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Effect of exposure duration and light spectra on nighttime melatonin suppression in adolescents and adults

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Abstract

This study investigated how light exposure duration affects melatonin suppression, a well-established marker of circadian phase, and whether adolescents (13–18 years) are more sensitive to short-wavelength (blue) light than adults (32–51 years). Twenty-four participants (12 adolescents, 12 adults) were exposed to three lighting conditions during successive 4-h study nights that were separated by at least one week. In addition to a dim light (<5 lux) control, participants were exposed to two light spectra (warm (2700 K) and cool (5600 K)) delivering a circadian stimulus of 0.25 at eye level. Repeated measures analysis of variance revealed a significant main effect of exposure duration, indicating that a longer duration exposure suppressed melatonin to a greater degree. The analysis further revealed a significant main effect of spectrum and a significant interaction between spectrum and participant age. For the adolescents, but not the adults, melatonin suppression was significantly greater after exposure to the 5600 K intervention (43%) compared to the 2700 K intervention (29%), suggesting an increased sensitivity to short-wavelength radiation. These results will be used to extend the model of human circadian phototransduction to incorporate factors such as exposure duration and participant age to better predict effective circadian stimulus.

1. Introduction

It is now well established that the human circadian system is maximally sensitive to short-wavelength light^{1–10} and requires higher quantities of light to acutely suppress melatonin or to shift the timing of circadian rhythms compared to what the visual system requires to support visual performance.^{7,11–20} Several spectral weighting functions and one mathematical model have been proposed, based on data from published psychophysical studies of nocturnal melatonin suppression,^{8,12} to predict the circadian effectiveness of a light source. These functions vary in terms of their framework and their relative consideration of retinal neurophysiology.^{18,21–26} Research to date has primarily focused on the spectral sensitivity of the human circadian system, but it should be noted that exposure duration also has an impact on the amount of light needed to stimulate the circadian system. Early studies by McIntyre *et al.*^{27,28} showed that increasing light levels lead to increased

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melatonin suppression and required shorter durations of exposure for observing significant suppression.

Past studies have also shown that duration of exposure can influence the efficacy of a light treatment, with lower light level exposures of longer duration potentially having the same effect on the circadian system as higher light level exposures of shorter duration.^{29–33} Indeed, this dose relationship has long been applied in the treatment of seasonal affective disorder, which generally recommends that patients be exposed to 10 000 lux if the duration of treatment is 30 min and 2500 lux if the duration is 2 h.^{34–36} In contrast to these previously published studies, which employed a fixed spectrum and very high light levels, the present study investigated the impact of exposure duration on melatonin suppression from two light spectra (cool and warm) delivering lower light levels that are more representative of those commonly experienced in indoor environments.

It is well established that the classical photoreceptors (rods and cones) provide input to the intrinsically photosensitive retinal ganglion cells (ipRGCs), which are the main conduit of electrical signals from the retina to the suprachiasmatic nuclei (SCN), where the master clock is located.^{22–26} Lucas *et al.*²⁶ proposed a toolbox to allow researchers to report the effective irradiance experienced by each of the photoreceptors (i.e. rods, cones and ipRGCs) involved in non-visual responses. While this toolbox can be used for equating the stimulus–response relationships employed in different studies, as well as for relating research findings to lighting conditions in the field, it does not provide an indication of the circadian system's absolute response to a given light stimulus. Rea *et al.* have proposed a model of human circadian phototransduction that is based on fundamental knowledge of retinal neurophysiology and neuroanatomy, including the operating characteristics of circadian phototransduction (converting light into neural signals), from response threshold to saturation.^{18,37} The ipRGCs are the central elements in the phototransduction model, consistent with electrophysiological and genetic knockout studies.^{22–27} The model also accounts for neural input from the outer plexiform layer of the retina, consistent with studies showing that signals from rods and cones provide photic information to the ipRGCs.

In a relatively recent study, Gooley *et al.*³⁸ exposed participants to equal photon densities of 460-nm and 555-nm narrowband light for 6.5 h, showing that the degree of melatonin suppression was constant under the 460-nm light but that exposure to the 555-nm light elicited a response that was strong initially, yet diminished over time. The authors concluded that the relative contribution of cones and ipRGCs to the activation of non-visual light responses changes with varying exposure durations. The lack of resolution on how photoreceptors interact in response to light and how exposure duration may influence circadian system response calls for further investigation.

Another area requiring further investigation is whether the same light stimulus can differentially affect the circadian systems of participants from varying age groups, such as adolescents and adults.^{39–44} In a study conducted in a residential environment, Higuchi *et al.*⁴⁵ reported that overhead fluorescent room lighting (approximately 140±83 lux at the eye) effectively suppressed melatonin in primary school children but not in middle-aged adults. Recently, researchers at the Lighting Research Center (LRC) at Rensselaer Polytechnic

Institute conducted evening/nighttime studies investigating the impact of self-luminous devices on melatonin levels in different age groups. One study involving 13 young adult participants showed that even though the use of self-luminous devices did not significantly suppress melatonin after 1 h, suppression was statistically significant after a 2-h exposure.³⁹ A follow-up study involving 20 adolescent participants demonstrated that both 1-h and 2-h exposures to self-luminous devices significantly suppressed melatonin by 23% and 38%, respectively.⁴⁰ In view of the consistent protocol between the two studies, the authors suggested that a higher measured suppression could be accounted for by an enhanced sensitivity to light at night, particularly short-wavelength light, among adolescents. This hypothesis is well supported by studies conducted by Crowley *et al.*,^{41,42} who showed that the temporal alignment of the endogenous circadian pacemaker in adolescents can be quite different from that in older individuals due to the maturation of biological processes regulating sleep–wake systems and alteration by prevalent psychosocial demands.

Thus, the goals of the present study were to better understand the effect of extended light exposure durations on the suppression of melatonin, a well-established marker of circadian phase, and to explore whether adolescents may be more sensitive to short-wavelength light than middle-aged adults.

2. Materials and methods

2.1. Participant selection

A total of 24 participants (19 females and 5 males) took part in the study. Twelve of the participants (nine females and three males) were adolescents with an age range of 13–18 years and a mean±standard deviation (SD) age of 16.5±1.9 years. The ages of the 12 adult participants ranged 32–51 years, with a mean±SD age of 46±5.2 years. The mean±SD Munich Chronotype Questionnaire scores⁴⁶ recorded for the adolescents and adults were 3.67±0.89 and 2.27±1.35, respectively, which suggests that both groups of participants were neither extreme larks (early persons) nor extreme owls (late persons). All participants were pre-screened for major health problems, such as bipolar disorder, seasonal depression, cardiovascular disease, diabetes and high blood pressure. Participants were excluded from the study if they were taking over-the-counter melatonin or any prescription medications such as blood pressure medicine, antidepressants, sleep medicine or beta-blockers, or reported any eye diseases such as cataracts and glaucoma.

Given that all participants were either in school or regularly employed, they were able to follow a consistent sleep–wake schedule (bedtimes no later than 23:00 and wake times no later than 07:30) during the week before the study night to maintain their melatonin circadian rhythm. Participants were also required to refrain from caffeine consumption for 12 h prior to the start of each study night. None of the participants reported difficulties in complying with the schedule or sleep-related disturbances over the course of the study.

The present study conformed to 45 CFR 46 and international ethical standards⁴⁷ and was reviewed, approved and monitored by Rensselaer Polytechnic Institute's Institutional Review Board. Informed consent was obtained from all study participants and/or their legal guardians.

2.2. Experimental conditions

The participants were required to come to the laboratory on three nights, each separated by at least one week to allow a wash-out period between the conditions. Over the course of the study, all participants were exposed to two spectrally distinct white light sources with correlated colour temperatures (CCTs) of 2700K and 5600K (Figure 1), which were delivered at a constant light level to provide a circadian stimulus (CS) of 0.25 as calculated using the model proposed by Rea *et al.*¹⁸

Using empirical, light-induced nocturnal suppression data from Brainard *et al.*¹² and Thapan *et al.*,⁸ the Rea *et al.*¹⁸ model characterizes the absolute and spectral sensitivities of the human circadian system to light, as measured by acute melatonin suppression. The spectral irradiance at the cornea is first converted into circadian light (CL_A), reflecting the spectral sensitivity of the circadian system, and then, secondly, transformed into a CS value that reflects the absolute sensitivity of the circadian system. Thus, CS is a measure of the *effectiveness* of the retinal light stimulus for the human circadian system from threshold (CS=0.1) to saturation (CS=0.7). The model does not take the light exposure's duration into account, however, and assumes a duration of 1 h. The photometric characteristics of the experimental conditions are provided in Table 1, and the α -opic irradiances^{26,48,49} for the experimental conditions are provided in Table 2. Although the 5600 K light source had a peak spectral sensitivity around 450 nm, it did not present risk for blue-light hazard, as the light sources were placed overhead, behind diffusers, and the participants were not asked to look directly at the light-emitting diodes (LEDs).⁵⁰

Finally, all of the participants were exposed to a dim light control condition, which permitted a baseline observation of the natural rise in participants' melatonin levels over the course of the study night.

2.3. Lighting apparatus

The stimulus for both white light sources was provided and controlled through overhead LED lighting (BeveLED2.0, 2200–6000 K, USAI Lighting, New Windsor, NY, USA). Spectrally neutral diffusers covered the luminaires to eliminate any potential glare and provide a uniform distribution. Calibration of the light stimulus was performed using a tripod-mounted illuminance meter (Model X-91 Broadband Lightmeter, Gigahertz-Optik, Haverhill Rd, Amesbury, MA, USA) to verify the light levels at the participants' eyes (i.e. vertical illuminance (E_V)). Target light levels were computed using the free online CS calculator,⁵¹ created by LRC researchers, to provide a target CS of 0.25 throughout the exposure period. For calibration purposes, it was assumed that the participants' line of sight was parallel to the floor. During each 4-h data collection period, light levels at the eye were spot-checked at each saliva sample time (see Protocol section) using a spectrometer (Model USB650 Red Tide Spectrometer, Ocean Optics, Winter Park, FL, USA) and the tripod-mounted illuminance meter. The mean $\pm$ SD CS values recorded across the two spectra were 0.25 $\pm$ 0.005 for the adolescents and 0.25 $\pm$ 0.011 for the adults.

2.4. Protocol

All participants for each study night were from the same age group and experienced the same experimental condition in a single laboratory. All participants arrived at the laboratory by 22:30 and remained in dim light (<5 lux at the eye level) for 30 min, followed by a 4-h exposure to one of the three experimental conditions listed in Table 1. In order to counter any potential subject-expectancy effect, no information concerning the pre-decided order of exposure to the three conditions was provided to the participants, although subjective assessments were not conducted to ascertain whether the participants could differentiate between the two lighting interventions. Over the course of each study night, five saliva samples were collected from each participant; the first sample was taken immediately before the beginning of the experimental condition after a 30-min dim light exposure, and four additional samples were taken thereafter at 1-h intervals (Figure 2). At 03:00, after the final saliva sample was collected, participants were released to go home.

Participants were free to operate their personal electronic devices (i.e. computers, tablets, cell phones) and were required to perform a similar task (e.g. browse the internet, watch a video or movie, read an e-book, etc.) on all study nights. All displays were covered with orange-tinted media that filtered out radiation <525nm (Roscolux #21 golden amber, Rosco Laboratories, Stamford, CT, USA) to prevent participants from receiving additional circadian-effective stimulus from their self-luminous devices. Periodic visual monitoring was carried out to ensure compliance with the experimental protocol and confirm that none of the participants closed their eyes.

Saliva samples were collected using the Salivette system (Sarstedt, Nümbrecht, DE), wherein the participant chews on a plain cotton cylinder, which is then placed in a test tube and centrifuged for 5 min at 1000 *g*. Each saliva sample was immediately frozen (−20°C). The frozen samples for each participant were assayed in a single batch using melatonin radioimmunoassay kits (Direct Melatonin RIA, ALPCO, Salem, NH, USA). The reported sensitivity of the saliva sample assay was 1.4 pg/mL and the intra- and inter-assay coefficients of variability were 12.1% and 13.2%, respectively.

2.5. Data analysis

Melatonin suppression for each condition was determined by comparing melatonin concentration levels collected during the dim light night (study night 1), the control condition, to those collected at the same time (T2–T5) on each lighting intervention night (study nights 2 and 3). For each study night, melatonin concentrations from T2–T5 were first normalized to T1, and the melatonin suppression at each time (T2–T5) was then calculated using the following formula

$$\text{Percent suppression} = 1 - \left(\frac{Mn}{Md} \right) \quad (1)$$

where M_n is the normalized melatonin concentration at each time on respective intervention nights (study nights 2 and 3) and M_d is the normalized melatonin concentration at each time on the dim light control night (study night 1).

Using melatonin suppression and variance data from previous studies,^{39,40} an a priori power calculation (SD=0.15) revealed that 12 participants in each group had an effect size of 0.8 and a power of 99.9% to significantly detect 25% melatonin suppression and a power of 93.4% to significantly detect 15% melatonin suppression. Statistical analyses were performed using mixed repeated measures analysis of variance (ANOVA). Age was used as a between factor. Exposure duration and light source spectrum were used as within factors. Further evaluation for the main effects and interactions was performed using post hoc two-tailed paired samples Student's *t*-tests. Tests were considered statistically significant if the resulting *p* value was <0.05. Bonferroni corrections were applied when needed. All data generated or analysed during this study are included in this published article.

3. Results

The ANOVA revealed a significant main effect of exposure duration ($F_{3,66}=21.99$, $p<0.05$, $\eta_p^2=0.50$), indicating that a longer duration exposure suppressed melatonin to a greater degree during participants' biological night (Figure 3). Examining the main effect of time, post hoc two-tailed paired samples *t*-tests using the Bonferroni correction indicated that the melatonin suppression after a 1-h exposure was significantly lower than after a 3-h exposure ($t_{47}=-7.01$, $p<0.05$), resulting in mean $\pm$ SEM suppression of 21% $\pm$ 4% and 41% $\pm$ 4%, respectively. Melatonin suppression after a 1-h exposure was also significantly lower than after a 4-h exposure ($t_{47}=-8.54$, $p<0.05$), the latter resulting in a mean $\pm$ SEM percentage suppression of 45% $\pm$ 3%. Furthermore, the mean melatonin suppression after a 2-h exposure (mean $\pm$ SEM=32% $\pm$ 5%) was also significantly lower than the values recorded after 3-h ($t_{47}=-5.38$, $p<0.05$) and 4-h ($t_{47}=-5.51$, $p<0.05$) exposures. There was no significant difference between the melatonin suppression observed after 3-h and 4-h exposures ($t_{47}=-2.87$, $p>0.05$).

The ANOVA also revealed a significant main effect of light source spectrum ($F_{1,22}=10.51$, $p<0.05$, $\eta_p^2=0.32$) and a significant interaction between spectrum and participant age ($F_{1,22}=9.14$, $p<0.05$, $\eta_p^2=0.29$), as shown in Figure 4. The mean $\pm$ SEM melatonin suppression over the entire 4-h exposure was 33% $\pm$ 2% after adults experienced the 2700K source and 33% $\pm$ 3% after they experienced the 5600K source. For the adolescent participants, the mean $\pm$ SEM melatonin suppression over the entire 4-h period was 29% $\pm$ 4% after exposure to the 2700K source and 43% $\pm$ 4% after exposure to the 5600K source. Examining the significant interaction between spectrum and participant age, post hoc two-tailed paired samples *t*-tests showed that melatonin suppression did not differ between the two spectra for the adults ($t_{47}=-0.24$, $p>0.05$), but for adolescents, melatonin suppression was significantly greater after exposure to the 5600K source than the 2700K source ($t_{47}=-7.01$, $p<0.05$), even though the circadian stimulus CS (0.25) was the same. The effect size for this analysis ($d=0.54$) exceeded Cohen's⁵² criterion for a medium effect ($d=0.50$).

Although no other interactions were statistically significant, we were interested in examining the overall pattern of suppression for each age group at each saliva sample time. Figure 5 shows melatonin suppression for the adults and adolescents at each saliva sample time for both intervention spectra. Specifically, post hoc two-tailed paired samples *t*-tests showed that melatonin suppression did not significantly differ ($p>0.05$) between the two spectra for the adults at all four saliva sample times (T2–T5). For the adolescents, melatonin suppression did not significantly differ ($p>0.05$) between the two spectra after a 1-h exposure (T2), but it was significantly greater after exposure to the 5600K source than the 2700K source after 2-h ($t_{11}=-5.79$, $p<0.05$), 3-h ($t_{11}=-3.12$, $p<0.05$) and 4-h ($t_{11}=-4.18$, $p<0.05$) exposures.

4. Discussion

This study set out to better understand how light exposure duration affects melatonin suppression and to determine whether adolescents are more sensitive to short-wavelength light than adults. The results showed that melatonin suppression tends to saturate with increasing duration of exposure for both age groups and for both light source spectra, especially after 3 h (T4). More specifically, the rate of suppression (i.e. the mean absolute percent suppression per minute of exposure) for the first hour (35%) was almost twice as effective compared to the overall 4-h exposure (19%) and over five times as effective compared to the last hour (7%). This trend of a non-linear circadian response to light exposures of varying durations is consistent with the results from Chang *et al.*³⁰ and St. Hilaire *et al.*,³² who investigated the effect of exposure duration on the phase shifting of dim light melatonin onset, a well-established marker of circadian phase. Specifically, Chang *et al.*³⁰ reported that per minute of exposure, a 12-min light pulse was found to be over five times more effective at phase delaying the circadian pacemaker than a 4-h light pulse. Similarly, St. Hilaire *et al.*³² reported that the amplitude of the phase response curve for a 1-h light exposure was approximately 40% of that for a 6.7-h exposure while representing only 15% of the latter light exposure's duration.³² Our results are also consistent with those of Gooley *et al.*,³⁸ who showed a similar response for melatonin suppression, particularly for a narrow-bandwidth light source peaking close to 460 nm. More specifically, Gooley *et al.* showed that melatonin suppression increased during the first 3-h exposure period, remained the same between the 3-h through 4.5-h exposure periods and then decreased with an exposure duration that was greater than 4.5 h. A similar trend was observed with the melatonin data reported from the present study, wherein post hoc *t*-tests revealed that suppression after a 3-h exposure (T4) did not significantly differ from the suppression after a 4-h exposure (T5). Furthermore, absolute melatonin suppression data after a 4-h exposure from the present study (approximately 45%) appears to be comparable to melatonin suppression after a 4.5-h exposure (third quadrant) from the Gooley *et al.* study (approximately 60%), when matched for effective CS ($CS=0.21$ (13.2 log photons $\text{cm}^{-2} \text{s}^{-1}$)).³⁸

As revealed by the significant interaction between age and spectrum, as well as by post hoc *t*-tests, the overall mean melatonin suppression for adults (see Figure 4) did not significantly differ for the two spectra, which were matched to provide equal CS. The adolescents, however, consistently (T2–T5) and significantly (T3–T5) suppressed more melatonin when exposed to the 5600K source compared to the 2700K source (see Figure 5). A similar trend

was reported by Gabel *et al.*,⁵³ who showed that melatonin suppression after exposure to both a warm (2800K (250 lux)) and a blue-enriched (9000K (250 lux)) light source was similar for adults (n=12, mean±SEM age=63.6±1.3 years) but more pronounced for young adults (n=26, mean±SEM age=25.0±0.6 years) after exposure to the blue-enriched light. (It should be noted, however, that as the study's authors calibrated the lighting intervention in terms of equivalent photopic light levels (lux) rather than equivalent CS levels, the higher melatonin suppression recorded for the 9000 K source should be expected). Figueiro *et al.*⁴⁰ also reported that a 1-h exposure to self-luminous devices delivering a very low CS of 0.04 (at approximately 6500 K) was sufficient to significantly suppress melatonin by 23% in adolescents.

The greater melatonin suppression after exposure to the 5600K lighting intervention in the adolescents group is not likely to be due to differences in circadian phase between the two groups. First, melatonin suppression for the adolescents after a 1-h exposure to the 2700K (18%) lighting intervention was very similar to the melatonin suppression experienced by the adults after the 2700K (18%) and 5600K (19%) interventions. Second, although adolescents are known to have a later circadian phase, the fact that they were attending school and maintaining a regular sleep–wake schedule may have helped to keep them in an earlier circadian phase. In fact, absolute melatonin levels measured prior to the light exposure (following the initial 30-min exposure to dim light) were not significantly different between the two age groups across all three study nights (Table 3). These findings indicate that all participants received the light stimulus at the same circadian phase.

Another possible explanation for this increased sensitivity to short-wavelength light might be age-related changes in lens transmittance and macular pigment density.⁵⁴ Although it has been shown that spectral retinal illuminance losses due to ageing are most pronounced at shorter wavelengths,⁵⁵ decreases in non-visual responses are predominantly governed by loss of absolute sensitivity associated with ageing rather than changes in spectral sensitivity.⁵⁶ When age is taken into account, the current CS model predicted effective CS of 0.31 and 0.33 for adolescents (as opposed to a CS of 0.25 for adults) after a 1-h exposure to the 2700K and 5600K sources, respectively, based on pupil-weighted spectral lens transmittance data provided by Turner and Mainster.⁵⁵ But the measured melatonin suppression (19%) for the adolescents following a 1-h exposure to the 2700K source instead more closely corresponded to the suppression measured in the adults (18%). Therefore, adolescents' enhanced sensitivity to the 5600K source cannot be explained by lens transmittance alone. A recent study by Najjar *et al.*⁵⁷ also showed that even though the lens transmittance was significantly lower in older subjects compared to young subjects, melatonin suppression was not significantly different between the age groups when matched for a photic stimulus at 480 nm.^{53,57–61}

The present results are inconsistent with the Gooley *et al.*³⁸ hypothesis that at the start of a lighting exposure episode, cones and ipRGCs both substantially affect the non-visual response, whereas for longer exposure durations, the non-visual response is mainly a response by the ipRGCs, as demonstrated by the lack of interaction between light spectra and exposure duration. The light levels of the 555-nm light source employed in that study (approximately 11.40–14.18 log photons cm⁻² s⁻¹) had a significantly lower CS

($CS_{max}=0.17$) than the 460-nm light source ($CS_{max}=0.55$). Therefore, one explanation for these results would be that the stimulus provided by the 555-nm source was not sufficiently strong to counteract the normal rise in melatonin levels at night and thus failed to maintain suppression over extended exposure hours. As the CS measure accounts for the spectral and absolute sensitivities of melatonin suppression, and is therefore a more accurate predictor of the response, the lower CS delivered by the 555-nm source employed in the Gooley *et al.* study provides a better rationale for their results and should be considered as an alternate explanation. Further studies should be designed to specifically test, a priori, the hypothesis of Gooley *et al.*³⁸

A few limitations are worth noting. First, participants' light exposures in the week prior to the laboratory session were neither monitored nor controlled. Given that all participants were full-time employees or students with regular schedules, however, it was assumed that they would receive consistent light exposures during all three weeks of the study. Second, participants were free to move their gaze, so the retinal light exposures were undoubtedly more variable than if the participants had been asked to look into a Ganzfeld sphere and keep their chin on a chin rest. This may have impacted the participants' actual received retinal light exposures because the study protocol did not mandate that participants maintain a near horizontal line-of-sight, which was the basis for the calibration of the overhead lighting. As a result, the actual light exposures at the eye were likely reduced because participants generally were looking downward to view their portable electronic devices (see Protocol section). These behavioural factors may explain why the resulting mean $\pm$ SEM melatonin suppression following a 1-h exposure to the 2700K and 5600K sources for the adults was 18% $\pm$ 6% and 19% $\pm$ 5%, respectively, and for the adolescents was 19% $\pm$ 7% and 28% $\pm$ 5%, respectively, rather than the predicted 25% suppression.

To address that difference, eye-level spot measurements were performed at each participant's angle of gaze throughout the experiment using a spectrometer and illuminance meter (see Lighting apparatus section). As these measurements were found to be consistent with the target CS of 0.25, adjustments to the experimental lighting were deemed unnecessary. For the adults, the spot-measured CS values across all sample times ranged 0.22–0.27 (mean $\pm$ SD=0.25 $\pm$ 0.013) and 0.25–0.26 (mean $\pm$ SD=0.25 $\pm$ 0.006) for the 2700K and 5600K sources, respectively. For the adolescents, the spot-measured CS values across all sample times ranged 0.24–0.26 (mean $\pm$ SD=0.25 $\pm$ 0.005) and 0.24–0.26 (mean $\pm$ SD=0.25 $\pm$ 0.006) for the 2700K and 5600K sources, respectively. Lack of continuous measurements throughout each experimental session is, however, a limitation of this study.

Third, although a gender disparity existed among the study's participants (79% females), it should be noted that past studies have reported no differences in measures of circadian preference and sleep with respect to sex.^{62,63}

Broadly speaking, the results of this study will be helpful for making lighting recommendations when considering non-visual responses for general applications such as offices, schools, residences and healthcare facilities, where the respective populations spend several hours in a designated space. Most of the light levels employed in this study are well

within the range of the IES-recommended light levels for these spaces and therefore would allow easy translation of the results to field expectations.¹⁴

Finally, data from the current study will be used to extend the model developed by Rea *et al.*¹⁸ by incorporating additional factors such as exposure duration and participant age to better predict effective CS. The extended-model will have numerous application benefits such as specifying ideal light stimuli for shift-work spaces, wherein the target light levels will be high enough to accommodate visual requirements but lower than the threshold required to have an extended effect on the circadian system from long-duration light exposures. For the near future, we recommend considering adolescents' higher sensitivity when developing lighting designs for residences, childcare facilities and schools, as well as when designing specifications for self-luminous devices.

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Figure 1.
The relative spectral power distributions for the two lighting interventions used in this study

Figure 2.

The protocol used in this study, showing the relationship between the successive study nights (listed in order of occurrence), lighting conditions and saliva sample times

Figure 3.

The significant main effect of duration of exposure. The bars show the combined mean melatonin suppression (T2–T5) for both age groups and the two intervention spectra (both providing CS=0.25). The error bars represent SEM, and the asterisks denote statistical significance

Figure 4.

The significant interaction between spectrum and participant age. The bars show mean melatonin suppression over the entire 4-h light exposure, by age group and intervention spectrum (both providing CS=0.25). The error bars represent SEM, and the asterisk denotes statistical significance

Figure 5. Mean melatonin suppression recorded for the adults and adolescents during exposure to each of the two intervention spectra at saliva sample times. The error bars represent SEM, and the asterisks denote statistical significance

Table 1

Specifications of the experimental conditions employed in this study

Experimental condition	Vertical illuminance (E_v) ^a (lux)	Circadian stimulus	Photon flux density (log photons $\text{cm}^{-2} \text{s}^{-1}$)
Dim light (control)	<5	0	<12.50
2700 K	295	0.25	14.46
5600 K	209	0.25	14.28

^a E_v indicates light levels calibrated for delivery at participants' eyes.

Table 2

Calculations of the five α -opic irradiances for the experimental conditions employed in this study, following the SI-compliant approach recommended by the CIE

Experimental condition	Cyanopic irradiance ^a ($\mu\text{W} \cdot \text{cm}^{-2}$)	Melanopic irradiance ^a ($\mu\text{W} \cdot \text{cm}^{-2}$)	Rhodopic irradiance ^a ($\mu\text{W} \cdot \text{cm}^{-2}$)	Chloropic irradiance ^a ($\mu\text{W} \cdot \text{cm}^{-2}$)	Erythroopic irradiance ^a ($\mu\text{W} \cdot \text{cm}^{-2}$)
Dim light (control)	0	0	<0.1	<0.5	<1.0
2700 K	4.8	13.8	21.0	37.2	51.3
5600 K	13.0	20.9	26.2	32.3	35.0

CIE: International Commission on Illumination; SI: International System of Units (SI).

^aBased on the spectral irradiance distributions of the light sources, α -opic irradiances are calculated using the CIE's SI-compliant version^{48,49} of the Lucas *et al*²⁶ toolbox.

Table 3

Mean±standard deviation of absolute salivary melatonin levels prior to light exposure, following a 30-min exposure to dim light for the experimental conditions and age groups

Experimental condition	<u>Mean±SD absolute salivary melatonin level (pg/mL)</u>	
	Adults	Adolescents
Dim light (control)	8.2±4.5	8.5±3.2
2700 K	7.5±4.5	8.9±3.3
5600 K	7.5±4.0	9.1±3.5