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.

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 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
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 (cross-over 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 ($\beta$ 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 to a sleep disorder self-assessment questionnaire [37] and those with BMI (weight/height$^2$) $>30$ kg/m$^2$ (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], $\alpha = 5\%$, $\beta = 10\%$, 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 ($\geq$ 18.5 kg/m$^2$ and <25.0 kg/m$^2$), 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
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. 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.

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

![METHODS Diagram](image-url)
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 4000 K and 40 W. The participants remained seated in a smaller area (4.50 m × 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 1

<table>
<thead>
<tr>
<th>SWC Days of the week</th>
<th>Monday</th>
<th>Tuesday</th>
<th>Wednesday</th>
<th>Thursday</th>
<th>Friday</th>
<th>Test</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>SWC</td>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
<td>Mean</td>
<td>Mean</td>
<td>Mean</td>
<td></td>
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<tr>
<td>Week baseline</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sleep latency (min)</td>
<td>7.6</td>
<td>6.9</td>
<td>8.5</td>
<td>7.1</td>
<td>6.4</td>
<td>5.6</td>
<td></td>
</tr>
<tr>
<td>Mid-sleep period (h)</td>
<td>04:19</td>
<td>01:06</td>
<td>04:14</td>
<td>01:17</td>
<td>03:51</td>
<td>00:59</td>
<td></td>
</tr>
<tr>
<td>Total sleep time (h)</td>
<td>05:23</td>
<td>01:34</td>
<td>06:03</td>
<td>01:42</td>
<td>05:28</td>
<td>01:12</td>
<td></td>
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<tr>
<td>Nocturnal awakenings (min)</td>
<td>16.1</td>
<td>8.7</td>
<td>16.9</td>
<td>15.7</td>
<td>16.6</td>
<td>12.7</td>
<td></td>
</tr>
<tr>
<td>Sleep efficiency (%)</td>
<td>82.5</td>
<td>19.4</td>
<td>85.8</td>
<td>11.6</td>
<td>87.5</td>
<td>8.1</td>
<td></td>
</tr>
<tr>
<td>Naps (min)</td>
<td>13.6</td>
<td>8.7</td>
<td>5.8</td>
<td>14.0</td>
<td>10.2</td>
<td>5.5</td>
<td></td>
</tr>
<tr>
<td>Week with intervention at 19:00</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sleep latency (min)</td>
<td>5.4</td>
<td>4.1</td>
<td>10.8</td>
<td>4.6</td>
<td>13.3</td>
<td>6.1</td>
<td></td>
</tr>
<tr>
<td>Mid-sleep period (h)</td>
<td>04:24</td>
<td>01:28</td>
<td>04:06</td>
<td>01:18</td>
<td>04:40</td>
<td>01:05</td>
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<tr>
<td>Total sleep time (h)</td>
<td>05:44</td>
<td>02:01</td>
<td>05:36</td>
<td>01:20</td>
<td>05:47</td>
<td>01:38</td>
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<tr>
<td>Nocturnal awakenings (min)</td>
<td>19.2</td>
<td>15.0</td>
<td>16.0</td>
<td>11.3</td>
<td>20.8</td>
<td>12.7</td>
<td></td>
</tr>
<tr>
<td>Sleep efficiency (%)</td>
<td>80.2</td>
<td>22.5</td>
<td>86.0</td>
<td>9.5</td>
<td>83.0</td>
<td>9.8</td>
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<tr>
<td>Naps (min)</td>
<td>4.5</td>
<td>2.6</td>
<td>11.0</td>
<td>5.8</td>
<td>10.1</td>
<td>6.3</td>
<td></td>
</tr>
<tr>
<td>Week with intervention at 21:00</td>
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<td></td>
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<td></td>
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<td></td>
</tr>
<tr>
<td>Sleep latency (min)</td>
<td>15.1</td>
<td>3.8</td>
<td>10.1</td>
<td>10.8</td>
<td>07.9</td>
<td>6.5</td>
<td></td>
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<tr>
<td>Mid-sleep period (h)</td>
<td>04:55</td>
<td>01:33</td>
<td>04:13</td>
<td>01:11</td>
<td>04:18</td>
<td>01:04</td>
<td></td>
</tr>
<tr>
<td>Total sleep time (h)</td>
<td>06:34</td>
<td>01:22</td>
<td>05:17</td>
<td>01:13</td>
<td>05:42</td>
<td>01:16</td>
<td></td>
</tr>
<tr>
<td>Nocturnal awakenings (min)</td>
<td>19.4</td>
<td>11.9</td>
<td>18.3</td>
<td>13.5</td>
<td>14.9</td>
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<tr>
<td>Sleep efficiency (%)</td>
<td>83.8</td>
<td>9.1</td>
<td>84.2</td>
<td>13.0</td>
<td>87.0</td>
<td>8.0</td>
<td></td>
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<tr>
<td>Naps (min)</td>
<td>25.7</td>
<td>13.8</td>
<td>13.5</td>
<td>6.0</td>
<td>15.6</td>
<td>8.2</td>
<td></td>
</tr>
</tbody>
</table>

*p Day of intervention.
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 ($F = 3.03; p = 0.05$). At 19:00 h bright light exposure, the participants sleepiness levels increased as expected, at KSS time tests (20:30, 22:00 h).

### Table 2

<table>
<thead>
<tr>
<th>KSS Days of the week</th>
<th>Monday</th>
<th>Tuesday</th>
<th>Wednesday</th>
<th>Thursday</th>
<th>Friday</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
</tr>
<tr>
<td>Week baseline</td>
<td></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>19:00</td>
<td>4.2</td>
<td>1.4</td>
<td>4.1</td>
<td>1.3</td>
<td>3.9</td>
</tr>
<tr>
<td>20:30</td>
<td>4.8</td>
<td>1.2</td>
<td>5.1</td>
<td>1.3</td>
<td>5.0</td>
</tr>
<tr>
<td>22:00</td>
<td>5.6</td>
<td>1.2</td>
<td>5.3</td>
<td>1.3</td>
<td>5.2</td>
</tr>
<tr>
<td>Week with intervention at 19:00</td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>19:00</td>
<td>3.5</td>
<td>0.7</td>
<td>4.2</td>
<td>1.5</td>
<td>3.4</td>
</tr>
<tr>
<td>20:30</td>
<td>7.0</td>
<td>2.8</td>
<td>4.8</td>
<td>1.3</td>
<td>3.9</td>
</tr>
<tr>
<td>22:00</td>
<td>5.0</td>
<td>0.0</td>
<td>5.2</td>
<td>1.2</td>
<td>4.3</td>
</tr>
<tr>
<td>Week with intervention at 21:00</td>
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<tr>
<td>19:00</td>
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<td>1.1</td>
<td>4.4</td>
<td>1.6</td>
<td>4.1</td>
</tr>
<tr>
<td>20:30</td>
<td>5.2</td>
<td>1.6</td>
<td>4.5</td>
<td>1.5</td>
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</tr>
<tr>
<td>22:00</td>
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<td>1.9</td>
<td>5.3</td>
<td>1.4</td>
<td>4.4</td>
</tr>
</tbody>
</table>

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

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. 

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.
But at 21:00 h, bright light exposure, the intermediate morning-evening 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 \( (p < 0.01; \text{Fig. 5}) \).

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. Lavioie 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 variables 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 class time 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 classroom lighting during the three class time 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 class time (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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