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Exposure to bright light during evening class hours increases alertness among working college students

Liliane Teixeira ^{a,*}, Arne Lowden ^b, Andréa Aparecida da Luz ^c, Samantha Lemos Turte ^c, Claudia Roberta Moreno ^c, Daniel Valente ^a, Roberta Nagai-Manelli ^c, Fernando Mazzilli Louzada ^d, Frida Marina Fischer ^c

- ^a National School of Public Health, FIOCRUZ, 1480 Leopoldo Bulhões St., Office 17, Rio de Janeiro 21041-210, RJ, Brazil
- ^b Stress Research Institute, Stockholm University, SE-106 91 Stockholm, Sweden
- ^c Department of Environmental Health, School of Public Health, University of São Paulo (USP), 715 Doutor Arnaldo Avenue, São Paulo 01246-904, SP, Brazil
- d Department of Physiology, Federal University of Paraná, 92 Coração de Maria St., Road BR116, km 95, Jardim Botânico, Curitiba, PR, Brazil

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ABSTRACT

Objective: To evaluate the effects of exposure to bright light on sleepiness during evening hours among college students.

Methods: Twenty-seven healthy college students, all males, with ages ranging from 21 to 24 years, working during the day and studying in the evening, participated in this study. During the 3 week study, the students wore actigraphs and recorded levels of sleepiness. In a crossover design, on the second and third weeks, the students were exposed to bright light (BL) at either 19:00 or 21:00 h. Salivary melatonin samples were collected before and after BL exposure. ANOVA test for repeated measurements were performed.

Results: After BL exposure, sleepiness levels were reduced at 20:30 and 22:00 h (F = 2.2; p < 0.05). ANOVA showed statistical differences between time (F = 4.84; p = 0.04) and between day and time of BL exposure (F = 4.24; p = 0.05). The results showed effects of melatonin onset at 20:00 and 21:30 h and sleepiness levels (F = 7.67; p = 0.02) and perception of sleepiness and intervention time (F = 6.52; p = 0.01).

Conclusion: Controlled exposure to BL during evening hours increased alertness among college students. The effects of BL on sleepiness varied according to the time of melatonin onset.

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1. Introduction

The advent of electricity changed living and working conditions. The exposure to artificial light at increasingly later hours, delays the bedtime of the population, even though, there is need to wake up early on workdays [1,2]. This discrepancy between social and biological times, called "social jetlag" by Wittmann et al. [3], may cause partial sleep deprivation on workdays/schooldays, and is also associated with poor sleep quality, daytime sleepiness, insomnia, cognitive difficulties, and obesity [4]. Partial sleep deprivation may become chronic, producing more serious consequences including cardiovascular and gastrointestinal disorders [5].

Previous studies have shown that dim light, such as 180 lux light exposure, is already sufficient to cause phase shifts in the timing of the human circadian clock [6]. Intensities and duration of

E-mail address: lilianeteixeira@ensp.fiocruz.br (L. Teixeira).

light are sufficient to alter circadian phase, and/or amplitude of circadian rhythms [7-10].

Several studies have evaluated the effects of artificial light on the sleep—wake cycle, melatonin, and body temperature. Louzada and Menna-Barreto [11] observed that the delay in bedtime in adolescents living in an urban area, is greater than in adolescents living in a rural area without electricity. Likewise, Harada [12], Kubota et al. [13] and Ruger et al. [14] observed that nocturnal light exposure delays the melatonin secretion rhythm and temperature, delaying the propensity to sleep.

Honma and Honma [15] showed that the same light stimulus acts differently depending of the timing on the circadian clock. For example, a day (phase) prolongation is observed in the presence of light stimuli at the end of the natural day, potentially causing a delay in the expression phase of biological rhythms. Also, if a light stimulus is received in the late dark phase, an advance of the rhythm was observed, being one early description of phase responses in humans to light [1,7,8,10].

Other authors have also observed that light exposure early in the day is associated with earlier bedtime [16–18]. In the clinical

^{*} Corresponding author. Address: Centre of Studies of Worker's Health and Human Ecology, Sergio Arouca National School of Public Health, Oswaldo Cruz Foundation, 1480 Leopoldo Bulhões st., Office 17, Rio de Janeiro 21041-210, RJ, Brazil. Tel.: +55 21 25982808.

area, bright light has frequently been used to treat individuals with seasonal depression [19,20], insomnia [21], and daytime sleepiness [22]. In the workplace, bright light purportedly controls the degree of alertness in shift and night workers [23,24], and short exposure to bright sunlight improves the state of physiological alertness, although this effect is not more powerful than that of a short nap [25]. In addition to delaying or advancing bedtimes, bright light exposure interferes with levels of sleepiness and alertness [26]. Meanwhile, bright light exposure decreases the impact of sleep deprivation on sleepiness levels and increasing alertness [25,27,28]. Also, experimental studies using bright light treatment in young people have been conducted. Duffy et al. [29] observed in an experimental setting that treatment with bright light (10,000 lux for 20 min/h for 5 h over three consecutive days) was followed by an adaptation of the body temperature phase to daily activity times. Furthermore, according to Lavoie et al. [30], young people submitted to light treatment (3000 lux from 00:30 to 04:30 h) showed suppression of melatonin secretion and an increase in peripheral body temperature.

Sleepiness levels in healthy individuals display a daytime variation, with the highest values upon waking, in the early afternoon (a prime time for napping), and close to bedtime [31]. Still, partial sleep deprivation, as occurs on classdays or workdays, may increase the sleepiness levels, facilitating a sleep episode [32,33]. Among college students a usual practice is to work during the day and attend evening classes [34]. Due to these demands, working college students show an irregular sleep pattern along the week and a sleep rebound during free days [35,36]. As a consequence, they report excessive daytime sleepiness, difficulties in maintaining attention and poor performance [32]. We are not aware of any publications in the field or intervention studies using bright light exposure to reduce sleepiness among full daytime working class students, those chronically sleep deprived, or enrolled in evening classes.

The hypothesis of the present study was: does bright light exposure during evening hours, in a school environment, result in reducing sleepiness, as it has been observed in experimental laboratory studies where social constraints have been controlled? In this case, are there any response differences according to bright light exposure time (19:00 or 21:00 h)?

The aim of this study is to evaluate the effects of exposure to bright light on sleepiness during evening hours among college students.

2. Methods

2.1. Type of study

This was an intervention study while using a convenience sample. Subjects were randomly divided into groups, and exposed to bright light on second and third weeks according to model (crossover design; Fig. 1).

2.2. Sample selection criteria

Study participants were male college students enrolled in evening classes. The study selected students who had been working for more than 3 months and with similar workweeks (approximately 36–40 h/week). This criterion aimed to minimize differences during the analyses of awakening times and the effects of prolonged working hours.

Individuals were excluded if they were using any chronic medication, including the use of sleep medication (β blockers, calcium antagonists or calcium channel blockers, anti-inflammatory drugs, anxiolytics, benzodiazepines, or sleep-inducing drugs and melatonin) that could affect sleep patterns. The sample also excluded

individuals that reported sleep disorders according a sleep disorder self-assessment questionnaire [37] and those with BMI (weight/height²) >30 kg/m² (obese).

The research protocol required that subjects abstained from alcohol 48 h prior to the intervention weeks and abstained 12 h prior to the start of study from caffeine, theobromine, and cigarettes, and throughout the study.

This study used a pretest to estimate a relevant sample size. The sample size was calculated based on the mean and standard deviations for salivary melatonin among the working college students before and after the first exposure to bright light. Using the Hulley and Cummings table [38], α = 5%, β = 10%, and T = 1.0, the minimum sample size was estimated to 21 students to reach enough power to detect changes for bright light exposure given at 19:00 and 21:00 h.

2.3. Description of study population

Twenty-three male students participated in this study, mean age of 22.2 years (Standard deviation = 1.3 years). Two participants were married, one of whom had a child. Monthly family income was US\$ 1,730,00. In relation to body mass index (BMI): two individuals were underweight, 11 eutrophic (\geqslant 18.5 kg/m² and <25.0 kg/m²), and 8 overweight. Regarding physical activity, 10 students defined themselves as sedentary (43.5%), irregularly active (30.4%), active (13.0%) and very active (8.7%). Two subjects were smokers, and one was a former smoker.

As for the sleep-related variables, only two students described the place where they slept as unpleasant (8.7%). As for chronotype according to the scores of Horne & Östberg questionnaire [39], 15 subjects (65.2%) were intermediate types, seven subjects(30.5%) were evening types and only one (4.3%) was a morning type subject.

A major share of the subjects had begun working after 16 years of age (74.0%). The main workplace was the office (47.8%), and most worked as interns (65.3%). As for the length of the workday, seven (30.4%) worked more than 8 h/day and 16 worked (69.6%) between 6 to 8 h.

2.4. Ethical issues

The students were contacted personally and read the informed consent form. They all agreed to participate in the study voluntarily, completing and signing the form. The informed consent form, as the whole study, was approved by the Ethics Committee of the School of Public Health, University of São Paulo.

2.5. Data collection

Data were collected from August 11, to October 14, 2008. No data collection took place on weeks that included holidays. The data collection followed a fixed schedule pattern, since the students' routine remain unchanged, working approximately 8 h per day and attending college in the evening hours (19:30–23:10 h). The subjects answered a comprehensive questionnaire on living and work conditions, health symptoms and sleep habits.

In the first study week (baseline week – without bright light intervention), data on the sleep–wake cycle (actigraph, daily activities diaries), and the Karolinska Sleepiness Scale were collected by participants. Also, salivary melatonin samples were collected in order to determine the melatonin onset (DLMO). On Wednesdays, during the second and third weeks, participants were submitted to a 8000 lux, 20-min white bright light exposure, as to avoid changes in students' routine.

The second stage included gathering records on melatonin rhythm and the sleep-wake cycle of the students, before, during, and after bright light exposure, using subjective methods (daily

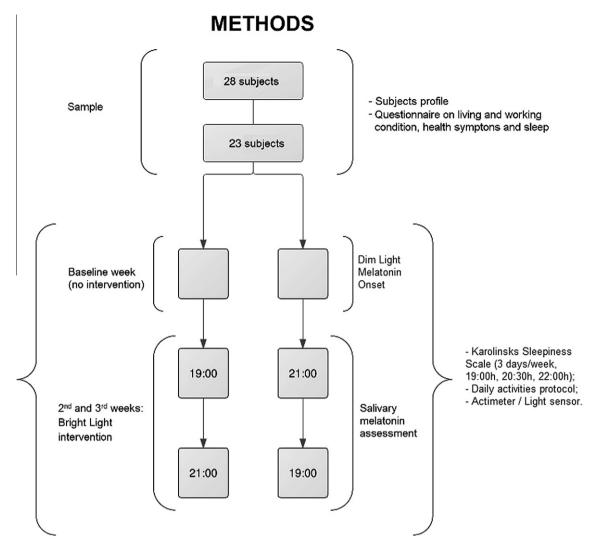


Fig. 1. Data collection protocol.

activities diaries and Karolinska Sleepiness Scale) and objective methods (salivary melatonin and actigraphy).

The actigraph (MicroMini-Motionlogger Actigraph, Ambulatory Monitoring, Inc®) was worn on the non-dominant wrist for 21 consecutive days. To increase the precision of the onset and end of the nighttime sleep phase, naps, and sleep latency, the students simultaneously completed the activity diaries (adapted version of Knauth's protocol) [40]. This study used the algorithm proposed by Sadeh [41] and analyzed the following sleep—wake cycle variables: subjective nighttime sleep latency (estimated time the student took to fall asleep), nighttime sleep duration or total bedtime, mid-sleep period, duration of awakenings during sleep, duration of naps, and sleep efficiency (percentage of total bedtime in which the individual was sleeping, excluding nocturnal awakenings and sleep latency). Nocturnal awakenings and naps were defined as events more than 5 minutes. Each student also received a lapel light sensor (MicroMini Light Sensor, Ambulatory Monitoring Inc®).

The Karolinska Sleepiness Scale (KSS) was used for self-assessment of the student's alertness [31]. The KSS contains nine points, varying from extremely alert (represented by the number 1) to very sleepy, fighting off sleep, and great effort to stay awake (number 9). Self-rated alertness was reported on Tuesdays, Wednesdays, and Thursdays at 19:00, 20:30, and 22:00 h.

To determine the dim light melatonin onset (DLMO), samples of salivary melatonin were collected hourly on a Wednesday evening,

(18:30-22:00 h), one week prior to the bright light exposure. This procedure followed a model suggested by Levy et al. [42]. On this evening, the students remained in a dimly lit room (<10lux) watching films. The subjects were not allowed to eat or drink liquids except water, during the data collection. Beginning at 19:00 h, saliva samples were taken every hour. Samples were taken using a Salivette polyester fiber swab (Sarstedt, Inc.), on which the individual deposits a saliva sample. The saliva was centrifuged and frozen for subsequent analysis. Salivary melatonin concentration was measured in duplicate using the radioimmunoassay method (Melatonin Direct RIA - BA 3300, Labor Diagnostika Nord GmbH & Co. KG, Nordhorn, Germany). Onset of melatonin secretion was defined as doses 4 pg/ml and higher of salivary melatonin [42]. The participants were divided in two categories: those showing melatonin secretion increase earlier, around 20:00 h (N = 12), and those at a later time, around 21:30 h (N = 11).

Since the students went to bed significantly later on weekends compared to work days, we decided to set bright light exposure on Wednesday evening to avoid excessive sleepiness due to weekend spillover effect.

The data collection took place one hour after sunset at 19 h (along with the start of classes), and at 21 h, the interval time between classes.

The students were divided in two groups: Group A was exposed to bright light at 19:00 h in the second week and at 21:00 h in the

Table 1Means and standard deviations for variables related to the sleep–wake cycle in working college students during the study weeks.

SWC	Days of the week											
	Monday		Tuesday		Wednesday*		Thursday		Friday		Test	р
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD		
Week baseline												
Sleep latency (min)	7.6	6.9	8.5	7.1	6.4	5.6	5.6	4.0	10.4	3.6	1.88	0.76
Mid-sleep period (h)	04:19	01:06	04:14	01:17	03:51	00:59	04:00	01:20	05:31	01:45	1.33	0.27
Total sleep time (h)	05:23	01:34	06:03	01:42	05:28	01:12	05:14	02:20	05:28	02:30	0.09	0.76
Nocturnal awakenings (min)	16.1	8.7	16.9	15.7	16.6	12.7	14.7	6.1	35.4	20.6	2.83	0.59
Sleep efficiency (%)	82.5	19.4	85.8	11.6	87.5	8.1	77.6	29.7	75.1	27.4	1.46	0.83
Naps (min)	13.6	8.7	5.8	14.0	10.2	5.5	10.5	2.6	7.3	5.3	1.96	0.06
Week with intervention at 19:00												
Sleep latency (min)	5.4	4.1	10.8	4.6	13.3	6.1	12.3	4.6	11.5	5.2	4.92	0.30
Mid-sleep period (h)	04:24	01:28	04:06	01:18	04:40	01:05	05:04	01:34	05:09	01:10	1.86	0.20
Total sleep time (h)	05:44	02:01	05:36	01:20	05:47	01:38	05:34	01:42	05:43	01:48	0.04	0.84
Nocturnal awakenings (min)	19.2	15.0	16.0	11.3	20.8	12.7	22.4	17.2	26.4	15.2	3.47	0.48
Sleep efficiency (%)	80.2	22.5	86.0	9.5	83.0	9.8	83.0	12.0	78.9	13.5	4.3	0.38
Naps (min)	4.5	2.6	11.0	5.8	10.1	6.3	6.6	4.2	10.9	7.0	1.66	0.80
Week with intervention at 21:00												
Sleep latency (min)	15.1	3.8	10.1	10.8	07.9	6.5	17.0	23.9	12.0	17.0	1.79	0.77
Mid-sleep period (h)	04:55	01:33	04:13	01:11	04:18	01:04	04:46	01:14	04:49	00:54	0.50	0.55
Total sleep time (h)	06:34	01:22	05:17	01:13	05:42	01:16	05:55	01:31	04:08	02:22	0.40	0.57
Nocturnal awakenings (min)	19.4	11.9	18.3	13.5	14.9	11.9	20.6	13.7	17.9	7.7	3.47	0.48
Sleep efficiency (%)	83.8	9.1	84.2	13.0	87.0	8.0	83.7	6.9	71.0	35.5	2.4	0.66
Naps (min)	25.7	13.8	13.5	6.0	15.6	8.2	24.8	13.6	13.3	8.6	4.2	0.38

^{*} Day of intervention.

third week. Group B was exposed to bright light at 21:00 h in the second week and at 19:00 h in the third week.

During the data collection, a classroom at the university was used. All the original light bulbs were replaced by Philips lamps, Super 84 4,000 K and 40 W. The participants remained seated in a smaller area (4.50 m \times 3.40 m), and exposed to bright light, (8000 lux) at the level of the eyes, during 20 minutes (this duration was established after bright light exposure pretest showed a significant sleepiness reduction). The area was enclosed with white curtains and the ceiling was covered with white paper. During the data collection, saliva samples were taken to measure salivary melatonin at 19:00, 21:00 h and right after the bright light exposure (19:20 or 21:20 h).

According to the sensor light measurements, the mean values of indoor light exposure during the evenings (19:00–23:00 h) including those preceding and following bright light exposure did not exceed 300 lux.

2.6. Data analysis

Descriptive analysis was performed with sociodemographic data.

The sleep-wake cycle, sleepiness levels and salivary melatonin were initially submitted to descriptive analysis. It was calculated means and standard deviation for each weekdays (Monday through Friday), during the three weeks of the experiment. The data were submitted to the Shapiro-Wilk normality test, and the variables displayed normal distribution. The statistical test ANOVA for repeated measurements was used to compare the mean sleepiness levels within and between weeks, before and after the intervention.

3. Results

Table 1 describes the sleep–wake cycle in the 3 weeks of the study protocol. The mid sleep after the bright light exposure at 19:00 and 21:00 h showed a mean delay of 24 and 28 min, respectively as some individuals showed a small delay of their sleep onset.

However, no significant differences were showed in the sleep—wake cycle comparing within and between the 3 weeks data were collected. An association between morningness–eveningness chronotype and melatonin onset was found (r = 0.72; p = 0.03).

3.1. Sleepiness perception (KSS values) during the week of bright light exposure

3.1.1. At 19:00 h

After exposure to bright light at 19:00 h, the mean levels of sleepiness (KSS) showed lower values on Wednesday at 20:30 and 22:00 h (F = 2.2; p < 0.05), and on Thursday, the day following the exposure day, at 19:00 and 20:30 h (F = 10.16, p < 0.01), when compared to those values of the baseline week (previous week), on Wednesday and Thursday (F = 6.70; p < 0.01) (Table 2).

3.1.2. At 21:00 h

During the previous evenings of bright light exposure (Mondays and Tuesdays) at 21:00 h, the Karolinska Sleepiness Scale levels (KSS) were not statistically different to those values on the baseline week (no bright light exposure) (Table 2).

3.2. Comparison of the 2 weeks of bright light exposure regarding sleepiness (KSS)

Comparing the average values of sleepiness on the night of exposure to bright light (Wednesdays, 19:00 and 21 h), it was observed that there were statistically significant differences in the values of KSS according to the hours of exposure (F = 4.84, p = 0.04).(Fig. 2).

3.3. Bright light exposure and sleepiness according to melatonin onset

Sleepiness was influenced by melatonin onset (F = 7.67; p = 0.02) and bright light exposure time (F = 6.52; p = 0.01). Exposure at 19:00 h for students with melatonin onset around 20:00 h was not sufficient to reduce sleepiness. Meanwhile, for students with melatonin onset around 21:30 h, the effect of bright light exposure was significant, i.e., the mean values of sleepiness

Table 2
Means and standard deviations for subjects Karolinska Sleepiness Scale, measured at 19:00 h, 20:30 h, and 22:00 h of the three study weeks, on the intervention days and days before and after the intervention.

KSS	Days of the week										
	Monday		Tuesday		Wednesday		Thursday		Friday		
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	
Week baseli	ine										
19:00	4.2	1.4	4.1	1.3	3.9	1.6	4.5	2.1	4.0	1.2	
20:30	4.8	1.2	5.1	1.3	5.0	1.7	4.9	1.9	4.9	1.1	
22:00	5.6	1.2	5.3	1.3	5.2	1.2	5.1	1.5	5.4	1.3	
Week with i	intervention at 19	:00									
19:00	3.5	0.7	4.2	1.5	3.4	1.2	3.6	1.3	3.8	1.0	
20:30	7.0	2.8	4.8	1.3	3.9	1.1	3.9	1.0	4.4	1.3	
22:00	5.0	0.0	5.2	1.2	4.3	1.4	5.1	1.7	4.7	1.5	
Week with	intervention at 21	:00									
19:00	4.8	1.1	4.4	1.6	4.1	1.4	3.9	1.7	4.3	1.6	
20:30	5.2	1.6	4.5	1.5	4.4	1.4	4.0	1.5	4.8	1.4	
22:00	5.0	1.9	5.3	1.4	4.4	1.7	5.1	1.4	4.7	1.7	

Sleepiness × hour: F = 10.16; p < 0.01.

Sleepiness \times intervention day: F = 6.70; p < 0.01.

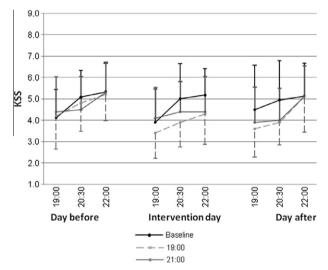


Fig. 2. Means and standard deviations of Karolinska Sleepiness Scale. Data collection were at 19:00, 20:30, and 22:00 h in the three study weeks (baseline and 19:00 and 21:00 h intervention), on the intervention days and days before and after the intervention. Sleepiness \times hour: F = 4.84; p = 0.04. Sleepiness \times intervention days: F = 4.24; P = 0.05.

remained the same throughout the evening at the three recording times (p = 0.03; Fig. 3).

The time of bright light exposure affected students sleepiness (F = 3.03; p = 0.04). Participants who showed melatonin onset around 20:00 h maintained alertness when they were exposed to bright light at 21:00 h, over the next hour (p = 0.01). The effect of bright light exposure among those with melatonin onset around 21:30 h was also significant, i.e., sleepiness levels remained the same after bright light exposure (p = 0.04; Fig. 4).

Significant differences on sleepiness in other weekdays were not found when compared earlier and later melatonin onset individuals.

3.4. Bright light exposure and sleepiness according to morningness-eveningness types

An influence of diurnal preference upon Karolinska Sleepiness Scale was detected on the 21:00 h bright light exposure (F = 2.73; p = 0.05). At 19:00 h bright light exposure, the participants sleepiness levels increased as expected, at KSS time tests (20:30, 22:00 h).

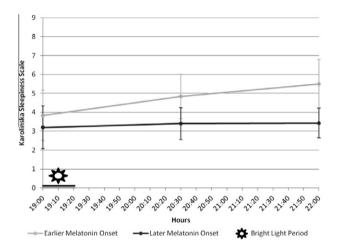


Fig. 3. Means and standard deviations of Karolinska Sleepiness Scale. Data collection were at 19:00, 20:30, and 22:00 h on the day of bright light exposure at 19:00 h, of earlier (about 20:00) and later (about 21:30) Melatonin Onset groups. Melatonin onset \times intervention time: F = 7.67; p = 0.02. Sleepiness \times intervention time: F = 6.52; p = 0.01.

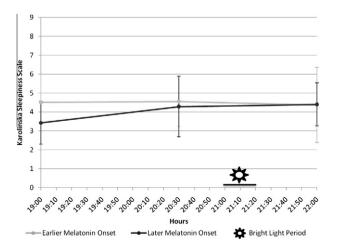


Fig. 4. Means and standard deviations of Karolinska Sleepiness Scale. Data collection were at 19:00, 20:30, and 22:00 h on the day of bright light exposure at 21:00 h, of earlier (about 20:00) and later (about 21:30) Melatonin Onset groups. Sleepiness \times intervention time: F = 3.03; p = 0.04.

But at 21:00 h, bright light exposure, the intermediate morningevening types participants showed a gradual increase of the sleepiness KSS level (p = 0.02); whereas for the evening types sleepiness levels decreased at 22:00 h test (p < 0.01; Fig. 5).

4. Discussion

The main objective of the study was to evaluate the effect of intense white light exposure during evening class hours on reported sleepiness among working male college students. The results of this study showed a positive effect of the bright light exposure reducing sleepiness. Former studies conducted with youngsters showed contradictory results. Lavoie et al. [30] did not observe any improvement in self-evaluated alertness, electroencephalographic data, or performance tests, our results showed an effect of bright light exposure on sleepiness, without masking significantly the sleep-wake cycle. However, Harada [12] found positive results reducing sleepiness by using 2000 lux for three hours (from 19:30 to 22:30 h) in five secondary school students. According to Harada, the students showed a reduction in salivary melatonin as compared to the control group.

Our study supports previous findings [43] that the effects of bright light exposure in the evening hours may vary with the time of melatonin onset and consequently with the decline in alertness.

Non-significant changes of the sleep-wake cycle were more likely to occur because participants were not on a free schedule, and thus had fixed work and study hours. In spite of this schedule there was no significant delay in mid-sleep after bright light exposure comparing within and between weeks; individual differences should be taken into account as some participants did show a delay on their sleep onset. This is important in order to avoid further reduction of the sleep time, as these working college students were already partially deprived of sleep.

Still interestingly is the correlation between the subject's individual chronotype and the time of bright light exposure. Bright light at 21:00 h was sufficient to reduce sleepiness only in the eveningness subjects. Griefahn et al. [44] obtained similar results detecting a major DLMO delay for evening-type shiftworkers.

Carskadon et al. [45] suggested that the circadian pacemakers' sensitivity to light can change during pubertal development, accentuating the tendency towards a delay in the sleep phase. According to these researchers, adolescence is marked by increased light sensitivity in the late light phase and decreased light sensitivity in the late dark phase. Greater sensitivity to light at the end of the day, together with decreased homeostatic sleep pressure, results in increased likelihood of a phase shift in this phase of development [45]. Thus, this variation in the circadian pacemakers' response is one of the proposed components to account for the phase shift observed in adolescence. The same effect may have occurred with some participants in the current study, although they were already young adults, 21 years or older. This fact mediated by interindividual variations may explain the overall limited effect on sleepiness found in the present study.

The mean light intensity levels to which the students were exposed before the intervention may have been sufficient to exert some masking effect on the individuals' circadian system. Some authors support this hypothesis, like Boivin & Czeisler [46], who showed that the circadian clock is sensitive to low light intensities, for example, 180 lux, and is further capable of adapting to certain conditions of light stimulation that persists for some time [47]. Studies by Boivin et al. [6] and Klerman et al. [48] showed that light's resetting effect and its intensity does not necessarily follow a linear relationship between the magnitude of the effect and the light intensity. In the present study no significant differences associated with exposure to indoor evening light (300 lux) and vari-

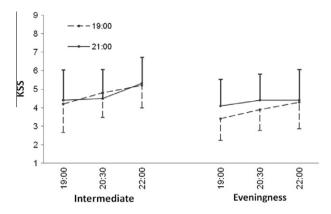


Fig. 5. Means and standard deviations of the Karolinska Sleepiness Scale (KSS) according to Hörne & Östberg Morningness—eveningness questionnaire. Results of three-time KSS tests (19:00, 20:30 and 22:00 h) during the bright light exposure intervention evenings (19:00 and 21:00 h). Sleepiness \times chronotype: F = 2.73; p = 0.05.

ables related to the sleep-wake cycle, sleepiness, and melatonin onset were observed.

Some authors point to a correlation between light intensity variable and melatonin suppression. According to Smith et al. [47], differential prior exposure in two groups (0.5 lux versus 200 lux) during the light phase showed greater melatonin suppression in the group previously exposed to 0.5 lux, when subsequently exposed to 200 lux in the early dark phase. According to Rufiange et al. [49], greater daytime exposure to natural light correlates with decreased melatonin suppression by evening light exposure. These results show that prior light exposure history alters the nocturnal melatonin secretion pattern and thus the circadian clock is capable of adapting to light exposure conditions [47]. Thus, analysis of prior light exposure could reveal a possible adaptation by the young subject's circadian clock. Furthermore, if the young subjects were exposed to bright natural light during the day, they might be less susceptible to the effect of sleepiness reduction by exposure to artificial light in the evening hours.

4.1. Limitations of the study

Data on natural light exposure was limited. This was due to the partial compliance by participants using the light sensor during daytime. Thus, the analysis of the entire photic history that could explain part of individual differences responding to bright light exposure was impaired.

Despite laboratory studies, the ability to control a larger number of variables; we did find similar results in this field study.

4.2. Recommendations

Educational programs on sleep hygiene usually fail to approach issues related to natural and artificial light exposure and its effects on the sleep-wake cycle and sleepiness. To the extent that students may recognize the effects of light exposure at different moments in the day, including the evening, some behaviors could be avoided, such as exposure to bright light near bedtime. Another important possibility would be to intervene in the school settings by improving classrooms lighting during some moments of evening classtime and at other times, in case daytime natural light is not adequate or sufficient for maintaining alertness.

The biological and social causes of sleep deficits resulting from the double workload (i.e., working and studying) should also be discussed in university courses (among faculty, students, and the administration) and directly with the students themselves and their families. Other studies should be performed using different forms of class-room lighting during the three classtime shifts (morning, afternoon, and evening). In larger student samples, it would be possible to analyze the shifts in sleep—wake cycle patterns, sleepiness patterns, and performance over the course of the wake phase in both male and female working and non-working college students.

5. Conclusion

Under the conditions of this experiment, exposure to bright light during evening classtime (at 19:00 and 21:00 h) produced an increase alertness among working college students. The effectiveness of bright light to reduce sleepiness was a function of the time of melatonin onset.

Conflict of interest

The ICMJE Uniform Disclosure Form for Potential Conflicts of Interest associated with this article can be viewed by clicking on the following link: http://dx.doi.org/10.1016/j.sleep.2012.08.017.

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References

- Khalsa SB, Jewett M, Cajochen C, Czeisler CA. A phase-response curve to single bright light pulses in human subjects. J Physiol 2003;549:945–52.
- [2] Zeitzer JM, Dijk DJ, Kronauer RE, Brown EN, Czeisler CA. Sensitivity of the human circadian pacemaker to nocturnal light: melatonin phase resetting and suppression. J Physiol 2000;526:695–702.
- [3] Wittmann M, Dinich J, Merrow M, Roenneberg T. Social jetlag: misalignment of biological and social time. Chronobiol Int 2006;23:497–509.
- [4] Reilly JJ, Armstrong J, Dorosty AR, Emmett PM, Ness A, Steer C, et al. Early life risks factors for obesity in childhood: cohort study. BJM 2005;330:1357–64.
- [5] Foster RG, Wulff K. The rhythm of rest and excess. Nat Rev Neurosci 2005;6:407–14.
- [6] Boivin DB, Duffy JF, Kronauer RE, Czeisler CA. Dose-response relationships for resetting of human circadian clock by light. Nature 1996;379:540-2.
- [7] Czeisler CA, Johnson MP, Duffy JF, Brown EN, Ronda JM, Kronauer RE. Exposure to bright light and darkness to treat physiologic maladaptation to night work. NE J Med 1990;322:1253–9.
- [8] Honma K, Honma S, Nakamura K, Sasaki M, Endo T, Takahashi T. Differential effects of bright light and social cues on reentrainment of human circadian rhythms. Am J Physiol 1995;268:528–35.
- [9] Jewett ME, Kronauer RE, Czeisler CA. Light-induced suppression of endogenous circadian amplitude in humans. Nature 1991;350:59–62.
- [10] Minors DS, Waterhouse JM, Wirz-Justice A. A human phase-response curve to light. Neurosci Lett 1991;133:36–40.
- [11] Louzada F, Menna-Barreto L. Sleep-wake cycle in rural populations. Biol Rhythm Res 2004;35:153–7.
- [12] Harada T. Effects of evening light conditions on salivary melatonin of Japanese junior high school students. J Circadian Rhythms 2004;2:4.
- [13] Kubota T, Uchiyama M, Suzuki H, Shibui K, Kim K, Tan X, et al. Effects of nocturnal bright light on saliva melatonin, core body temperature and sleep propensity rhythms in human subjects. Neurosci Res 2002;42:115–22.
- [14] Ruger M, Gordijn CM, Breersma D, Vries B, Daan S. Acute and phase-shifting effects of ocular and extraocular light in human circadian physiology. J Biol Rhythms 2003;18:409–19.
- [15] Honma K, Honma S. A human phase-response curve for bright light pulses. Jpn J Psychiatry Neurol 1988;42:167–8.
- [16] Dumont M, Carrier J. Daytime sleep propensity after moderate circadian phase shifts induced with bright light exposure. Sleep 1997;20:11–7.
- [17] Roenneberg T, Wirz-Justice A, Merrow M. Life between clocks: daily temporal patterns of human chronotypes. J Biol Rhythms 2003;18:80–90.

- [18] Samkova L, Vondrasova D, Hajek I, Illnerova H. A fixed morning awakening coupled with a low intensity light maintains a phase advance of the human circadian system. Neurosci Lett 1997;224:21–4.
- [19] Levy AJ, Bauer VK, Cutler NL, Sack RL, Ahmed S, Thomas KH, et al. Morning vs evening light treatment of patients with winter depression. Arch Gen Psychiatry 1998:55:890–6.
- [20] Winkler D, Pjrek E, Praschak-Rieder N, Willeit M, Pesawas L, Konstantinidis A, et al. Actigraphy in patients with seasonal affective disorder and healthy control subjects treated with light therapy. Soc Biol Psychiatry 2005;58:331–6.
- [21] Espie CA, Inglis SJ, Tessier S, Harvey L. The clinical effectiveness of cognitive behaviour therapy for chronic insomnia: implementation and evaluation of a sleep clinic in general medical practice. Behav Res Ther 2001;39:45–60.
- [22] Bootzin RR, Stevens SJ. Adolescents, substance abuse, and the treatment of insomnia and daytime sleepiness. Clin Psychol Rev 2005;25:629–44.
- [23] Czeisler CA, Kronauer RE, Allan JS, Duffy JF, Jewett ME, Brown EN, et al. Bright light induction of strong (type 0) resetting of the human circadian pacemaker. Science 1989;244:1328–33.
- [24] Eastman CI, Martin SK. How to use light and dark to produce circadian adaptation to night shift work. Ann Med 1999;31:87–98.
- [25] Kaida K, Takahashi M, Haratani T, Otsuka Y, Fukasawa K, Nataka A. Indoor exposure to natural bright light prevents afternoon sleepiness. Sleep 2006;29:462–9.
- [26] Cajochen C. Alerting effects of light. Sleep Med Rev 2007;11:453-64.
- [27] Leproult R, Colecchia EF, L'Hermite-Balériaux M, Van Cauter E. Transition from dim to bright light in the morning induces an immediate elevation of cortisol levels. J Clin Endocrinol Metab 2001;86:151–7.
- [28] Phipps-Nelson J, Redman JR, Dijk DJ, Rajaratnam SM. Daytime exposure to bright light, as compared to dim light, decreases sleepiness and improves psychomotor vigilance performance. Sleep 2003;26:695–700.
- [29] Duffy JF, Kronauer RE, Czeisler CA. Phase-shifting human circadian rhythms: influence of sleep timing, social contact and light exposure. J Physiol 1996:495:289–97.
- [30] Lavoie S, Paquet J, Selmaoui B, Rufiange M, Dumont M. Vigilance levels during and after bright light exposure in the first half of the night. Chronobiol Int 2003:20:1019–38.
- [31] Åkerstedt T, Gillberg M. Subjective and objective sleepiness in the active individual. Int J Neurosci 1990;52:29–37.
- [32] Carskadon MA, Dement WC. Nocturnal determinants of daytime sleepiness. Sleep 1982;5:S73-81.
- [33] Teixeira LR, Lowden A, Turte SL, Nagai R, Moreno C, Latorre RC, et al. Sleep and sleepiness among working and non-working high school evening students. Chronobiol Int 2007;24:99–113.
- [34] Fischer FM, Oliveira DC, Teixeira LR, Teixeira MCTV, Amaral MA. Efeitos do trabalho sobre a saúde de adolescentes [Effects of work on the health of adolescents]. Ciência & Saúde Coletiva 2003;8:973–84.
- [35] Machado ERS, Varella VBR, Andrade MMM. The influence of study's schedule and work on the sleep-wake cycle of college students. Biol Rhythm Res 1998;29:578-84.
- [36] Tsai LL, Li SP. Sleep patterns in college students. Gender and grade differences. J Psych Res 2004;56:231–7.
- [37] Pires MLN, Silva AAB, Mello MT, Pompeia S, Giglio S, Tufik S. Sleep habits and complaints of adults in the city of São Paulo, Brazil, in 1987 and 1995. Braz J Med Biol Res 2007;40:1505–15.
- [38] Hulley SB, Cummings SR. Designing clinical research. Baltimore: Williams & Wilkins; 1988.
- [39] Horne JA, Östberg O. A self-assessment questionnaire to determine morningness-eveningness in human circadian rhythms. Int J Chronobiol 1976;4: 97-110.
- [40] Knauth P, Kieswetter E, Ottman W, Karvonen M, Rutenfranz J. Time-budget studies of policement in weekly or swiftly rotating shift systems. Appl Ergon 1983:14:247–52.
- [41] De Souza L, Benedito-Silva AA, Pires ML, Poyares D, Tufik S, Calil HM. Further validation of actigraphy for sleep studies. Sleep 2003;26:81–5.
- [42] Levy AJ, Cutler NL, Sack RL. The endogenous melatonin profile as a marker of circadian phase position. J Biol Rhythms 1999;14:227–36.
- [43] Cajochen C, Krauchi K, Von Arx MA, Mori D, Graw P, Wirz-Justice A. Daytime melatonin administration enhances sleepiness and theta/alpha activity in the waking EEG. Neurosci Lett 1996;207:1–5.
- [44] Griefahn B, Kuenemund C, Robens S. Shifts of the hormonal rhythms of melatonin and cortisol after a 4-hours bright light pulse in different diurnal types. Chronobiol Int 2006;23:659–73.
- [45] Carskadon MA, Acebo C, Jenni OG. Regulation of adolescent sleep. NY Acad Sci 2004:1021:276–91.
- [46] Boivin DB, Czeisler CA. Resetting of circadian melatonin and cortisol rhythms in humans by ordinary room light. Neuroreport 1998;9:779–82.
- [47] Smith KA, Schoen MW, Czeisler CA. Adaptation of the human pineal melatonin suppression by recent photic history. J Clin Endocrinol Metab 2004;89:3610–4.
- [48] Klerman EB, Dijk DJ, Kronauer RE, Czeisler CA. Simulations of light effects on the human circadian pacemaker: implications for assessment of intrinsic period. Am J Physiol 1996;270:271–82.
- [49] Rufiange M, Beaulieu C, Lachapelle P, Dumont M. Circadian light sensitivity and rate of retinal dark adaptation in indoor and outdoor workers. J Biol Rhythms 2007;22:454–7.