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## Evening light exposure to computer screens disrupts human sleep, biological rhythms, and attention abilities

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### ABSTRACT

The use of electronic devices with light-emitting screens has increased exponentially in the last decade. As a result, humans are almost continuously exposed to unintentional artificial light. We explored the independent and combined effects of two aspects of screen illumination, light wavelength, and intensity, on sleep, its biological regulation, and related functional outcomes. The  $2 \times 2$  repeated-measure design included two independent variables: screen light intensity (low ([LI] versus high [HI])) and wavelength (short [SWL] versus long [LWL]). Nineteen participants (11F, 8M; mean age 24.3 [ $\pm 2.8$ ] years) underwent four light conditions, LI/SWL, HI/SWL, LI/LWL, and HI/LWL, in counterbalanced order. Each light exposure lasted for two hours (21:00–23:00), following which participants underwent an overnight polysomnography. On each experimental night, oral temperature and urine samples (for melatonin analysis) were collected at multiple time points. Each morning, participants filled out questionnaires and conducted a computerized attention task. Irrespective of light intensity, SWL illumination significantly disrupted sleep continuity and architecture and led to greater self-reported daytime sleepiness. SWL light also altered biological rhythms, subduing the normal nocturnal decline in body temperature and dampening nocturnal melatonin secretion. Light intensity seemed to independently affect sleep as well, but to a lesser degree. Both light intensity and wavelength negatively affected morning attention. In sum, light wavelength seems to have a greater influence than light intensity on sleep and a wide-range of biological and behavioral functions. Given the widespread use of electronic devices today, our findings suggest that screen light exposure at evening may have detrimental effects on human health and performance.

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Chronobiology; cognitive function; digital media; intensity; light; melatonin; sleep; thermoregulation; wavelength

## Introduction

In 2012, the American Medical Association (AMA) issued a resolution stating that light at night constitutes environmental pollution because it disrupts daily biological cycles, including the sleep/wake cycle (AMA, 2012), in part due to the suppression of nighttime MLT secretion (Hätonen et al., 1999; Lewy et al., 1980; Rüger et al., 2003; Zeitzer et al., 2005). Moreover, the extent of MLT suppression caused by bright light exposure has been shown to be dependent on the intensity and wavelength of the light (Blask, 2009; Skene et al., 1999), as well as on the duration of the light exposure (Chang et al., 2012). Studies that examined the effects of exposure to bright light at night have revealed that peripheral and core body

temperature at night do not drop as expected after bright light exposure (Bunnell et al., 1992; Kubota et al., 1998; Rüger et al., 2006). Furthermore, exposure to bright Artificial Light at Night (ALAN) has been shown to lead to longer sleep latencies (Bunnell et al., 1992; Komada et al., 2003; Lavoie et al., 2003) and reduced sleep quality (Cajochen et al., 1998; Kubota et al., 1998; Tzischinsky & Lavie, 1997).

Light is also known to have a therapeutic effect. Exposure to bright light has been shown to improve affective disorders and circadian rhythms sleep disorders (Even et al., 2008; Lam & Levitt, 1999). “Light Therapy” is also used successfully for chronobiological malalignment treatments, such as Delayed Sleep Phase Syndrome, Advanced Sleep Phase Syndrome, jet lag, and shift-work disorders (Dagan,

2002). Thus, different time of bright light exposure can delay or advance natural sleep/wake rhythms by altering natural MLT secretion patterns.

Whether detrimental or beneficial, the academic and clinical interest in ALAN exposure has been on the rise in the last three decades. In recent years, exposure to bright light has been increasing exponentially, in large parts of our world because of unintentional illumination from electronic screens that emit light directly into the eyes. Millions of computers, tablets, TVs, and smart-phones are bought worldwide every month and the usage time of these devices is increasing constantly. Most of these devices are equipped with Light Emitting Diode (LED) screens, exposing users to ongoing SWL light exposure. Previous studies have shown that light from electronic devices can alter MLT secretion (Chang et al., 2015; Figueiro et al., 2011; Wood et al., 2013), thermoregulation (Higuchi et al., 2003, 2005), sleep physiology and sleepiness measures (Chang et al., 2015; Grønli et al., 2016; Higuchi et al., 2005), cognitive performance (Cajochen et al., 2011), and mood (Sroykham & Wongsawat, 2013). However, none of these studies have attempted to isolate the unique and/or additive effects of the different light properties (wavelength and intensity) on biological, chronobiological, and behavioral measures. Moreover, no study has examined both the physiological and functional consequences of this light exposure.

This study paradigm will allow us to examine the above gaps in the literature. We hypothesize that high light intensity and SWL emerging from computer screens in the evening will equally and independently disrupt MLT secretion, temperature regulation, sleep quantity and quality, when compared to lower intensity and LWL illumination. We also expect that diverse combinations of these light properties will differently affect the above measures, with the most detrimental combination being SWL and high intensity light. We expect that evening light exposure will affect the behavioral indices similarly.

## Methods

### Participants

Participants were 19 healthy men and women with a mean age of  $24.3 \pm 2.8$  years (range: 20–29 years), with regular sleep patterns, measured using the Pittsburgh Sleep Quality Index (PSQI) score of <5 for inclusion

(Buysse et al., 1989; Shochat et al., 2007), and a normative sleep-wake cycle, as indicated by the Morningness–Eveningness Questionnaire (MEQ) (Horne & Ostberg, 1976; Lavie & Segal, 1989). Sleep quality and continuity were measured for one week using wrist actigraphy (Respironics Model II, Philips, Inc. USA). Only participants with 6–8 hours of nightly sleep, regular sleep/wake patterns, and no sleep/wake schedule problems proceeded to the experimental phase of the study stage. Participants with a BMI under 18 or above 25 were excluded, as well as those with any history of medical, neurological, psychiatric conditions, or sleep disorders (confirmed by polysomnography), or any regular medication intake (excluding contraceptives for female participants). Participants with ocular damage, such as to their field of vision, color blindness, or impaired functioning of the pupil in reaction to light, were excluded, however use of eyeglasses or contact lenses to correct vision was allowed. All participants signed informed consent prior to participation in the study. The study was approved by the Helsinki Committee of Assuta Medical Center and Maccabi Health Services.

### Measurements

Three physiological and three behavioral measures were collected during the course of the study.

### Physiological measures

**Polysomnography.** The sleep testing room was a standard test room at the Sleep Medicine Research Center at Assuta Medical Center. Standard in-lab polysomnography was conducted using the Somnoscreen-PSG type sleeping test instrument (Somnomedics, Germany). Sleep channels included: electroencephalography (EEG), electro-oculography (EOG), leg and chin electromyography (EMG), nasal flow, chest and diaphragm breathing, snoring, electrocardiography (EKG), heart rate, blood oxygen saturation, and body position. Sleep data processing was performed by skilled and trained sleep technicians in accordance with the Rechtschaffen and Kales criteria (Rechtschaffen & Kales, 1968). We calculated sleep continuity parameters, i.e. latency to stage 1 (SL1) and stage 2 (SL2), percent wake after sleep onset (%WASO), index of awakenings, total sleep time (TST), time in bed (TIB), and sleep efficiency



(SE) and sleep architecture parameters, i.e. percent stage 1 (%S1), stage 2 (%S2), REM (%REM), and SWS (%SWS), index of sleep stage changes, and REM onset latency (ROL).

**Melatonin.** Urine samples were collected for analyzing melatonin levels by measuring 6-sulfahydroxymelatonin (6-SMT) concentration, the major metabolite of the hormone in urine (De Almeida et al., 2011). The quantitative determination of 6-SMT in urine was completed by a solid phase enzyme-linked immunosorbent assay (ELISA # RE54031; IBL, Hamburg; Germany) as described previously (Zubidat et al., 2008). 6-SMT concentrations (ng/mL) were spectrophotometrically determined by ELISA microplate reader at 450 nm with reference wavelength 650 nm (PowerWave HT, Bioteck, Winooski; USA) and analyzed by Gen5TM Data Analysis Software (Version 2, Bioteck, Winooski, USA). All urine samples were frozen ( $-20^{\circ}\text{C}$ ) immediately after collection.

Urine samples were collected on all experimental days at three time points: 21:00, 23:00 and immediately following wake time. As the first morning sample concentration of 6-SMT has been extensively used as an estimate of overnight melatonin secretion (McMullan et al., 2013), we used this sample to represent the maximum MLT secretion (100%) per participant. The night samples (at 21:00 and 23:00) were transformed using the formula ( $[\text{value at } 21:00 \text{ or } 23:00 / \text{value at wake time}] * 100$ ) to reflect the percentage change in MLT secretion per participant, from pre-exposure (baseline) to post-exposure (23:00). Data from one participant is not included in the analyses due to a technical failure in melatonin ELISA analysis.

**Body temperature.**  $T_b$  was measured orally, using an electronic oral thermometer (Domotherem, UEBE Medical GMBH, Germany). Body temperature ( $T_b$ ) was taken at six time points, three on the testing night: 21:00 h, 23:00 h, and immediately prior to bedtime, and three the following morning, at 0, 60, and 120 minutes post-awakening.

### **Behavioral measures**

**The Epworth sleepiness scale.** The Epworth sleepiness scale (ESS) is a self-administered 8-item questionnaire to assess daytime sleepiness (Johns, 1991). Respondents were asked to rate, on a 4-point

scale, their usual chances of dozing off or falling asleep while engaged in eight different activities of varying levels of activity/passivity. The ESS score ranges from 0 to 24, with higher scores reflecting greater daytime sleepiness.

**The brief symptom inventory questionnaire.** The BSI is a brief psychological self-report symptom scale consists of 53 statements of problems and complaints. Respondents reported the extent to which each item has caused discomfort in the past month (Derogatis & Melisaratos, 1983). A General Severity Index (GSI) and nine subscale index scores (Somatization, Obsessive-compulsive, Interpersonal sensitivity, Depression, Anxiety, Hostility, Phobic anxiety, Paranoid ideation, Psychoticism) were calculated for each participant.

**Conner's continuous performance test-III.** The Conner's continuous performance test-iii (CPT-III) test is a computerized continuous performance task, examining inattentiveness, impulsivity, sustained attention and vigilance (Multi Health Systems, Inc., Toronto, Canada) (Conners, 2008). Individuals are seated in front of a computer screen and requested to press the spacebar as fast as they can when they see any letter on the screen, other than the letter X. Test duration is 14 minutes and includes 360 trials, used to compute T-scores that assess various aspects of the respondents' attention, including detectability ( $d'$ ), a measure how well the respondent discriminates non-targets (e.g. the letter X) from targets (e.g. all other letters), error rates (omissions, commissions, and perseverations), and reaction time statistics (Hit Reaction Time [HRT] and HRT Standard Deviation [HRT-SD]).

### **Procedure**

Recruitment ads were placed in social network websites stating basic inclusion criteria and study details. Interested persons were initially interviewed via phone to rule out major exclusion criteria (e.g. age, general health, and sleep patterns). Persons who were eligible and interested in participating were invited to the sleep laboratory at Assuta Medical Center (Tel Aviv, Israel) for in-lab screening. At the screening visit, all participants signed informed consent and filled out intake questionnaires, including demographic and

health questionnaires, the PSQI, and the MEQ. Participants then received an actigraph and sleep diaries for one week to assess the quality and quantity of their sleep and sleep-wake patterns and schedules. Following the home screening, participants were scheduled for four non-consecutive in-lab testing nights across two consecutive weeks (on Sunday and Wednesday nights of each week). Therefore, the wash-out period between consecutive light exposures was two to three days. For the duration of the 2-week experimental period, all participants were requested to sleep in accordance with their normal sleep schedule, both at home and in the laboratory.

A repeated measures design was used, with two independent variables: screen light intensity and light wavelength. Light intensity at two levels: low (LI) – 80 lux ( $35 \text{ mw/cm}^2$ ) and high (HI) – 350 lux ( $160 \text{ mw/cm}^2$ ) and a dominant light wavelength at two levels: short (SWL) – 460 nm and long (LWL) – 620 nm. Each participant underwent all four experimental light conditions, LI/SWL, HI/SWL, LI/LWL, and HI/LWL, in randomized and counterbalanced order. Light intensity and wavelength levels were measured and adjusted throughout the study using a light metering device (AvaSpec-2048-FT-SDU; Avantes, Inc., Eerbeek, The Netherlands). Our dependent variables included (1) three physiological measures: oral temperature, melatonin (6-SMT in urine), and sleep parameters (continuity and architecture) and (2) three behavioral measures: sleepiness (ESS), affect (BSI), and attention (CPT-II). All dependent variables were measures following each light manipulation.

Participants arrived at the sleep laboratory at 21:00 h on all experimental nights. The bedroom was about  $12 \text{ m}^2$  in size and included a desk with a 22-inch computer LED screen (Model 226V4L, Philips, USA) and a bed. The screen was placed at a distance of about 60 cm from the participant and at eye level. The room was dark and the room temperature was set to  $22^\circ\text{C}$ . Participants sat in front of the computer screen for two hours and performed onscreen tasks between 21:00 h and 23:00 h. Tasks were reading texts and answering related questions, writing exercises and solving verbal and arithmetic problems. Participants were not informed of the differing screen light conditions and were told that the purpose of the study was to examine the effect of the tasks on their sleep.

During exposure, the subjects were allowed to eat light food and drink non-caffeinated beverages; they could use the restroom before evening testing and were then asked to remain in their assigned bedroom for the duration of the testing period. Following the light exposure, participants were connected to the sleep testing system by a skilled technician and asked to go to bed. Bedtimes and wake times were based on the average sleep/wake time as indicated in the individual actigraphy reports. The sleep period (or TIB) was held constant per participant for the duration of all experimental nights. Approximately 30 minutes following awakening, participants filled out the ESS and the BSI questionnaires and performed the CPT task.

As detailed above, oral temperature was taken at six time points and urine samples were collected at three time points across the experimental night and morning. This protocol was repeated for each of the four testing nights. Upon completion of the study protocol participants were given monetary compensation for their participation in the study.

### **Statistical analysis**

Two-way (wavelength  $\times$  intensity) repeated measures (RM) ANOVAs were performed to evaluate mean value differences for all sleep parameters, the ESS and BSI questionnaires and for each dimension of CPT-III. Three-way (wavelength  $\times$  intensity  $\times$  time) RM ANOVAs were performed to evaluate mean value differences in melatonin and temperature indices. Post-hoc Tukey tests were performed for significant ANOVAs. Two-tailed  $p$ -values below 0.05 were considered significant. Statistical analyses were performed using SPSS, version 20 (SPSS Inc., Chicago, IL, USA).

## **Results**

### **Sleep continuity**

Exposure to SWL illumination significantly shortened TST, increased %WASO and the nocturnal awakening index, and decreased SE compared with LWL. Neither intensity, nor interaction between intensity and wavelength affected these sleep continuity measures. Both SWL and high intensity illumination independently prolonged SL1 and SL2, with no interaction found between wavelength and intensity. By virtue of the



experimental protocol, no significant main effects or interaction effects were found for TIB. Summary statistics are presented in Table 1.

### Sleep architecture

SWL significantly increased %S1 and %S2 sleep and increased the index of sleep state changes. Neither intensity nor interaction between intensity and wavelength affected the sleep architecture measures. Both SWL and high intensity illumination significantly decreased %SWS, with no interaction found between wavelength and intensity. No significant main effects or interaction effect were found for ROL or %REM. Summary statistics are presented in Table 1.

### Body temperature

As expected, we also discovered a main effect of time on  $T_b$  ( $F_{(5,90)} = 4.51, p < .01$ ), reflective of the natural circadian curve. A significant 2-way interaction of wavelength and time on  $T_b$  was also noted ( $F_{(5,90)} = 6.17, p < .001$ ). Follow-up analyses revealed  $T_b$  was significantly higher at 23:00 and prior to bedtime under SWL conditions, compared with LWL conditions, irrespective of light intensity (Figure 1). No other main or interaction effects were noted.

### Melatonin

A main effect of time was revealed ( $F_{(1,17)} = 11.52, p < .01$ ), representing expected circadian variations in melatonin secretion over time. A significant 2-way interaction of wavelength and time on melatonin secretion was also noted ( $F_{(1,17)} = 5.01, p < .05$ ), with follow-up analyses showing the suppression of melatonin levels being greater under exposure to SWL-conditions compared with LWL-conditions, irrespective of light intensity (Table 2). No other significant main or interaction effects were found.

### Subjective sleepiness

During exposure to SWL-conditions, participants reported significantly higher subjective sleepiness on the ESS compared to the LWL-conditions ( $F_{(1,18)} = 4.80, p < .05$ ). Light intensity did not affect sleepiness ( $F_{(1,18)} = 3.88, p = ns$ ) and no interaction between

wavelength and intensity was found ( $F_{(1,18)} = 0.52, p = ns$ ) (Figure 2).

### Mood

In the mornings following exposure, participants in the SWL ( $F_{(1,18)} = 3.71, p < .10$ ) and HI ( $F_{(1,18)} = 3.04, p < .10$ ) conditions exhibited greater negative emotion on the BSI General Severity Index (GSI), however these findings did not reach statistical significance (Figure 3). No interaction between wavelength and intensity was noted for the GSI ( $F_{(1,18)} = 0.74, p = ns$ ) and no main or interaction effects were found for any of the nine BSI subscale scores (range:  $F_{(1,18)} = 0.00-7.15, p = ns$ )

### Attention

A significant main effect was revealed for SWL when compared with LWL light-exposures for detectability ( $d'$ ) and error rate (omissions only). In addition, higher light intensity led to longer HIT reaction times, when compared with low intensity light. No other main or interaction effects were found (Table 3).

### Discussion

To the best of our knowledge, this is the first experimental design exploring the independent and combined effects of two main features of electronic screen illumination, wavelength and intensity, on sleep and physiological regulation of the sleep/wake cycle. Moreover, we explored possible functional outcomes of variable illumination conditions, including subjective sleepiness, mood, attention and concentration abilities. Our results show that although intensity of light negatively affects sleep and related physiological variables, light wavelength seems to have a greater influence on these physiological functions and their behavioral consequences.

Our results demonstrate that evening exposure to SWL-illumination from computer screens disrupts sleep continuity and quality. Specifically, this type of light exposure lengthened sleep latency, reduced sleep duration, increased the number of nocturnal awakenings and time awake at night, and decreased sleep efficiency. In regards to sleep architecture, the

**Table 1.** Summary statistics of the sleep parameters in the 4 experimental conditions: SWL-High, SWL-Low, LWL-High, and LWL-Low.

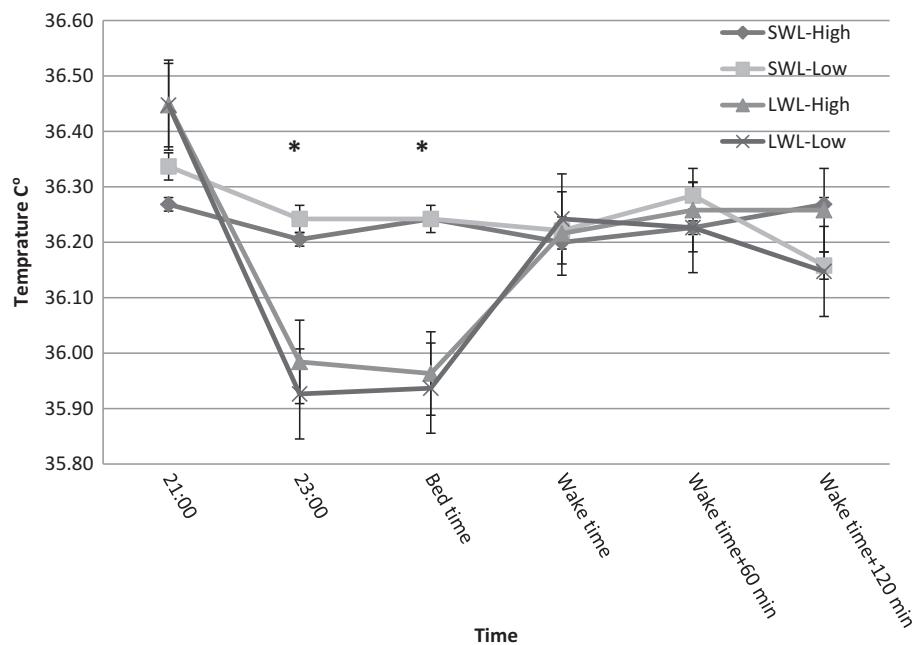
	SWL-High mean ( $\pm$ SD)	SWL-Low mean ( $\pm$ SD)	LWL-High mean ( $\pm$ SD)	LWL-Low mean ( $\pm$ SD)	$F(1,18) =$	P-value
TIB (min)	393.8 (23.7)	398.0 (24.4)	396.5 (26.0)	398.1 (23.3)	$W = 0.18$ $I = 1.31$ $W \times I = 0.03$	ns ns ns
TST (min)	362.0 (27.3)	371.1 (23.8)	378.4 (23.4)	379.7 (25.1)	$W = 21.45$ $I = 3.70$ $W \times I = 0.85$	p < .001 ns ns
SL1 (min)	11.9 (6.1)	8.9 (6.3)	6.2 (4.8)	4.5 (2.3)	$W = 17.9$ $I = 5.17$ $W \times I = 1.25$	p < .001 p < .05 ns
SL2 (min)	15.6 (7.0)	12.8 (8.3)	9.2 (5.4)	7.0 (4.8)	$W = 14.03$ $I = 5.71$ $W \times I = 0.18$	p < .001 p < .05 ns
ROL (min)	81.4 (24.6)	80.8 (21.7)	80.2 (23.5)	84.4 (20.9)	$W = 0.09$ $I = 0.12$ $W \times I = 0.24$	ns ns ns
%WASO	7.6 (2.3)	6.7 (3.0)	4.5 (1.9)	4.6 (2.3)	$W = 24.4$ $I = 0.25$ $W \times I = 0.67$	p < .0001 ns ns
SE (%)	92.4 (2.3)	93.3 (3.0)	95.5 (1.9)	95.4 (2.3)	$W = 24.5$ $I = 0.30$ $W \times I = 0.83$	p < .001 ns ns
Awakening index	3.1 (1.1)	3.0 (1.2)	2.3 (0.8)	2.4 (1.1)	$W = 18.3$ $I = 0.00$ $W \times I = 0.08$	p < .001 ns ns
%S1	3.3 (1.9)	3.7 (2.2)	2.5 (1.4)	2.9 (2)	$W = 5.18$ $I = 2.14$ $W \times I = 0.00$	p < .05 ns ns
%S2	49.9 (4.9)	48.3 (6.9)	46.1 (6.7)	44.0 (6.2)	$W = 5.57$ $I = 3.61$ $W \times I = 0.04$	p < .05 ns ns
%REM	18.3 (4.8)	18.1 (4.2)	18.1 (3.4)	18.8 (3.4)	$W = 0.05$ $I = 0.13$ $W \times I = 0.24$	ns ns ns
%SWS	21.0 (4.2)	23.2 (5.9)	28.7 (5.7)	29.7 (5.5)	$W = 42.55$ $I = 4.54$ $W \times I = 0.55$	p < .001 p < .05 ns
Sleep stage change Index	10.1 (2.7)	10.3 (3.2)	9.2 (2.2)	9.1 (2.7)	$W = 5.15$ $I = 0.02$ $W \times I = 0.11$	p < .05 ns ns

Wavelength ( $W$ ), intensity ( $I$ ), interaction of wavelength by intensity ( $W \times I$ ), time in bed (TIB), total sleep time (TST), latency to stage 1 (SL1) and stage 2 (SL2), REM onset latency (ROL), percent wake after sleep onset (%WASO), sleep efficiency (SE), and percent stage 1 (%S1), stage 2 (%S2), REM (%REM), and SWS (%SWS).

most pronounced effects were seen in NREM sleep, with exposure to SWL screen-light increasing lighter sleep stages at the cost of deep sleep (reducing SWS). Two recent studies compared the effects of eReaders (iPads with SWL illumination) versus printed books; they also revealed a negative effect of SWL-illumination, emerging from e-screens on sleep (Chang et al., 2015; Grønli et al., 2016). The results of our study found greater effects on sleep quality and quantity parameters, which in our opinion may be a function of either the timing or the length of the exposure with our light manipulation lasting two hours and occurring later in the evening (21:00–23:00), when compared with the above studies in which subjects

experienced either earlier or shorter exposures. Another issue that may explain these differences may be the screen size, which in our study was 22 inches while iPads are approximately 10 inches in size. Additional research is needed to examine whether different light features like screen size or conditions like timing of evening exposure may differently affect sleep.

While light wavelength seemed to have significant and wide-ranging effects on sleep, the intensity of the light seemed to affect sleep as well, but to a lesser degree. High intensity illumination prolonged sleep latency and significantly decreased SWS but did not affect any other sleep continuity or structure



**Figure 1.** Body temperature ( $T_b$ ) across the 4 experimental conditions.

Mean ( $\pm$ SEM) body temperature ( $T_b$ ) ( $^{\circ}$ C) across the 4 experimental conditions: SWL-High, SWL-Low, LWL-High, LWL-Low at 6 points across the testing night: 21:00, 23:00, Bedtime, Wake time, Wake time +60 min, and Wake time +120 min. (\* $p < .001$ ).

**Table 2.** 6-SMT melatonin metabolite raw data (pg/ml) and normalized (%).

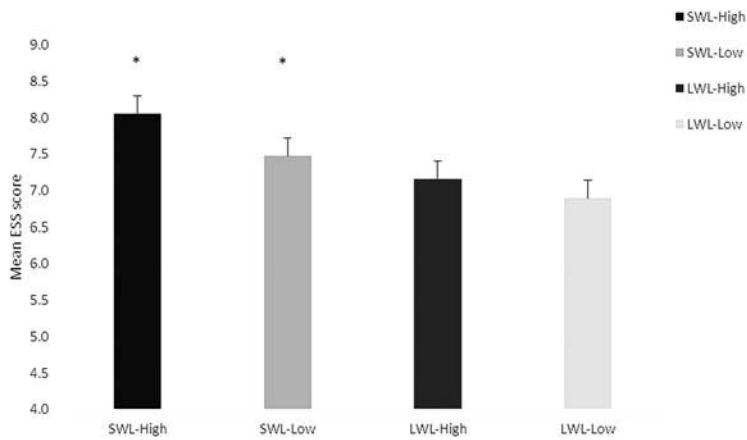
	21:00		23:00		$\Delta$ (%) (23:00–21:00)	Wake time	
	Raw (pg/ml)	norm (%)	Raw (pg/ml)	norm (%)		Raw (pg/ml)	norm (%)
	Mean ( $\pm$ SD)	Mean ( $\pm$ SD)	Mean ( $\pm$ SD)				
SWL-high	58.4 (50.2)	25.0 (19.5)	67.9 (23.5)	29.1 (22.2)	4.1	273.3 (137.3)	100.0
SWL-low	35.8 (23.5)	12.7 (8.6)	64.5 (49.5)	24.8 (22.3)	12.1	283.2 (107.6)	100.0
LWL-high	47.8 (48.4)	21.6 (23.4)	97.0 (76.6)	40.8 (33.9)	19.2	262.2 (139.6)	100.0
LWL-low	62.0 (100.3)	20.3 (27.3)	110.6 (88.6)	44.8 (35.6)	24.5	260.5 (119.4)	100.0

Means (SD) for raw data (pg/ml) and normalized (%) 6-SMT melatonin metabolite at 21:00, 23:00 and Wake time across the 4 experimental conditions: SWL-High, SWL-Low, LWL-High, and LWL-Low.  $\Delta$  (%) represents the change in percentage between 21:00 and 23:00. ( $p < .05$ ).

parameters. Our results are consistent with previous reports on the effects of screen illumination on sleep which revealed prolonged sleep latency (Chang et al., 2015; Higuchi et al., 2005). However, in contrast to our findings several recent studies did not find effects of screen illumination on other measures of sleep continuity or architecture (Grønli et al., 2016; Heath et al., 2014; Rangtell et al., 2016). A possible explanation for these differences may be the magnitude of the light intensity emitted from the screen. In the above-mentioned studies electronic screens with light intensity levels of 60–80 lux, which is comparable to our low intensity condition, in contrast to our high intensity condition which was about fourfold that level, approximately 350 lux. Thus, the degree of light intensity may differentially affect sleep ability and quality;

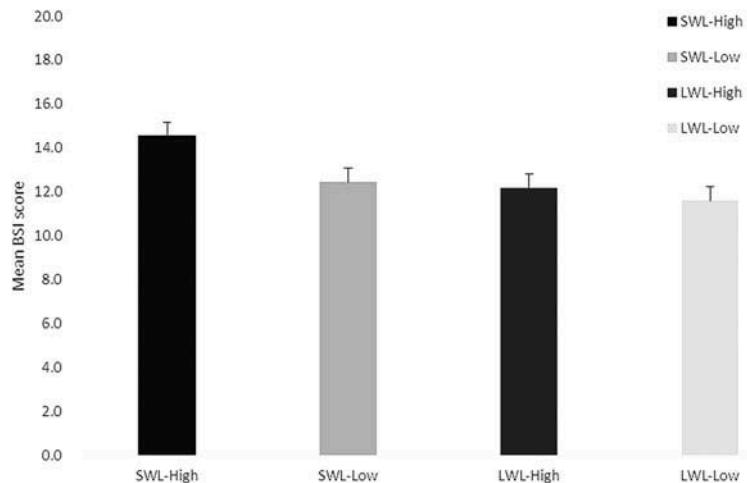
however future studies are needed to explore the exact nature of this relationship.

To the best of our knowledge, this is the first study showing that SWL light emitted from computer screens prior to bedtime can alter thermoregulation and secretion of melatonin, two central markers of human chronobiology. Consistent with our study hypothesis, we found that exposure to SWL-illumination from computer screens for two hours in the evening disrupts the normal nocturnal drop in peripheral body temperature, while exposure to LWL illumination had no effect on the circadian temperature curve. Although we did not find an effect of light intensity on the  $T_b$ -curve, studies have shown that higher light intensities may lead to higher nocturnal  $T_b$  values when compared with lower intensities,



**Figure 2.** Morning Epworth Sleepiness Scale (ESS) Scores across the four experimental conditions .

Means ( $\pm$ SEM) for morning Epworth Sleepiness Scale (ESS) Scores across the 4 experimental conditions: SWL-High, SWL-Low, LWL-High, and LWL-Low. \* $p < .05$ .



**Figure 3.** BSI General Index score across the 4 experimental conditions.

Means ( $\pm$ SEM) of BSI General Index score across the four experimental conditions: SWL-High, SWL-Low, LWL-High, and LWL-Low.

however results remain inconsistent (Higuchi et al., 2003, 2005). Moreover, our study paradigm allowed us to isolate the effects of light intensity and wavelength on melatonin secretion. Consistent with previous studies (Cajochen et al., 2011; Wood et al., 2013), we observed that evening exposure to SWL, but not LWL light, via computer screens, suppressed melatonin secretion. Contrary to our hypothesis however, light intensity alone did not affect melatonin secretion.

As the vast majority of electronic device screens (e.g. computers, tablets and smart-phones) today use LED technology, emitting primarily SWL-light, the detrimental effects of this unintentional “light

pollution” on our natural bodily functions may have health and functional consequences that need to be further explored. Moreover, these findings may have applicability to sleep hygiene recommendations, as only reducing brightness of electronic screens does not seem to protect from the harmful effects of the SWL-lighting emitted from these screens. In other words, when we instruct people to reduce the brightness of their electronic screens at night, it may not be sufficient to eliminate the negative effects of these light sources on sleep and thermoregulation. These findings may be further supported by the known presence of retinal photo-receptors particularly sensitive to wavelength input (Freedman et al.,

**Table 3.** Summary statistics for the CPT-III dimensions.

	SWL-High mean ( $\pm$ SD)	SWL-Low mean ( $\pm$ SD)	LWL-High mean ( $\pm$ SD)	LWL-Low mean ( $\pm$ SD)	$F(1,18) =$	<i>P</i> -value
d'	53.80 (12.05)	52.95 (12.35)	46.84 (9.29)	47.84 (8.17)	$W = 5.34,$ $I = 0.10$ $W \times I = 0.95$	$p < .05$ <i>ns</i> <i>ns</i>
Omissions	57.74 (18.14)	54.95 (17.23)	47.11 (6.14)	47.16 (4.91)	$W = 6.02$ $I = 1.13$ $W \times I = 0.71$	$p < .05$ <i>ns</i> <i>ns</i>
Commissions	49.26 (9.51)	49.47 (9.17)	49.26 (8.73)	49.05 (9.12)	$W = 0.05$ $I = 0.00$ $W \times I = 0.07$	<i>ns</i> <i>ns</i> <i>ns</i>
Perseverations	48.538 (5.79)	48.47 (4.13)	49.53 (5.13)	49.26 (8.35)	$W = 1.25$ $I = 0.40$ $W \times I = 0.01$	<i>ns</i> <i>ns</i> <i>ns</i>
HRT	48.37 (5.87)	47.42 (6.64)	48.42 (5.77)	46.95 (5.46)	$W = 0.10$ $I = 4.30$ $W \times I = 0.24$	<i>ns</i> $p < .05$ <i>ns</i>
HRT-SD	40.47 (7.37)	39.89 (8.02)	40.58 (7.22)	38.21 (6.79)	$W = 0.82$ $I = 2.97$ $W \times I = 1.53$	<i>ns</i> <i>ns</i> <i>ns</i>

Mean T-scores (SD) and summary statistics for the CPT-III dimensions, d' (detectability), omissions, commission, perseverations, HRT (Hit Reaction Time) and HRT-SD (HRT Standard Deviation) across the four experimental conditions: SWL-High, SWL-Low, LWL-High, and LWL-Low.

1999; Lucas et al., 1999), possibly affecting circadian clocks located in the SCN and thus regulation of chronobiological functions.

Specific features of light seemed to differently affect behavioral measures. Attention, a central function of our cognitive abilities, was found impaired in the morning after the light exposure. Specifically, SWL-exposure seemed to affect accuracy of response, i.e. reduced ability to discriminate targets from non-targets and high omission rates, while light intensity slowed reaction times but did not affect performance accuracy. Moreover, SWL-illumination, but not light intensity, led to greater subjective sleepiness, the morning after exposure. An interesting study by Cajochen and colleagues (Cajochen et al., 2011) examined the acute behavioral response of screen exposure and showed that use of LED screens (SWL-illumination) led to increased evening alertness and decreased sleepiness immediately following exposure, when compared with non-LED screens (LWL-illumination). However, our results may indicate that these immediate after-effects of LED screen exposure may have subsequent “hang-over effects” the morning after, i.e. reduced attention ability and increased sleepiness. In recent years, use of electronic devices with LED screens has become pervasive and constant, with pronounced increases in use among children and adolescents (Gradisar et al., 2013). These findings

may be particularly worrisome in youth with still evolving attention and learning capacities.

In our study, mood was not significantly affected by light intensity or wavelength, although there was tendency for greater reports of negative mood in the morning after exposure to both the SWL and high intensity light conditions. Although we did not find effects of light on day-after morning affect, it is possible that our mood measure was not sensitive enough to detect milder mood changes in healthy participants.

In contrast to our hypothesis, we did not find any interaction between wavelength and intensity on any of our main outcome measures, physiological or behavioral. We found this result to be quite unexpected; as these light characteristics are emitted from the same light source, we would have expected additive effects of wavelength and intensity on some if not all our main outcomes. A possible hypothesis for this finding may be the existence of distinct systems or separate neural pathways responsible for processing of wavelength versus intensity of light input. This idea may be supported by findings that receptors sending information from the eye to the SCN are specific to different light features, such as wavelength or intensity, (Brainard et al., 2001; Hatter et al., 2002) and thus their processing and effects may be independent as well. Further studies are needed to explore this and other hypotheses.

There are several limitations to this study: First, we used a 22-inch computer LED screen. Future studies

should assess different types and sizes of electronic LED screens (e.g. tablets, smartphones) in order to examine the generalizability of our results to other devices. In addition, we examined only one night of each light exposure condition. As the exposure to LED screens is ongoing and often occurs on a nightly basis, future studies should explore the chronic effect of this “light pollution” from electronic devices. Additionally, we do not collect data on the amount or type of daytime light participants were exposed to prior to the experimental nights. Previous findings have shown differential daytime light exposure may affect nighttime light sensitivity in some individuals (Hébert et al., 2002). Future studies should measure 24-hour light exposure to examine possible effects on sleep and circadian measures. Lastly, our sample included only 19 participants; larger replication studies are needed to confirm our findings.

In sum, the results of our study suggests the possible existence of a “chain reaction” of physiological changes emerging from exposure to ALAN, i.e. reduced melatonin profiles coupled with subdued  $T_b$  rhythms, and reduced quality and quantity of sleep. These changes may directly or indirectly lead to next morning behavioral and functional deficits, such as greater sleepiness and inattention. As larger and larger segments of the population are exposed to “light pollution” emitted from these devices, effects on our health, cognition, and daily function may be significant and pervasive.

## Declaration of interest

The authors report no conflicts of interest. The authors alone are responsible for the content and writing of this article.

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## References

- American Medical Association. (2012). AMA Adopts New Public Health Policies at Annual Meeting. Available from: <http://www.cisionwire.com/american-medical-association/r/ama-adopts-new-public-health-policies-at-annual-meeting,c927593>.
- Blask DE. (2009). Melatonin, sleep disturbance and cancer risk. *Sleep Med Rev*. 13:257–64.
- Brainard GC, Hanifin JP, Greeson JM, et al. (2001). Action spectrum for melatonin regulation in humans: Evidence for a novel circadian photoreceptor. *J Neurosci*. 21:6405–12.
- Bunnell D, Treiber S, Phillips N, Berger R. (1992). Effects of evening bright light exposure on melatonin, body temperature and sleep. *J Sleep Res*. 1:17–23.
- Buysse DJ, Reynolds CF, Monk TH, et al. (1989). The pittsburgh sleep quality index: A new instrument for psychiatric practice and research. *Psych Res*. 28:193–213.
- Cajochen C, Frey S, Anders D, et al. (2011). Evening exposure to a light-emitting diodes (LED)-backlit computer screen affects circadian physiology and cognitive performance. *J Appl Physiol*. 110:1432–38.
- Cajochen C, Kräuchi K, Danilenko KV, Wirz-Justice A. (1998). Evening administration of melatonin and bright light: Interactions on the EEG during sleep and wakefulness. *J Sleep Res*. 7:145–57.
- Chang A-M, Aeschbach D, Duffy JF, Czeisler CA. (2015). Evening use of light-emitting eReaders negatively affects sleep, circadian timing, and next-morning alertness. *Proc Natl Acad Sci*. 112:1232–37.
- Chang AM, Santhi N, St Hilaire M, et al. (2012). Human responses to bright light of different durations. *J Physiol*. 590:3103–12.
- Connors CK. (2008). Connors 3rd Edition manual. Toronto, Canada: Multi-Health Systems.
- Dagan Y. (2002). Circadian rhythm sleep disorders (CRSD). *Sleep Med Rev*. 6:45–55.
- De Almeida EA, Di Mascio P, Harumi T, et al. (2011). Measurement of melatonin in body fluids: Standards, protocols and procedures. *Child's Nerv Syst*. 27:879–91.
- Derogatis LR, Melisaratos N. (1983). The brief symptom inventory: An introductory report. *Psychol Med*. 13:596–605.
- Even C, Schroder CM, Friedman S, Rouillon F. (2008). Efficacy of light therapy in nonseasonal depression: A systematic review. *J Affective Disord*. 108:11–23.
- Figueiro MG, Wood B, Plitnick B, Rea MS. (2011). The impact of light from computer monitors on melatonin levels in college students. *Neuroendocrinol Lett*. 32:158–63.
- Freedman MS, Lucas RJ, Soni B, et al. (1999). Regulation of mammalian circadian behavior by non-rod, non-cone, ocular photoreceptors. *Science* (80). 284:502–04.
- Gradisar M, Wolfson AR, Harvey AG. (2013). The sleep and technology use of Americans: Findings from the National Sleep Foundation's 2011 Sleep in America poll. *J Clin Sleep Med*. 9:1291–99.
- Grønli J, Byrkjedal IK, Bjørvatn B, et al. (2016). Reading from an iPad or from a book in bed: The impact on human sleep. A randomized controlled crossover trial. *Sleep Med*. 21:86–92.
- Hätönen T, Alila-Johansson A, Mustanoja S, Laakso ML. (1999). Suppression of melatonin by 2000-lux light in humans with closed eyelids. *Biol Psych*. 46:827–31.
- Hatter S, Liao HW, Takao M, et al. (2002). Melanopsin-containing retinal ganglion cells: Architecture, projections, and intrinsic photosensitivity. *Science*. 295:1065–70.

- Hébert M, Martin SK, Lee C, Eastman CI. (2002). The effects of prior light history on the suppression of melatonin by light in humans. *J Pineal Res.* 33:198–203.
- Heath M, Sutherland C, Bartel K, et al. (2014). Does one hour of bright or short-wavelength filtered tablet screen light have a meaningful effect on adolescents' pre-bedtime alertness, sleep, and daytime functioning? *Chronobiol Int.* 31:496–505.
- Higuchi S, Motohashi Y, Liu Y, et al. (2003). Effects of VDT tasks with a bright display at night on melatonin, core temperature, heart rate, and sleepiness. *J Appl Physiol.* 94:1773–76.
- Higuchi S, Motohashi Y, Liu Y, Maeda A. (2005). Effects of playing a computer game using a bright display on pre-sleep physiological variables, sleep latency, slow wave sleep and REM sleep. *J Sleep Res.* 14:267–73.
- Horne JA, Ostberg O. (1976). A self-assessment questionnaire to determine morningness-eveningness in human circadian rhythms. *Int J Chronobiol.* 4:97–110.
- Johns M. (1991). A new method for measuring daytime sleepiness: The epworth sleepiness scale. *Sleep.* 14:540–45.
- Komada Y, Tanaka H, Yamamoto Y, et al. (2003). Effects of bright light pre-exposure on sleep onset process. *Psychiatry Clin Neurosci.* 54:365–66.
- Kubota T, Uchiyama M, Hirokawa G, et al. (1998). Effects of evening light on body temperature. *Psychiatry Clin Neurosci.* 52:248–49.
- Lam RW, Levitt AJ. (1999). Canadian consensus guidelines for the treatment of seasonal affective disorder. Clinical & Academic Publishing. Vancouver, Canada.
- Lavie P, Segal S. (1989). Twenty-four-hour structure of sleepiness in morning and evening persons investigated by ultra-short sleep-wake cycle. *Sleep.* 12:522–28.
- Lavoie S, Paquet J, Selmaoui B, et al. (2003). Vigilance levels during and after bright light exposure in the first half of the night. *Chronobiol Int.* 20:1019–38.
- Lewy AJ, Wehr TA, Goodwin FK, et al. (1980). Light suppresses melatonin secretion in humans. *Science.* 210:1267–69.
- Lucas RJ, Freedman MS, Muntz M, et al. (1999). Regulation of the mammalian pineal by non-rod, non-cone, ocular photoreceptors. *Science.* 284:505–07.
- McMullan CJ, Schernhammer ES, Rimm EB, et al. (2013). Melatonin secretion and the incidence of type 2 diabetes. *Jama.* 309:1388–96.
- Rangtell FH, Ekstrand E, Rapp L. (2016). Two hours of evening reading on self-luminous tablet vs. reading physical book does not alter sleep after day time bright light exposure. *Sleep Med.* 23:111–18.
- Rechtschaffen A, Kales A. (1968). A manual of standardized techniques and scoring system for sleep stages of human subjects. Washington, DC: US Gov Print Off; NIH Public.
- Rüger M, Gordijn MC, Beersma DG, et al. (2003). Acute and phase-shifting effects of ocular and extraocular light in human circadian physiology. *J Biol Rhythms.* 18:409–19.
- Rüger M, Gordijn MCM, Beersma DGM, et al. (2006). Time-of-day-dependent effects of bright light exposure on human psychophysiology: Comparison of daytime and nighttime exposure. *Am J Physiol Regul Integr Comp Physiol.* 290:R1413–R1420.
- Shochat T, Tzischinsky O, Oksenberg A, Peled R. (2007). Validation of the pittsburgh sleep quality index Hebrew translation (PSQI-H) in a sleep clinic sample. *Isr Med Assoc J.* 9:853–56.
- Skene DJ, Lockley SW, Thapan K, Arendt J. (1999). Effects of light on human circadian rhythms. *Reprod Nutr Dev.* 39:295–304.
- Sroykham W, Wongsawat Y. (2013). Effects of LED-backlit computer screen and emotional self-regulation on human melatonin production. *Proc Annu Int Conf IEEE Eng Med Biol Soc EMBS:*1704–1707.
- Tzischinsky O, Lavie P. (1997). The effects of evening bright light on next-day sleep propensity. *J Biol Rhythms.* 12:259–65.
- Wood B, Rea MS, Plitnick B, Figueiro MG. (2013). Light level and duration of exposure determine the impact of self-luminous tablets on melatonin suppression. *Appl Ergon.* 44:237–40.
- Zeitzer JM, Khalsa SB, Boivin DB, et al. (2005). Temporal dynamics of late night photic stimulation of the human circadian timing system. *Am J Physiol Regul Integr Comp Physiol.* 289:R839–844.
- Zubidat AE, Nelson RJ, Haim A. (2008). Urinary adrenalin and cortisol secretion patterns of social voles in response to adrenergic blockade under basal conditions. *Physiol Behav.* 93:243–49.