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Inhibition of Heart Rate Variability during Sleep in Humans by 6700 K Pre-sleep Light Exposure

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Abstract Two different spectral analyses of heart rate (HR) variability (HRV) were performed on seven young male subjects to evaluate the effects of different color temperatures of light exposure (6700 K, 5000 K, 3000 K) before sleep on cardiac vagal activity. In investigating HRV, we used an ordinary fast Fourier transform (FFT) and coarse graining spectral analysis (CGSA), which selectively extracts random fractal components from a given time series. The results showed that suppressions of HR during sleep after 6700 K light exposure were more inhibited than the other two lighting conditions. Increases in high-frequency (HF) components of HRV during sleep were also inhibited by 6700 K pre-sleep lighting. These results indicate that pre-sleep exposure to light of a higher color temperature may inhibit the enhancement of cardiac vagal activity during sleep. Moreover, significant HF alterations were shown in fractal-free HF (not in ordinary HF) components by CGSA. Because the HF component originates from respiratory sinus arrhythmia with periodical fluctuations, CGSA may be an appropriate approach for HRV evaluation during sleep. *J Physiol Anthropol 26(1): 39–43, 2007* http://www.jstage.jst.go.jp/browse/jpa2
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Introduction

There is accumulating neurological evidence suggesting that bright light affects autonomic outflows, enhances sympathetic activity and suppresses parasympathetic activity through the suprachiasmatic nucleus (SCN) in the hypothalamus (Niijima et al., 1993; Scheer et al., 2003). Recent findings also indicate that light containing much shorter wavelengths of ca. 6500 K with a high color temperature influences the nervous systems even when the subjects are exposed to the same light intensity; i.e. light of a high color temperature enhances sympathetic and/or suppresses parasympathetic activities more than the other light sources (i.e., 5000 K or 3000 K) at the same light intensity (Mukae and Sato, 1992; Kobayashi and Sato, 1992; Yasukouchi and Ishibashi, 2005). It is well known that exposure to bright light at night induces suppression of melatonin secretions and elevations of core temperature, blood pressure and heart rate (HR) (Burgess et al., 2001; Higuchi et al., 2005; Tsunoda et al., 2001; Yokoi et al., 2006). However, little is known about the effect of exposure to light of a high color temperature before sleep on autonomic activities during sleep, although Morita and Tokura (1996) have previously documented the effects of pre-sleep high color temperature exposure on core temperature and melatonin in humans. Moreover, Katsuura et al., (2002), have reported that time-dependent changes in physiologically suitable color temperature in humans are similar to changes in natural sunlight. Therefore, artificial lights of a high color temperature would not be appropriate for nighttime illuminance for humans. The homeostatic ability of autonomic modulation to artificial light exposure at nighttime should be considered when perspectives of the techno-adaptability of humans are involved.

One of the reliable non-invasive indexes of monitoring cardiac autonomic activities is heart rate variability (HRV) (Pagani et al., 1986; Pomeranz et al., 1985; Sayers, 1973). Through spectral analyses of HRV, beat-by-beat observations of the fluctuation in HR intervals have revealed the major involvement of high-frequency (HF) and low-frequency (LF) components. The causal factor of the HF component, which serves as a reliable index of cardiac vagal activity, is respiratory sinus arrhythmia (RSA). Details on the physiological mechanism and significance of RSA have previously been described (Berntson et al., 1993; Hayano et al., 1996). Although RSA displays periodical fluctuations in HRV that coincide well with the respiratory cycle, spontaneous HRV contains aperiodic fluctuations, where the power spectrum exhibits fractal components (Kobayashi and Musha, 1982). Distinguished HF components in a power spectrum of HRV are measured by controlling the respiration of subjects in a certain fashion (Grossman et al., 1991; Kobayashi, 1998), albeit the approach is impractical under normal sleep-in conditions. The

aperiodic (or fractal) component has to be abbreviated in HRV evaluations during sleep to yield a specifically useful outcome, especially the detailed analysis of RSA. Therefore, we resorted to the coarse graining spectral analysis (CGSA) method, which is a technique for extracting random fractal components from a given time series (Yamamoto and Hughson, 1991; Yamamoto and Hughson, 1993), for HRV analysis.

In this study, we examined the effects of pre-sleep exposure to light of a high color temperature on cardiac vagal activity using HRV, and compared the reliability of HRV findings during sleep between an ordinary FFT method and the CGSA approach.

Methods

Seven young healthy male adults (mean age: 21.0±2.1 years) participated in this study with prior consent. The subjects were asked to wear short pants and sleeveless shirts during the experiment, and abstain from naps before the experiment. All studies were conducted from July to November.

Subjects were exposed to experimental lighting conditions consisting of three different color temperatures (6700 K, 5000 K, 3000 K) before sleep. Each condition of the light sources (Table 1) was irradiated on a randomized subject on a different day.

The investigations were performed in a climatic chamber without windows. The climatic chamber was controlled at an ambient temperature of 25±0.1°C with a relative humidity of 50±1%. The experimental protocol of this study is shown in Fig. 1A. During the first 1-hr period (18:00–19:00 hr), subjects were similarly exposed to control lighting (a dim light of 10-lux intensity; the illuminance meter was placed in the center of the chamber at a horizontal position 900 mm above the floor) for each exposure condition. Subjects washed themselves (19:00–19:30 hr) with warm water (ca. 40°C) provided in a bathroom adjacent to the experimental chamber before exposure to a certain light stimulus for 7.5 hr (19:30–02:00 hr) under each lighting condition. The intensity of each light exposure was adjusted to 1000 lux. The subjects were instructed to rest on a sofa and to remain awake during the light exposure. They were allowed to read without the use of any device equipped with other light sources (e.g., mobile computer, video game) as described previously (Higuchi et al., 2005; Higuchi et al., 2003). Supper (a uniformly prepared ordinary Japanese meal for all experimental conditions) was provided at 20:30 hr. The subjects slept in a bed in near darkness from 02:00 to 09:00 hr. Subjects were accommodated in the chamber for one night before the experiment, with all procedures and conditions for habituation. Subjects acclimatized to a sleep-wake pattern 10 days before the experiments were asked to maintain the same sleep-wake cycle until termination of the experimental period.

Electrocardiography (ECG) was performed on subjects with an amplifier (AB-621G, Nihon Kohden, Japan). ECG signals were measured on-line using a personal computer with an analog-to-digital conversion rate of 1 kHz per channel by 12-bit AD converter (ADM-5298BPC, Microscience, Japan). The R-R interval sequences (i.e., HRV) were obtained by detecting the peak of the R wave in the ECG. Unequal R-R intervals were interpolated into 6-Hz equidistant data at 5-min. intervals (1800 data points). The data were first employed for the calculation of the mean R-R interval. The HR was obtained by dividing 60000 msec by the mean R-R interval. In calculating the HRV spectra, linear trends were eliminated by linear regression before FFT and CGSA were used for 10 time-shifted subsets of 1024 data points with the Hunning window. The algorithm of the CGSA was based on the method of Yamamoto and Hughson (1993). CGSA provides aperiodic (or fractal) components from the total power spectra. The fractal-free (i.e., periodic, or harmonic) components were obtained by subtracting the total power spectra from the aperiodic components. The ordinary HF and LF components were estimated by FFT, while the fractal-free HF and LF components were monitored by CGSA for each time interval. The HF and LF components were respectively integrated from 0.15 to 0.50 Hz and from 0.05 to 0.15 Hz of the power spectra, and were averaged at 20-min intervals for variance homogeneity.

Table 1 Specification of light sources for exposure

	Type	Model number	Ra	*x*-axis	*y*-axis
Control	Incandescent lamp	LDS100V38WWK	100	0.463	0.415
3000 K	Fluorescent lamp	FHF32EX-L-H	84	0.438	0.392
5000 K	Fluorescent lamp	FHF32EX-N-H	84	0.349	0.349
6700 K	Fluorescent lamp	FHF32EX-D-H	84	0.316	0.319

The significant differences between the values under different lighting conditions for each time interval were determined by the two-tailed multiple t-test with Bonferroni correction (three comparisons under three different conditions). Differences were considered significant when $p<0.05$.

Results

Because the subject sample was relatively small, the relative changes from the control lighting condition in each different lighting condition were used for analysis. No significant differences in the baseline control values were found between any two of the three conditions. Before translation into relative changes, no significant differences were observed between the lighting conditions, although significant changes during sleep compared with pre-sleep in HR and HRV were obtained.

Based on the relative changes of HR (Fig. 1B), significant differences were verified between the different lighting conditions. The HR in the midst of the sleep period after 6700 K light exposure was higher than that of 3000 K light exposure. In addition, the HR of 6700 K light exposure scored the highest at the end of the sleep period compared with those subjected to 5000 K and 3000 K light exposures. In short,

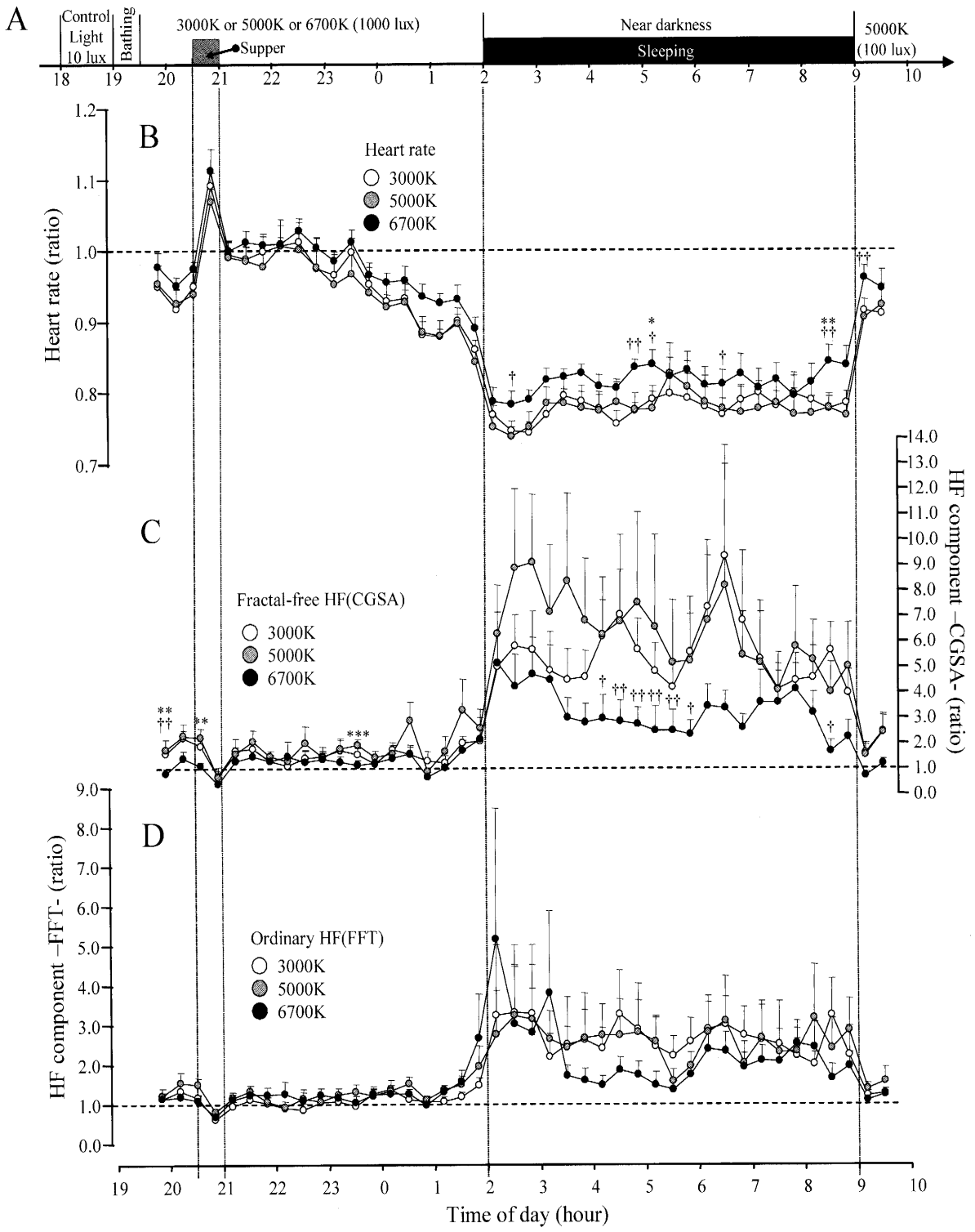

Fig. 1 Experimental protocol (A) and relative changes in (B) heart rate (vs. control lighting condition) under different lighting conditions, (C) fractal-free high-frequency (HF) component by coarse graining spectral analysis (CGSA) and (D) ordinary HF component by fast Fourier transform (FFT). Values are expressed as the mean standard errors (SE) of 7 human subjects, where significant differences of $p<0.01$ (***), <0.05 (**), or <0.1 (*) and $p<0.05$ (††), or <0.1 (†) in data of 6700 K exposure were compared with 5000 and 3000 K light, respectively.

suppressions of HR during sleep after 6700 K light exposure were more likely to be inhibited than during the other lighting conditions.

From the relative changes of fractal-free HF determined by CGSA (Fig. 1C), significant differences between the different lighting conditions were verified. The fractal-free HF components during 6700 K light exposure were lower than those during 3000 K and 5000 K light exposures. The fractal-free HF components in the midst of the sleep period after 6700 K light exposure were lower than that after 3000 K exposure. From these findings, increases of fractal-free HF components were inhibited under 6700 K light exposure.

With reference to the relative changes of ordinary HF by FFT (Fig. 1D), significant differences were not observed between the lighting conditions for each time interval.

Furthermore, no significant differences in the ordinary LF, or fractal-free LF were verified between the lighting conditions for each time interval.

Discussion

The results of this study suggest that pre-sleep light exposure to different color temperatures may modify cardiac vagal activities during sleep, even when the light intensity of the different color temperatures remains unchanged. Exposure of 6700 K light with a high color temperature, or relatively short-wavelength light, would suppress cardiac vagal activity during sleep compared with a 3000 K light source. Morita and Tokura (1996) have observed marked suppressions of nocturnal increases of melatonin secretion by 6500 K light exposure compared with 3000 K light exposure before sleep. Nocturnal increases of melatonin secretion are strongly inhibited by short-wavelength light (Brainard et al., 2001; Cajochen et al., 2005). Cajochen et al. (2005) have also reported that exposures to short-wavelength light (460 nm) inhibit nocturnal HR suppressions. Moreover, a study with administrations of exogenous melatonin suggests that melatonin elicits a direct effect on cardiac autonomic activity, thereby increasing cardiac vagal activities (Nishiyama et al., 2001). Furthermore, Kozaki et al. (2005) have demonstrated considerable attenuation of slow-wave sleep (SWS) with 6700 K pre-sleep light exposure. During SWS, the HF components tend to increase (Bonnet and Arand, 1997; Trinder et al., 2001; Tsunoda et al., 2001). Pre-sleep exposure to light of a high color temperature may inhibit cardiac vagal control during sleep, although the causal factor of decreased HF components remains unclear.

We evaluated the cardiac vagal activities using HF components derived from the spectral analysis of HRV. The results of this study indicate that the fractal-free HF component showed a significant correlation with the HR results. In contrast, the effects of color temperature on ordinary HF components were not significant. The ordinary HF component contained a fractal (or aperiodic) component. The HF component originates from RSA, which is a periodical fluctuation of the heartbeat that synchronizes with the respiratory rhythm (Berntson et al., 1993; Hayano et al., 1996). Therefore, the following possibility may be considered with respect to the results: aperiodic components of HF masked the alteration of RSA. Certain studies have shown that no significant effects are exerted on ordinary HF components when human subjects are exposed to bright light stimulations at night (Burgess et al., 2001; Tsunoda et al., 2001). The results of this study indicate that relative changes in the fractal-free HF components before and after sleep were more remarkable than in the ordinary HF. The improved signal-to-noise ratio of HF components could provide the significant differences between the different lighting conditions. CGSA may be an appropriate approach for HRV evaluation during sleep, especially with detailed analysis of the HF components.

In summary, pre-sleep exposure to light of a high color temperature may inhibit enhancements of cardiac vagal activity during sleep. Attenuation of vagal control is one of the risk factors in coronary heart disease (Tsuji et al., 1994). The present study was limited by expression of the results in changes relative to the control value, although relative values do reflect intra-individual variations. Further studies to investigate the effects of inter-individual variations on the inhibitions of cardiac vagal control on exposure to light of a high color temperature are warranted.

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