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Acute exposure to evening blue-enriched light impacts on human sleep

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SUMMARY

Light in the short wavelength range (blue light: 446–483 nm) elicits direct effects on human melatonin secretion, alertness and cognitive performance via non-image-forming photoreceptors. However, the impact of blue-enriched polychromatic light on human sleep architecture and sleep electroencephalographic activity remains fairly unknown. In this study we investigated sleep structure and sleep electroencephalographic characteristics of 30 healthy young participants (16 men, 14 women; age range 20–31 years) following 2 h of evening light exposure to polychromatic light at 6500 K, 2500 K and 3000 K. Sleep structure across the first three non-rapid eye movement non-rapid eye movement – rapid eye movement sleep cycles did not differ significantly with respect to the light conditions. All-night non-rapid eye movement sleep electroencephalographic power density indicated that exposure to light at 6500 K resulted in a tendency for less frontal non-rapid eye movement electroencephalographic power density, compared to light at 2500 K and 3000 K. The dynamics of non-rapid eye movement electroencephalographic slow wave activity (2.0–4.0 Hz), a functional index of homeostatic sleep pressure, were such that slow wave activity was reduced significantly during the first sleep cycle after light at 6500 K compared to light at 2500 K and 3000 K, particularly in the frontal derivation. Our data suggest that exposure to blue-enriched polychromatic light at relatively low room light levels impacts upon homeostatic sleep regulation, as indexed by reduction in frontal slow wave activity during the first non-rapid eye movement episode.

INTRODUCTION

Light impacts directly upon human physiology and behaviour, such as clock gene expression (Cajochen *et al.*, 2006), hormonal secretion (Brainard *et al.*, 2001; Cajochen *et al.*, 2005), brain activity (Lockley *et al.*, 2006; Vandewalle *et al.*, 2007a) and cognition (Chellappa *et al.*, 2011a; Vandewalle *et al.*, 2007b), through its alerting effects and/or its resetting properties on the endogenous circadian pacemaker (Chellappa *et al.*, 2011b). These effects are acute (seconds for brain responses) or extend beyond the light exposure (hours for hormonal secretion), and display maximal sensitivity to light at the short wavelength range (446–483 nm) (Berson *et al.*, 2002). Thus, these effects may be mediated not only by classical rod and cone photopigments (Güler *et al.*, 2008), but are also more likely to reflect melanopsin-based photoreception (Tsai *et al.*, 2009). While the effects of light on the circadian rhythms of melatonin (Khalsa *et al.*, 2003) and core body temperature (Kubota *et al.*, 2002; Rüger *et al.*, 2006) are fairly well established, surprisingly little is known about how light affects sleep architecture and electroencephalographic (EEG) power density during sleep.

Morning bright light shortens sleep duration, without effects on non-rapid eye movement (NREM) sleep homeostasis (Dijk *et al.*, 1989) or on REM sleep parameters (Carrier and Dumont, 1995). Evening light decreases sleep propensity (Carrier and Dumont, 1995) and increases NREM sleep Stage 2 latency (Cajochen *et al.*, 1992; Carrier and Dumont,

1995). Recently, it was found that evening polychromatic light (both blue-enriched and white polychromatic light) resulted in a longer REM sleep latency in older participants after three evenings of light exposure compared to evenings without light exposure. Furthermore, in the same study, longer sleep latency and shorter phase angle of entrainment (interval between the circadian dim light melatonin onset and habitual bedtime) were reported after evening light exposure (Münch *et al.*, 2011). In other words, polychromatic light impacted directly upon the phase relationship between the circadian timing system and the timing of sleep.

So far, the wavelength dependency of light on sleep has been described in only one study, in which monochromatic blue light (460 nm), compared to green light (550 nm), altered the dynamics of NREM sleep EEG activity, such that slow wave activity (SWA: 0.75–4.5 Hz) was reduced in the first sleep cycle with a rebound in the third sleep cycle (Münch *et al.*, 2006). However, it remains unknown if blue-enriched polychromatic light impacts on human sleep structure and EEG activity. Here, three different polychromatic light settings were used in the evening prior to sleep: blue-enriched light condition (light at 6500 K), non-blue-enriched light (light at 2500 K) and incandescent light (3000 K; for a detailed description of the light settings, see Methods). Our main hypothesis was that exposure to evening blue-enriched light, compared to light at 2500 K and 3000 K (administered by commercially available compact fluorescent lamps), will result in an attenuation of NREM SWA, particularly at the beginning of the night, when sleep pressure is at its maximum. The use of two different non-blue-enriched light sources (2500 K and 3000 K) was due to the naturalistic design of this study, whereby light at 2500 K contains a lower irradiance at the short wavelength compared to light at 6500 K, while light at 3000 K is a broadband white light source. We hypothesized that the effects of both light sources on sleep would not differ, as their spectral composition is fairly similar (for more information of the light settings, see Chellappa *et al.*, 2011a).

MATERIAL AND METHODS

Study participants

Thirty volunteers [16 men, 14 women; age range 20–31 years; mean ± standard deviation (SD): 25.2 ± 3.1, years] were included into the study. All participants were non-smokers, were not extreme chronotypes and were free from medical, psychiatric and sleep disorders, as assessed by medical examination and questionnaires. An ophthalmologic examination was carried out prior to the study to exclude volunteers with visual impairments. For a detailed description of the screening criteria, see Chellappa *et al.* (2011a). One week prior to the study, participants were requested to abstain from excessive alcohol and caffeine consumption (i.e. at most, five alcoholic beverages per week and one cup of coffee or one caffeine-containing beverage per day). Furthermore, they were instructed to keep a regular sleep–wake schedule (bedtimes and wake times within ± 30 min of self-selected target time). Although short (<6.5 h) and long (>8.5 h) sleep duration were not an exclusion criterion, all participants had an average sleep duration of 7–8 h of sleep. Compliance was verified by wrist actigraphy (Actiwatch L; Cambridge Neurotechnologies, Cambridge, UK) and by self-reported sleep logs. All participants gave written informed consent. The study was approved by the local ethics committee (Ethikkommissionbeider Basel, Switzerland) and conformed to the Declaration of Helsinki.

Design and procedure

A balanced cross-over design study was carried out during the winter season (January–March) to minimize the effects of outdoor light levels, and included three segments separated by a 1-week intervening period. Considering the volunteers' habitual bedtimes, the protocol started 10 h after usual wake-up time in the early evening (i.e. 18:00 hours) and ended the next day, after usual wake-up time (i.e. 08:00 hours). Participants spent 1.5 h under dim light conditions (<8 lux), 2 h under complete darkness, 2 h of light exposure (either by a compact fluorescent lamp with 6500 K or 2500 K or by an incandescent light bulb at 3000 K), and a post-light period of approximately 45 min under dim light until habitual sleep time. Each protocol was conducted at the same time of day (evening), and same time-length of the protocol (*c.* 6 h in total) for all participants. Illumination levels were set to 40 lux on the white wall at the central point in the field of view. All photometric measurements of illumination levels, light colours and spectra were taken at 120 cm height, with the measurement plane vertically at 125 cm distance from the wall, in correspondence with eye location and with orientation in the sitting position. The light measure at that location was typically between 25 and 32 lux. The intensity and spectral composition of the light conditions are illustrated in Table 1.The treatment order (6500 versus 2500 K versus 3000 K) was counterbalanced to avoid possible order effects of the light conditions. The detailed study protocol and the light settings are described elsewhere (Chellappa *et al.*, 2011a).

Data acquisition

Polysomnographic recordings (Vitaport-3 digital recorder; TEMEC Instruments BV, Kerkrade, the Netherlands) comprised eight EEGs, two electro-oculograms, one submental electromyogram and one electrocardiogram. All signals were low-pass-filtered at 30 Hz (fourth-order Bessel type anti-aliasing, total 24 dB Oct^{-1}) at a time constant of 1.0 s. After online digitization by using a range 610 microV, 12-bit AD converter (0.15 microV bit^{-1}) and a sampling rate at 128 Hz for the EEG, the raw signals were stored on a Flash RAM card (Viking, Rancho Santa Margarita, CA, USA) and

Table 1 Intensity and spectral composition of the polychromatic light settings

Light condition	*Lamp type*	*Correlated colour temperature CCT (K)*	*Illuminance on eye level (lx)**	*Photon density (380–780 nm) (photons m^{-2} s^{-1})***	*V (λ)-weighted photon density (photons m^{-2} s^{-1})***	*Melanopic-weighted photon density (photons m^{-2} s^{-1})***	*Irradiance (380–780 nm) ($mW\ m^{-2}$)*	*V (λ)-weighted irradiance ($mW\ m^{-2}$)*	*Melanopic-weighted irradiance ($mW\ m^{-2}$)*
CFL blue-reduced	Duluxstar Mini Twist 13 W/825 (warm comfort white, 2500 K)	2481	27	2.27E + 16	1.14E + 16	1.79E + 16	76.9	39.5	7.4
Incandescent reference	Incandescent lamp (classic A 75 W 3000 K)	2581	29.8	6.76E + 16	1.26E + 16	3.32E + 16	203.2	43.6	13.6
CFL daylight blue-enriched	Duluxstar Mini Twist 13 W/825 (cool daylight 6500 K)	5984	27.6	2.52E + 16	1.13E + 16	6.96E + 16	91.7	40.4	29.5

*Measurement of light reflected from illuminated wall that corresponds to horizontal gaze towards the wall (120cm).
**Spectral data corresponds to the visible range from 380-780 nm.

downloaded later to a PC hard drive. Sleep stages were scored visually per 20-s epochs (Vitaport Paperless Sleep Scoring Software), according to Rechtschaffen and Kales (1968), by a single experienced polysomnography technician, blind to the light conditions. NREM sleep was defined as the sum of NREM Stages 2, 3 and 4. Slow wave sleep (SWS) was defined as the sum of NREM sleep Stages 3 and 4. EEG artefacts were detected by an automated artefact algorithm (CASA, 2000; PhyVision BV, Gemert, the Netherlands). Spectral analysis was conducted using a fast-Fourier transformation (FFT; 10% cosine 4-s window), which yielded a 0.25 Hz bin resolution. EEG power spectra were calculated during NREM sleep and REM sleep in the frequency range from 0 to 32 Hz. Finally, artefact-free 4-s epochs were averaged across 20-s epochs. Here, we report EEG data derived from eight derivations (F3, F4, C3, C4, P3, P4, O1, O2) referenced against linked mastoids (A1, A2) in the range of 0.50–20 Hz.

Statistics

The statistical packages SAS (version 9.1; SAS Institute, Cary, NC, USA) and STATISTICA (version 6.1; StatSoft, Tulsa, OK, USA) were used. Visually scored sleep stages were expressed as percentages of total sleep time (TST) or in minutes (sleep latencies, total sleep time). To examine sleep EEG power density in the range of 0.50–20 Hz during NREM sleep for the light conditions, all-night EEG power density during NREM sleep was computed for frontal (F3, F4), central (C3, C4), parietal (P3, P4) and occipital (O1, O2) derivations for each 0.25 Hz frequency bin. Comparisons across light conditions were made with a general linear model, with factors 'light condition' (6500 K versus 2500 K versus 3000 K) and 'derivation' (frontal, central, parietal, occipital). For the factor 'derivation', the corresponding two derivations (i.e. frontal = F3 and F4) were averaged per participant, given that there were no lateralization effects.

NREM–REM sleep cycles were defined according to Feinberg and Floyd (1979). Because not all study participants completed four sleep cycles, all sleep data analyses were confined to the first three NREM–REM sleep cycles. Thereafter, each sleep cycle was subdivided into 10 time intervals of equal length during NREM sleep and into four time intervals during REM sleep (percentiles). For the analysis of sleep cycles, we used a general linear model [one-, two- and three-way analysis of variance (ANOVA)] with main factors 'light condition', 'derivation' and 'cycle', and the interaction of factors 'light condition' versus 'derivation', 'light condition' versus 'cycle' and 'light condition' versus 'cycle' versus 'derivation'. Because no significant effects were observed for the three-way interaction, but rather for the interaction of 'light condition' versus 'cycle' (for each derivation separately), we then computed Tukey's *post hoc* multiple comparisons test for this specific interaction. All *P*-values derived from r-ANOVAs were based on Huynh–Feldt's (H-F) corrected degrees of freedom (significance level: $P < 0.05$).

To estimate the decline of NREM SWA across sleep cycles, a non-linear regression analysis was calculated separately for each participant. To investigate the decay of NREM EEG SWA power density, we first performed a detection of each quarter Hz within the SWA frequency range (0.75–4.5 Hz) that had a significant interaction of the factors 'light condition' and 'cycle' (i.e. 2.0–4.0 Hz). This SWA range was then utilized to assess SWA decay (on absolute values). An exponential decay function was fitted as follows: SWA = SWA~ + (SWA0–SWA~).exp(–rt), whereby SWA0 = intercept on the *y*-axis, SWA = EEG SWA power per sleep cycle, SWA~ = horizontal asympote for time $t = \sim$, r = slope of decay and t = time of each NREM sleep cycle mid-point.

RESULTS

Sleep structure

A one-way ANOVA with the factor 'light condition' indicated that exposure to three different light settings did not elicit significant differences for sleep structure across the entire night, except for a tendency for wake (%TST, $F_{2,12} = 2.5$; $P = 0.09$) (Table 2), with less wakefulness during sleep following light exposure at 6500 K than light at 3000 K. Analysis of sleep structure per sleep cycle (cycles 1–3) revealed no significant differences across the three light settings, except for NREM sleep Stage 1, where the interaction 'light condition' versus 'cycle' was significantly different, with more NREM sleep Stage 1 following light at 6500 K than at 3000 K, during the first NREM–REM sleep cycle ($F_{4,83} = 4.4$, $P < 0.05$, *P*-value corrected for multiple comparisons).

Sleep EEG power density

A two-way r-ANOVA with the factors 'light condition' and 'derivation' for all-night NREM sleep EEG power density revealed a tendency for the interaction of factors 'light condition' and 'derivation' in the frequency bins of 1.75–3.25 Hz ($F_{6,168} > 2.5$; $P < 0.1$). The main factor 'light condition' revealed no significance, while the main factor 'derivation' yielded significance in the frequency ranges of 0.50–5.0 Hz, 6.75–7.75 Hz, 9.25–12.0 Hz and 13.0–15.5 Hz ($F_{3,84} > 3.6$; $P < 0.05$). Exposure to light at 6500 K resulted in a tendency for less frontal NREM EEG power density in the range of 1.75–3.25 Hz, in comparison to light at 2500 K and 3000 K ($P < 0.1$; Tukey–Kramer test). All-night REM sleep EEG power density did not differ significantly across the three light conditions (data not shown).

Analysis of NREM EEG power density per sleep cycle yielded a significant main factor of 'light condition' from 2.25 Hz to 4.25 Hz ($F_{4,86} > 1.0$; $P < 0.05$), with light at 6500 K resulting in less EEG activity in these frequency bins, compared to light at 3000 K and 2500 K. A two-way ANOVA with the interaction of factors 'light condition' and 'cycle', for each derivation separately, elicited significant differences in the frequency bins from 2.0 to 4.0 Hz ($F_{4,86} > 1.2$; $P < 0.05$). *Post hoc* comparisons of this two-way interaction revealed significant differences for the first NREM–REM sleep cycle, such that light at 6500 K resulted in less EEG power density in the frequency range from 2.0 to 4.0 Hz (SWA range),

Table 2 Visually scored sleep stages subsequent to light exposure at 6500 K, 3000 K and 2500 K ($n = 30$)

Stages	*Blue-enriched light (6500 K)*	*Classic light (3000 K)*	*Warm light (2500 K)*	P*
TST (min)	378.0 ± 7.9	390.7 ± 3.1	382 ± 6.3	0.3
SE (%)	94.6 ± 0.6	95.1 ± 0.4	92.5 ± 1.3	0.1
Wake (%)	2.8 ± 0.6	3.3 ± 0.4	5.5 ± 1.3	0.09
Stage 1 (%)	11.3 ± 0.5	11.2 ± 0.6	10.9 + 0.6	0.9
Stage 2 (%)	52.7 ± 0.9	52.5 ± 1.3	53.1 ± 1.3	0.9
Stage 3 (%)	10.5 ± 0.6	10.6 + 0.5	11.0 ± 0.6	0.8
Stage 4 (%)	7.1 ± 1.0	7.3 ± 1.1	7.0 + 1.0	0.9
SWS (%)	17.6 ± 1.0	17.9 ± 1.1	18.0 ± 1.1	0.9
NREM (%)	70.4 ± 0.9	70.6 ± 1.1	71.2 ± 0.9	0.8
REM (%)	18.2 ± 0.9	18.2 ± 0.9	17.9 ± 1.0	0.9
MT (%)	2.2 ± 0.1	2.1 ± 0.1	2.1 ± 0.1	0.7
Arousal (%)	5.0 ± 1.1	5.4 ± 1.1	7.6 ± 1.4	0.1
SL1, min	6.1 ± 1.2	6.2 ± 1.1	6.4 ± 1.1	0.8
SL2, min	8.1 ± 1.1	8.3 ± 1.1	8.4 ± 1.4	0.8
RL, min	64.9 ± 7.2	65.3 ± 8.3	66.1 ± 8.7	0.9

TST, total sleep time; SE, sleep efficiency [(Stages 1–4 REM)/(time after lights off – time lights on) × 100]; Wake, wakefulness after lights off (% TST); SWS, slow wave sleep (Stages 3 + 4) (% TST); NREM, non-rapid eye movement sleep (Stages 2–4) (% TST); REM, rapid eye movement sleep (% TST); MT, movement time (% TST); Arousal, wake + movement time (% TST); SL1, sleep latency to Stage 1; SL2, sleep latency to Stage 2; RL, REM sleep latency (after sleep onset).

*P (one-way repeated-measures analysis of variance). Values are depicted as mean ± standard error of the mean ($n = 30$).

relative to light at 3000 K and 2500 K ($P < 0.05$; Tukey–Kramer test).

Dynamics of SWA during NREM sleep

To investigate the dynamics of SWA (2.0–4.0 Hz) across sleep cycles, we analysed SWA for each percentile during NREM sleep cycles. A two-way r-ANOVA with factors 'light condition' and 'percentiles' performed separately for each derivation yielded a significant interaction for only the frontal derivation ($F_{18,265} = 1.86$; $P = 0.04$). *Post hoc* comparisons yielded significantly less frontal NREM SWA during the first sleep cycle [time intervals 4–8 (percentiles) during NREM sleep)] following exposure to light at 6500 K ($P < 0.05$; Tukey–Kramer test) (Fig. 1).

Given the differential SWA levels across the three light conditions, we computed the exponential decay function for NREM SWA, first on an individual basis and then by computing the average parameters for all-night duration of NREM sleep EEG. The fitted regression curves for mean SWA per NREM–REM sleep cycle are illustrated in Fig. 2. A one-way ANOVA performed on the rate of SWA dissipation across the three light conditions (for each derivation separately) revealed no significant differences. A two-way r-ANOVA on the predicted amount of SWA derived from the non-linear exponential function yielded a main effect of 'cycle' ($F_{3,75} = 44.08$; $P < 0.01$) and 'light condition' ($F_{2,45} = 4.23$; $P = 0.02$), with less SWA following light at 6500 K compared to light at 3000 K, for only the frontal derivation. The interaction of these factors did not yield significance.

DISCUSSION

Our data indicate that exposure to 2 h of evening blue-enriched light impacted significantly on the temporal dynamics of sleep EEG activity, such that frontal NREM SWA was reduced during the first NREM–REM sleep cycle, relative to light at 2500 K and 3000 K.

Light and sleep: circadian and homeostatic processes

One probable explanation for the blue-light-induced reduction in NREM EEG power density in the SWA range, at the beginning of the night, could be through its possible alerting effects. The alerting properties of light during wakefulness rely crucially upon factors such as prior wakefulness, environmental light history and endogenous circadian phase (Chellappa *et al.*, 2011b). Sleep is regulated by the interaction of a homeostatic and a circadian component (Borbély, 1982), and the duration of prior wakefulness is critical when considering sleep intensity. In our study, prior wakefulness was similar for all light conditions, thus the differential levels of NREM SWA cannot be ascribed to differences in prior wake duration. Furthermore, this study was conducted during the winter season, when outdoor light levels are lowest at this latitude in Switzerland. While we assume that the amount of prior light exposure was comparable among our participants, we did not control for this factor, and thus we cannot rule out the effects of individual prior light history. The effects of photic history on the light resetting capacity of the human circadian pacemaker are relatively unknown. A recent study investigated the effects of prior exposure to 6–8 weeks of

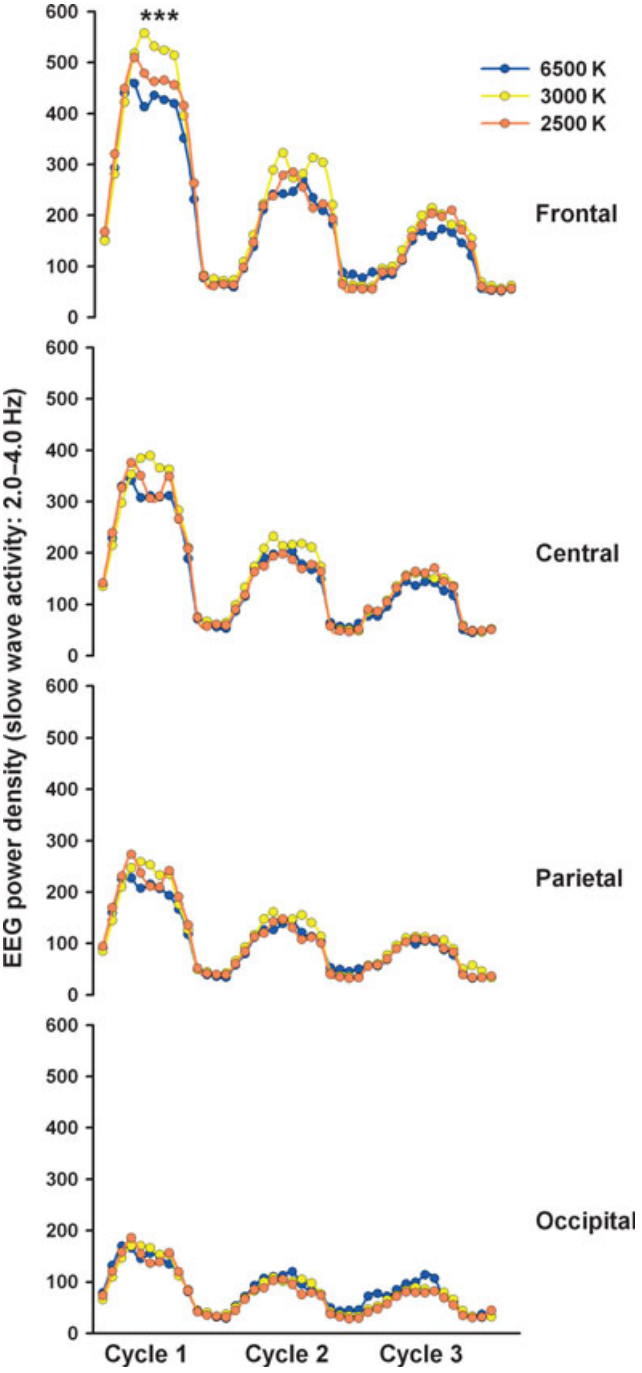

Figure 1. Dynamics of absolute values of slow wave activity (SWA: 2.0–4.0 Hz) per non-rapid eye movement (NREM)–REM sleep cycles 1–3 for frontal, central, parietal and occipital derivations, following light exposure at 6500 K (blue lines), at 3000 K (yellow lines) and at 2500 K (orange lines) ($n = 30$; $^*P < 0.05$).

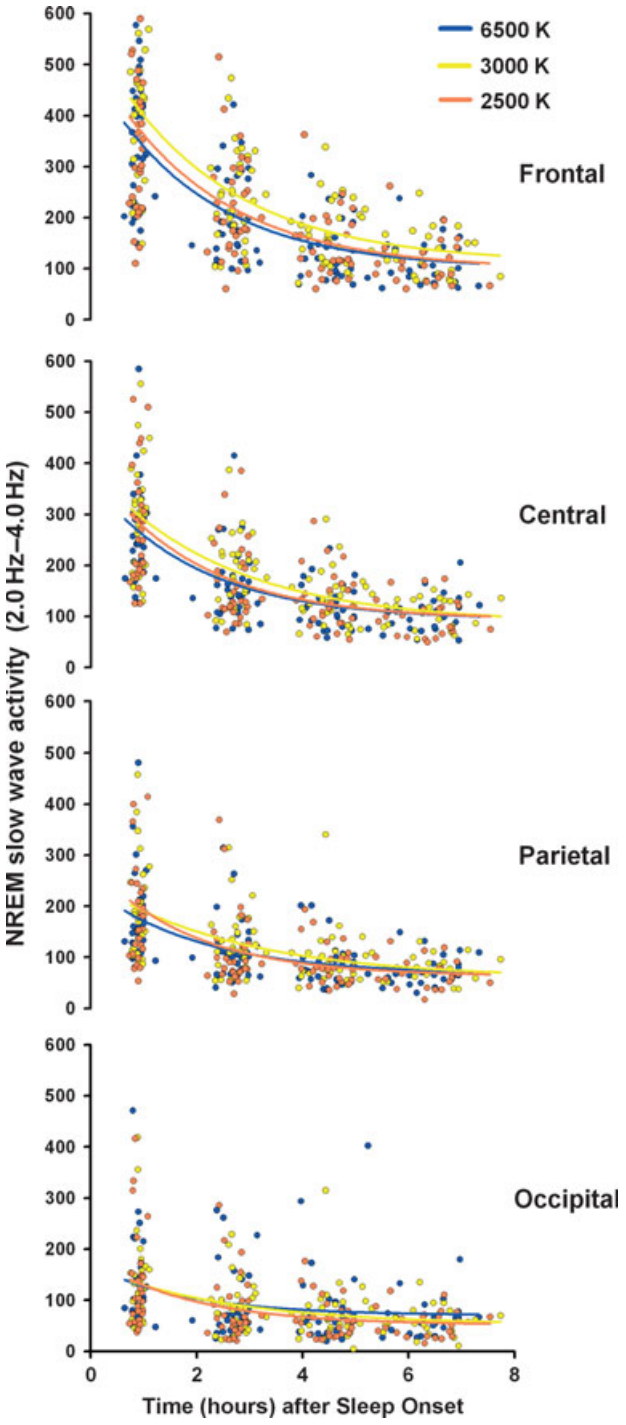

Figure 2. Time–course of absolute non-rapid eye movement (NREM) slow wave activity (SWA: 2.0–4.0 Hz) per NREM–REM sleep cycles 1–3 for frontal, central, parietal and occipital derivations, following light exposure at 6500 K (blue lines), at 3000 K (yellow lines) and at 2500 K (orange lines). Lines represent an exponential decay function [SWA = SWA~ + (SWA0–SWA~).exp(–rt)] of NREM SWA fitted to the data separately for the three light conditions. Data are plotted by time (in hours) after sleep onset (NREM sleep Stage 2) (n = 30).

polychromatic light with reduced, intermediate or enhanced efficacy with respect to the photopic and melanopsin systems (Santhi *et al.*, 2012). Interestingly, the authors observed a significant suppression of nocturnal melatonin and delayed sleep onset following exposure to bright blue- and blue-enriched light, relative to blue-depleted light and near darkness conditions, suggesting that these effects were mediated presumably by the melanopsin rather than the photopic system. With respect to endogenous circadian phase, none of the participants was an extreme chronotype, which reduced the interindividual variability in morningness–eveningness preference. Extreme chronotypes exhibit variations in endogenous circadian phase, i.e. earlier for extreme morning types and later for extreme evening types (Mongrain *et al.*, 2004). Therefore, we assume that our light effects on sleep were not due to interindividual differences in circadian phase.

Light and sleep: neuronal networks

Evening exposure to bright polychromatic light alters the dynamics of NREM SWA, with lower levels of SWA in the first NREM–REM sleep cycle and higher levels during the fourth cycle relative to dim light (Cajochen *et al.*, 1992). Here we show that the magnitude of these effects depend on the light's wavelength, such that light at 6500 K attenuated NREM SWA significantly at the begining of the night compared to light at 2500 K and 3000 K.

Acute light-induction of sleep in mice seems to be modulated by the melanopsin-based photoreception (Tsai *et al.*, 2009), thus reflecting a strong photic input to sleep. The neuronal networks underlying the responses to light involve intrinsically photosensitive retinal ganglion cells that project onto the suprachiasmatic nuclei (SCN), subparaventricular zone and the ventrolateral pre-optic area (VLPO) (Hattar *et al.*, 2002). Thus, direct photic input can alter SCN and VLPO activity. The VLPO innervates the major nuclei of the ascending monoaminergic and histaminergic pathways implicated in wakefulness (Lin *et al.*, 1996), and galanin- and gamma-aminobutyric acid (GABA)-releasing neurons within the VLPO have been characterized as sleep-active neurons that may promote sleep actively by inhibiting the ascending arousal systems (Saper *et al.*, 2001). It is tempting to speculate that blue-enriched light may elicit a stronger wake-promoting signal onto the SCN and VLPO, which contains sleep-promoting neurons, by shifting the balance of the inhibitory interaction towards sleep promotion and arousal inhibition (Tsai *et al.*, 2009). Ultimately this may impinge on human sleep structure and EEG activity.

The topographical distribution of NREM SWA indicated a significant decrease for only the frontal derivation. The homeostatic increase in the SWA range is most predominant in the frontal cortex (Cajochen *et al.*, 1999; Finelli *et al.*, 2000), indicating a higher vulnerability of this brain area to increased sleep pressure or neuronal tiredness due to use-dependent factors, such as synaptic overload (Tononi and Cirelli, 2006). In this scenario, blue-enriched light may counteract this increased susceptibility of the frontal cortex

to higher sleep pressure, resulting in less frontal NREM SWA at the beginning of sleep.

One limitation in our study is the absence of a control night, when no light pulses were conducted. Thus, the extent to which light affects sleep structure and sleep EEG power density remains unclear. Interestingly, we could partly confirm the effects of bright polychromatic light (Cajochen *et al*., 1992) and monochromatic blue light (460 nm) (Münch *et al*., 2006) with reduced NREM SWA in the first sleep cycle, although we did not observe the rebound of SWA in latter sleep cycles. This suggests that blue-enriched polychromatic light may exhibit acute effects on the initial sleep episode, which do not persist in later stages of sleep. Furthermore, we tested specifically the acute effect of light on sleep, which does not allow for the assessment of long-term effects. Chronic exposure to light may elicit long-lasting effects on sleep structure, consolidation and sleep EEG activity, although the magnitude and direction of these changes remain to be explored. Future studies focusing on the long-term non-visual light effects on human sleep are needed to address this question, and may enable a better future understanding of the significance of light effects onto sleep for real-life scenarios, which is currently unavailable.

The present data indicate that light impinges acutely onto NREM sleep EEG power density (slow wave activity) in a wavelength-dependent manner. We speculate that these findings may be mediated by a light-induced reduction in accumulated sleep pressure, as indexed by less frontal NREM SWA. Our data also indicate that, even at low ambient light levels (*c*. 40 lux), light can impact directly upon sleep EEG characteristics. Therefore, the use of commercially available compact fluorescent lights with different colour temperatures may impact significantly upon circadian physiology and sleep. In our study, prior light exposure was controlled stringently, such that all participants were under dim light (1.5 h) and darkness (2 h) before exposure to the light settings. Controlling for prior light history enhances sensitivity to light exposure, by enabling the melanopsin (and also rods and cones) photopigment system to achieve a stable state of photo-equilibrium through a reduction in the 'bleaching effect' of previous light exposure (Mure *et al*., 2009). Nevertheless, caution should be made when generalizing these results into real-life conditions, given this stringent control for 'photic memory'. Taken together, these data have implications for our understanding of the non-image forming effects of light on sleep regulation in humans.

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CONFLICT OF INTEREST

No conflicts of interest declared.

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