



## Brief Communication

# Let there be no light: the effect of bedside light on sleep quality and background electroencephalographic rhythms



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## ABSTRACT

**Objectives:** Artificial lighting has been beneficial to society, but unnecessary light exposure at night may cause various health problems. We aimed to investigate how whole-night bedside light can affect sleep quality and brain activity.

**Patients and methods:** Ten healthy sleepers underwent two polysomnography (PSG) sessions, one with the lights off and one with the lights on. PSG variables related to sleep quality were extracted and compared between lights-off and lights-on sleep. Spectral analysis was performed to rapid eye movement (REM) sleep and non-REM (NREM) sleep epochs to reveal any light-induced differences in background brain rhythms.

**Results:** Lights-on sleep was associated with increased stage 1 sleep (N1), decreased slow-wave sleep (SWS), and increased arousal index. Spectral analysis revealed that theta power (4–8 Hz) during REM sleep and slow oscillation (0.5–1 Hz), delta (1–4 Hz), and spindle (10–16 Hz) power during NREM sleep were decreased in lights-on sleep conditions.

**Conclusions:** Sleeping with the light on not only causes shallow sleep and frequent arousals but also has a persistent effect on brain oscillations, especially those implicated in sleep depth and stability. Our study demonstrates additional hazardous effect of light pollution on health.

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## 1. Introduction

Exposure to light at night is now considered to be ordinary. Artificial light certainly has beneficial aspects; it has extended the length of productive days of work and recreational activities. However, when it becomes unreasonably excessive, it can be considered as light pollution, negatively affecting human physiology [1]. It may disturb circadian organization; influence neuroendocrine systems; and cause many diseases, such as obesity [2], diabetes mellitus, depression, and even cancers [1].

Artificial lighting also is commonplace in bedrooms, and individuals with poor sleep hygiene often deliberately or unintentionally fall asleep with lights on. For example, one may fall asleep late night with the television light on, and children who are afraid of the dark may ask their parents to keep their lights on during sleep. Light exposure may affect sleep quality, but there have been no systemic comparative studies with objective measures. If any dif-

ference exists, it may be reflected in background brain oscillations. Here we performed two whole-night polysomnography (PSG) sessions, one with lights off and one with lights on, to investigate the effect of light on sleep quality and brain activity.

## 2. Methods

Ten young healthy volunteers (4 women; mean age, 27 years; range, 21–34 years) participated in our study, which was reviewed and approved by the local internal review committee. All participants gave fully informed written consent. They were all healthy sleepers without major health problems, including no neurologic, psychiatric, or endocrine disorders. Interviews and the Pittsburgh Sleep Quality Index [3] suggested normal and regular sleep-wake habits. First the PSG was performed with the lights off, and then the results of the PSG were reviewed to rule out participants with potential signs of sleep disorders. A doctor specializing in sleep medicine (Koo DL) reviewed the recordings and found that none of the participants had any indications of sleep problems. The obtained data were not used for analysis, due to the possibility of the first-night effect. Participants were divided into two groups: one group participated in a PSG study with the lights off first and then

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with lights on after a mean interval of 1 week, and the other group participated in a PSG study in the opposite order. To simulate artificial light commonly used in the participants' bedrooms, we used a commercially available fluorescent lamp (40 lux, 30-cm long), located approximately 1 m away from the participants' eyes (above the head and perpendicular to body). The light was kept on during the entire session. All experiments were performed in a sleep laboratory.

Participants were asked not to drink alcohol or caffeinated beverages and to sleep and wake at regular habitual hours starting from a week before the study until the end of the experiment. Whole-night PSG was recorded by Embla N7000 system. PSG variables that were appropriate in evaluating sleep quality were collected (Table 1). Electroencephalographic (EEG) signals (200 Hz, 0.5–70 Hz band-pass filter; F3/F4/C3/C4/O1/O2 referenced to contralateral mastoids) were collected with electrooculogram, electromyogram, and electrocardiogram. The lights were turned off at 11:00 pm and participants were asked to wake up on their own will. Sleep architecture was scored by an experienced technician who was blind to the night light conditions in 30-s epochs according to the standard criteria [4].

Sleep-staged epochs were divided into three 10-s epochs for EEG analysis. Low-frequency ocular artifacts were eliminated by excluding epochs scored as wakefulness. Epochs with high-frequency muscle artifacts were removed with a previously published method [5]. Subsequently, artifact-free epochs labeled either as nonrapid eye movement (NREM) (stage 2 sleep [N2] and slow-wave sleep [SWS]) or rapid eye movement (REM) sleep were multiplied by Hanning window and were submitted to discrete Fourier transformation with Goertzel algorithm, which calculated spectral components at predetermined frequencies (0.5–30 Hz with 0.1-Hz step) [6]. The mean power of discrete frequency bands (slow oscillations, 0.5–1 Hz; delta frequency bands, 1–4 Hz; theta frequency bands, 4–8 Hz; spindle frequency bands, 10–16 Hz; low beta frequency bands, 16–25 Hz; and high beta frequency bands, 25–30 Hz) was computed for frontocentral electrodes.

For statistical comparison, the nonparametric Wilcoxon signed rank test was used. To account for multiple comparisons concerns in EEG parameters, Bonferroni correction was used when appropriate. A *P* value of <.05 was considered significant.

### 3. Results

All participants demonstrated normal patterns of sleep. With the lights on, nine participants reported higher scores in subjective

**Table 1**  
Summary of polysomnographic findings.

PSG variables	Lights off	Lights on	<i>P</i> value
Total bedtime (min)	471.2 ± 9.9	469.7 ± 9.8	.5903
TST (min)	434.1 ± 13.0	433.0 ± 8.3	.8387
Sleep efficiency (%)	93.1 ± 2.0	92.9 ± 1.0	.6365
Stage N1/TST (%)	8.6 ± 1.7	10.2 ± 1.9	.0438
Stage N2/TST (%)	54.2 ± 2.5	56.6 ± 1.9	.2287
SWS/TST (%)	15.1 ± 2.5	11.3 ± 2.8	.0003
NREM sleep duration/TST (%)	78.1 ± 1.9	78.6 ± 1.3	.5956
REM sleep duration/TST (%)	21.8 ± 1.8	21.9 ± 1.3	.5956
Latency to N1 (min)	6.1 ± 1.1	6.3 ± 1.3	.9321
Latency to N2 (min)	11.8 ± 2.1	11.5 ± 2.5	.9561
Latency to REM (min)	78.1 ± 7.5	81.4 ± 9.3	.7296
WASO/TST (%)	4.8 ± 1.0	7.1 ± 1.9	.0948
Arousal index (n/h)	9.1 ± 0.4	12.9 ± 1.2	.0032

**Abbreviations:** PSG, polysomnography; min, minutes; TST, total sleep time; N1, stage 1 sleep; N2, stage 2 sleep; SWS, slow-wave sleep; NREM, nonrapid eye movement sleep; REM, rapid eye movement sleep; WASO, wake after sleep onset; n/h, number per hour.

Number consists of mean ± standard error of the mean.

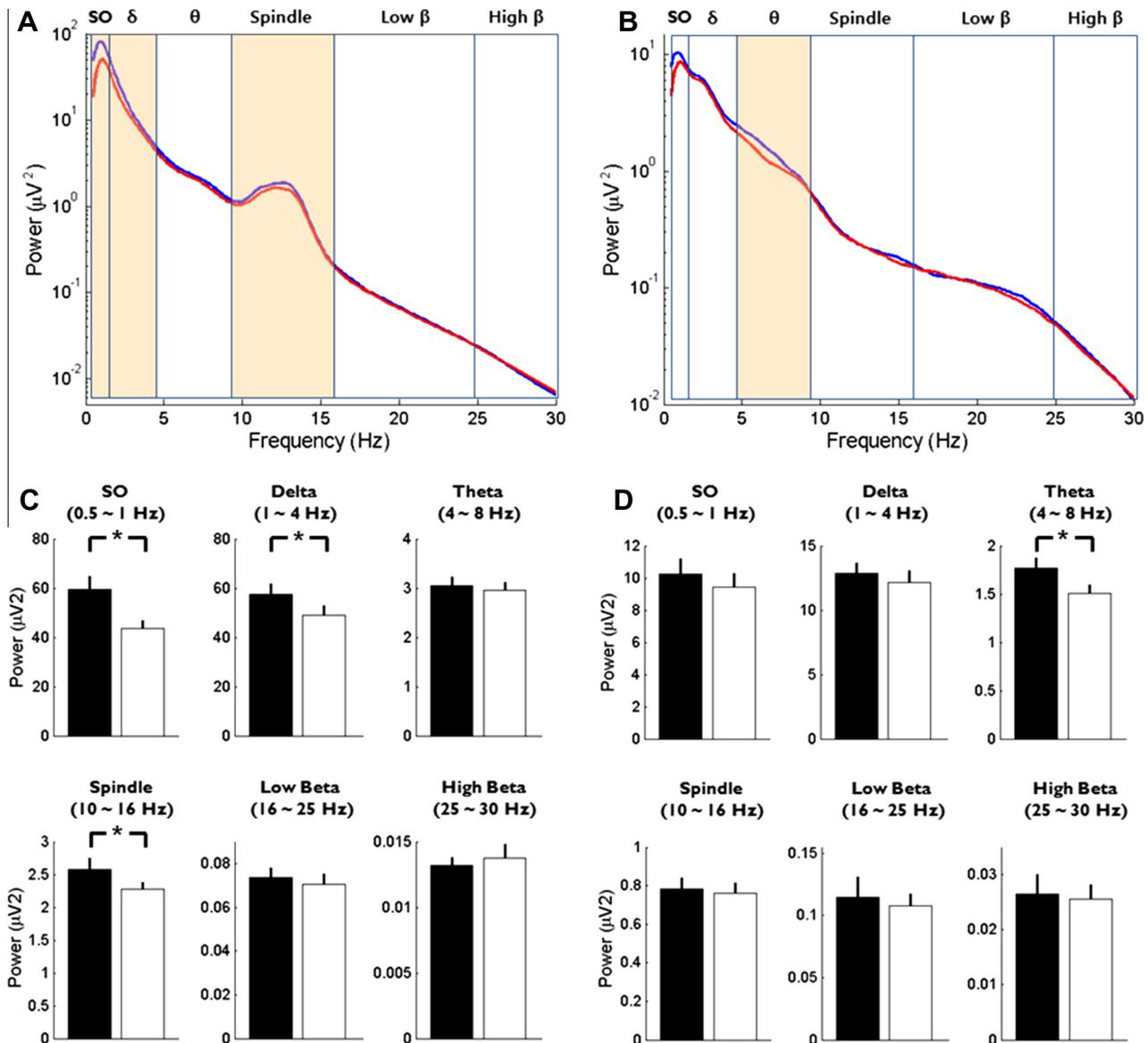
feeling of frequent arousals and shallow sleep. We extracted PSG variables (Table 1) that were appropriate in evaluating sleep quality. There was no significant difference in total bed time, total sleep time, or sleep efficiency between lights-off and lights-on sleep. However, constant exposure to dim light affected sleep architecture, increasing the proportion of stage 1 sleep (N1) (*P* < .05) and decreasing the proportion of SWS (*P* < .001). No differences in NREM and REM sleep duration were found. Lighting did not affect sleep onset or sleep latency to specific stages. We observed differences in sleep maintenance parameters (i.e., wake after sleep onset showed an increasing trend [*P* < .1]), and the number of arousals per hour significantly increased (*P* < .01) in lights-on sleep.

Spectral analysis revealed that nocturnal light exposure had persistent effects on EEG rhythms, differentially affecting NREM and REM sleep stages. Power spectral density on NREM epochs (Fig. 1A) demonstrated that lighting decreased slow-wave activity (SWA) (0.5–4 Hz) and spindle range. Slow oscillations, delta frequency bands, and spindle frequency bands were significantly decreased with the light on (Fig. 1C; *P* < .05). During REM sleep (Fig. 1B), theta activity was decreased (Fig. 1D; *P* < .05). We also performed further analysis on individual slow waves and spindles detected by automated algorithms (see [Supplementary Materials](#) for details). The amplitude, the up and down slope of slow waves, were decreased with the lights on (Suppl. Fig. 1A). For spindles, there was a marked decrease in the incidence and integrated activity (Suppl. Fig. 1B).

### 4. Discussion

Unintentional or intentional light exposure during sleep is becoming increasingly more common due to the widespread use of artificial lighting, and its negative effect on health is a major theme of research. Whole-night light exposure demonstrated acute effects on sleep architecture and brain activity, including shallow sleep, frequent arousals, and reduction of power in SWA and spindle frequency bands during NREM sleep and theta frequency bands during REM sleep.

Light is the major synchronizer of circadian rhythm for a number of physiologic variables, such as alertness, body temperature, and hormones [7]. Light stimulates retinal ganglion cells [8]. This stimulation triggers nonvisual response with direct connection to the central circadian pacemaker and suprachiasmatic nuclei of the anterior hypothalamus, which suppresses melatonin secretion from the pineal gland [9]. Suppressed melatonin may pose an impact on homeostatic sleep regulation and affect the synchrony of neural activity in neocortex and thalamocortical networks demonstrated as diminished SWA and spindles, which are markers of sleep depth [10–12]. Reduced spindle incidence and power may be affected by reduced SWA itself, which is known to group spindle activity [11]. Close correlation with spindle rate and sleep stability provides additional support that sleeping with the lights on is associated with shallow sleep and that melatonin may be involved in circadian regulation of sleep spindles [13]. Similar experimental designs and melatonin level measurements may reveal more about the possible relationship with melatonin, brain oscillations, and sleep disruption. Suprachiasmatic nuclei also project to the basal forebrain, which produces acetylcholine and affects neocortical synchrony for maintenance of cortical activation. Lights during NREM sleep may hinder the inhibition of acetylcholine release, causing reduced SWA and spindle frequency bands [14]. It is unclear as to how light during sleep affected REM theta sleep; the power of theta activity, an EEG hallmark during REM sleep, also may be regulated by melatonin or cholinergic neurons in basal forebrain, which plays important role in REM theta sleep generation [15]. Because SWA and spindle activity during NREM sleep and theta activity during REM sleep strongly are implicated in



**Fig. 1.** Results of spectral analysis of whole-night electroencephalographic recordings. Grand average of power spectra across all analyzed channels and all participants during nonrapid eye movement (NREM) sleep. The blue solid line represents lights-off condition and the red solid line represents lights-on condition. Power (y axis) is scaled in logarithmic scale. Pink-colored boxes represent frequency bands that are statistically different between two light conditions (A). Letter B represents the same occurrence (A) but during rapid eye movement (REM) rather than NREM sleep (B). Comparison of functional frequency band power during NREM sleep. The black bar represents lights-off condition and the white bar represents lights-on condition. Bars scale up to the mean and black solid lines stand for standard error of the mean (C). Stars (\*) correspond to statistically significant results. Letter D represents the same occurrence (C) but during REM rather than NREM sleep (D). (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

memory consolidation [11,16], it is tempting to hypothesize the potentially negative effect of bedside lights in the protection of previously learned items. Our study provides first-line evidence that light exposure during the night can alter neurophysiology during sleep. Future studies are warranted to deepen the understanding of the neuronal mechanisms and their behavioral or cognitive effects on sleep.

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#### Conflict of interest

The ICMJE Uniform Disclosure Form for Potential Conflicts of Interest associated with this article can be viewed by clicking on the following link: <http://dx.doi.org/10.1016/j.sleep.2013.09.007>.

#### References

- [1] Stevens RG, Blask DE, Brainard GC, Hansen J, Lockley SW, Provencio I, et al. Meeting report: the role of environmental lighting and circadian disruption in cancer and other diseases. *Environ Health Perspect* 2007;115:1357–62.
- [2] Fonken LK, Workman JL, Walton JC, Weil ZM, Morris JS, Haim A, et al. Light at night increases body mass by shifting the time of food intake. *Proc Natl Acad Sci U S A* 2010;107:18664–9.
- [3] Buysse DJ, Reynolds CF, Monk TH, Berman SR, Kupfer DJ. The Pittsburgh Sleep Quality Index (PSQI): a new instrument for psychiatric research and practice. *Psychiatry Res* 1989;28:193–213.

- [4] Iber C, Ancoli-Israel S, Chesson A, Quan SF. The AASM manual for the scoring of sleep and associated events: rules, terminology, and technical specifications. Westchester (IL): American Academy of Sleep Medicine; 2007.
- [5] Brunner DP, Vasko RC, Detka CS, Monahan JP, Reynolds 3rd CF, Kupfer DJ. Muscle artifacts in the sleep EEG: automated detection and effect on all-night EEG power spectra. *J Sleep Res* 1996;5:155–64.
- [6] Cho JR, Koo DL, Joo EY, Yoon SM, Ju E, Lee J, et al. Effect of levetiracetam monotherapy on background EEG activity and cognition in drug-naïve epilepsy patients. *Clin Neurophysiol* 2011;123:883–91.
- [7] Minors DS, Waterhouse JM, Wirz-Justice A. A human phase-responsive curve to light. *Neurosci Lett* 1992;133:36–40.
- [8] Gooley JJ, Lu J, Fischer D, Saper CB. A broad role for melanopsin in nonvisual photoreception. *J Neurosci* 2003;23:7093–106.
- [9] Brainard GC, Hanifin JP, Greeson JM, Byrne B, Glickman G, Gerner E, et al. Action spectrum for melatonin regulation in human: evidence for a novel circadian photoreceptor. *J Neurosci* 2001;21:6405–12.
- [10] Cajochen C, Krauchi K, Danileko KV, Wirz-Justice A. Evening administration of melatonin and bright light: interactions on the EEG during sleep and wakefulness. *J Sleep Res* 1998;7:145–57.
- [11] Marshall L, Helgadottir H, Molle M, Born J. Boosting slow oscillations during sleep potentiates memory. *Nature* 2006;444:610–3.
- [12] Bayer L, Constantinescu I, Perrig S, Vienne J, Vidal PP, Muhlethaler M, et al. Rocking synchronizes brain waves during a short nap. *Curr Biol* 2011;21:R461–2.
- [13] Dang-Vu TT, McKinney SM, Buxton OM, Solet JM, Ellenbogen JM. Spontaneous brain rhythms predict sleep stability in the face of noise. *Curr Biol* 2010;20:R626–7.
- [14] Douglas CL, Baghdoyan HA, Lydic R. Prefrontal cortex acetylcholine release, EEG slow waves, and spindles are modulated by M2 autoreceptors in C57BL/6J mouse. *J Neurophysiol* 2002;87:2817–22.
- [15] Lee MG, Hassani OK, Alonso A, Jones BE. Cholinergic basal forebrain neurons burst with theta during waking and paradoxical sleep. *J Neurosci* 2005;25:4365–9.
- [16] Nishida M, Pearsall J, Buckner RL, Walker MP. REM sleep, prefrontal theta, and the consolidation of human emotional memory. *Creb Cortex* 2009;19:1158–66.