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Effects of Evening Exposure to Light from Organic Light-Emitting Diodes on Melatonin and Sleep

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Background and Purpose Organic light-emitting diodes (OLEDs) emit less blue light than traditional light-emitting diodes (LEDs), but the effects of OLED light exposure (LE) on melatonin and sleep have not been evaluated.

Methods Twenty-four healthy subjects (age 26.9±5.7 years; including 18 females) with the intermediate chronotype were exposed to three different light conditions [4,000 K 150 lux OLED LE, 4,000 K 150 lux LED LE, and dim light (DL) at <10 lux] for 6.5 h from 17:30 to 24:00, in a random order and with a 1-week interval. Participants entered the unit for the experiment at 16:00, and their daylight was measured by actigraphy from 8:00 to 16:00 during each session. Saliva samples for melatonin were taken every hour from 18:00 to 24:00. Sleep was monitored by polysomnography, and vigilance was evaluated by psychomotor vigilance test upon awakening.

Results Melatonin onset occurred at 21:11±01:24, 21:20±01:19, and 21:36±01:16 in the DL, OLED, and LED conditions, respectively. Melatonin onset was significantly delayed under LED LE compared to DL (p=0.007) but did not differ under OLED LE (p=0.245). Melatonin suppression, sleep parameters, and vigilance were similar among the three light conditions. The accumulated amount of daytime light in each session was negatively correlated with the melatonin onset time under the DL (rho=-0.634, p=0.002) and OLED (rho=-0.447, p=0.029) conditions, not under the LED condition (p=0.129).

Conclusions Melatonin onset under OLED LE was not significantly delayed compared to DL. Exposure to sufficient daylight may advance melatonin onset even when a subject is exposed to OLED LE in the evening.

Key Words light, melatonin, sleep, circadian rhythm.

INTRODUCTION

In modern societies people are exposed to significantly more artificial light at nighttime.[1] Exposure to light in the evening and early night suppresses the release of melatonin and can shift circadian phases to later times.[2,3] These effects of light are dependent on the light intensity, wavelength, and duration of exposure.[4-9] Exposure to blue light (i.e., wavelengths of 446–483 nm) can be particularly detrimental, since the transmission of light information to the primary circadian pacemaker in the brain is mediated by the intrinsically photosensitive retinal ganglion cells of the eyes that contain melanopsin, which are most sensitive to blue light.[10-13]

Light-emitting diodes (LEDs) are abundantly used for indoor illumination, partly for energy-saving reasons,[14] and their emission spectrum is rich in blue light. Harmful effects of exposure to artificial light at night on human health are being increasingly recognized,[15-18] with the International Energy Agency formally releasing a report confirming the potential

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hazards of blue LEDs to human eyes and the circadian rhythm in 2014.[19]

Organic LEDs (OLEDs), which emit less blue light, are energy-efficient and more physiologically friendly form of lighting.[20-22] OLED light exposure (LE) may decrease the adverse health outcomes of artificial light at night, but no studies have investigated the impact of OLED LE at night on sleep quality and the circadian system. The impacts of OLED LE on melatonin secretion and sleep quality need to be measured in order to determine whether OLED LE represents an improvement over LED LE.

We aim to compare the effects of OLED LE and LED LE during the hours before bedtime on melatonin secretion and objective sleep quality. We hypothesized that evening OLED LE would result in less melatonin suppression, smaller circadian phase delays, and better sleep quality and daytime vigilance when compared to LED LE.

METHODS

Ethical approval

The screening and study procedures were approved and monitored by the Institutional Review Board of the Samsung Medical Center (IRB No. 2017-08-125) and were conducted in compliance with the Declaration of Helsinki and the Good Clinical Practice guidelines. Informed written consent was obtained from all study participants prior to enrollment, and they were compensated financially for their participation.

Subjects and study screening

This study involved 24 healthy adults with the intermediate chronotype as defined by a morningness-eveningness questionnaire. Participants with chronic medical or psychological conditions and sleep disorders including sleep-disordered breathing with an apnea-hypopnea index exceeding 15/h in polysomnography (PSG) were excluded from the study. Participants completed a 7-day sleep-wake diary, and all of them typically slept between 23:00 and 08:00. They also completed questionnaires about sleep and mood, including the Pittsburgh Sleep Quality Index (PSQI), Beck Depressive Inventory (BDI), and Insomnia Severity Scale (ISI).

Participants were required to avoid unusual LE (e.g., going out during their habitual sleep time) during the evening and nighttime for a minimum of 1 week prior to testing. They were instructed to maintain a regular sleep schedule for 1 week prior to participating in this study. Sleep and LE as monitored by wrist actigraphy (Actiwatch-Spectrum Pro, Philips/Respironics, Bend, OR, USA) were obtained from all subjects from 1 day before each session. The average LE during the daytime from 08:00 to 16:00 before being admitted to the unit was measured by actigraphy.

Study protocol

Each subject visited the unit for three separate sessions, each separated by 1 week. The study protocol is summarized in Fig. 1. Participants were admitted to the light-control unit around 16:00. They consumed the provided meal and brushed their teeth by 17:30. During their stay they avoided consuming alcohol- and caffeine-containing foodstuffs, and were allowed to drink only water. After applying PSG electrodes, the 6.5-h experimental LE was initiated. Participants were instructed to sit on a chair and to remain awake during the LE. Watching television or using electrical devices was prohibited in order to avoid any additional LE. Saliva samples were collected hourly from 18:00 to 24:00. The participants went to bed to sleep with lights off at around 24:00, and woke up between 06:00 and 07:00 ad libitum. Psychomotor vigilance test (PVT) was performed upon awakening for 5–10 min. A research assistant kept watch on the wakefulness and procedures of the participants using video and EEG monitoring in the control room of the light-control unit throughout the study.

Light conditions

Three light conditions were tested: 4,000 K OLED LE, 4,000 K LED LE, and dim light (DL; <10 lux). The light sources

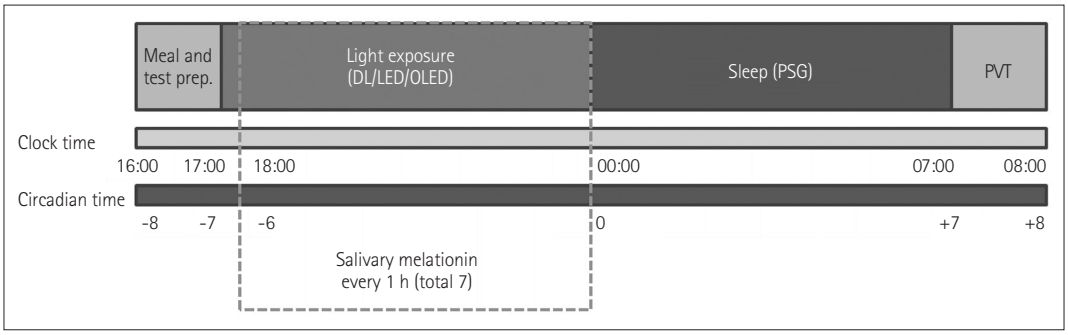

Fig. 1. Study protocol. DL: dim light, LED: light-emitting diode, OLED: organic LED, PSG: polysomnography, PVT: psychomotor vigilance test.

were ceiling-mounted light panels (LL167RS1-74P1, 300×300 mm, 24 W, LG display, Seoul, Korea; LED Edge panel, 315×315 mm, 20 W, SIGMA LED, Incheon, Korea). The spectral power distributions of the light produced by OLEDs and LEDs are shown in Supplementary Fig. 1 (in the online-only Data Supplement). The three conditions of experimental LE were performed in a random order with a 1-week interval between each condition. The illuminance level of both the OLED light and the LED light at eye level in a sitting position was maintained at 150 lux. A uniform illumination environment was produced in the room. The light illuminance was measured using a lux meter (Testo 540, Testo, Lenzkirch, Germany). Subjects were in darkness (<0.02 lux, <0.00006 W/m^2) during the scheduled sleep time.

Outcome measures of melatonin onset time and suppression

Salivary melatonin was collected using polyester swab Salivettes (Sarstedt, Nümbrecht, Germany). Samples were stored in the freezer until delivery to the laboratory for measuring melatonin concentrations, which was performed using liquid chromatography-tandem mass spectrometry. The minimum detectable dose of melatonin (analytical sensitivity) was determined to be 0.5 pg/mL. The melatonin onset time was calculated as the time when the melatonin concentrations exceeded the mean of three low consecutive values (before the increase) plus twice the standard deviation.[23]

Melatonin suppression was assessed using the area under the time-melatonin concentration curve (AUC) from 18:00 to 24:00 (immediately before lights off) under LED LE or OLED LE, compared with the AUC under DL during the same period. Melatonin suppression was calculated as a percentage using the formula 100-[(AUC_{LE}/AUC_{DL})×100)].

Outcome measures of sleep and vigilance

Subjects underwent PSG (Embla N7000 & RemLogic, Embla, Denver, CO, USA) each night following LE. Sleep staging and arousals were scored according to the 2017 updated guidelines of the American Academy of Sleep Medicine.[24]

PVT was conducted using automated psychometric measures that are commercially available from Joggle Research (https://admin.joggleresearch.com). The following PVT metrics were obtained: mean reaction times, errors (pressing the wrong button or failing to release the button for 3 s or longer), and lapses (reaction times ≥355 ms).

Statistical analysis

All statistical analyses were performed using SPSS (version 18.0 for Windows, SPSS Inc., Chicago, IL, USA). Data were tested for normality using the Shapiro-Wilk test. The paired *t*-test was used to compare melatonin suppression between LED LE and OLED LE. Repeated-measures ANOVA was used to compare the average LE during daytime before the experiment, AUC, melatonin onset time, PSG, and PVT parameters among the three light conditions. If significant differences among the three light conditions were detected by the repeated-measures ANOVAs, we performed paired-samples *t*-tests to compare parameters between pairs of light conditions (i.e., DL vs. OLED, DL vs. LED, and OLED vs. LED). Associations between LE during the daytime, melatonin onset time, and AUC were evaluated using Spearman correlation coefficients. Bonferroni correction was applied in multiple comparisons, with significance defined as $p<0.017$, while $p<0.05$ was considered indicative of statistical significance in other tests.

RESULTS

Subject characteristics

The 24 subjects in this study were aged 26.9±5.7 years (mean ±standard deviation), and included 18 females. They did not have any definite sleep-related complaints (PSQI score=5.3±2.4 and ISI score=4.8±3.4) or depressive mood (BDI score=6.0±3.9). Their sleep logs indicated that the habitual sleep time was between 23:53±00:52 and 07:20±01:37. The accumulated amounts of daylight (8:00−16:00) did not differ between the three conditions (Table 1).

Melatonin onset time and suppression

The melatonin onset time differed significantly between the three groups, at 21:11±01:24, 21:20±01:19, and 21:36±01:16 in the DL, OLED, and LED conditions, respectively ($p=0.011$) (Fig. 2 and Table 1). The melatonin onset time was significantly delayed for LED LE compared to DL ($p=0.007$), whereas it was not delayed for OLED LE ($p=0.287$), and showed a trend toward a delay for LED LE compared to OLED LE ($p=0.078$). A negative correlation was observed between the daylight amount and melatonin onset time in the DL (rho=-0.634, $p=0.002$) and OLED (rho=-0.447, $p=0.029$) conditions, but not in the LED condition ($p=0.129$). The melatonin onset time, delay, and suppression under the different light conditions are summarized in Fig. 3 and Table 1.

The mean AUC did not differ among the three light conditions ($p=0.207$). The median suppression percentages for melatonin for OLED LE and LED LE were 3.2% and 12.6%, respectively. Melatonin suppression was not significantly different between OLED and LED lighting ($p=0.208$). The accumulated daylight was not correlated with AUC in each condition ($p>0.05$).

Table 1. Salivary melatonin suppression, melatonin onset time, and daylight in the three study conditions

	DL	OLED	LED	*p*
AUC, pg/mL×h	39.9±47.0	38.8±54.5	32.4±40.7	0.207
Melatonin suppression, %	-	3.2 (-19.8, 20.7)	12.6 (-2.9, 43.4)	0.208
Melatonin onset time	21:11±01:24	21:20±01:19	21:36±01:16	0.011*
Melatonin onset delay, min	-	9±35	25±37	0.078
Log of total amount of accumulated LE during daytime (8:00–16:00) before the experiment	14.8±4.6	15.6±4.7	16.4±4.6	0.356
Correlation between LE before the experiment and melatonin onset time†	-0.634†	-0.447†	-0.342	

Data are mean±standard-deviation or median (first, third quartile) values.
*p<0.05 in repeated-measures ANOVA, †Spearman correlation coefficients (rho values), †p<0.05.
AUC: area under the time-melatonin concentration curve, DL: dim light, LE: light exposure, LED: light-emitting diode, OLED: organic LED.

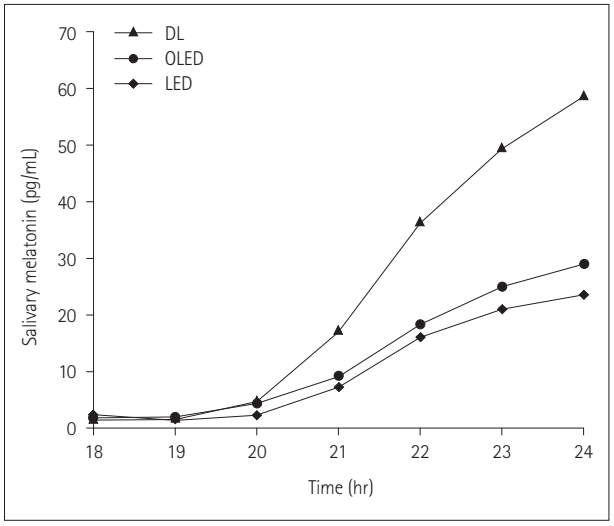

Fig. 2. Representative melatonin profiles. The melatonin profiles from 18:00 to 24:00 from one representative individual under the three light conditions (DL, OLED, and LED). DL: dim light, LED: light-emitting diode, OLED: organic LED.

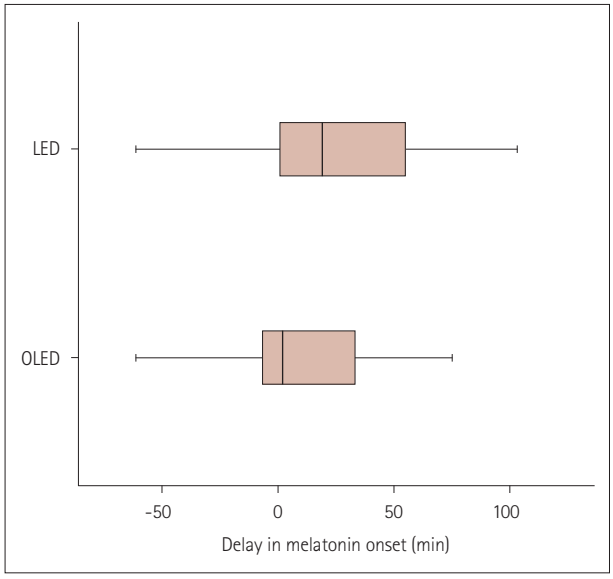

Fig. 3. Melatonin phase shifts under OLED LE and LED LE compared to DL. Values on the χ-axis are differences between melatonin onsets for LED LE and OLED LE compared to DL. Each box plot shows the median, first and third quartiles, and range. Melatonin onset for LED LE was significantly delayed compared to DL (p=0.007), whereas OLED LE did not significantly delay melatonin onset compared to DL (p=0.287). DL: dim light, LE: light exposure, LED: light-emitting diode, OLED: organic LED.

Sleep and vigilance

The PSG parameters did not differ significantly among the conditions (Table 2). In PVT, the number of lapses (5.8±4.1 vs. 2.8±2.3) and the reaction time (1/response time=3.7±0.7 vs. 4.0±0.5) were lower for LED LE than for DL (Table 2). The numbers of errors were similar among the conditions.

DISCUSSION

This is the first study of humans to compare the effects of OLED LE and LED LE in the evening on the melatonin profile, sleep quality, and vigilance. We found that melatonin onset occurred significantly later for LED LE than for DL, whereas it was not delayed for OLED LE. More daylight exposure was related to earlier melatonin onset under OLED LE as well as DL, while there was no relation under LED LE.

OLED light has several benefits as a future lighting source.[25] First, the spectrum of an OLED contains a smaller proportion of blue light, which has the greatest influence on the melanopsin in intrinsically photosensitive retinal ganglion cells. Second, the spectral power distribution of OLEDs is closer to natural sunlight compared to other light sources, and hence produces a more-comfortable environment. Third, OLED light panels emit diffuse light from their surface, which helps to reduce fatigue in the human eye. It is presumed that OLEDs represent a human-friendly type of lighting that will disturb the circadian rhythm less and facilitate sleep.

In terms of melatonin onset, OLED LE showed a beneficial effect compared to LED LE in this study. Light with a high

Table 2. Sleep parameters and vigilance after LE in the three conditions

	DL	OLED	LED	*p*
Polysomnography				
Sleep latency, min	5.6±5.1	6.3±5.3	5.7±5.3	0.732
Sleep efficiency, %	91.9±6.2	91.3±8.9	93.2±6.1	0.283
WASO, %	6.6±5.5	7.1±8.2	5.4±5.1	0.287
Stage N1, %	10.8±5.2	10.3±6.5	10.1±6.1	0.763
Stage N2, %	50.3±8.9	50.4±7.3	52.0±6.8	0.385
Stage N3, %	18.4±9.5	17.6±8.2	16.4±7.5	0.139
REM sleep, %	20.5±5.8	21.7±5.4	21.5±6.9	0.608
Total AI, /h	14.8±5.7	14.2±8.4	14.0±6.7	0.721
Spontaneous AI, /h	9.3±5.0	8.6±5.9	8.5±5.5	0.560
Psychomotor vigilance test				
Number of lapses	5.8±4.1	4.1±3.5	2.8±2.3	0.004*
1/response time, /s	3.7±0.7	3.7±0.6	4.0±0.5	0.039*
Errors	1.4±1.9	1.6±1.5	2.0±3.2	0.579

Data are mean±standard-deviation values.
*$p<0.05$ in repeated-measures ANOVA.
AI: arousal index, DL: dim light, LE: light exposure, LED: light-emitting diode, OLED: organic LED, REM: rapid eye movement, WASO: waking after sleep onset.

color temperature suppresses melatonin secretion more, but the effects of different light sources with the same high color temperature on melatonin secretion remain unclear.[6,8] In the present study, the light intensity (150 lux), color temperature (4,000 K), and exposure time (18:00–24:00) were identical for the OLED and LED conditions. The present results are consistent with technical reports on the beneficial effects of OLEDs with smaller amounts of blue light on the circadian rhythm.[20-22]

Exposure to daylight during the phase-advance period (in the morning) may advance melatonin onset.[26] A prior LE history affects light sensitivity to the circadian rhythm.[27-29] The present study is not a 24 hours simulation study, and involved participants in a laboratory environment from early evening to the next morning (covering about 18 hours). Instead, all of the subjects were instructed to wear an actigraphy device for 1 day before entering the laboratory, and we demonstrated that the accumulated amount of daylight during the study did not differ significantly among the tested conditions. A particularly interesting finding was that exposure to sufficient daylight advanced the melatonin onset time even for OLED LE during the phase-delay period (in the evening and nighttime). This suggests that OLEDs represent a promising light source for minimizing the detrimental effects on nighttime melatonin secretion when combined with sufficient exposure to daylight.

We also found that sleep quality as measured by PSG and morning vigilance were not impaired under OLED LE or LED LE, which was contrary to our expectations. Very few studies have assessed the effects of LE in the evening before bedtime on PSG parameters and vigilance after sleep. Our group previously revealed an association between sleeping with lights on and increased the amount of N1-stage sleep and the arousal index and decreased N3-stage sleep.[30] These conditions clearly differ from the present study in that the subjects were exposed to light throughout the sleep. Chang et al.[31] showed that reading an e-book before bedtime delayed sleep latency, decreased rapid-eye-movement sleep, and subjective morning alertness compared to reading a printed book. However, in that study the participants remained in the laboratory for the entire 2-week study period, and so their daytime LE was completely controlled, which differed from the conditions in our field study.

We can speculate as to why the sleep and vigilance parameters in the present study did not differ among the different light sources. First, the participants were allowed to perform their usual activities of daily living before entering the unit. LE during the daytime and various daytime activities including exercise were performed as usual. Their homeostatic pressure might have increased to initiate and maintain normal sleeping despite LE in the evening. Sleep quality is influenced by the daytime physical conditions and emotional stress. Second, the amount of light from the ceiling-mounted light panels delivered to the eyes would not be consistent. The subjects spent most of the time in the unit reading books in the head-down position. Television and other electrical devices were prohibited in the unit. At the beginning of each session, we set the light intensity as 150 lux at the eye level when participants looked ahead to the front while seated. However, the amount of light delivered to eyes might vary with the angled

position of the head during the 6 hours of LE. Inconsistency in the amount of light entering the retina might have reduced the ability to identify the effects of artificial light on sleep quality and vigilance after sleep.

We admit that an experimental setting with the strict control of daylight, daytime activity, and a fixed head position toward light sources throughout the evening would be more appropriate for obtaining accurate results, especially for sleep and vigilance. However, the present experimental setting might better reflect real lighting environments during the evening and nighttime, despite this approach reducing the statistical power for identifying significant differences. In addition, evening LE under a specific condition was performed for only one night during each session in this study, with such short-term exposure possibly being insufficient for detecting alterations of the circadian rhythm and sleep in this real-life condition. Future researches involving repeated measurements or larger numbers of subjects should make further attempts to identify significant impacts of light sources during evening on sleep and vigilance.

Finally, a ceiling light panel producing an illuminance of 150 lux seems not be too bright to interfere with the circadian rhythm and sleep in young and healthy participants, even during the evening. Insomnia patients and the elderly might be more sensitive to the light level at nighttime. Moreover, females accounted for more than 70% of the subjects in this study, and sex differences in the sensitivity to light and melatonin onset time could have introduced bias into the results. Not obtaining information about the menstrual cycles of the female subjects was also a limitation of this study. Further researches should be designed that involve various populations in order to clarify the effects of different types of lighting on sleep according to age and comorbid sleep disorders.

In conclusion, we have demonstrated that evening and early-night OLED LE exerts weaker effects on the melatonin phase delay than does LED LE. Exposure to sufficient daylight may advance melatonin onset even when a subject is exposed to OLED light in the evening. These findings suggest that OLEDs are a suitable light source in the evening to minimize the detrimental effects of artificial light on the melatonin onset time.

Supplementary Material

The online-only Data Supplement is available with this article at https://doi.org/10.3988/jcn.2020.16.3.401.

Author Contributions

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Conflicts of Interest

The authors have no potential conflicts of interest to disclose.

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