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Light isn't just for vision anymore: implications for transportation safety Part II

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16. Abstract

Humans are a diurnal species, programmed to be awake during the day and asleep at night. Therefore, it is not surprising that sleepiness plays an important role in vehicles accidents. In principle, light can be used to increase alertness at night and thereby possibly reduce sleep-related traffic accidents. We recruited 16 subjects to participate in a within-subjects experiment to investigate whether exposures to low levels of blue light, predicted to reliably stimulate the circadian system, would positively affect alertness and night-time driving performance without significantly decreasing visibility. Subjects participated in the study during the day and at night. During each session, subjects were asked to perform a driving simulator task for 3.5 hours while exposed, in a counterbalanced manner, to two levels of blue light (6.5-8.5 lux and 2.5-4.5 lux at the cornea of a 436 nm light) with a dim red light exposure (<2 lux at the cornea of a 630 nm light) in between. In addition to measuring driving performance (velocity, throttle and steering), we collected saliva samples for melatonin assay, objective alertness (EEG and ECG) and self-report sleepiness (Karolinska Sleepiness Scale). Disability glare calculations were performed. Neither the high nor the low level of blue light suppressed nocturnal melatonin production. Results did not show a significant effect of light on alpha power, beta power and heart rate. Throttle was significantly reduced after exposure to the higher blue light level, but no other effects on driving performance were observed. Subjects felt sleepier at night than during the day, but this increase in subjective sleepiness did not result in poorer driving performance. A higher threshold of the circadian system to shorter wavelengths of light was found. An important next step would be to better understand the retinal mechanisms associated with this lower-than-expected response by the circadian system to shorter-wavelength blue light. It is also recommended that a more formal assessment of the impact of disability glare on driving visibility at night from a longer wavelength blue light (e.g., 470 nm), known to positively impact the circadian system, be made.

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EXECUTIVE SUMMARY

In 1998, nearly 30% of all fatal accidents involving large trucks occurred during hours of darkness, according to the Federal Motor Carrier Safety Administration's *Large Truck Crash Profile: The 1998 National Picture* (*20*). In about 1.5% of crashes involving large trucks, police reported that drivers visibly appeared to be fatigued or very tired (*20*). More than 7% of single-vehicle fatal truck accidents were reported as having driver drowsiness or sleeping as a related factor (*20*). The National Highway Safety Administration (NHSTA) reports that 56,000 automobile accidents per year are caused by drivers falling asleep at the wheel. According to the 1990 *World Almanac* (17), each accident involving a fatality or very serious injury results in a cost of nearly $1.5 million, simply accounting for wage losses, medical expenses and insurance administration.

Humans are a diurnal species, programmed to be awake during the day and asleep at night (21). Therefore, it is not surprising that sleepiness plays an important role in vehicles accidents. The most common preventive action taken by sleepy drivers is to stop driving, change the environment in the vehicle by opening the windows or turning on a loud radio, or consume caffeinated products. Although the preferred preventive action is to stop driving, it is known that this course of action does not always happen due to work demand. In principle, light can be used as a non-pharmacological treatment for increasing alertness at night and thereby possibly reducing sleep-related traffic accidents. Recent research has begun to illustrate the many ways that light and lighting systems affect humans in terms of circadian photobiology, including the characteristics of light necessary to regulate the circadian system.

One major barrier before circadian photobiology can be considered in transportation application is how light for the circadian system can be introduced without significantly increasing disability glare and reducing night-time visual performance. The goal of the present study was to investigate whether exposure to very low levels of blue light, predicted to reliably stimulate the circadian system, can positively affect night-time driving performance without significantly decreasing visibility.

We recruited 16 subjects to participate in a within-subjects experiment. Subjects were asked to participate in the study during the day and at night. During each session, subjects were asked to perform a driving simulator task for 3.5 hours while exposed to two levels of blue light (peak wavelength at 436 nm; 6.5-8.5 lux and 2.5-4.5 lux) with a dim red lighting condition (less than 2 lux at the cornea of a 630 nm light) between each blue light exposure. All lighting conditions were presented to the subjects in a counterbalanced manner. In addition to measuring driving performance, we collected saliva samples for melatonin assay, objective alertness via electroencephalogram (EEG) and subjective sleepiness (Karolinska Sleepiness Scale). Disability glare calculations

were performed to investigate the impact of blue light exposures on visibility. Neither the high nor the low level of blue light suppressed nocturnal melatonin production. Results did not show a significant effect of light on alpha and beta powers, heart rate, and driving simulator performance (velocity, throttle and steering). Subjects reported feeling sleepier at night than during the day, but this increase in subjective sleepiness did not result in poorer driving performance. Results suggest a higher than expected threshold of the circadian system to shorter wavelengths of light, suggesting that the phototransduction mechanisms for this higher-than-predicted threshold for alertness are still unknown.

BACKGROUND

Circadian rhythms are influenced by exogenous factors, the most important of which is the 24-h light/dark cycle serving as the primary synchronizer of the circadian clock to the solar day. The endogenous period of the human circadian system is not exactly 24 hours in most individuals; rather it is slightly longer, 24.2 hours on average (11). This deviation from a 24-hour cycle actually provides a means for the internal clock system to be continuously realigned to the seasonally changing light/dark cycle of the external environment. This continuous adjustment results in greater precision in controlling the timing, or phase of the rhythm, because every day the internal rhythm is 'reset' to the correct solar time. This daily resetting is called entrainment. Light and dark patterns are conveyed from the retina to the suprachiasmatic nuclei (SCN) via the retino-hypothalamic tract (RHT) (22). Depending upon when it is applied in the 24-hour cycle, light can phase advance or phase delay human circadian rhythms (18). Light given in the early subjective night will result in a phase delay, while light applied in the late subjective night will result in a phase advance. In addition to the phase shifting effects on circadian rhythms, light can have an acute effect in human behavior and physiological functions, such as melatonin levels, brain activities, and subjective sleepiness.

Relative to the human visual system, the human circadian system has much higher threshold for activation (26*, 33*) and has a peak spectral sensitivity in shorter wavelengths, close to 450 nm (7*, 24, 25, 28-31*). Thus, properly tuning the spectral power distribution of a light source to those wavelengths maximally effective for the circadian system will lead to significant reductions in the "brightness" of light exposure needed to affect the circadian system. The visual system is served by a fovea that is primarily concerned with fine spatial resolution and by the peripheral retina that largely mediates detection, but not recognition of objects. The circadian system is diffusely distributed across the retina and largely indifferent to light distribution. Most significantly perhaps, the visual system is very fast to respond (fractions of a second) whereas the circadian system requires longer exposure for activation (several minutes) (5). Finally, the circadian system is differentially sensitive to light depending upon the time of day (18), whereas the visual system is capable of processing suprathreshold stimuli with little regard to the time of day.

Performance and circadian rhythms in humans

Alertness and performance are strongly influenced by the timing of the circadian clock. Although performance measures show circadian patterns, different circadian patterns are associated with different levels of mental load (3,14). For example, repetitive tasks show higher performance during the day, but with lower levels in the morning and evening, while short-term memory declines throughout the day (19). In general, however, performance is lowest when minimum core body temperature (MCBT) occurs, about 1.5h prior to normal waking (19). MCBT is used as a marker of the circadian clock. Core body temperature follows a circadian pattern, with peak occurring late afternoon/early evening and trough occurring in the second half of the night (18). It has also been shown that performance is affected by the duration of the time awake (15, *32*). In sleep deprivation studies, performance rapidly decreases during the hours immediately following awakening followed by a gradual leveling out at low levels after 40 to 72 hours of being awake (*1*). Other studies indicate that when sleep duration is less than 7 hours night after night, a steadily growing impairment in performance is observed day after day (*2*).

Impact of light on performance and alertness

It is increasingly clear that relatively brief exposures to light (15 to 30 minutes) can have a positive impact on alertness, wakefulness and performance of certain tasks, even if for only a temporary amount of time, in individuals who are not sleep deprived. Badia and colleagues (4) exposed subjects to 90-min. blocks of alternating bright (5000 to 10,000 lx) and dim (50 lx) light during day-time and night-time hours. Body temperature, alertness [measured using electrophysiological brain activity measure by electroencephalogram (EEG)] and performance were higher after exposure to bright light than after exposure to dim light during the night-time hours, but not during day-time hours. Campbell and Dawson (10) exposed subjects to bright (1000 lx) and dim (10 and 100 lx) ambient light for 8 h at night. Subjective and objective measures of alertness were higher after exposure to bright light than after exposure to dim light.

Boyce and colleagues (6) submitted subjects undergoing a night shift routine over three successive nights to four different lighting conditions (using white light): low (250 lx), high (2800 lx), increasing (from 200 to 2800 lux over an 8-h shift), and decreasing (from 2800 to 200 lx over an 8-h shift). They found that the two "early bright light" conditions (high and decreasing) improved performance of certain types of tasks, increased level of arousal, improved quality of sleep as the number of nights on shift work increased, and delayed the time the subjects went to bed after the night shift. Figueiro and colleagues (*12*) exposed night-shift nurses working in a newborn intensive care unit to 15 min of bright white light (at least 500 lx at the eye) or dim light (less than 100 lx at the eye). They showed that subjective feelings of wakefulness, alertness, and overall well-being were improved after brief periodic exposures to bright white light.

Cajochen and colleagues (8) exposed subjects to illuminances ranging from 3 to 9100 lx of a white light for 6.5 h during the early night. They found an acute alerting response to light as assessed by EEG, as well as a reduction in self-reported sleepiness. More

recently, Cajochen and colleagues (9), building on knowledge that light has an alerting effect at night and on the knowledge that the circadian system is maximally sensitive to short-wavelength radiation (blue light), were able to show that much lower irradiances of monochromatic short-wavelength (blue) light (5 lx of blue light at a peak wavelength of 460 nm for a duration of about 40 min.) increased objective and subjective alertness.

More recently, Figueiro and colleagues (13) demonstrated that subjective (Norris Scale) and objective (EEG) measures of alertness are highly correlated and both measures increase monotonically with four blue (peak wavelength at 470 nm) light levels (5, 10, 20 and 40 lx at the cornea). Moreover, they demonstrated that objective measures of alertness were highly correlated with predictions of melatonin suppression for the same circadian stimulus (CS) calculated using a mathematical model of human circadian phototransduction (*27*), suggesting that the SCN play a role in light's alerting effects in humans.

GOALS OF THE PROJECT

This project was concerned with the acute effects of light on night-time alertness. We proposed to determine if low illuminance levels of blue light at the cornea could be used as a non-pharmacological tool to increase alertness and reduce sleepiness during simulated driving at night. The plan was to employ a short-wavelength light source that was an effective stimulus for the circadian system but simultaneously a minimally effective disability glare source. Based on circadian stimulus and disability glare calculations, a filtered clear mercury source with a peak emission at 436 nm was chosen to provide two levels of illuminance, 6.5-8.5 and 2.5-4.5 lux, at the cornea.

METHODOLOGY

Light Source

In order to select the optimum light source for the proposed study, it was necessary to select a source that maximized the magnitude of the circadian stimulus while minimizing its photopic illuminance. The magnitude of the circadian stimulus (CS) calculator was determined from the mathematical model of human circadian phototransduction developed by Rea and colleagues (*27*). Four spectral sensitivity functions are used in the model: the scotopic luminous efficiency function, V'(λ) (38), based on rod sensitivity, V10(λ) based upon the S, M and L cone fundamentals (34), the S-cone fundamental (34), and a standard photo-opsin emulating melanopsin contained within the intrinsically photosensitive retinal ganglion cell (ipRGC; (6)), and having a peak spectral response at 480 nm and a half-bandwidth of 95 nm. Briefly, in the model the cone fundamentals form a spectrally opponent (blue vs. yellow)(39) input to the ipRGC which sends circadian light signals to the SCN. The modeled rod response suppresses output from the ipRGC when the blue-yellow opponent signal is positive, with diminishing suppression at higher irradiance levels as rods become more fully bleached. A negative blue-yellow opponent

signal, however, produces a response determined solely by the ipRGC (no S- or L+M-cone response) and eliminates input and no rod suppression). The calculator determines CS based on the circadian phototransduction mechanisms discussed above, the spectral power distribution (SPD) of the light source and the light level at the cornea.

Figueiro and colleagues (40) showed that one hour exposure to 18 lx at the cornea of an LED peaking at 470 nm resulted in 35% melatonin suppression. Calculations were made to determine whether photopic illuminance levels could be reduced if a light source peaking in shorter wavelengths was used to stimulate the circadian system. These calculations suggested that the shorter the peak wavelength, the lower the photopic light levels needed to achieve the same criterion response (i.e., 35% melatonin suppression) for the circadian system. Largely for practical reasons, a filtered clear mercury vapor (Hg) light source peaking at 436 nm was chosen for the study; the SPD of the unfiltered 175W Hg source (General Electric, HR175A39) is shown in Figure 1.

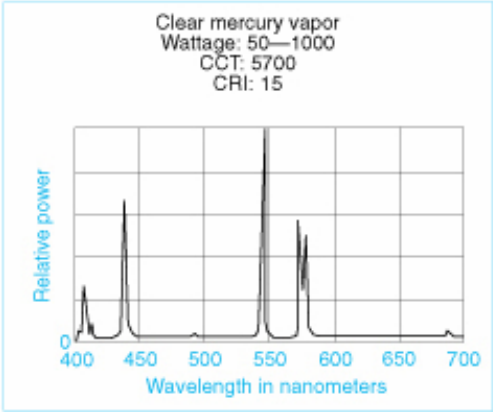

Figure 1: SPD of the unfiltered light source used in the experiment

Lighting Equipment

Two Hg lamps were placed inside wooden housings that were built for the experiment (Figure 2). Vent holes were provided on the top of each of the wooden housing to avoid overheating the lamps. An aluminum plate was screwed under the vents using a ¼ inch spacer to avoid any light leakage through the vent holes while still maintaining a convective air circulation within the housing. There was an opening in front of the wooden housing where aluminum extrusions were mounted along the front edges. Four sliding windows were built from black foam board. A filter (Manufacturer: Lee Filters, # 120) was attached to sliding windows to filter out the longer wavelengths, so that subjects were exposed to only the 436 nm wavelength. Figure 3 shows the SPD of the filtered Hg light source. Two of the sliding windows had a smaller aperture and they were used to reduce light output from the box, reducing the light reaching the subjects' corneas. The inner surface of the box was painted white and a matt aluminum reflector was mounted behind the lamps to increase the overall efficiency of the light box. The wooden housing was supported by two wooden legs. The housing could slide up and down along the legs

to allow for fine adjustment of the light levels. The control gears of the two lamps were mounted on one of the legs.

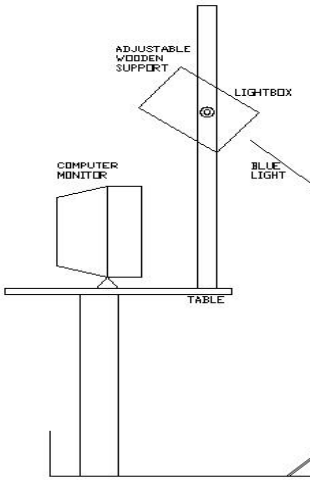

Figure 2: Schematic of the light boxes built for the experiments.

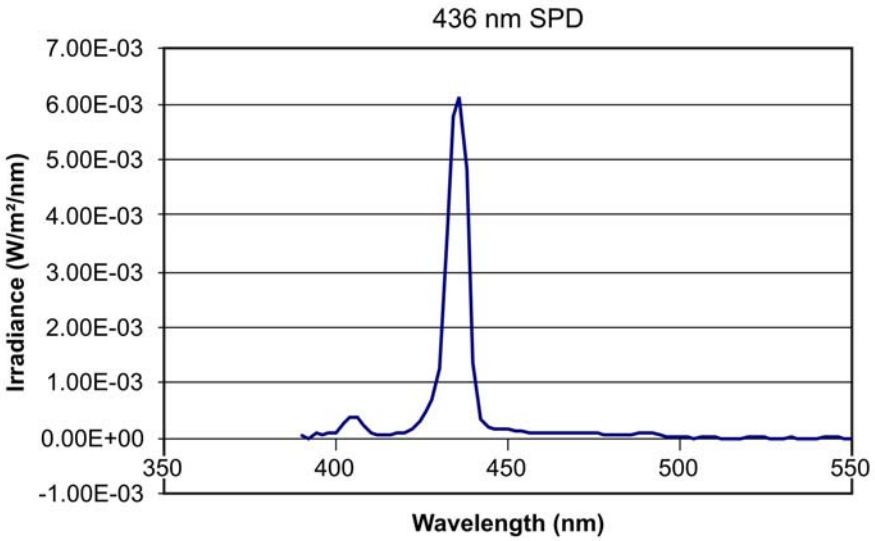

Figure 3: SPD of the filtered light source used in the experiment

Subjects were exposed to two levels of blue light (B1 and B2) and a dim red light in between blue light exposures (the red light was less than 2 lux at the cornea of a light emitting diode (LED) peaking at 630 nm). Although the dim red light was always of the same irradiance, it was referred to D1 when exposure was just prior or just after B1 and D2 when exposure was just prior of just after exposure to B2. The two blue light levels shown in Table 1 were determined based on *a priori* predictions of melatonin suppression using calculations from the model of human circadian phototransduction by Rea and

colleagues (27). As shown in Table 1, CS values needed to achieve target melatonin suppression levels were determined. The values of CS were scaled so that 1000 lux of CIE Illuminant A (an incandescent blackbody radiator at 2856 K) is equivalent to 1000 circadian light units (CLA). The human circadian system response to light (i.e., CLA), as measured by acute nocturnal melatonin suppression follows a logistic function (33). This response function was used to transform the CLA values into circadian stimulus values (CSA). CSA is considered to be a better measure of the effectiveness of the light stimulus for the human circadian system because it is defined in terms of the circadian system's input-output relationship with light, including both threshold and saturation.

Luminance, L (cd/sq.m)	Light condition	Target Illuminance (lux)	CLA value	CSA value	Predicted melatonin suppression (%)
2.00	High (B2)	6.5	416	519	38
0.85	Low (B1)	2.5	140	175	21

Table 1. Photometric and calculated light levels used in the experiment.

Once the set up was completed, a CCD spectrometer (Photo research Inc., PR-705.) was used to verify the SPD and the irradiance of the blue light from the light box. The CCD spectrometer was calibrated at the Lighting Research Center (LRC) using the National Institute of Standards and Technology (NIST) calibrated standards of luminance and spectral irradiance. The calibrated white reflectance standard (dimension: 5 2/8inch x 5 2/8 inch; Labsphere, model SR 099) used had a reflectance of 98%. The spectroradiometer and the diffuse white reflectance standard were used to calibrate the Gigahertz illuminance meter's readings. The Gigahertz illuminance meter was used to verify the light levels subjects were being exposed to during the experiment. The range of measured illuminance during the experiment was between 6.5 to 8.5 lux at the cornea for B2 and between 2.5 and 4.5 lux at the cornea for B1. The variation in light levels was due to subjects positioning while performing the driving task. The experimenter was instructed to monitor subjects' movements during the experiment to reduce this variability as much as possible.

Glare Calculations

A two-step process was used to calculate disability glare. First, the veiling luminance was calculated using the following formula proposed by Fry (16):

$$L_v = 9.2 \times \sum_{i=1}^{n} \frac{E_i}{\theta_i \times (\theta_i + 1.5)} \qquad \text{(Equation 1)}$$

Where L_v is the veiling photopic luminance (in cd/m²),
E_i is the photopic illuminance at the eye from the i^{th} light source (in lx),
θ_i is the visual angle for the i^{th} light source (in degrees).

For the purpose of this calculation, E_i was considered a constant value for a stimulus specification. Since light levels at the cornea varied slightly, an illuminance of 8 lux was used in the calculations. This light level was selected because, although the target light levels were 6.5 lux, the range of light levels that subjects were actually exposed to was between 6.5 and 8.5 lux, so 8 lux would be close to the higher range. Theta is the visual angle for the light source and is directly proportional to the vertical distance between the position of the eye and the light box, while inversely proportional to the horizontal distance between the eye and the light box. In this experiment, the vertical distance was 13 inches and the horizontal distance was 15 inches; thus, theta was 41 degrees. The Lv was found to be 0.042 cd/m^2.

Once the veiling luminance was determined, the impact of disability glare (or veiling luminance) on visual performance was determined using the definition from the Illuminating Engineering Society of North America (23). According to the IESNA, disability glare can be predicted by comparing the luminance of the target with reference to its background or by using the following formula:

$$\text{Visual task contrast (VTC)} = \frac{L_t - L_b}{L_b + L_v} \qquad \text{(Equation 2)}$$

Where L_t is target luminance,
L_b is background luminance
L_v is veiling luminance

For the purpose of our calculations, the luminance of the target on the road was assumed to be 0.2 cd/m^2. A reasonable background luminance for nighttime driving in rural locations is 0.1 cd/m^2. The veiling luminance calculated above is 0.042 cd/m^2
Using the formula above, the VTC was 70.30%, suggesting that 8 lux at the cornea would result in a 30% reduction in VTC.

The same glare calculations were performed using an illuminance of 4 lux. Again, although the target light levels were 2.5 lux, subjects were exposed to a range that varied from 2.5 to 4.5. The VTC was 83%, suggesting that 4 lux at the cornea would result in a 17% reduction in VTC.

The driving simulator

The driving simulators were setup at the LRC, as shown in Figure 4 below, to accommodate four subjects at a time. The driving simulator comprised of a desktop computer system with a 20 inch CRT monitor connected to the computer, a mouse and keyboard. To simulate driving conditions, a driving kit (Thrust master) was connected to the computer. The driving kit included a steering wheel with gear shift controls, a brake pedal and an accelerator pedal.

The driving simulator software (RACER 0.5.7) was installed in the computer. RACER was freely downloaded from the internet and is a robust program using open source architecture. In order to ensure that the subjects had different driving experiences during testing, two tracks named, CAMPAGNE (CN) and HIGHWAY (HW) were selected for the study. The CN track provided a visually appealing graphical environment with frequent turns and narrow roads. The HW track was selected to mimic a highway driving condition and provided a dull and boring driving environment to the subject (Figure 5).

Figure 4: Driving simulator with subjects separated by a divider in the centre. The Left side subject is exposed to B2 while the right side subject is exposed to B1 light condition.

Figure 5: Left: Highway (HW) track; Right: Campagne (CN) track

The driving simulator was programmed to automatically record all driver responses and events including steering (degrees turned), throttle (% open, where foot off of pedal = 0, foot all the way on pedal = 1), velocity (meters per second), and braking (% open, where foot off of breaks = 0, foot all the way on the brakes = 1).

EEG Data Collection

The Biosemi ActiveTwo system with active electrodes was used for EEG recordings. This system is battery powered, minimizing electrical interference from alternating current (ac) during recording sessions. Electrodes were placed on subjects' scalps according to the International 10-20 system at Oz, Pz, Cz, and Fz (42). Two additional electrodes serving as virtual reference electrodes for those attached to the scalp were attached to the right and to the left earlobes. Another electrode was placed approximately 5 cm below the left clavicle to measure an electrocardiogram (ECG) signal.

Saliva Sample Collection

Saliva collection was done using the Salivette system from Alpco Diagnostics. This system consists of a centrifuge vessel with a suspended insert in which a cotton swab is placed. To collect the saliva the cap was removed and then a subject put the tube against their lips and took the cotton swab into their mouth without ever touching it with their hands. The subject then chewed the swab to impregnate it with saliva. Depending on the subject, this can take between 30 seconds and 2 minutes. Between 1-2 ml of saliva was required for the analyses. After the subject was done chewing they then spit the cotton back into the suspended insert and the cap on the tube was replaced.

After the saliva sample was collected the tubes were placed in a centrifuge and spun at 3,500 RPM for 5 minutes. This causes the saliva to come out of the cotton swab and to collect in the bottom of the centrifuge vessel. When the tubes were finished being spun the suspended insert was removed and thrown away and the cap was replaced on the salivette tube. At this point the tubes were frozen for transport to a laboratory for melatonin assays.

Subjective ratings

The 9-point Karolinska Sleepiness Scale (KSS) was used to probe self-report of sleepiness. A score of 1 denoted extremely alert, 5 denoted neither alert nor sleepy and 9 denoted very sleepy with great effort to keep awake.

Subjects

Sixteen subjects between the age of 18 and 50 (nine male and seven female), who possessed a valid driver's license and had experience driving were recruited for the study. Two female subjects were not able to complete the protocol due to motion sickness during the experiment. Interested candidates were asked to complete the Munich Chronotype Questionnaire and selection of the subjects was based on their answers. As a part of the experimental protocol we chose candidates of the moderate early type who would regularly go to bed before 01:00. Subjects with major health problems, such as cardiovascular disease, diabetes, or high blood pressure, or subjects taking over the counter melatonin or any prescription medication, such as blood pressure medicine, antidepressants, sleep medicine, hormone replacement therapy or beta blockers were

excluded from the study. Subjects were asked to keep a regular schedule on the day/night before the experiment and were not allowed to drink caffeine on the day of the experiment.

Procedures

All experimental sessions took place from November 2008 to March 2009. The protocol was approved the Rensselaer Polytechnic Institute Review Board (IRB). At the start of the experiment, the protocol was explained to participants and informed consent was obtained from subjects. As shown in Table 2, each group participated in one daytime and one nighttime session, separated by at least one week. Table 2 details the lighting conditions as well as the order of the presentation of the track conditions that each subject experienced in each trial. Within the same session, every subject was presented all lighting conditions (B1, B2, and a dim red light in between each blue light exposure) in a counterbalanced manner. During each 50-minute trial, subjects experienced both course types (CN and HW) for 25 minutes each. The counterbalancing was performed to counteract the fatigue and practice effect. In addition, the first trial of each session was performed in dim red light and considered a practice trial and was not included in the analyses of the results.

Table 2: Experimental design: CN = Campagne course; HW – Highway course; B1 – low level of blue light; B2 – High level of blue light; D1 – dim red light prior to or after B1; D2 – dim red light prior to or after B2.

	Day time Testing - Nov 12th, 2008									
Subject-1	**Dim (Practice)**		**B1**		**D1**		**B2**		**D2**	
	11:00	11:25	12:00	12:25	13:00	13:25	14:00	14:25	15:00	15:25
	CN	HW	CN	HW	CN	HW	CN	HW	CN	HW
Subject-2	**Dim (Practice)**		**D1**		**B1**		**D2**		**B2**	
	11:05	11:30	12:05	12:30	13:05	13:30	14:05	14:30	15:05	15:30
	CN	HW	CN	HW	CN	HW	CN	HW	CN	HW
Subject-3	**Dim (Practice)**		**B1**		**D1**		**B2**		**D2**	
	11:10	11:35	12:10	12:35	13:10	13:35	14:10	14:35	15:10	15:35
	HW	CN	HW	CN	HW	CN	HW	CN	HW	CN
Subject-4	**Dim (Practice)**		**D1**		**B1**		**D2**		**B2**	
	11:15	11:40	12:15	12:40	13:15	13:40	14:15	14:40	15:15	15:40
	HW	CN	HW	CN	HW	CN	HW	CN	HW	CN

	Night time testing - Nov 18th,2008									
Subject-1	**Dim (Practice)**		**B1**		**D1**		**B2**		**D2**	
	23:00	23:25	0:00	0:25	1:00	1:25	2:00	2:25	3:00	3:25
	CN	HW	CN	HW	CN	HW	CN	HW	CN	HW
Subject-2	**Dim (Practice)**		**D1**		**B1**		**D2**		**B2**	
	23:05	23:30	0:05	0:30	1:05	1:30	2:05	2:30	3:05	3:30
	CN	HW	CN	HW	CN	HW	CN	HW	CN	HW
Subject-3	**Dim (Practice)**		**B2**		**D2**		**B1**		**D1**	
	23:10	23:35	0:10	0:35	1:10	1:35	2:10	2:35	3:10	3:35
	HW	CN	HW	CN	HW	CN	HW	CN	HW	CN
Subject-4	**Dim (Practice)**		**D2**		**B2**		**D1**		**B1**	
	23:15	23:40	0:15	0:40	1:15	1:40	2:15	2:40	3:15	3:40
	HW	CN	HW	CN	HW	CN	HW	CN	HW	CN
	Day time Testing-Dec 10th, 2008									
Subject-5	**Dim (Practice)**		**B2**		**D2**		**B1**		**D1**	
	11:00	11:25	12:00	12:25	13:00	13:25	14:00	14:25	15:00	15:25
	CN	HW	CN	HW	CN	HW	CN	HW	CN	HW
Subject-6	**Dim (Practice)**		**D2**		**B2**		**D1**		**B1**	
	11:05	11:30	12:05	12:30	13:05	13:30	14:05	14:30	15:05	15:30
	CN	HW	CN	HW	CN	HW	CN	HW	CN	HW
Subject-7	**Dim (Practice)**		**B2**		**D2**		**B1**		**D1**	
	11:10	11:35	12:10	12:35	13:10	13:35	14:10	14:35	15:10	15:35
	HW	CN	HW	CN	HW	CN	HW	CN	HW	CN
Subject-8	**Dim (Practice)**		**D2**		**B2**		**D1**		**B1**	
	11:15	11:40	12:15	12:40	13:15	13:40	14:15	14:40	15:15	15:40
	HW	CN	HW	CN	HW	CN	HW	CN	HW	CN
	Night time testing - Dec16th, 2008									
Subject-5	**Dim (Practice)**		**B2**		**D2**		**B1**		**D1**	
	23:00	23:25	0:00	0:25	1:00	1:25	2:00	2:25	3:00	3:25
	CN	HW	CN	HW	CN	HW	CN	HW	CN	HW
Subject-6	**Dim (Practice)**		**D2**		**B2**		**D1**		**B1**	
	23:05	23:30	0:05	0:30	1:05	1:30	2:05	2:30	3:05	3:30
	CN	HW	CN	HW	CN	HW	CN	HW	CN	HW
Subject-7	**Dim (Practice)**		**B1**		**D1**		**B2**		**D2**	
	23:10	23:35	0:10	0:35	1:10	1:35	2:10	2:35	3:10	3:35
	HW	CN	HW	CN	HW	CN	HW	CN	HW	CN
Subject-8	**Dim (Practice)**		**D1**		**B1**		**D2**		**B2**	
	23:15	23:40	0:15	0:40	1:15	1:40	2:15	2:40	3:15	3:40
	HW	CN	HW	CN	HW	CN	HW	CN	HW	CN

	Day time Testing - Mar 4th, 2009									
Subject-9	**Dim (Practice)**		**B1**		**D1**		**B2**		**D2**	
	11:00	11:25	12:00	12:25	13:00	13:25	14:00	14:25	15:00	15:25
	CN	HW	CN	HW	CN	HW	CN	HW	CN	HW
Subject-10	**Dim (Practice)**		**D1**		**B1**		**D2**		**B2**	
	11:05	11:30	12:05	12:30	13:05	13:30	14:05	14:30	15:05	15:30
	CN	HW	CN	HW	CN	HW	CN	HW	CN	HW
Subject-11	**Dim (Practice)**		**B1**		**D1**		**B2**		**D2**	
	11:10	11:35	12:10	12:35	13:10	13:35	14:10	14:35	15:10	15:35
	HW	CN	HW	CN	HW	CN	HW	CN	HW	CN
Subject-12	**Dim (Practice)**		**D1**		**B1**		**D2**		**B2**	
	11:15	11:40	12:15	12:40	13:15	13:40	14:15	14:40	15:15	15:40
	HW	CN	HW	CN	HW	CN	HW	CN	HW	CN
	Night time testing - Mar 10th, 2009									
Subject-9	**Dim (Practice)**		**B1**		**D1**		**B2**		**D2**	
	23:00	23:25	0:00	0:25	1:00	1:25	2:00	2:25	3:00	3:25
	CN	HW	CN	HW	CN	HW	CN	HW	CN	HW
Subject-10	**Dim (Practice)**		**D1**		**B1**		**D2**		**B2**	
	23:05	23:30	0:05	0:30	1:05	1:30	2:05	2:30	3:05	3:30
	CN	HW	CN	HW	CN	HW	CN	HW	CN	HW
Subject-11	**Dim (Practice)**		**B2**		**D2**		**B1**		**D1**	
	23:10	23:35	0:10	0:35	1:10	1:35	2:10	2:35	3:10	3:35
	HW	CN	HW	CN	HW	CN	HW	CN	HW	CN
Subject-12	**Dim (Practice)**		**D2**		**B2**		**D1**		**B1**	
	23:15	23:40	0:15	0:40	1:15	1:40	2:15	2:40	3:15	3:40
	HW	CN	HW	CN	HW	CN	HW	CN	HW	CN

	Day time Testing-Mar 25th, 2009									
Subject-13	**Dim (Practice)**		**B2**		**D2**		**B1**		**D1**	
	11:00	11:25	12:00	12:25	13:00	13:25	14:00	14:25	15:00	15:25
	CN	HW	CN	HW	CN	HW	CN	HW	CN	HW
Subject-14	**Dim (Practice)**		**D2**		**B2**		**D1**		**B1**	
	11:05	11:30	12:05	12:30	13:05	13:30	14:05	14:30	15:05	15:30
	CN	HW	CN	HW	CN	HW	CN	HW	CN	HW
Subject-15	**Dim (Practice)**		**B2**		**D2**		**B1**		**D1**	
	11:10	11:35	12:10	12:35	13:10	13:35	14:10	14:35	15:10	15:35
	HW	CN	HW	CN	HW	CN	HW	CN	HW	CN
Subject-16	**Dim (Practice)**		**D2**		**B2**		**D1**		**B1**	
	11:15	11:40	12:15	12:40	13:15	13:40	14:15	14:40	15:15	15:40
	HW	CN	HW	CN	HW	CN	HW	CN	HW	CN
	Night time testing - Mar 18th, 2009									
Subject-13	**Dim (Practice)**		**B2**		**D2**		**B1**		**D1**	
	23:00	23:25	0:00	0:25	1:00	1:25	2:00	2:25	3:00	3:25
	CN	HW	CN	HW	CN	HW	CN	HW	CN	HW
Subject-14	**Dim (Practice)**		**D2**		**B2**		**D1**		**B1**	
	23:05	23:30	0:05	0:30	1:05	1:30	2:05	2:30	3:05	3:30
	CN	HW	CN	HW	CN	HW	CN	HW	CN	HW
Subject-15	**Dim (Practice)**		**B1**		**D1**		**B2**		**D2**	
	23:10	23:35	0:10	0:35	1:10	1:35	2:10	2:35	3:10	3:35
	HW	CN	HW	CN	HW	CN	HW	CN	HW	CN
Subject-16	**Dim (Practice)**		**D1**		**B1**		**D2**		**B2**	
	23:15	23:40	0:15	0:40	1:15	1:40	2:15	2:40	3:15	3:40
	HW	CN	HW	CN	HW	CN	HW	CN	HW	CN

The day session for each group began at 11:00 and continued until 15:40 while the night sessions began at 23:30 and ended at 03:40. Each group consisted of four subjects. The subjects arrived at the LRC 45 minutes prior to the start of the session. They were seated comfortably and EEG electrodes were placed on their scalp according to the International 10-20 system. Electrodes EXG1 and EXG2 were affixed to the right and left earlobes respectively to act as references for the scalp signals. EXG3 through EXG6 were affixed at Oz, Pz, Cz, and Fz on the scalp. EXG7 was placed approximately 1-2cm behind the right eye, and EXG8 was placed approximately 5cm below the left clavicle to measure an electrocardiogram (ECG) signal.

Near the end of each dim and each blue light exposure period, the scalp electrodes on each subject were attached to the EEG recording system. Three minutes of continuous data were collected. The subjects were asked to fixate their opened eyes on a specific marked point on the monitor. Subjects were visually monitored by an experimenter to ensure compliance with the protocol. As soon as the EEG data collection was completed each subject was asked to chew on a cotton salivette for the saliva sample and complete the subjective sleepiness assessment (KSS). A five minute break was provided to each subject at the completion of each trial.

During the experiment, one of the experimenters monitored the light level at the eye of each subject using the calibrated photometer to ensure that they received the targeted amount of blue light (B1 or B2). The experimenters also manually adjusted the window shields over the luminaire to alternate between the lighting conditions (B1, B2 and dim) using the window light blocking shield. For the dim light conditions, a windowless light blocking shield (made of craft paper) was pulled over the blue window shield to completely block out the light at the desired cubicle.

The RACER driving simulator automatically saved the driving history of each subject. After each experiment session, the simulator data were retrieved from the computer and analyzed as detailed below.

RESULTS

Saliva melatonin

Frozen plasma samples were sent to an independent laboratory for melatonin radioimmunoassay (Pharmascan, Osceola, WI). Melatonin levels were expected to be low during the daytime and high during the nighttime sessions. Melatonin levels after blue light exposures at night were expected to be lower than after dim red light exposures. A two (time of day) by four (lighting conditions) analyses of variance (ANOVA) was conducted to determine whether melatonin levels were significantly lower after exposures to the two levels blue light compared to the dim red light at night. As expected, the ANOVA showed a significant main effect of time of day ($F_{1,12} = 36.8$; $p<0.0001$). As expected, melatonin levels during the daytime were significantly lower than during the nighttime. Unexpectedly, the ANOVAs showed no significant main effect of lighting conditions or a significant interaction between time of day and lighting conditions, suggesting that the blue light stimulus was not strong enough to suppress the natural rise of melatonin during the nighttime.

Percent melatonin suppression was calculated comparing melatonin levels just prior to (or just after) light exposure to melatonin levels just after (or just prior to) light exposure. Melatonin suppression was only calculated for the nighttime data because melatonin levels are already low during the daytime and there should be no suppression by light. Melatonin suppression after exposure to B1 was 8% while suppression after exposure to B2 was only 1%. These results were not consistent with predictions from the model (see Table 1), which indicated that melatonin suppression after exposure to B2 would be on average 38% and 21% after exposure to B1. Possible reasons for this discrepancy will be discussed later in this report.

In order to reduce subject variability, the data were normalized by multiplying each subject's data set to a normalizing factor. The normalizing factor was determined for each subject by using the ratio of the average of all subjects' data set to the average of each subject's data set. Each subject's original data set was then multiplied by the ratio determined using their own data set. Again, the two by four ANOVA revealed only a significant main effect of time of day ($F_{1,13} = 72.9$; $p<0.0001$). There was no statistical

main effect of lighting conditions or a significant interaction between time of day and lighting conditions.

EEG and ECG

The EEG signals were sampled at 16384 Hz and then low-pass filtered and downsampled to 2048 Hz for electronic storage by the Biosemi system. All subsequent EEG data processing and analyses were performed with Matlab, version R2008a by The Mathworks^{TM}. The signals recorded from the two reference channels were averaged and these values were subtracted from those obtained from all of the other channels. The direct current (dc) offset of each channel was eliminated by subtracting the mean value of each channel from itself. A low-pass finite impulse response (FIR) filter (f 3dB = 50 Hz) was applied and the data were downsampled to 512 Hz. Then a high-pass, 3rd order Butterworth filter (f 3dB = 4 Hz) was applied to the downsampled signals from each channel to eliminate slow trending in the data.

Another program divided the filtered data into 5-second epochs. Eye blink artifacts were eliminated by removing epochs from all channels where voltage fluctuations of any epoch exceeded +/- 100 μV. A Blackman window followed by a fast Fourier transform (FFT) was then applied to the data segments. This process yielded spectral power distributions from 1 to 50 Hz. The power spectra for each one-minute segment were then combined to give an average spectral power distribution for each 3-minute trial. The relative power levels for the three minutes in the alpha (8-12 Hz), beta (12-30 Hz), gamma (30-50 Hz), theta (5-7 Hz), and alpha-theta (5-9 Hz) ranges were calculated as a percentage of overall power from 1-50 Hz. Reported here are the results for alpha and beta frequencies.

The ECG data were digitally processed the same way as the EEG data up to the high-pass filtering. For the ECG analysis, the high-pass filtering -3dB cut-off was lowered to 0.2 Hz. Heart rates corresponding to the filtered ECG data were determined by two methods: 1) by taking the FFT of the ECG, whereby the frequency having the peak power within the range from 40 to 120 beats/minute is the heart rate, and 2) determining the elapsed time between the QRS complexes (the successive peaks, Q then R then S, in the ECG signal) of the ECG. The QRS complexes were located by the first derivative of the ECG falling below a negative threshold value after individual normalization of first derivative of the ECG.

A two (time of day) by four (lighting conditions) by four (channels) ANOVA was performed on alpha and beta frequencies. If alertness increased after blue light exposure, it was expected that alpha would be reduced and beta would be increased after exposure to blue light compared to dim light. There was a significant main effect of channel for alpha ($F_{3,13} = 29.86$, $p<0.0001$) and beta ($F_{3,13} = 3.47$, $p<0.0025$). There was no significant main effect of time of day, lighting conditions or a significant interaction between the variables for either alpha and beta frequencies.

In order to reduce subject variability, the data from every subject were normalized with respect to the grand mean. The data from each subject were normalized by the ratio of the mean for that subject to the overall mean. The effectiveness of each level of blue light compared to dim light was based upon the difference between B1 and D1 and between B2 and D2 using the normalized values for each subject. This difference was calculated to determine whether there was an increase in beta and a decrease in alpha after blue light exposure compared to dim light exposure. Again, a two (time of day) by two (lighting conditions) by four (channels) ANOVA was performed. The ANOVA revealed only a significant main effect of time of day ($F_{1,13} = 5.4$; $p = 0.037$) for beta power (Figure 6). A Student's one tail paired t-test revealed a significant higher ($p = 0.0002$) beta power during the daytime than at night. There was no statistical main effect of lighting conditions or a significant interaction between time of day and lighting conditions for beta or alpha power frequencies.

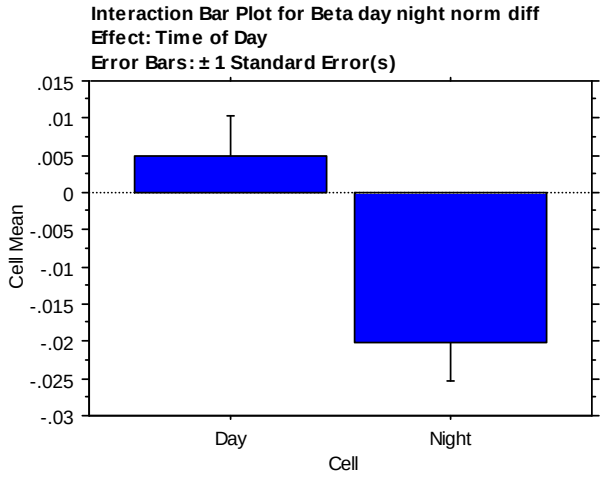

Figure 6: Main effect of time of day for beta power difference (B1-D1 and B2-D2). Beta power was significantly higher during the day than at night.

A two (time of day) by four (lighting conditions) ANOVA was performed on the two measures of heart rate (FFT and QRS). Although heart rate was higher during the day than at night as well as after blue light exposure compared to dim light, there were no significant main effects or interactions between variable for either dependent measure (FFT and QRS). The data were again normalized to reduce subject variability and the differences between B1 and D1 and between B2 and D2 were calculated. Although not significantly different, QRS heart rate difference was greater for B2-D2 than for B1-D1 ($F_{1,13} = 2.3$; $p = 0.15$), suggesting a greater increase in heart rate after exposure to B2 (Figure 7). A post-hoc one tail paired t-test revealed an almost significant difference ($p = 0.056$) between B2-D2 and B1-D1.

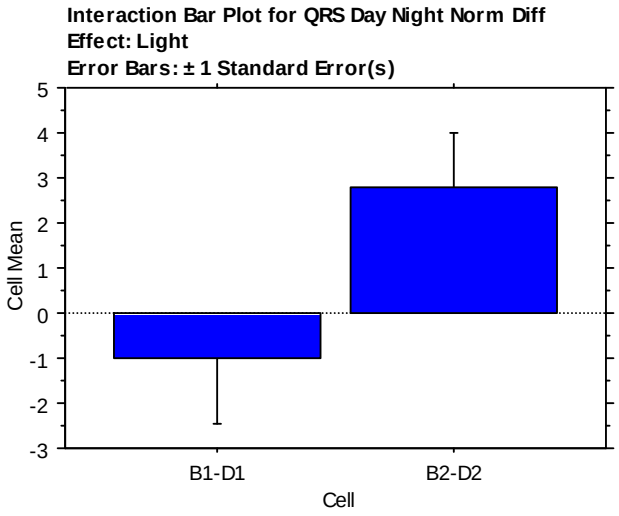

Figure 7: QRS normalized difference. Heart rate difference was greater after exposure to B2 than after dim light. A positive number suggests an increase in heart rate afterB2 than after D2.

Driving performance

The following measures were obtained from the driving simulator data: 1) velocity, which measured the average speed (meters/sec) and the average standard deviation speed during the 25-minute period they drove in each course type (CN or HW); 2) average steering and the average standard deviation steering, measured in degrees, which is the change from a vertical position towards the left or right, and 3) average throttle and the average standard deviation throttle, which is the percentage pressure on pedal (foot off = 0%, foot all the way on pedal = 100%). All of the analyses were performed using with Matlab, version R2008a by The Mathworks^{TM}.

A two (time of day) by two (course type) by four (lighting conditions) ANOVA was performed for velocity (average and standard deviation), steering (average and standard deviation), and throttle (average and standard deviation). There was a significant main effect for course type for average velocity ($F_{1,13} = 120.9$; $p < 0.0001$), stdev velocity ($F_{1,13} = 22.14$; $p = 0.0004$), average steering ($F_{1,13} = 95.1$; $p < 0.0001$), stdev steering ($F_{1,13} = 105.9$; $p < 0.0001$), average throttle ($F_{1,13} = 31.9$; $p < 0.0001$), and stdev throttle ($F_{1,13} = 55.7$, $p < 0.0001$). Average velocity and throttle were higher when subjects were performing the driving task in the HW course than in the CN course, while average steering was greater in the CN than in the HW course. There was no statistical significant main effect of lighting condition or a main interaction between the measures, although an almost statistical significant main effect of lighting condition was found for throttle ($F_{3,13} = 2.8$, $p = 0.053$). A paired one tail t-test revealed that throttle during exposure to B2 was significantly lower ($p = 0.032$) than after exposure to D2. Although not statistically

significant, there was a decrease in average and standard deviation of steering after exposure to B2 compared to all other lighting conditions.
In order to reduce subject variability, the data for each subject were normalized as they were for the EEG data and the differences between driving performance (velocity, steering and throttle) after B1 and D1 and after B2 and D2 were calculated for each subject using the normalized values. A two (time by day) by two (lighting conditions, B1-D1 and B2-D2) by two (course type) ANOVA was performed for each driving performance measures. There was a significant main effect of course type ($F_{1,13} = 4.9$; $p = 0.046$) for throttle. Throttle difference between blue light and dim light exposures was greater for the CN course than for the HW course. An almost significant main effect of lighting conditions ($F_{1,13} = 3.3$; $p = 0.09$) was found. A one-tail paired t-test showed a significant difference between B1-D1 and B2-D2 ($p = 0.035$). The negative difference found for B2-D2 suggests a reduced throttle after exposure to B2, which did not happen after exposure to B1 (Figure 8).

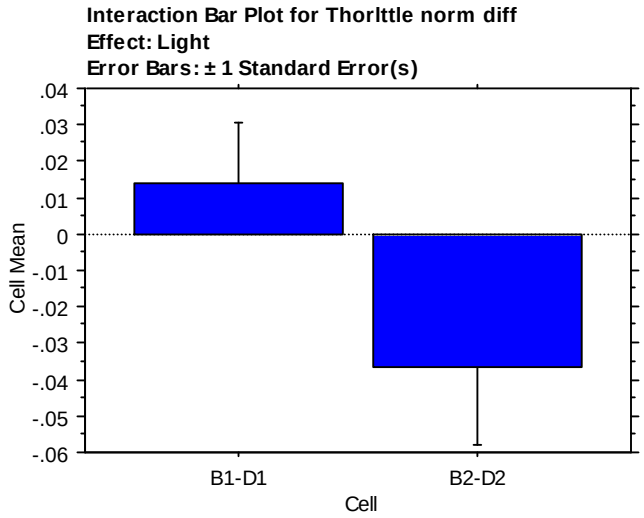

Figure 8: Normalized throttle difference. A negative number suggests that throttle was reduced after exposure to B2 than after D2.

Subjective Sleepiness (KSS)

Subjective sleepiness scores were submitted to a two (time of day) by four (lighting conditions) ANOVA. There was a significant main effect of time of day ($F_{1,13} = 9.7$, $p = 0.008$). As expected, a paired one tail t-test revealed significantly ($p < 0.0001$) higher sleepiness score during the nighttime than during the daytime (Figure 9). There was no main effect of lighting condition or interaction between time of day and lighting conditions. Although not statistically significant, self-reports of sleepiness were lower after dim light exposures than after blue light exposures.

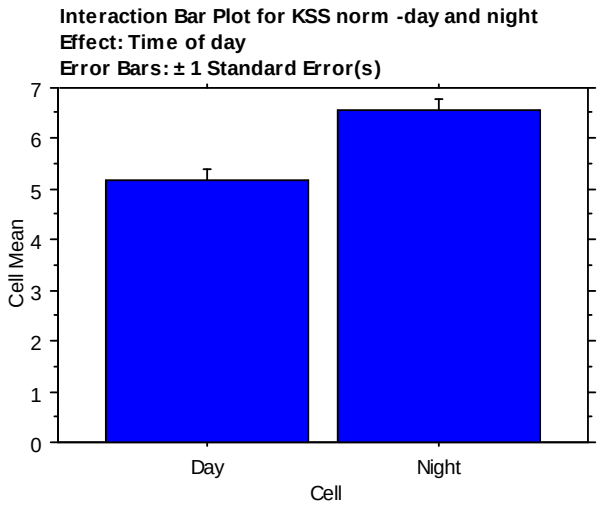

Figure 9: KSS scores for day and night. A significant higher score (more sleepiness) was reported at night than during the day.

DISCUSSION

The present study was carried out to investigate the possibility of using low levels of blue light to increase nighttime alertness with minimum impact on nighttime driving visibility. A light source peaking close to the maximum sensitivity of the circadian system (λ_{max} = 436 nm) was used so that photopic light levels, and thus disability glare, could be reduced while still maintaining effective stimulation to the circadian system to induce alertness.

Consistent with human physiology (2, 15), sleep pressure was higher at night than during the day. Subjects reported feeling more tired while driving at night than during the day and beta power, a measurement of alertness and cognition, was also significantly higher during the day than at night. Although not statistically significant, alpha power was higher at night than during the day, suggesting, again, that subjects were feeling sleepier at night than during the day. This increase in sleepiness did not, however, seem to negatively affect simulated driving performance. In order to understand these results, it may be important to differentiate between fatigue and sleepiness. Fatigue is gradually cumulative and has been associated with unwillingness to maintain effort, which then leads to reduction in performance. Fatigue has been shown to reduce performance in driving episodes that require sustained attention for a long period of time. Sleepiness is related to circadian and homeostatic processes and tends to be greater at night, when the alerting signal from the circadian system is lower and the sleep pressure is higher. Philip and colleagues (35) showed that normally-rested drivers did not exhibit a significant decrement in performance due to fatigue generated by extensive driving during the day. They attributed these results to the driving breaks given during the experiment. Driving breaks seem to reduce fatigue and have been associated with a reduction in traffic accidents. Philips et al. (35) were able to show, however, that sleep deprivation had a

clear impact on driving skills, although they found a large inter-subject variability. Some subjects had a large decrement in performance, while others were not at all affected by sleep deprivation. In the present study, the lack of a significant difference between performance during day and night might have been due to the counterbalancing of the experimental conditions, which was designed to eliminate the fatigue effect. Another possible explanation is the fact that the daytime session was held close to the timing when alertness circadian rhythm shows a mid-afternoon drop (14, 15).

The non-expected low nocturnal melatonin suppression by the blue light was puzzling. For this counterbalanced design, a steady rise of melatonin levels across time of night was observed for all lighting conditions (D1, D2, B1, B2), suggesting that the blue light exposures were not effective at suppressing melatonin. At this juncture, we believe the explanation for these results is related to poor model predictions of melatonin suppression at very short wavelengths. Previous studies conducted at the Lighting Research Center (36, 37) also showed lower than predicted melatonin suppression after exposure to a 445 nm light. Consistently, Brainard et al (41) investigated the impact of a 420 nm and a 460 nm light on acute melatonin suppression. Their results suggest that the 420 nm light was less effective in suppressing melatonin than expected for a single opsin curve. It is yet unknown why the circadian system seems to exhibit a higher than expected threshold for these shorter wavelengths of light. Further investigation on the retinal mechanisms associated with this phenomenon is recommended.

Regarding the impact of disability glare from blue light exposure on driving performance, the results presented here suggest that a 30% reduction in VTC may not lead to a significant decrement in driving performance because there was no significant main effect of lighting conditions on most of the driving performance measures. In other words, driving performance during light exposure did not significantly differ from driving performance in dim light. One could argue, however, that the (nearly significant) reduced throttle after exposure to B2 compared to D2 may have been due to disability glare or that the overall lack of significant improvement in driving performance after blue light exposure was due to reduced visibility compared to dim light. This is likely not the case because the other measures of alertness (EEG and heart rate) were not significantly changed by blue light exposures, suggesting no impact of blue light on alertness. It is important to point out, however, that 30% reduction in VTC may not have impacted driving performance in simulated driving conditions, but it is not known how this reduction in VTC will impact nighttime driving performance in real-life situations.

CONCLUSION

Previous studies have demonstrated that 30 lux of 470 nm blue light increases subjective and objective alertness at night, but this was the first study to investigate the impact of a shorter wavelength (peak at 436 nm) with the same predicted level of circadian system stimulation might have on nocturnal alertness and driving performance. Whereas disability glare will be reduced at shorter wavelengths for a criterion response by the circadian system (e.g., 35% melatonin suppression), the present results did not show a significant impact of the short-wavelength stimulus on nocturnal alertness and

performance. Since the short-wavelength stimulus was not a strong stimulus for the circadian system, as demonstrated by its inability to suppress melatonin, it is logical to expect that the impact of light on alertness and performance would have been either non-existent or reduced. Although recent studies by the LRC suggest that light-induced alertness at night may not be mediated only by the circadian system, the levels used in this experiment seem to have been below threshold for activation by any of the light-induced alertness pathways. An important next step would be to better understand the retinal mechanisms associated with this lower-than-expected response by the circadian system to shorter wavelength blue light and make adjustments to the mathematical model of human circadian phototransduction accordingly. From a more practical perspective, it is also recommended that a more formal assessment of the impact of disability glare on driving visibility at night from a longer wavelength blue light (e.g., 470 nm), known to positively impact the circadian system.

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REFERENCES

1. Akerstedt, T. and J.E. Froberg, *Interindividual differences in circadian patterns of catecholamine excretion, body temperature, performance, and subjective arousal.* Biol Psychol, 1976. **4**(4): p. 277-92.
2. Akerstedt, T. and S. Folkard, *The three-process model of alertness and its extension to performance, sleep latency, and sleep length. Chronobiology International*, Vol. 14, 1997, pp. 115-123.
3. Akerstedt, T. Altered sleep/wake patterns and mental performance. *Physiology & Behavior*, Vol. 9, 2006, pp.209-218.
4. Badia, P., B. Myers, M. Boecker, J. Culpepper, and J. R. Harsh JR. Bright Light Effects on Body Temperature, Alertness, EEG and Behavior. *Physiology and Behavior*, Vol. 50, 1991, pp. 583-588.
5. Berson, D. M., F. A. Dunn, and M. Takao. Phototransduction by Retinal Ganglion Cells that Set the Circadian Clock. *Science*, Vol. 295, 2002, pp. 1070-1073.
6. Boyce, P. R., J. W. Beckstead, N. H. Eklund, R. W. Strobel, and M. S. Rea. Lighting the Graveyard Shift: The Influence of a Daylight-Simulating Skylight on the Task Performance and Mood of Nightshift Workers. *Lighting Research and Technology*, Vol. 29, 1997, pp. 105-134.
7. Brainard, G. C., J. P. Hanifin, J. M. Greeson, B. Byrne, G. Glickman, E. Gerner, and M. D. Rollag. Action Spectrum for Melatonin Regulation in Humans: Evidence for a Novel Circadian Photoreceptor. *Journal of Neuroscience*, Vol. 21, 2001, pp. 6405-6412.
8. Cajochen, C., J. M. Zeitzer, C. A. Czeisler, and D. J. Dijk. Dose-Response Relationship for Light Intensity and Ocular and Electroencephalographic Correlates of Human Alertness. *Behavioral Brain Research*, Vol. 115, 2000, pp. 75-83.
9. Cajochen, C., M. Munch, S. Kobialka, K. Krauchi, R. Steiner, P. Oelhafen, S. Orgul, and A. Wirz-Justice. 2005. High Sensitivity of Human Melatonin, Alertness, Thermoregulation, and Heart Rate to Short Wavelength Light. *Journal of Clinical Endocrinology and Metabolism*, Vol. 90, 2005, pp. 1311-1316.
10. Campbell, S. S., and D. Dawson. Enhancement of Nighttime Alertness and Performance with Bright Ambient Light. *Physiology and Behavior*, Vol. 48, 1990, pp. 317-320.
11. Czeisler, C.A., et al., *Stability, precision, and near-24-hour period of the human circadian pacemaker.* Science, 1999. **284**(5423): p. 2177-81.
12. Figueiro, M. G., M. S. Rea, P. Boyce, R. White, and K. Kolberg. The Effects of Bright Light on Day and Night Shift Nurses' Performance and Well-Being in the NICU. *Neonatal Intensive Care*, Vol. 14, 2001, pp. 29-32.
13. Figueiro, M.G., J.D. Bullough, A. Bierman, C.R. Fay, M.S. Rea. On light as an alerting stimulus at night. *Acta Neurobiol Exp* 67, Vol. 2, 2007, pp. 171-178.
14. Folkard, S. Circadian performance rhythms: some practical and theoretical implications. *Philosophical Transactions of the Royal Society of London, Series B Biological Sciences*, Vol. 327, 1990, pp. 543-553.
15. Folkard, S. and P. Totterdell. "Time since sleep" and "body clock" components of alertness and cognition. *Acta Psychiatrica Belgica*, Vol. 94, 1994, pp. 73-74.

16. Fry, G. A. Evaluating Disability Effects of Approaching Automobile Headlights. In *Highway Research Bulletin* 89, HRB, National Research Council, Washington, D.C., 1954, pp. 38-42.
17. Hoffman, M. S. (ed.). *The World Almanac and Book of Facts*. Pharos Books, New York, N.Y., 1990.
18. Jewett, M. E., D. W. Rimmer, J. F. Duffy, E. B. Klerman, R. E. Kronauer, and C. A. Czeisler. Human Circadian Pacemaker is Sensitive to Light Throughout Subjective Day Without Evidence of Transients. *American Journal of Physiology*, Vol. 273, 1997, pp. R1800-R1809.
19. Kleitman, N. and D.P. Jackson. Body temperature and performance under different routines. *Journal of Applied Physiology*, Vol. 3, 1950, pp. 309-328.
20. *Large Truck Crash Profile: The 1998 National Picture*. Federal Motor Carrier Safety Administration, Washington, D.C., 2000.
21. Moore-Ede, M. C., F. M. Sulzmann, and C. A. Fuller. *The Clocks That Time Us.* Harvard University Press, Cambridge, Mass., 1982.
22. Moore, R., *Organization of the mammalian system*. Circadian clocks and their adjustment, ed. D. Chadwick and K. Ackrill. 1995, England: John Wiley and Sons. 88-106.
23. Rea, M. S. (ed.). *IESNA Lighting Handbook: Reference and Application*, 9th ed. Illuminating Engineering Society of North America, New York, N.Y., 2000.
24. Rea, M. S., J. D. Bullough, and M. G. Figueiro. Human Melatonin Suppression by Light: A Case for Scotopic Efficiency. *Neuroscience Letters*, Vol. 299, 2001, pp. 45-48.
25. Rea, M. S., J. D. Bullough, and M. G. Figueiro. Phototransduction for Human Melatonin Suppression. *Journal of Pineal Research*, Vol. 32, 2002a, pp. 209-213.
26. Rea, M. S., M. G. Figueiro, and J. D. Bullough. Circadian Photobiology: An Emerging Framework for Lighting Practice and Research. *Lighting Research and Technology*, Vol. 34, 2002b, pp. 177-190.
27. Rea, M., et al. A model of phototransduction by the human circadian system. *Brain Research Reviews*, Vol. 50, 2005, pp. 213-228.
28. Thapan, K., J. Arendt, and D. J. Skene. An Action Spectrum for Melatonin Suppression: Evidence for a Novel Non-Rod, Non-Cone Photoreceptor System in Humans. *Journal of Physiology*, Vol. 535, 2001, pp. 261-267.
29. Warman, V. L., D. J. Dijk, G. R. Warman, J. Arendt, and D. J. Skene. Phase Advancing Human Circadian Rhythms with Short Wavelength Light. *Neuroscience Letters*, Vol. 342, 2003, pp. 37-40.
30. Wright, H.R., and L. C. Lack. Effect of Light Wavelength on Suppression and Phase Delay of the Melatonin Rhythm. *Chronobiology International*, Vol. 18, 2001, pp. 801-808.
31. Wright, H. R., L. C. Lack, and D. J. Kennaway. Differential Effects of Light Wavelength in Phase Advancing the Melatonin Rhythm. Journal of Pineal Research, Vol. 36, 2004, pp. 140-144.
32. Wright, K.P., Jr., J.T. Hull, and C.A. Czeisler, *Relationship between alertness, performance, and body temperature in humans.* Am J Physiol Regul Integr Comp Physiol, 2002. **283**(6): p. R1370-7.

33. Zeitzer, J. M., D. J. Dijk, R. Kronauer, E. Brown, and C. Czeisler. Sensitivity of the Human Circadian Pacemaker to Nocturnal Light: Melatonin Phase Resetting and Suppression. *Journal of Physiology*, Vol. 526, 2000, pp. 695-702.
34. Smith, V.C., Pokorny, J. Spectral sensitivity of the foveal cone photopigments between 400 and 500 nm. Vision Research 1975, 15:161-171.
35. Philip, P., P. Sgaspe, N. Moore *et al*. Fatigue, sleep restriction and driving performance. *Accident Analysis and Prevention*, Vol. 37, 2005, pp. 473-478.
36. Qi, Rui. Influence of Blue and Red Light on Melatonin Suppression. Masters Thesis, Lighting Research Center, Rensselaer Polytechnic Institute, Troy, NY, 2008
37. Miller, D.E. Changes in Spectral Sensitivity of the Human Circadian System. Masters Thesis, Lighting Research Center, Rensselaer Polytechnic Institute, Troy, NY, 2009
38. Commission International de l'Eclairage (CIE). Light as a True Visual Quantity: Principles of Measurement. Commission Internationale de l'Eclairage 1978, Paris.
39. Ingling, C.R., Martinez-Uriegas, E. The relationship between spectral sensitivity and spatial sensitivity for the primate r-g X-channel. Vision Research 1983, 23:1495-1500.
40. Figueiro, M. G., J. D. Bullough, R. H. Parsons, and M. S. Rea. Preliminary Evidence for Spectral Opponency in the Suppression of Melatonin by Light in Humans. Neuroreport, Vol. 15, 2004, pp. 313-316.
41. Brainard, G. C., Sliney, D., Hanifin, J. P., Glickman, G., Byrne, B., Greeson, J. M., Jasser, S., Gerner, E., Rollag, M. D. (2008). Sensitivity of the Human Circadian System to Short-Wavelength (420-nm) Light. J Biol Rhythms. 23(5), 379-386.
42. American Electroencephalographic Society: Guidelines for standard electrode position nomenclature. J Clin Neurophysiol 1991, 8:200-202.