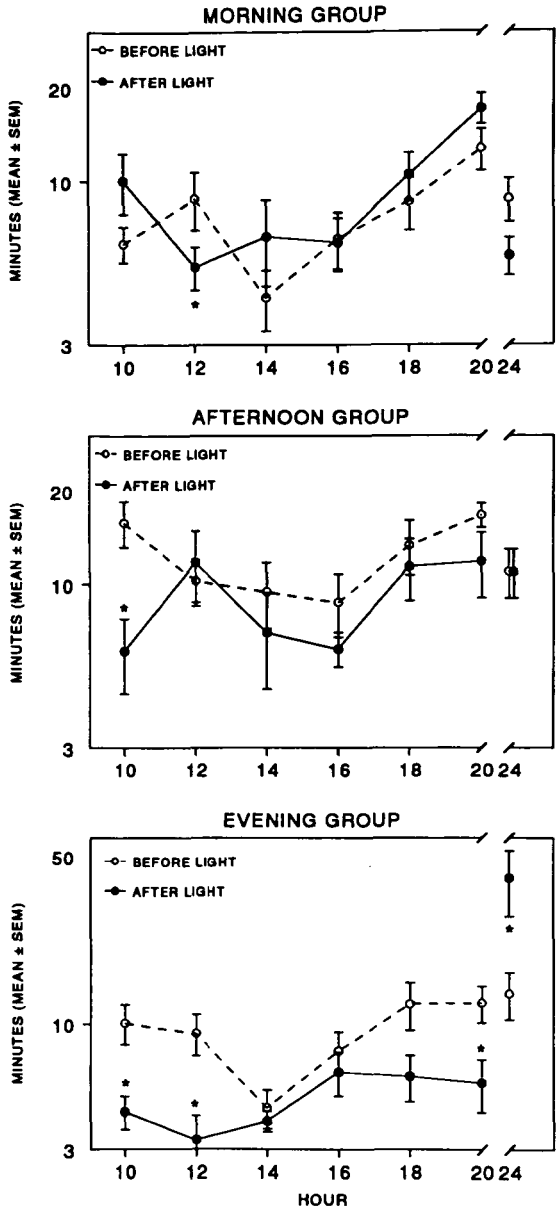

FIG. 3. Mean sleep latencies in minutes (and SEM) for each sleep-latency test, before and after bright light treatment, for each group of subjects. Mean sleep latencies at midnight (bedtime) are also shown. Untransformed data are represented on a log scale (*$p < 0.05$, post-hoc paired t tests).

Changes in mean sleep latencies were more pronounced at the beginning and at the end of the day, and ANOVAs conducted separately for each test indicated a significant Group × Day interaction for the 1000 hours ($p < 0.001$), 1200 hours ($p < 0.05$), and 2000 hours ($p < 0.05$) tests. As predicted by the Group × Day × Hour interaction, group differences were not the same at each of these times of day. Tukey tests revealed large differences ($p < 0.01$) between the Morning group and the two other groups at 1000 hours. At 1200 hours, the difference was only between the Evening and Afternoon groups ($p < 0.05$), whereas at 2000 hours it was between Morning and Evening groups ($p < 0.05$). ANOVAs conducted for each test on pre-treatment scores showed no difference between the three groups before bright-light administration except for the 1000 hours test ($p = 0.04$) when the mean sleep latency of the Morning group (6.33 minutes) was significantly shorter than the one of the Afternoon group (15.86 minutes, $p < 0.05$). Significant results of the post-hoc paired t tests within each group are shown in Fig. 3.

Sleep latency at midnight

The last daytime sleep latency test was performed at 2000 hours. There is no measure of sleep propensity available at 2200 hours, but since bedtime was fixed at midnight, the night-sleep latency can be used to give a measure of sleep propensity 4 hours after the last MSLT. This night-sleep latency is illustrated at 2400 hours in Fig. 3, along with daytime sleep latencies.

The ANOVA showed a significant Group × Day interaction [$F(2,20) = 5.4$, $p < 0.01$] with a different effect of treatment between the Evening group and the two other groups (Tukey tests, $p < 0.05$). Contrary to daytime sleep latencies, bedtime sleep latency tended to decrease in the Morning group (from 8.8 to 5.8 minutes, $p < 0.08$) and increased in the Evening group (from 13.4 to 40.4 minutes, $p < 0.05$); mean sleep latency remained the same in the Afternoon group (11.0 minutes).

DISCUSSION

Timed bright light exposure was used to change the phase of the circadian oscillator while the timing of the sleep episode was kept constant. Since the most sensitive part of the phase–response curve is located during the habitual sleep episode (19), only moderate phase shifts were expected. The circadian rhythm of core body temperature recorded during constant routines confirmed that significant phase advance and phase delay were achieved after bright light treatment in the Morning and Evening groups, respectively. As predicted by the phase–response curve, the circadian phase remained unchanged after bright light exposure in the afternoon.

Bright light exposure had a different effect on daytime sleep propensity according to the timing of bright light administration, and this interaction varied according to the time of day. After bright light treatment, mean sleep latencies decreased in the Afternoon and Evening groups and did not change significantly in the Morning group. The differences between the three

groups were more pronounced at the beginning and at the end of the day.

Since the afternoon bright light exposure was designed to act as a control condition, the decrease in mean sleep latency in this group suggests that the protocol itself increased the physiological sleep tendency. However, the sleep patterns during the nights preceding the daytime sleep-latency tests were similar, and total sleep time remained almost identical before and after the light treatment in all three groups. Alternatively, since the subjects had relatively low values on the pre-treatment MSLTs, it is possible that the requirement of a high sleep efficiency on the screening night in the inclusion criteria has caused a selection bias toward chronically sleep-deprived people (23). After 7 days in the laboratory, habituation and boredom may have contributed to unmasking a high physiological sleep tendency (24).

Since the three groups were not significantly different before the light treatment (except for the timing of bright light, they were subjected to the same protocol) this high physiological tendency must have affected the three groups equally. However, the results show that its effects on daytime sleep propensity may have combined differently with the circadian phase shifts produced with morning and evening bright light exposure. First, except for the 1200 hours test, sleep latencies did not decrease in the Morning group. Second, it is noteworthy that the sleep latencies in the Evening group were very short after the light treatment (Fig. 2).

Models of sleep–wake regulation suggest that the interaction between the circadian and homeostatic components is necessary to maintain a stable level of wakefulness in the daytime since the increase of the circadian drive to wakefulness counteracts the increase of the homeostatic sleep tendency (2,25). Circadian contribution to wakefulness is usually very low upon awakening in the morning, and wake propensity depends mostly on the quality of recuperation (the homeostatic component) (25). Given a high physiological sleep tendency, an optimal level of wakefulness can be very difficult to maintain in the morning, and high sleep propensity will be easily unmasked in an unstimulating environment. In the present study, evening bright light caused a delay in the circadian increase in wake propensity that, combined with the unmasking of a high physiological sleep tendency, may have contributed to the increased sleep propensity in the first two tests. On the other hand, the advance of the circadian increase in wake propensity may have helped the Morning group's subjects to maintain the same level of wakefulness in the morning.

Two important phase times of daytime sleep propensity are located in the evening: first, the early evening minimum in sleep propensity (SPMIN), located about 8 hours before the temperature minimum and, second, the "sleep gate", when circadian sleep propensity starts to increase, located about 3 hours after SPMIN (6,11,25,26). According to the phase of the temperature minimum estimated during the constant routines, SPMIN was expected to move from about 2138 hours before the light treatment to about 2024 hours after the treatment in the Morning group and to go from around 2113 hours to around 2251 hours in the Evening group. Similarily, the sleep gate was expected to advance to before midnight in the Morning group but to be delayed until after 1:00 a.m. in the Evening group. After the light treatment, no early evening minimum in sleep propensity was observed in the daytime sleep latencies of the Evening group. The last sleep latency test, conducted at 2000 hours, was probably too early to measure this zone, and the significant decrease in mean sleep latencies in the last test is consistent with a delay of the zone of low sleep propensity. Moreover, the increase of 27 minutes in mean sleep latency at midnight strongly suggests that bedtime occurred before the zone of low sleep propensity was over. Conversely, the trends to longer sleep latencies at 2000 hours and to shorter latencies at midnight in the Morning group are consistent with an earlier phase of both SPMIN and the sleep gate after the morning bright light treatment.

To our knowledge, there is no other study on the effects of an altered circadian phase in a stable sleep–wake cycle on daytime sleep propensity. However, the interpretation that these effects would be more easily observed in the presence of high sleep propensity is indirectly corroborated by a study comparing sleep propensity in subjects with morning or evening chronotypes, who were following similar sleep–wake schedules (27). According to their chronotype, it can be assumed that morning subjects had an advanced circadian phase compared to the evening subjects. In the first part of the study, sleep propensity was measured without sleep deprivation and there was no difference between the two groups of subjects. However, when the measures were repeated after a night of sleep deprivation, the results were completely different: compared to evening-type subjects, subjects with an early chronotype slept much less during the day, suggesting that an early circadian phase may attenuate the increased sleep propensity caused by sleep deprivation.

In conclusion, modest circadian phase shifts induced with bright light exposure have an effect on daytime sleep propensity, as measured by the MSLT. When the timing of the sleep–wake cycle is constant, a circadian phase shift alters the interaction between the homeostatic and circadian components of sleep propensity. The consequences of this change will be more appar-

ent when the physiological sleep tendency is high. In that case, as suggested by the present results, a phase delay will increase daytime sleep propensity, particularly in the morning and in the early evening, while a circadian phase advance may help to maintain a normal level of wakefulness. The positive effects of modest phase advances caused by morning bright light may have useful clinical applications in patients suffering from a low recuperative sleep.

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REFERENCES

1. Borbély AA, Achermann P, Trachsel T, Tobler I. Sleep initiation and initial sleep intensity: interactions of homeostatic and circadian mechanisms. *J Biol Rhythms* 1989;4:149–60.
2. Edgar DM, Dement WC, Fuller CA. Effect of SCN lesions on sleep in squirrel monkeys: evidence for opponent processes in sleep–wake regulation. *J Neurosci* 1993;13:1065–79.
3. Dijk DJ, 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;15:3526–38.
4. Weitzman ED, 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: 1018–30.
5. Carskadon MA, Dement WC. Sleepiness and sleep state on a 90-min schedule. *Psychophysiology* 1977;14:127–33.
6. Lavie P. To nap, perchance to sleep—Ultradian aspects of napping. In: Dinges DF, Broughton RJ, eds. *Sleep and alertness: chronobiological, behavioral, and medical aspects of napping.* New York: Raven Press, 1989:99–120.
7. Richardson GS, Carskadon MA, Orav EJ, Dement WC. Circadian variation of sleep tendency in elderly and young adult subjects. *Sleep* 1982;5:S82–94.
8. Czeisler CA, Weitzman ED, Moore-Ede MC, Zimmerman JC, Knauer RS. Human sleep: its duration and organization depend on its circadian phase. *Science* 1980;210:1264–7.
9. Zulley J, Wever R, Aschoff J. The dependence of onset and duration of sleep on the circadian rhythm of rectal temperature. *Pflugers Arch* 1981;391:314–8.
10. Strogatz SH, Kronauer RE, Czeisler CA. Circadian regulation dominates homeostatic control of sleep length and prior wake length in humans. *Sleep* 1986;9:353–64.
11. Lack LC, Lushington K. The rhythms of human sleep propensity and core body temperature. *J Sleep Res* 1996;5:1–11.
12. Czeisler CA, et al. Bright light resets the human circadian pacemaker independent of the timing of the sleep–wake cycle. *Science* 1986;233:667–71.
13. Lewy AJ, Sack RL, Miller LS, Hoban TM. Antidepressant and circadian phase-shifting effects of light. *Science* 1987;235:352–3.
14. Drennan M, Kripke DF, Gillin JC. Bright light can delay human temperature rhythm independent of sleep. *Am J Physiol* 1989;257:R136–41.
15. Dijk DJ, Visscher CA, Bloem GM, Beersma DGM, Daan S. Reduction of human sleep duration after bright light exposure in the morning. *Neurosci Lett* 1987;73:181–6.
16. Terman M, Remé CE, Rafferty B, Gallin PF, Terman JS. Bright light therapy for winter depression: potential ocular effects and theoretical implications. *Photochem Photobiol* 1990;51:781–92.
17. Horne JA, Ostberg O. A self-assessment questionnaire to determine morningness–eveningness in human circadian rhythms. *Int J Chronobiol* 1976;4:97–110.
18. Carrier J, Dumont M. Sleep propensity and sleep architecture after bright light exposure at three different times of day. *J Sleep Res* 1995;4:202–11.
19. Czeisler CA, et al. Bright light induction of strong (type 0) resetting of the human circadian pacemaker. *Science* 1989;244: 1328–33.
20. Brown EN, Czeisler CA. The statistical analysis of circadian phase and amplitude in constant-routine core-temperature data. *J Biol Rhythms* 1992;7:177–202.
21. Carskadon MA, Dement WC, Mitler MM, Roth T, Westbrook PR, Keenan S. Guidelines for the multiple sleep latency test (MSLT): a standard measure of sleepiness. *Sleep* 1986;9(4): 519–24.
22. Rechtschaffen A, Kales A. *A manual of standardized terminology, techniques and scoring system for sleep stages of human subjects.* Washington, DC: Public Health Service, U.S. Government Printing Office, 1968.
23. Roehrs T, Timms V, Zwyghuizen-Doorenbos A, Buzenski R, Roth T. Polysomnographic, performance, and personality differences of sleepy and alert normals. *Sleep* 1990;13:395–402.
24. Carskadon MA, Dement WC. The multiple sleep latency test: what does it measure. *Sleep* 1982;5:S67–72.
25. Dijk DJ, Czeisler CA. Paradoxical timing of the circadian rhythm of sleep propensity serves to consolidate sleep and wakefulness in humans. *Neurosci Lett* 1994;166:63–8.
26. Strogatz SH, Kronauer RE, Czeisler CA. Circadian pacemaker interferes with sleep onset at specific times each day: role in insomnia. *Am J Physiol* 1987;253:R172–8.
27. Lavie P, Segal S. Twenty-four-hour structure of sleepiness in morning and evening persons investigated by ultrashort sleep–wake cycle. *Sleep* 1989;12:522–8.