The effect of narrowband 500 nm light on daytime sleep in humans

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Abstract

Naps frequently take place during the daytime under some ambient light. People are commonly advised to wear eyeshades, or use black-out curtains while sleeping, as light is thought to inhibit sleep. Little is known, however, about how light during daytime sleep may affect the quality or architecture of that sleep. The present within-subjects design administered green narrowband light via light masks to 17 young adults (23.2 ± 4.7 years) during four 90-minute afternoon naps. Subjects were exposed to each of four light conditions that approximate the intensity of 1) physiological darkness (~0 lx), 2) moonlight (~1 lx), 3) typical indoor lighting (~80 lx) and 4) indirect outdoor light (~6400 lx). All subjects were able to sleep in all lighting conditions, with no differences in sleep quality or architecture. Power analysis revealed sufficient power to detect meaningful differences. Sleep inertia measured upon waking showed a general effect of the nap, independent of condition. Although light has various alerting effects at night, 500 nm LED light presented via light mask does not appear to inhibit daytime sleep. The finding that this light had no effect on the ability of individuals to fall asleep or stay asleep during an afternoon nap may inform decisions regarding the use of the nap as a facilitator of schedule adjustment, and challenges the assumption of light as a barrier to napping.

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1. Introduction

Napping is an effective tool for managing sleep deprivation and dysrhythmia brought about by chronic or acute circadian disruption, both pervasive concerns that produce serious deficits in performance and health [1–3]. These negative physical and psychological symptoms are improved by daytime sleep and naps during nightshifts [4–7]. In healthy, well-rested subjects as well, napping can enhance cognitive performance across a range of memory tasks [8–12]. By necessity, naps often take place under some light exposure, and healthy sleep recommendations commonly include creating a darkened environment by means of eyeshades or black-out curtains [61–64]. Despite these recommendations, we are aware of no controlled empirical studies examining the effect of light intensity on the quality or architecture of daytime sleep. This is the aim of the current study.

Light is a powerful modulator of myriad physiological systems in human and non-human animals, altering mood, hormone secretion, fatigue, and phase of the circadian pacemaker [13–17]. Light may influence sleep through either of two mechanisms that jointly govern its timing and architecture [18,19]: first, a homeostatic process sensitive to prior waking time and sleep history and second, a circadian process that produces a daily rhythm in sleep propensity. Effects of light on the latter process are well established: light early and late at night resets the circadian clock in opposite directions, whereas light during the day has little resetting effect. With regard to homeostatic sleep processes, light affects a variety of measures of alertness in human subjects, including awake EEG, psychomotor vigilance task, self-reported sleepiness [13], and does so in a dose-dependent manner, with strongest effects at illuminations >1000 lx, and half the efficacy at common room lighting intensities of ~100 lx [20]. In contrast to circadian phase-resetting effects, alerting effects of light persist into the day [21–25], although null results have also been reported in one study of sleep-restricted subjects [26].

For ocular light to influence brain activity during sleep, it must necessarily pass through closed eyelids. Estimates of transmission of green light across adult human eyelids range from 0.3% to 2% measured psychophysically or physically, respectively [27–29]. Even with marked attenuation, light administered through eyelids during the last hours of nocturnal sleep via goggles shortened sleep durations of humans most likely as a result of a phase-shifted circadian pacemaker [30,31], although the possibility of subjects opening their eyes was not excluded. Endocrine actions of light during sleep are also suggested by regulation of human menstrual cycles with manipulations of bedroom lighting [32]. With respect to the alerting effects of light, bright flashes during sleep, designed as a smoke alarm for the deaf, can awaken some subjects [33], but not consistently, and more often while subjects are in REM sleep than slow-wave sleep. We are unaware of any polysomnographic studies of tonic light effects in sleeping humans. In nocturnal rodents, however, light clearly alters
both the timing and architecture of sleep. Mediated by intrinsically photosensitive retinal ganglion cells containing the photopigment melanopsin [34,35], sleep can be induced in mice by nighttime light pulses. In rats, daytime sleep in the presence of light has lower efficiency and reduced REM compared to that in darkness [36,37]. Importantly, a recent paper suggests that neonatal mice avert their gaze from light, even before their eyelids open and their visual system has developed [38].

Notwithstanding species differences, the various findings described above, as well as anecdotal experience, may underlie the conventional wisdom that light should be avoided during napping, a conclusion that poses a barrier to the implementation of napping without having been formally tested. Whether certain lighting conditions are more favorable for daytime sleep than others should be determined if naps are to be encouraged in the general population or used as facilitators of schedule adjustment in dysrhythmic or sleep-deprived individuals. We therefore presented human subjects with four intensities of light that approximate 1) physiological darkness (0 lx), 2) moonlight (-1 lx), 3) typical indoor lighting (-80 lx) and 4) indirect outdoor light (-6400 lx) to determine the effect of constant light on daytime sleep quality and architecture. Consistent with other studies of light effects in humans, narrowband light was selected to permit quantitative comparisons across studies. Green light of 500 nm was chosen specifically because this wavelength falls near the optimal sensitivity of photoreceptive mechanisms implicated in circadian and sleep physiology [34,35] thereby maximizing the potential efficacy of light delivered without raising concerns about potential damaging effects of shorter wavelength light. Furthermore, the present implementation expands on past work with the same light source wherein the stimuli was physiologically effective in human subjects [31].

2. Methods

2.1. Participants

All subjects gave their written informed consent, and the research protocol was approved by the University of California, San Diego Human Research Protections Program. Twenty-three UCSD graduate and undergraduate students with no medical conditions were recruited and screened in an in-person clinical interview for sleep and psychiatric disturbances (13 females, 10 males; 21.9 ± 4.1 y). Subjects were also screened for excessive Morningness and Eveningness using the Horne–Ostberg Morning and Eveningness Questionnaire [39] and for excessive daytime sleepiness using the Epworth Sleepiness Scale [40]. Additional exclusion criteria included the use of any medications, consumption of more than 300 mg of caffeine/day, current drug use, color-blindness, and individuals exhibiting an erratic sleep–wake schedule (e.g. shift workers).

2.2. Procedure

All naps were conducted in the laboratory. Subjects were asked to maintain regular sleep schedules consisting of weekly averages of no less than 7 h per night with consistent bedtimes before 1:00 am. Data from sleep diaries and actigraph watches were gathered for a baseline week prior to the first nap and for each night prior to subsequent naps to ensure compliance. Subjects were asked to refrain from alcohol and caffeine beginning at 12 pm the day prior to each nap. Additionally, subjects were asked to refrain from exercise for several hours before the nap, and diaries were examined for aberrant behavior upon arrival at the laboratory. In one instance, a subject was asked to reschedule a nap as he had exercised too close to nap time. On each day of treatment, subjects arrived to the laboratory 1 h prior to their nap to be hooked up to the polysomnography equipment, which included standard electroencephalographic (EEG), electro-oculographic (EOG) and electromyographic (EMG) measures for recording sleep. Nap start times were between 1:00 pm and 3:30 pm and were consistent within subject. During sleep, each participant was exposed to one of the four lighting conditions (in lux: 0, 1, 80, or 6400; in W/cm²: 0, 4.46 × 10⁻⁻⁷, 2.66 × 10⁻⁻⁵, or 4.46 × 10⁻⁻³ and in photon flux: 0, 1.12 × 10¹², 6.69 × 10¹³, or 1.12 × 10¹⁶, respectively) via goggles with a diffused green (500 nm) LED light previously shown to be safe for human subjects [31]. Subjects were instructed to keep their eyes closed for the duration of the exposure. Conditions were counterbalanced and each nap was separated by a minimum of 1 week and occurred on the same day of the week to control for prior sleep schedule (except in one case, where two naps were separated by 3 days). For approximately half of the subjects (n=8), a habituation nap was added to control for possible order effects. Participants were in bed for a period of 90 min in each condition, and sleep was monitored as it occurred.

Before and after each nap, subjects were administered the Karolinska Sleepiness Scale [41], and 11 out of the 17 subjects were also administered the Digit Subtraction Task, a measure of sleep inertia [42]. Following every nap, subjects completed post-nap questionnaires regarding the subjective experience of the nap and the light masks, including a measure of discomfort.

2.3. Actigraphy

Actigraphy data were collected using the Actiwatch-64 (Respironics; Bend, OR), a monitor that measures wrist movement and provides a measure of sleep duration. Data were scored using the automatic minor rest interval (AMRI) detection of the Respironics Actiware 5.52.0003 program, were sampled every 30 s, and computed Total Sleep Time was used to verify adherence to sleep schedule.

2.4. Polysomnography

Polysomnographic data were collected during naps using Astro-Med Grass Heritage Model 15 amplifiers with Grass Gamma software. A central EEG derivation was used, with scalp EEG and EOG electrodes referenced to unlinked opposite mastoids and submental muscle tone EMGs attached under the chin. A 60 Hz notch filter was also utilized to eliminate potential background noise. At the beginning of each recording, an internal 50 μV calibration signal was generated followed by impedance checks and biocalibrations. EEG data was digitized at a sampling rate of 256 Hz and were imported to Pass Plus waveform analysis software (Delta Software, St. Louis, MO). Data were scored blind to condition in 30 s epochs according to Rechtschaffen and Kales sleep staging criteria [43]. Sleep latencies were calculated relative to lights off and do not include additional time spent in bed during EEG impedance checks and biocalibrations before lights off.

2.5. Masks

Light was administered via modified Phototherapy Masks (see [31] for full description). The foam face pad and elastic straps were replaced with a cushioned gel mask to increase subjective comfort and for ease of sanitation. This modification resulted in a reduction of maximal intensity to approximately 6400 lx. Additionally, although the lux unit is used for measuring the effects of light on cone-based visual responses and is therefore not an ideal measure for a physiological response based on melanopsin photoreception, lux measures are retained throughout the manuscript because this unit is the most widely reported and most easily used as a benchmark for various commonly experienced lighting conditions (e.g., outdoors, bedroom etc). Light measurements were taken at the beginning, middle and end of the study using a Light Meter 840020 (Sper Scientific, Scottsdale, AZ) and wavelength of the emitted light was determined using a USB2000 spectrometer and Spectrasuite Software (OceanOptics, Inc).
2.6. Analysis

The effect of the four light intensities on sleep quality was tested using a repeated-measures ANOVA in SPSS (version 17, SPSS Inc., Chicago, IL). Planned tests included the effect of light on six dependent variables, three addressing sleep quality: Total Sleep Time (TST), Sleep Latency (SL) and Wake After Sleep Onset (WASO); and three addressing sleep architecture: percentage of Stage 2, Slow Wave Sleep and Rapid Eye Movement Sleep (%Stage2, %SWS and %REM, respectively). Other dependent measures included the percent of Stage 1 sleep and REM Latency, and data from the questionnaires, including discomfort during the nap and alertness pre- and post-nap as measured objectively by the Digit Subtraction Task (DST) and subjectively by change scores in the Karolinska Sleepiness Scale (KSS).

Due to scheduling and technical difficulties, 6 subjects (4 females and 2 males) were missing data from one or more naps and were therefore not included in the final analysis. No subjects were excluded for being unable to sleep. Unless otherwise noted, all effects were tested at the 0.05 significance level.

3. Results

3.1. Polysomnography

All subjects were able to sleep for the majority of the 90 min rest period in all of the lighting conditions (mean±sd = 77.0±10.5). No main effects of light were found on any of the primary dependent measures of sleep quality (Total Sleep Time, F(3,48) = 1.63; Sleep Latency, F(3,30) = 0.81 and Wake After Sleep Onset, F(3,48) = 2.62, with Huynh–Feldt correction for violation of sphericity and all p values ns). Furthermore, light did not significantly affect the architecture of daytime sleep (% Stage2, F(3,48) = 1.01; %SWS, F(3,48) = 0.56; and % of Rapid Eye Movement Sleep, F(3,48) = 0.93 and all p values ns) (Fig. 1).

Post hoc tests of other sleep measures, including the effect of light on Stage 1, REM Latency, and Sleep Efficiency, also revealed no effect of light. Further, no outcomes differed when gender was included as a covariant (all p values ns), nor did they differ when those with habituation naps were compared to those without (all p values ns).

Visual inspection of the data plotted within subjects across conditions yielded no suggestion of a discernable pattern in any of the dependent measures, nor did visual inspection of the condition means. With 17 complete subjects, we had power of 0.87 to detect a 10 min difference in Total Sleep Time, power of 0.99 to detect a 5 min difference in Sleep Latency, and power of 0.99 to detect a difference of 10 min in Wake After Sleep Onset.

3.2. Minutes awake and prior sleep

Because naps took place at multiple time points in the afternoon (1:00 pm, 2:00 pm, or 3:30 pm) and there was some variability in wake times (sd = 91 min), both the number of minutes awake before the nap (mean = 358.9, sd = 100 min) and prior nocturnal sleep length (mean = 412.2, sd = 80 min) varied. As previous studies report effects of these measures on sleep architecture [44,45], Pearson product–moment correlation coefficients were calculated within each condition between all sleep variables and both prior night’s TST and the number of minutes of wakefulness. The percent of REM sleep in the nap and the number of minutes awake before the nap were correlated in Conditions 3 and 4 (r = −0.53 and −0.53, respectively, p < 0.05, Fig. 2), and were in the expected direction but did not reach significance in the other conditions. The data were therefore analyzed excluding the three subjects with the fewest number of minutes awake.

Fig. 1. No differential effects of light intensity on measures of daytime sleep. Estimated marginal means ± SEM of measures of sleep time (A–C) and sleep architecture (D–F) for each lighting condition (n = 17).
awake prior to naptime, and were also analyzed excluding the three subjects with fewest minutes of prior nocturnal sleep. The outcomes of the tests were not changed based on these exclusions, and therefore all complete subjects were included in the final analysis.

3.3. Discomfort, alertness, and sleep inertia

Light intensity did not affect the level of subjectively reported discomfort during the nap (mean ± SEM = 2.69 ± 0.41, 2.44 ± 0.44, 2.31 ± 0.44, and 3.06 ± 0.46 for 0 lx, 1 lx, 80 lx, and 6400 lx, respectively; F(3,30) = 0.70, ns) nor did it affect the level of alertness as measured by the KSS scores after the nap (mean ± SEM = 4.63 ± 0.63, 4.63 ± 0.60, 4.13 ± 0.93, and 4.38 ± 0.57 for 0 lx, 1 lx, 80 lx, and 6400 lx, respectively; F(3,30) = 0.22, ns), and by the change in KSS scores (post-pre; mean ± SEM = 0.00 ± 0.16, −0.06 ± 0.02, 0.50 ± 0.19, and −0.38 ± 0.14 for 0 lx, 1 lx, 80 lx, and 6400 lx, respectively; F(3,30) = 0.46, ns). The objective measure of alertness, correct responses on the DST, was analyzed using a two-way within-subject ANOVA with light and time objective measure of alertness, correct responses on the DST, was not significantly different across the four experimental conditions (F(3,45) = 1.08, ns) nor did it affect the level of alertness as measured by the KSS scores after the nap (mean ± SEM = 4.63 ± 0.63, 4.63 ± 0.60, 4.13 ± 0.93, and 4.38 ± 0.57 for 0 lx, 1 lx, 80 lx, and 6400 lx, respectively; F(3,45) = 0.22, ns) and by the change in KSS scores (post-pre; mean ± SEM = 0.00 ± 0.16, −0.06 ± 0.02, 0.50 ± 0.19, and −0.38 ± 0.14 for 0 lx, 1 lx, 80 lx, and 6400 lx, respectively; F(3,45) = 0.46, ns). The objective measure of alertness, correct responses on the DST, was analyzed using a two-way within-subject ANOVA with light and time objective measure of alertness, correct responses on the DST, was not significantly different across the four experimental conditions (F(3,45) = 1.08, ns) nor did it affect the level of alertness as measured by the KSS scores after the nap (mean ± SEM = 4.63 ± 0.63, 4.63 ± 0.60, 4.13 ± 0.93, and 4.38 ± 0.57 for 0 lx, 1 lx, 80 lx, and 6400 lx, respectively; F(3,45) = 0.22, ns). The objective measure of alertness, correct responses on the DST, was analyzed using a two-way within-subject ANOVA with light and time objective measure of alertness, correct responses on the DST, was not significantly different across the four experimental conditions (F(3,45) = 1.08, ns). The objective measure of alertness, correct responses on the DST, was analyzed using a two-way within-subject ANOVA with light and time objective measure of alertness, correct responses on the DST, was not significantly different across the four experimental conditions (F(3,45) = 1.08, ns) nor did it affect the level of alertness as measured by the KSS scores after the nap (mean ± SEM = 4.63 ± 0.63, 4.63 ± 0.60, 4.13 ± 0.93, and 4.38 ± 0.57 for 0 lx, 1 lx, 80 lx, and 6400 lx, respectively; F(3,45) = 0.22, ns) and by the change in KSS scores (post-pre; mean ± SEM = 0.00 ± 0.16, −0.06 ± 0.02, 0.50 ± 0.19, and −0.38 ± 0.14 for 0 lx, 1 lx, 80 lx, and 6400 lx, respectively; F(3,45) = 0.46, ns).

Fig. 3. Sleep inertia effect over all naps, with no effect of light intensity. Difference scores for number correct on the Digit Subtraction Task (post−pre). Negative values across conditions reflect the overall effect of time (subjects had sleep inertia following all naps), with no difference between lighting conditions.

4. Discussion

Unexpectedly, even the brightest 500 nm light did not interfere with the subjects’ ability to fall or stay asleep during an afternoon nap, suggesting that the reported alerting effects of light may not extend to daytime sleep in a laboratory environment. As noted above, we had reasonable statistical power to detect meaningful effects and therefore believe that any possible alerting effect was smaller than would be clinically significant. That is, if an individual has a 90 min opportunity to nap during the afternoon, the < 5 min difference in sleep onset that may be caused by lighting conditions would not likely be meaningful. Also of note is the fact that subjects did not report significantly more discomfort in the brighter conditions, nor did the level of alertness upon waking vary across conditions. These results have implications for both the use of light masks as a therapeutic treatment and for the use of daytime naps as treatment for dysrhythmia or sleep deprivation.

Several methodological issues warrant comment. First, as a green narrowband light was used, it is possible that our findings are specific to green light with 500 nm wavelength. With open eyes, humans are alert most by light between 435 and 530 nm [46–49], and circadian responses are optimized in a similar range [17,50–52]. The retinal photopigment, melanopsin, which mediates both circadian and sleep modulating-responses to nighttime light in rodents [34,35], exhibits peak absorption around 480 nm. Moreover, these exact light-emitting masks (unmodified) were previously shown to be physiologically effective in human subjects as measured by Grandner [31]. Thus, compared to broad-spectrum white light, the green narrowband light in the present study should be expected to nearly maximally activate the photoreceptive pathways already implicated in light-regulation of sleep, arousal and circadian phase.

Many, but not all, non-visual effects of light exhibit a logistic sigmoidal dependence on intensity. Use of light intensities spanning 3.5 orders of magnitude enables detection of such dependence as well as any effects that might be intensity-specific. Stimulus values were chosen for their representation of various scenarios that would be routinely encountered by nappers under non-laboratory conditions (e.g., sleeping in a completely darkened room (0 lx); in the presence of computer displays (1 lx); or on a hammock under a shade tree (6400 lx)). Given that 500 nm light is attenuated by the eyelids by 98 to 99.7% [27,29], our dimmest light condition (1 lx) would represent an effective retinal illuminance of 0.003–0.02 lx. In rodents, light of this intensity is too dim to suppress melatonin or markedly shift circadian phase, but is nonetheless physiologically relevant as it potently alters circadian period and waveform [53]. Analogous effects of very dim light are known in a variety of non-human mammals [54]. The brightest condition (6400 lx) delivered 19–128 lx to the retina. This intensity substantially exceeds the light intensity sufficient for many physiological effects of light, including nighttime melatonin suppression, enhanced alertness, and phase shifting [23,47,50,55,56]. Thus, we were prepared to observe threshold, logarithmic or intensity-specific effects of light on sleep measures. Instead, the lack of effect of any intensity suggests that sleep measures are insensitive to medium wavelength light at least as measured in healthy young adult in the laboratory during the day.

In spite of efforts to control the sleep schedules of the participants, a limitation of this study is the variation both in the amount of sleep subjects received prior to the nap and in the number of hours spent awake before the nap, with resultant variation in the amount of homeostatic pressure to sleep during the afternoon opportunity. These variables predicted subsequent napping performance within condition as it did in other studies [44,45], validating the sensitivity of our dependent measures. The counterbalanced, within-subjects design is a
strength of the present experiment and minimizes concerns of individual differences in baseline arousal levels and duration of prior nocturnal sleep, which we can’t exclude as relevant in settings outside the laboratory. In this population, which includes some mildly sleep-deprived subjects, it remains possible that more complex interactions of light on circadian phase, nocturnal sleep, or other variables could be of significance outside the laboratory. The average amount of sleep on the night prior to the nap was slightly less than 7 h, and although this is less than the 8 h ideal, it is consistent with the U.S. national average (National Sleep Foundation Sleep Poll, 2009) and therefore represents a “normally” rested population that may benefit from napping. Whether effects might be seen in a “fully” rested, but less representative, sample remains to be determined (i.e., mildly arousing bright light could potentially inhibit sleep in a well-rested subject). Issues of prior sleep time and wakefulness were addressed by exclusion of the most extreme cases from analysis. The results of the subsequent analyses, as noted earlier, did not differ. While the low sleep latencies reported in this study may reflect the somewhat restricted previous night’s sleep, it is important to note that subjects spent an additional 15–20 min spent in bed before lights off during EEG impedance checks and biocalibrations, yielding lower values than are reported elsewhere [57,58]. Additionally, as subjects were allowed to resume most of their regular activities throughout the study and only reported to the laboratory for each nap, it is possible that individual variation in activity level may have affected sleep variables in some fashion. However, subject diaries were screened for aberrant behavior prior to each nap.

The sleep inertia following a nap confirms earlier reports [42] and should be a consideration in any recommendations or personal decisions regarding napping. It is important to note that the effect was measured within the first 3 min of waking, and not thereafter. It is unknown if these effects would persist beyond that time frame. Additionally, it is possible that greater sleep inertia effects would be seen if measured at a different time of day.

Because nighttime sleep was not assessed in the present study, it is unclear whether the insensitivity of sleep to green light occurs throughout the circadian cycle or only during the day. In the circadian domain, light at night, but not in the day, induces robust phase shifting in humans (and thus the present treatments would be highly unlikely to exert any effect on circadian phase). Additionally, human perceptual thresholds for light [59], and various alerting effects of light (e.g. awake EEG and PVT) change across the course of the day [13], although daytime alerting effects have also been observed in subjects awake in dim light (< 10 lx) [23,60]. Whether the daytime effects on common measures of alertness (such as EEG power density) are dissociable from those on sleep quality and architecture remains to be determined. Regardless, the empirical results establish that even bright light in the afternoon does not interfere with intentional napping. Many participants in this study were not habitual nappers and indeed most expressed concern as to whether or not they would be able to sleep in the middle of the day in a novel environment with light shining in their eyes. Yet they did. Although the present lighting conditions were tested in healthy and reasonably-rested adults, napping may be of use to any individual suffering from mild dysrhythmia or sleep deprivation, and may be implemented in a wide range of lighting conditions. Additionally, in treating severe dysrhythmia, as seen in shift workers or jetlagged individuals, light could conceivably be administered during sleep to assist in shifting biological rhythms and subsequent sleep/wake cycles. Further research into the effects of light during daytime sleep may be useful, not only for those who use naps as a means to adjust to shift-work schedules or recover from jetlag, but to any individual who occasionally engages in daytime sleep. This study is a first and important step in understanding how light may interfere with daytime sleep, but should be repeated with polychromatic light to extend the generalizability of these findings to real world situations.

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