Daytime Exposure to Bright Light, as Compared to Dim Light, Decreases Sleepiness and Improves Psychomotor Vigilance Performance

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Study Objectives: This study examined the effects of bright light exposure, as compared to dim light, on daytime subjective sleepiness, incidences of slow eye movements (SEMs), and psychomotor vigilance task (PVT) performance following 2 nights of sleep restriction.

Design: The study had a mixed factorial design with 2 independent variables: light condition (bright light, 1,000 lux; dim light, < 5 lux) and time of day. The dependent variables were subjective sleepiness, PVT performance, incidences of SEMs, and salivary melatonin levels.

Setting: Sleep research laboratory at Monash University.

Participants: Sixteen healthy adults (10 women and 6 men) aged 18 to 35 years (mean age 25 years, 3 months).

Interventions: Following 2 nights of sleep restriction (5 hours each night), participants were exposed to modified constant routine conditions. Eight participants were exposed to bright light from noon until 5:00 pm. Outside the bright light exposure period (9:00 am to noon, 5:00 pm to 9:00 pm) light levels were maintained at less than 5 lux. A second group of 8 participants served as controls for the bright light exposure and were exposed to dim light throughout the entire protocol.

Measurements and Results: Bright light exposure reduced subjective sleepiness, decreased SEMs, and improved PVT performance compared to dim light. Bright lights had no effect on salivary melatonin. A significant positive correlation between PVT reaction times and subjective sleepiness was observed for both groups. Changes in SEMs did not correlate significantly with either subjective sleepiness or PVT performance.

Conclusions: Daytime bright light exposure can reduce the impact of sleep loss on sleepiness levels and performance, as compared to dim light. These effects appear to be mediated by mechanisms that are separate from melatonin suppression. The results may assist in the development of treatments for daytime sleepiness.

Key Words: Bright light, sleep loss, sleepiness, psychomotor vigilance performance, slow eye movements, electrooculogram, melatonin

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INTRODUCTION

EXCESSIVE SLEEPINESS IS NOW CONSIDERED THE MAJOR CAUSE OF ACCIDENTS IN ALL MODES OF TRANSPORTATION.[1,2] Shift workers, who work outside the regular 8:00 AM to 5:00 PM day, comprise approximately 20% of the population in an urban economy.[3] These individuals often report excessive daytime sleepiness[4,5] caused by their irregular sleep patterns and sleep loss. It is estimated that shift work reduces sleep time by on average 3 to 4 hours.[6]

One of the major determinants of the sleep-wake cycle is the circadian system. In mammals, circadian (~ 24-hour) rhythms are generated and maintained by a set of small bilaterally paired hypothalamic nuclei, the suprachiasmatic nuclei.[7] The light-dark cycle is the major synchronizing agent or time cue for circadian rhythms. Although it was once thought that bright light is required to phase shift the human circadian pacemaker, there is now considerable evidence that the pacemaker is sensitive to ordinary room light.[8,9] Light information is transmitted to the suprachiasmatic nuclei via a specialized pathway, the retinohypothalamic tract, from nonvisual photoreceptors in the retina.[10]

In addition to the synchronizing properties of light, some studies report that nocturnal exposure to bright light has a direct alerting effect in humans. For example, bright light is reported to reduce subjective assessments of sleepiness, increase sleep latency, and improve neurobehavioral performance.[11-17] In addition, nocturnal bright light exposure has been shown to reduce electrooculogram (EOG) correlates of sleepiness such as the incidences of slow eye movements (SEMs) and decrease low frequency electroencephalographic (EEG) activity.[16] However, these reports are controversial, as others have found that nocturnal bright light has no beneficial effect on neurobehavioral performance,[18-20] and 1 study has reported detrimental effects of nocturnal bright light.[20]

Exposure to bright light during the night also suppresses the synthesis and secretion of the pineal hormone melatonin.[21,22] Melatonin synthesis and secretion occur mainly during the dark phase of the light-dark cycle, and its rhythmic secretion is controlled by the suprachiasmatic nuclei via a sympathetic pathway.[23] Several lines of evidence indicate that the circadian rise in endogenous melatonin is closely associated with the onset of nocturnal sleepiness in humans,[24-28] and exogenous melatonin can also promote sleep and sleepiness.[29-33] Furthermore, the hyperthermic response to bright light, which occurs coincidentally with melatonin suppression, is inhibited by exogenous melatonin.[34,35] Therefore, it has been suggested that nocturnal bright light exposure may reduce sleepiness by its suppression of melatonin synthesis.[12] Supporting this proposition, Cajochen et al observed dose-dependent effects of nocturnal bright light exposure on sleepiness and neurobehavioral performance and showed that these effects were closely associated with suppression of plasma melatonin levels.[16]

The vast majority of previous studies examining the influence of bright light exposure have scheduled testing during the nighttime, in order to coincide with the circadian decline in performance and alertness levels. While the results of these studies may be relevant to night-shift work situations, the potential for bright light to be used to improve alertness and performance levels during the daytime has not been extensively studied. Badia and colleagues[12] examined the effects of daytime and

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nighttime bright light (between 5,000 and 10,000 lux) and dim light (50 lux) exposure on body temperature, alertness, EEG activity, and psychomotor performance. In contrast to nighttime bright light exposure, no significant effect of daytime bright light was observed. Similarly, in another study, daytime bright light exposure did not affect subjective alertness, sleep latencies, or psychomotor vigilance task (PVT) performance.[20]

A significant methodologic issue in testing the effects of bright light is the level of illuminance used in the dim light control condition and for background illumination. It is reported that half of the alerting effect of 9100 lux occurred at approximately 100 lux (ordinary room lighting).[16] In view of these findings, it is important that the effects of daytime bright light exposure be tested with very low background illumination and with low-intensity control condition.

The aim of the present study was to examine the effects of daytime bright light (~ 1,000 lux) exposure, compared to a dim light (< 5 lux) control group, on subjective and objective measures of sleepiness. The objective measure of sleepiness was SEMs, which have previously been shown to be highly correlated with subjective sleepiness and neurobehavioral performance.[36] To increase baseline daytime sleepiness levels, participants were exposed to 2 nights of sleep restriction (5 hours/night). To examine whether the alerting effects of bright light depended on melatonin suppression, salivary melatonin levels were measured.

METHODS

Participants

Sixteen healthy adults (10 women and 6 men) aged between 18 and 35 years (mean = 25 years, 3 months) were recruited by poster advertisements displayed at Monash University. All participants were in good physical and psychological health, as ascertained by general and medical self-report screening questionnaires, were not smokers, and consumed less than 300 mg of caffeine per day and less than 5 standard alcoholic drinks (50 g alcohol) per week. Volunteers who had engaged in transmeridian travel or shift work 3 months prior to the study were excluded from participation. The Horne-Ostberg Morningness-Eveningness Questionnaire[37] and the Pittsburgh Sleep Quality Index[38] were used to exclude individuals with extreme circadian rhythm types and low sleep quality, respectively.

Each participant was given an explanatory letter outlining the research procedures and its objectives, and written informed consent was obtained. The protocol was approved by the Standing Committee on Ethics in Research Involving Humans at Monash University. All participants completed the experiment and were paid $100 (Australian).

Design

The study had 2 treatment conditions: a bright light condition of approximately 1,000 lux and a dim light condition of less than 5 lux. Participants were randomly allocated to 1 of the 2 conditions. The study had a mixed factorial design with 2 independent variables: light condition (independent groups) and time of day (repeated measures). The dependent variables were subjective sleepiness, PVT performance, incidences of SEMs, and salivary melatonin levels.

Materials

Assessment of subjective sleepiness—The 9-point Karolinska Sleepiness Scale (KSS) was used to obtain subjective ratings of sleepiness.[39] The scale is considered a valid measure of subjective sleepiness, as it has been found to be sensitive to fluctuations due to sleep loss[40] and to circadian variations.[27]

Neurobehavioral performance—Neurobehavioral performance was assessed using the PVT.[41-43] The PVT is a small (< 250 g) hand-held device that measures several aspects of vigilance performance. The stimulus to which the participant must respond is presented by both visual and auditory signals. The visual stimulus consists of a timer that begins counting in milliseconds, and the auditory stimulus is a tone delivered through earphones.

Recording of SEM and EEG—EEG activity was derived from Fz, Cz, Pz, and Oz according to the International 10-20 System of electrode placement.[44] The EEG leads were referenced to the left mastoid. The SEMs were recorded by EOG, with the EOG electrodes applied to the outer canthi of each eye; the right EOG was placed slightly above the cantomeatal plane, and the left placed slightly below. Both leads were referenced to nasion. The EOG and EEG recordings were obtained through standard gold-cap Grass-type electrodes (Compumedics Pty Ltd, Australia). All signals were amplified and digitized online (8-bit AD converter; storage sampling rate at 250 Hz for EEG and 125 Hz for EOG) and were digitally low-pass filtered at 35 Hz and high-pass filtered at 0.3 Hz, on standard polysomnographic hardware and software (S-Series Sleep Monitoring System, Compumedics Pty Ltd, Australia). Raw signals were recorded directly to an IBM hard drive and later transferred to compact disk for long-term storage and analysis.

Melatonin collection and assay—Salivary melatonin levels were determined by radioimmunoassay.[45] Polypropylene 2.5-mL tubes were used to collect saliva samples (2 mL).

Lighting—Dim light (mean = 3.3 lux, range 1.6 lux - 5.0 lux) was delivered by standard lamps with 40-watt incandescent globes positioned behind the participant. The bright light (mean = 1,056 lux, range = 1,000 lux - 1,100 lux) was delivered using 2 separate light sources placed 1.5 meters in front of the participants at their eye level. Each light source consisted of 6 fluorescent tubes (*Thorn* 2L, 36 W) aligned vertically inside a purpose-built wooden box (53cm x 53cm x 27cm). Light levels were measured using a Lumacolor J17 luxmeter (Textronix, USA) by placing the sensor on the forehead of the participant in the angle of his or her gaze.

PROCEDURE

The experimental protocol consisted of 2 separate phases: a baseline phase and a laboratory phase.

Baseline Phase

During the first 9 days, participants were required to maintain a normal sleep-wake routine in their own environment. They were instructed to sleep from approximately 11:00 PM until 7:00 AM (± 30 minutes) and to maintain a sleep diary to confirm sleep and wake times. During the last 3 days of this schedule, participants were also required to telephone the experimenter just prior to and after sleep times to verify their sleep-wake schedule. On day 10, participants restricted their sleep to 5 hours, sleeping from 1:00 AM to 6:00 AM. They were telephoned by the experimenter at 12:55 AM and 6:05 AM to verify the sleep restriction. Participants were instructed to refrain from daytime napping during the study and to abstain from foods and beverages containing caffeine and alcohol from day 9 until completion of the laboratory phase.

Laboratory Phase

Participants were transported to the Monash University Sleep Laboratory at 5:00 PM on day 11. The lightproof, sound-attenuated, temperature and humidity-controlled sleep laboratory consisted of a recording room and 2 separate monitoring suites (bedroom, kitchen, recreation area, and bathroom). When participants arrived at the sleep laboratory, electrodes were applied to their scalp and face to facilitate polygraphic recordings of EEG and EOG activity. Impedance levels for EEG and EOG were maintained under 5 kOhms.

From 5:30 PM until the completion of the laboratory phase, a modified constant-routine protocol was imposed. The purpose of this protocol was to control and evenly distribute factors such as food intake, posture, and activity levels, which can influence sleepiness and performance.[46] Participants were required to remain awake in a semirecumbent position under dim light of less than 5 lux. Isocaloric snacks were pre-

sented every 2 to 3 hours. The PVT performance was assessed every hour. From 1:00 AM until 6:00 AM, participants were permitted to sleep. The purpose of this first part of the laboratory phase was to acclimatize participants to the laboratory environment, ensure that they were sufficiently practiced in the performance task, and control their sleep-wake behavior.

At 6:00 AM, participants were awakened. Tests of subjective sleepiness and performance were presented hourly from 9:00 AM until noon, half hourly from 12:30 PM until 5:00 PM, and again hourly from 5:00 PM until 9:00 PM. After each testing period, participants performed the Karolinska Drowsiness Test (KDT)[39] and provided a saliva sample. The KDT was used to facilitate artifact-free recording of EEG and EOG activity. During the KDT, participants were instructed to relax and fixate on a 5-cm black dot positioned 1 meter away at eye level for 4 minutes. They were then asked to close their eyes for a further 1 minute without moving. Saliva samples were immediately stored at -20°C until they were transported for radioimmunoassay to the University of Adelaide.[45]

From noon to 5:00 PM, participants were exposed to 1 of the 2 possible light conditions (ie, dim or bright). During bright light exposure, participants were asked to fixate at the light source every 10 minutes and between each individual performance tests, so that light exposure was controlled and maximized. During testing, participants held the PVT device in front of them, approximately 45 degrees below eye level. As previously mentioned, the light source was situated approximately 1.5 meters in front of the participant and was positioned at the participant's eye level. As the maximum duration of an individual performance test was 10 minutes, and all testing sessions were performed directly in front of the light source, exposure to the light source was relatively uninterrupted.

Following the laboratory phase, participants were transported to their homes.

Data Analysis

SPSS Version 11 (SPSS Inc, USA) was used for all statistical analyses. Data collected from 1 participant in the bright light group were excluded from all analyses due to the participant's lack of compliance with the study protocol. The EEG data will be reported elsewhere.

The polysomnographic recordings during each KDT (eyes open and closed portions) were visually inspected for incidences of SEMs in 30-second epochs, as described elsewhere.[16] In accordance with widely accepted scoring criteria, SEMs were classified as slow (<0.25 Hz) sinusoid horizontal waves (>100 μV) that lasted longer than 1 second.[40,47,48] The percentage of KDT epochs containing SEMs was then calculated.

Saliva samples collected each hour from 10:00 AM to 8:00 PM were assayed for melatonin. The reported sensitivity of the assay was 1 pg/mL. The interassay coefficient of variance was 15% and the intra-assay coefficient of variance was less than 10%.

Data that were collected at 30-minute intervals (between noon and 5:00 PM) were averaged to obtain hourly values.

Inspection of all PVT measures (ie, lapses, errors, and median and mean reaction times) revealed that the mean reaction-time measure was most affected by sleep loss. Accordingly, only this measure was used in subsequent analyses.

Initial inspection of raw data for KSS, PVT, and KDT measures revealed a high degree of interparticipant variability. Therefore, data were transformed to deviations from baseline for each participant.[49] Baseline was designated as the mean of the values obtained at 9:00 AM, 10:00 AM, and 11:00 AM for each variable. Absolute melatonin levels, obtained at noon, 1:00 PM, 2:00 PM, and 3:00 PM, were used for statistical analyses. Evaluation of systematic changes in each measure according to light group and time of day was carried out using a 2-way mixed factorial analysis of variance (ANOVA) on the transformed data during the light exposure period and the corresponding time points for the dim light group. Therefore, a significant main effect of light group would reflect a significant change in the parameters induced by the light treatment.

All data were normally distributed and showed homogeneity of variance between samples. To examine the relationship between measures of sleepiness and performance, Pearson Product Moment Correlation coefficients were calculated for each participant using raw data from 9:00 AM to 8:00 PM. This time period was selected for analysis because there were insufficient data points collected before, during, and after the light exposure period to yield sufficient statistical power. Mean correlation coefficients were obtained and 1-sample *t*-tests were performed separately for each light group to determine whether the mean correlation coefficients differed significantly from 0. Before performing the *t*-tests, skewness and kurtosis of the correlations were examined and found to be within acceptable limits.[50]

RESULTS

Sleepiness, Performance, and Melatonin Levels Prior to Light Exposure

Participants were randomly allocated to treatment groups. To determine whether differences existed prior to light exposure, independent groups *t*-tests were conducted on all dependent variables for the baseline period. No significant differences were observed between the bright (N = 7) and dim light (N = 8) groups for PVT reaction times [$t(13) = 0.622$, $P > .05$], SEMs [$t(13) = 0.381$, $P > .05$], or salivary melatonin levels [$t(13) = 0.613$, $P > .05$]. However, a significant difference was found between groups for KSS baseline data [$t(13) = 2.592$, $P < .05$], with the bright light group (N = 7) reporting higher levels of subjective sleepiness compared to the dim light group (N = 8).

Bright Light Effects on Subjective Sleepiness

While subjective sleepiness scores remained approximately equal to baseline at noon for both light conditions, sleepiness scores (KSS) in the bright light group (N = 7) decreased from 1:00 PM onward (Figure 1). In contrast, the dim light group (N = 8) displayed a moderate increase in sleepiness over the same period. The ANOVA revealed a significant main effect for light group during the light exposure period [$F(1,13) = 6.258$, $P < .05$]. There was no significant effect for time [$F(3,39) = 1.219$, $P > .05$] and no interaction [$F(3,39) = 0.588$, $P > .05$].

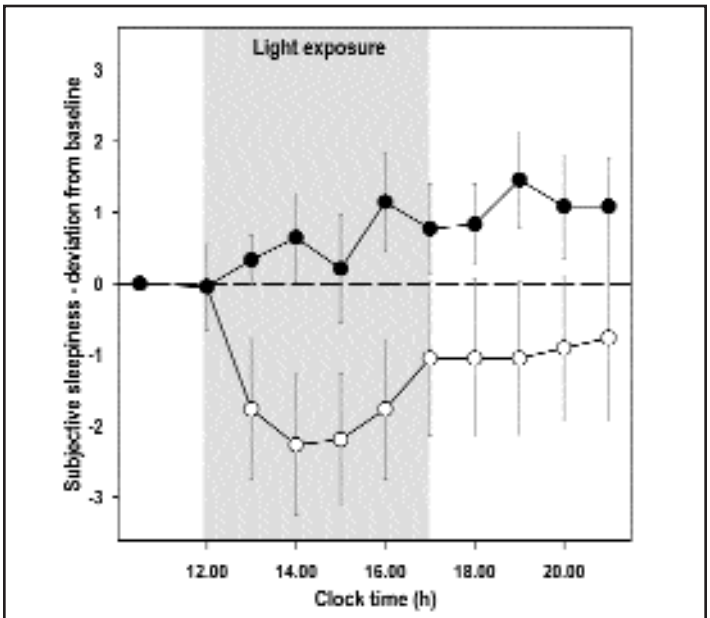

Figure 1—Mean (±SE) deviations from baseline of subjective sleepiness (Karolinska Sleepiness Scale) scores for the bright light group (○) (N = 7), exposed to approximately 1,000 lux between noon and 5:00 PM, and the dim light group (●) (N = 8), exposed to less than 5 lux throughout the study. The shaded area represents the period of bright light exposure. The dashed horizontal line represents the baseline, as determined from individual mean Karolinska Sleepiness Scale scores from 9:00 AM, 10:00 AM, and 11:00 AM. Negative deviations represent decreases in sleepiness relative to baseline. Positive deviations represent increases in sleepiness relative to baseline.

In view of the significant difference between baseline measures for the bright (N = 7) and dim (N = 8) light groups, it was deemed necessary to perform further analyses to confirm the effects of bright light. Independent group *t*-tests were conducted on KSS data obtained during and after light exposure. A significant difference was observed between the bright (N = 7) and dim light (N = 8) groups during the light exposure period [$t(13) = -2.502$, $P<.05$]; however no significant difference was observed between the groups following the light exposure period [$t(13) = -1.801$, $P>.05$].

Bright Light Effects on PVT

As for KSS data, PVT performance (mean reaction times) appeared to improve for the bright light group (N = 7) during the exposure period but appeared to become slower for the dim light group (N = 8) (Figure 2).

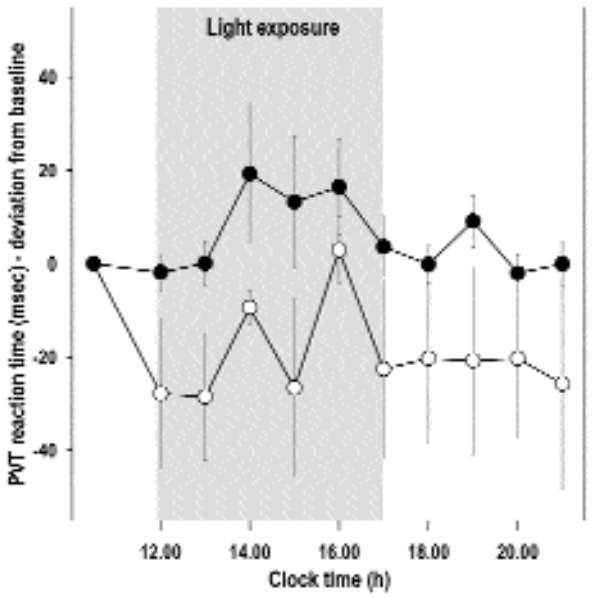

Figure 2—Mean (±SE) deviations from baseline of reaction time (Psychomotor Vigilance Task, PVT) performance (milliseconds) for the bright light (○) (N = 7) and dim light (●) groups (N = 8). Negative deviations indicate improvements in performance, and positive deviations indicate decrements in performance. Conventions as for Figure 1.

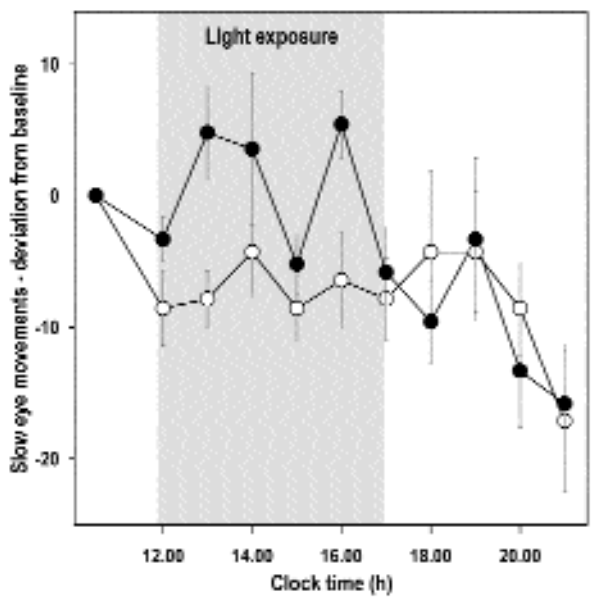

Figure 3—Mean (±SE) deviations from baseline of the percentage of epochs containing slow eye movements (SEMs) during Karolinska Drowsiness Tests for the bright light (○) (N = 7) and dim light (●) (N = 8) groups. Conventions as for Figure 1.

The ANOVA revealed a significant main effect for light group [$F(1,13) = 5.014$, $P<.05$]. There was no significant effect for time [$F(3,39) = 2.616$, $P>.05$] or a group-by-time interaction [$F(3,39) = 0.644$, $P>.05$].

Bright Light Effects on SEMs

The percentage of epochs containing SEMs for the bright light group (N = 7) remained constant over the light exposure period, whereas (N = 8) the percentage of epochs containing SEMs for the dim light group showed a marked increase first at 1:00 PM and then at 4:00 PM (Figure 3). Statistical analysis revealed a significant main effect for light group [$F(1,13) = 9.238$, $P<.01$] but no effect of time [$F(3,39) = 1.916$, $P>.05$] and no interaction between light group and time [$F(3,39) = 1.844$, $P>.05$].

Bright Light Effects on Salivary Melatonin Levels

Mean absolute melatonin levels were calculated for each participant over the bright light exposure period (N = 7) and for the equivalent time in the dim light condition (N = 8). The overall mean (±SE) absolute melatonin levels were 3.93 (±1.26) pg/mL for the bright light group and 5.63 (±2.98) pg/mL for the dim light group. An independent samples *t*-test confirmed that there was no significant difference in the melatonin levels between the 2 groups [$t(13) = -1.399$, $P>.05$, two tailed]. Similarly, absolute levels did not differ between the 2 light conditions [$F(1,13) = 1.958$, $P>.05$], did not differ across time [$F(5,65) = 1.479$, $P>.05$], and did not show an interaction between light group and time [$F(5,65) = .770$, $P>.05$].

Associations Between SEM, PVT, and KSS According to Light Group

The strongest association observed between the various measures in both light groups was for PVT performance (reaction times) and KSS (subjective sleepiness) (Table 1). One-sample *t*-tests revealed that these correlation coefficients differed significantly from 0 in both groups [$t_{bright}(6) = 3.391$, $P<.05$, $t_{dim}(7) = 3.092$, $P<.05$]. Positive associations between PVT performance and the percentage of KDT epochs containing incidences of SEMs were also observed in both light groups. However, these were not significant [$t_{bright}(6) = 1.307$, $P>.05$, $t_{dim}(7) = 0.803$, $P>.05$]. Likewise, no significant correlation was found between KSS and SEMs within either light group [$t_{bright}(6) = 0.578$, $P>.05$, $t_{dim}(7) = 0.017$, $P>.05$].

Associations Between Melatonin Levels and SEMs, PVT, and KSS According to Light Group

One-sample *t*-tests revealed correlation coefficients that did not differ significantly from 0 in both groups for associations between melatonin and SEMs [$t_{bright}(6) = 0.885$, $P>05$, $t_{dim}(7) = 1.383$, $P>.05$], PVT data [$t_{bright}(6) = -0.583$, $P>.05$, $t_{dim}(7) = -1.195$, $P>.05$], and KSS scores [$t_{bright}(6) = 0.857$, $P>.05$, $t_{dim}(7) = -.592$, $P>.05$].

Table 1—Mean (±SE) correlation coefficients between KSS and SEMs, PVT reaction times and SEMs, and PVT reaction times and KSS.

	Bright light group (N=7)	Dim light group (N=8)
KSS – SEMs	-0.09 (0.15)	-0.14 (0.11)
PVT reaction time – SEMs	0.17 (0.13)	0.08 (0.11)
PVT reaction time – KSS	0.38* (0.11)	0.45* (0.11)

* $p < .05$ as determined by 1-sample *t*-tests
KSS, Karolinska Sleepiness Scale; SEMs, slow eye movements; PVT, psychomotor vigilance test

DISCUSSION

The present study found that bright light exposure of approximately 1,000 lux, compared to dim light exposure of less than 5 lux, during the day reduced subjective sleepiness, reduced the percentage of KDT epochs containing SEMs, and improved reaction times on the PVT. These effects were independent of suppression of salivary melatonin levels. A significant correlation between PVT performance and subjective sleepiness was observed. However, the percentage of KDT epochs containing SEMs was not found to be correlated with subjective sleepiness or PVT performance.

The reduction in subjective sleepiness and PVT reaction times during daytime bright light exposure is consistent with findings of previous studies that have examined the effects of nocturnal bright light exposure.[11-16] In the present study, PVT performance appeared to improve almost immediately from the onset of bright light exposure. The data collected at this time were not statistically analyzed because the assumption in planning the analyses was that the effects of light exposure would take some time to manifest.[51] In contrast, changes in the KSS data did not become apparent until approximately 1 hour after the onset of bright light exposure. This implies that the manifestation of the effects of bright light exposure varies according to the measure utilized and may reflect the varying cognitive resources being used on each measure. Alternatively, although the PVT delivered both visual and auditory cues, participants may have relied predominantly on the visual cues, and, hence, the apparent immediate effect of bright light may result from the improvement in visual acuity. These issues should be examined in future research.

The duration of the alerting effect of bright light was not directly examined in this study. Although testing was continued for 4 hours after the light exposure had ceased, only data from the light exposure period were statistically analyzed in order to maximize statistical power and because participants had knowledge of clock time and hence the potential for 'end of experiment' effects in this portion of the data.

The finding that bright light exposure reduced the percentage of KDT epochs containing SEMs was consistent with previous findings following nocturnal exposure to bright light.[16] It appeared that the variability in the percentage of KDT epochs containing SEMs was considerably reduced in the bright light condition (N = 7) compared to the dim light condition (N = 8). Although individual subject variability may have contributed to the results obtained, visual inspection of the data following the light exposure period (ie, after 5:00 PM) indicates that the trend of SEMs were relatively comparable in both light groups, contrasting with the results obtained during the light exposure period. This supports a conclusion that bright light exposure was the major contributing variable reducing incidences of SEMs. One possible explanation for these results is that bright light restores the underlying circadian rhythm of alertness by offsetting the impact of sleep loss. It is also possible that the results reflect changes in a circadian rhythm of SEMs that is altered by light intensity and is distinct from, albeit related to, circadian rhythms of sleep propensity and alertness. These issues could be investigated in future studies.

One notable difference between the present findings and those of previous studies examining nocturnal bright light exposure is that in the present study suppression of salivary melatonin was not observed. It has been suggested that suppression of melatonin by bright light may be a mechanism responsible for improvements in subjective alertness and neurobehavioral performance.[11,12] This study's protocol was designed so that the timing of bright light exposure would coincide with the time of day when endogenous melatonin levels are low, and the results confirmed this was the case. Bright light exposure did not result in any systematic changes in absolute melatonin levels. While it is noted that conclusions to be drawn from this study may be limited by the sensitivity of the melatonin assay and the sample size, collectively the results indicate that the alerting effects of bright light exposure on subjective sleepiness, PVT performance, and incidences of SEMs occurred independently to melatonin suppression.

Studies examining the effects of nocturnal bright light exposure have consistently reported relationships between subjective assessments of sleepiness, incidences of SEMs, and psychomotor performance.[36,40,47] The present findings showed a strong association between subjective sleepiness and PVT performance, supporting the use of the KSS as an instrument for assessing subjective state-related changes in sleepiness. Furthermore, it suggests that under the conditions of the present investigation, variations in the KSS strongly reflect changes in neurobehavioral performance.

The present study did not find a significant association between SEMs and sleepiness or PVT performance. Previous studies report a significant association between these variables during the night,[36,40,47] at a time when the circadian rhythm of sleep propensity and the homeostatic drive for sleep are high. The SEMs are commonly associated with, and are often a precursor to, sleep onset and hence reflect sleep propensity.[47] In this study, the drive for sleep was increased by sleep restriction for 2 nights; however, this may have been insufficient to override the circadian rhythm of sleep propensity, which falls to comparatively low levels during the day. It is also possible that the calculation of correlations over the entire testing period of interest (9:00 AM-8:00 PM) obscured important relationships between SEMs and KSS and PVT variables before, during, and after the light exposure period, hence limiting the conclusions of this study. Future studies could address this issue by using larger sample sizes and increasing the number of data collection points. However, the results of this study raise a question about whether SEMs are an accurate indicator of daytime sleepiness resulting from sleep loss, a speculation that warrants more rigorous investigation. Research into this area has important implications for the development of technologies that alert an individual to the presence of excessive daytime sleepiness, for example when driving.

Although this study included a control dim light condition, it was not possible to administer bright light in a double-blind fashion. This gives rise to possible placebo or expectancy effects. Attempts to devise effective placebos for bright light studies have had limited success[52] because the nature of the stimulus is inherently difficult to disguise from normal-sighted individuals. In order to minimize possible expectancy effects, a between-subjects design was employed where participants were not informed about the predicted direction of results and were not provided with feedback regarding their performance relative to other participants. Two participants, 1 from each treatment condition, were always tested concurrently, and care was taken to ensure that all participants were treated identically. If expectancy or placebo effects were operating in the present data, it would have been anticipated that subjective assessments of sleepiness would have been influenced by light exposure before the objective measures (eg, PVT performance). In fact, a trend in the opposite direction was observed, suggesting that expectancy effects alone could not account for the differences observed between groups in the present study.

This study maintained background illumination levels at less than 5 lux in order to examine the comparative effects of bright light exposure (~1000 lux) on alertness. Although the dim light levels used in this study were considerably less than the bright light condition, a pilot test indicated that the dim light level did not cause discomfort to participants and did not appear to affect visual acuity. Given that participants were exposed to the dim light level throughout the laboratory phase (30 hours), it is likely that the observed effects reflect the alerting effect of bright light exposure as opposed to the detrimental effect of dim light exposure.

Understanding the effects of bright light on human performance may assist in the development of treatments for daytime sleepiness caused by sleep loss. Currently, treatments for this growing problem include ingestion of stimulants such as caffeine and taking 'power' naps; however, each has notable disadvantages. For example, caffeine can induce anxiety in some people and can be disruptive to sleep.[53,54] Napping is not always feasible in a working environment and may induce the detrimental effects of sleep inertia.[55,56] In contrast, providing appropriate light exposure may afford a more natural means of treating excessive daytime

sleepiness related to sleep loss.

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ABBREVIATIONS

ANOVA	analysis of variance
EEG	electroencephalogram
EOG	electrooculogram
KDT	Karolinska Drowsiness Test
KSS	Karolinska Sleepiness Scale
PVT	psychomotor vigilance task
SE	standard error
SEMs	slow eye movements

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