The potential of outdoor lighting for stimulating the human circadian system

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Prepared for: Alliance for Solid-State Illumination Systems and Technologies (ASSIST)

Publish Date: Revised December 11, 2012 (original May 13, 2010)

Abstract

The purpose of the present report is to provide a quantitative analysis of the impact of light at night, particularly from streetlights of different spectral power distributions, on the human circadian system. The Rea et al. (2005, 2012) model of human circadian phototransduction was used to estimate levels of circadian stimulation, as measured by melatonin suppression by light, from four typical outdoor light sources as might be experienced by people under different realistic scenarios. Under the practical application scenarios examined here, three of the four sources examined would not meaningfully stimulate the human circadian system after one hour of exposure, while one source (a 6900 K LED) is predicted to have a modest stimulating effect after a one-hour exposure (corresponding to 12 – 15% nocturnal melatonin suppression).

The approach taken was to determine whether sufficient light is incident on the retina to reach a working threshold for stimulating the circadian system and, thereby, to ascertain whether and to what degree outdoor lighting might stimulate the circadian system, as measured by melatonin suppression. Although the information presented represents a state-of-the-art analysis of light-induced nocturnal melatonin suppression, there are several limitations to this analysis due to the uncertain causal relationship between retinal light exposure at night and human health.

Introduction

Every species on earth exhibits a wide range of biological cycles that repeat approximately every 24 hours. These are known as circadian rhythms (circa – approximately; dies – day) and are exhibited at every level of biological systems, from timing of DNA repair in individual cells to behavioral changes, like the sleep-wake cycle. Circadian rhythms reflect the tight coupling between the intrinsic timing of the brain's internal clock in the suprachiasmatic nuclei of the hypothalamus (SCN) and the natural timing of the solar light-dark cycle. In fact, the light-dark cycle registered on the retina is the primary stimulus for setting the timing of a multitude of circadian rhythms exhibited by humans and most other mammals.

Light can be specified along five dimensions: quantity, spectrum, distribution, timing, and duration (Rea et al. 2002). Responses by the visual and circadian systems to changes along these dimensions reveal fundamental differences between their operating characteristics. Compared with the visual system, the human circadian system is relatively insensitive to light. Relative to the visual system, light effective for the human circadian system must be several orders of magnitude greater in quantity and prolonged for many minutes to produce a measurable response (Rea et al. 2002, McIntyre et al. 1989). The visual system and the human circadian system are both sensitive to short wavelengths (400 – 500 nm); however, the human circadian system is nearly blind to long-wavelength radiation (> 600 nm) that the visual system can, in fact, see very well. Unlike the visual system, the human circadian system is not concerned with image formation, so the light can be blurred or diffusely distributed over a large portion of the retina to provide stimulation. Thus, the human circadian system is biased against false positive responses to optical radiation—it needs to reliably know when it is day and when it is night. To do so, it exhibits a high threshold and a narrow spectral response to light, and it needs prolonged exposure to light, probably over a large portion of the retina. Moreover, the system is differentially sensitive to light over the course of the day; both the direction and the magnitude of response change depending upon when light is incident on the retina. Light exposure in the morning advances the timing of the SCN clock, whereas the same light exposure in the evening delays the timing of the clock—at midday, the system is much less sensitive to light exposure (Jewett et al. 1997, Khalsa et al. 2003).

Civilization has changed the natural light-dark cycle that humans experience. Buildings shield us from the weather as well as the bright daytime sky. Electric light sources not only provide illumination at night and throughout building interiors, they also provide self-luminous displays such as televisions and computer monitors. Epidemiologists and other medical researchers have expressed concern over electric lighting as a potential disruptor of the natural light-dark cycle (Stevens et al. 2007, Stevens 2009, Haus and Smolensky 2012). Indeed, a wide range of maladies from insomnia to breast cancer have been statistically associated with disruption of the natural 24-hour light-dark cycle (reviewed in Blask 2009). Further, animal studies have shown that tumor growth is faster when melatonin is suppressed by light at night (Blask et al. 2005). Other studies suggest that “jet-lagged” animals (i.e., subjected to irregular light-dark patterns) are at higher risk for cancer, cardiovascular disease, diabetes and obesity (Filipski et al. 2004, 2006; Fu and Lee 2003; reviewed in Rüger and Sheer 2009). Despite the absence of a causal connection between disrupted circadian rhythms and compromised health in humans, continued investigations of light-induced disruption of the human circadian system are clearly warranted (Reiter et al. 2009).

Considering the significance of the light-dark cycle for regulating biological functions, and the accumulation of evidence from epidemiological and animal studies linking circadian disruption

to compromised health and well-being, it is surprising that so little has been done to quantify light and dark in industrialized societies as they might affect the human circadian system. Given this paucity of photometric data, it is perhaps not surprising that so little has been done to parametrically study the impact of circadian disruption on health and well-being in people. However, without proper photometric data it is essentially impossible to draw valid inferences about the impact of lighting, both natural and fabricated, on human health and well-being.

Recently, a model of human circadian phototransduction (i.e., the conversion of optical radiation incident on the retina to neural signals sent to the SCN) has been developed (Rea et al. 2005, 2012). The model considers the necessary biophysical characteristics of optical radiation incident on the retina that influence human circadian phototransduction, as measured in terms of light-induced nocturnal melatonin suppression. More specifically, the model takes into account the spectral composition of the optical radiation, the absolute amount of radiation, the spatial distribution of irradiance on the cornea, and the duration of exposure necessary to evoke a particular biological response from the human circadian system. Validations of the model have been made (Figueiro et al. 2006a, Figueiro et al. 2007, Bullough et al. 2008, Figueiro et al. 2008).

Concerns have been raised by an advocacy group, the International Dark Sky Association (IDA), over light at night as it affects human health through stimulation of the circadian system (IDA 2009). The purpose of the present report is to provide a quantitative analysis of the impact of light at night, particularly from streetlights of different spectral power distributions, on the human circadian system. The Rea et al. (2005, 2012) model was used to estimate levels of circadian stimulation from four typical outdoor light sources as might be experienced by people under different realistic scenarios. Although perhaps obvious, it must be emphasized that stimulation of the human circadian system at night is not necessarily synonymous with health risk. For a meaningful discussion of the relationship between light at night and human health, it is nevertheless essential to first determine if and to what degree practical light sources used for nighttime illumination stimulate the human circadian system. If, under realistic scenarios, outdoor lighting systems could measurably stimulate the human circadian system, then the health concerns raised by the IDA may have merit. If, on the other hand, outdoor lighting minimally stimulates the human circadian system, then IDA's cautionary advice, while still potentially valid, is more speculative and less deserving of immediate social action in the context of all the other concerns that face society.

Problem statement

The IDA has drawn attention to the relative spectral composition of different outdoor light sources as a possible concern for human health. The concern stems from the epidemiological studies of rotating shift workers having increased cancer risks and of animal studies showing

that suppression of melatonin by light increases tumor growth, as previously noted above. Since the human circadian system is maximally sensitive to short wavelengths (440 – 460 nm) and since "white" light sources used for outdoor lighting typically have strong emissions at these short wavelengths, the IDA has made the argument that "white" light sources used in outdoor lighting may negatively impact human health. It is, however, impossible to draw any inferences about the impact of a given type of light source on the circadian system response, let alone on human health and well-being, without first providing a complete specification of the stimulus. In other words, knowing the relative spectral content of a source is only a very small part of the whole picture, and one that can easily mislead the non-expert. Therefore, discussions of the topic of light at night as it might affect human health and well-being must include the temporal-spatial-spectral distribution of optical radiation incident on the retina together with corresponding temporal-spatial-spectral and absolute sensitivity of the human circadian system.

Analytical approach

First, and notwithstanding the fact that some optical radiation does filter through closed eyelids (Hätönen et al. 1999, Jean-Louis et al. 2000, Bierman et al. 2011), the eyelids must be open to effectively stimulate the human circadian system at night by ambient electric lighting. Only optical radiation incident on the healthy, functional retina can stimulate both the visual and the circadian systems of people. To have a meaningful discussion of outdoor lighting then, it is necessary to have a much more detailed understanding of the light exposure on the retina than the relative spectral content of the source. Indeed, any discussion of a single aspect of optical radiation is a disservice to rational discussion of the impact of light at night on human health and well-being. For that reason, several scenarios of light exposures that might be experienced by people are presented using the best available information, recognizing again that the link between light at night as it stimulates the human circadian system is not synonymous with a link between light at night as it affects health and well-being. The approach taken here then is simply to determine whether sufficient light is incident on the retina to reach a working threshold for stimulating the circadian system and, thereby, to ascertain whether and to what degree outdoor lighting might stimulate the circadian system, as measured by melatonin suppression.

Figure 1 shows the spectral irradiances of four sources at 95 lx, one each for two commercially available "cool-white" LED sources, a sodium-scandium metal halide (MH) lamp, and a high-pressure sodium (HPS) lamp. Using the model of human circadian phototransduction by Rea et al. (2005, 2012), it is possible to compare the effectiveness of the different light sources at defined irradiances for suppressing a criterion amount of nocturnal melatonin for a known pupil area. Pupil areas for a young population (17 – 25 years of age) can be estimated (over a limited range of irradiance levels) from a model published by Berman et al. (1992) using the spectral

irradiance distributions at the cornea (i.e., light spectrum and amount). The spectral irradiances at the cornea from the "cool-white" LEDs, the MH, and the HPS lamps in Figure 1 can be therefore adjusted using the model by Berman and colleagues to scale the spectral irradiance distribution incident on the retina, which can then be input to the model of human circadian phototransduction to calculate a prediction of nocturnal melatonin suppression.

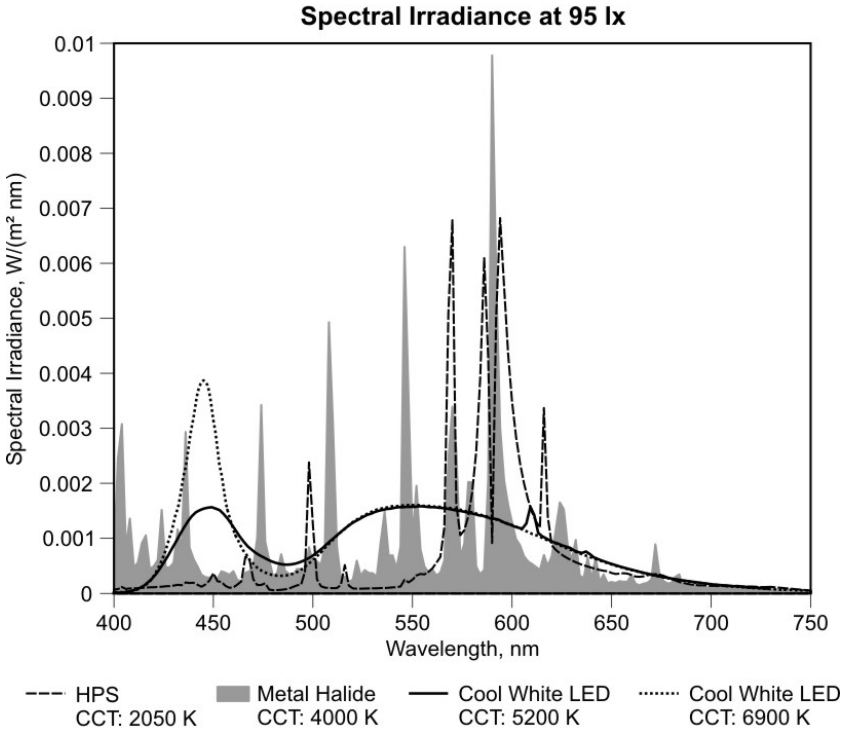

Fig. 1. Spectral irradiance distributions for a photopic illuminance of 95 lx.

Estimates of irradiance levels at the cornea from the "cool-white" LEDs, the MH and the HPS sources for three different conditions were considered: a reference condition comparable to what has been employed in controlled laboratory conditions, and two practical scenarios that could occur with an outdoor lighting installation (Figure 2). From those irradiances, and assuming a one-hour exposure with natural pupils, it was possible to estimate the degree to which the circadian system of a 20-year old would be stimulated, defined operationally for this exercise as percentage of nocturnal melatonin suppression.

For each of the following conditions, a 20-year-old person views each of the four light sources (Figure 1). The eye height of the observer is 5 ft. (1.5 m) above the ground, the luminaire mounting height is 27 ft. (8.2 m), and the lighting distribution and intensity are nominally based on a 150W, Type III, full cutoff luminaire (Figure 2).

Reference condition: The person directly views each luminaire from a point 5 ft. (1.5 m) from the vertical center line of the mounting pole, and the illuminance at the cornea is 95 lx.

Scenario 1: This same person is now looking down the road and is 10 ft. (3 m) away from the vertical center line of the mounting pole at the location where the luminaire would produce the maximum illuminance, 27 lx, at the cornea.

Scenario 2: This same person is 30 ft. (10 m) away from the vertical center line of the pole looking directly at the luminaire; the illuminance at the cornea is 18 lx.

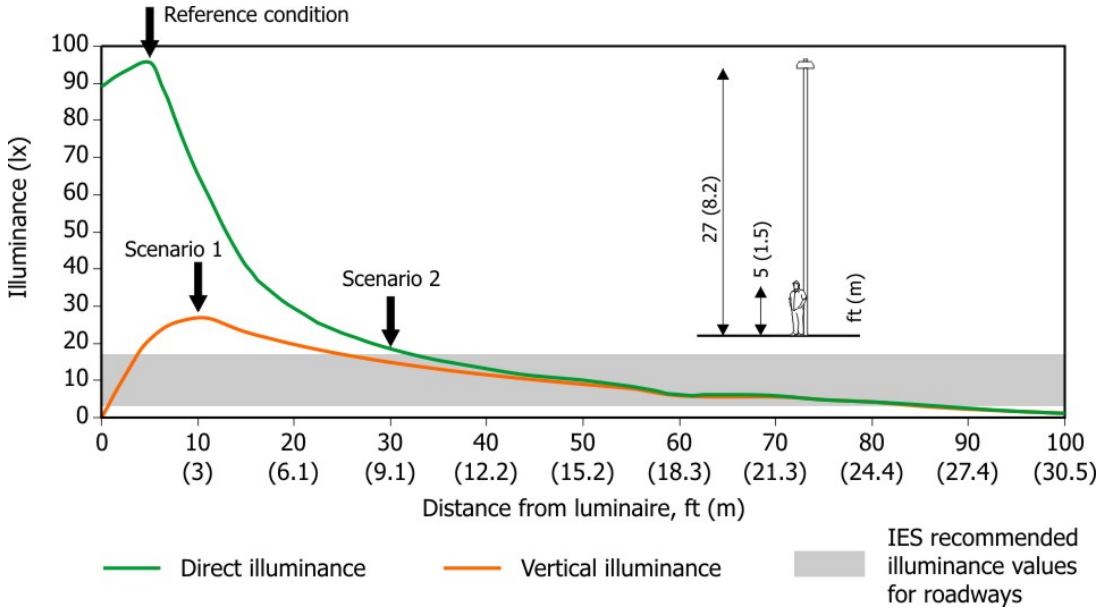

Fig. 2. Reference condition and two lighting scenarios (see text) used to calculate effective circadian light stimulation for four light sources. For calculation purposes, each of the four sources is nominally 150W and installed in a Type III distribution luminaire mounted on a 27 ft. (8.2 m) pole. The eye height of the observer is 5 ft. (1.5 m) above the ground. Illustrated is the horizontal illuminance, in lux (green line), and the vertical illuminance, in lux (orange line), at different distances from the pole. Also shown is the range of IESNA recommended horizontal illuminance values, in lux (shaded area), for roadway lighting (IESNA 2000[R2005]).

Figure 3 illustrates the results of the calculations. For the reference condition emulating a laboratory experiment, melatonin would be suppressed by 15% for the HPS source, 14% for the MH source, 21% for the 5200 K “cool-white” LED source, and 30% for the 6900 K “cool-white” LED source. Under the two more realistic scenarios, based upon the model calculations, the 20-year old would not have reliably suppressed nocturnal melatonin (above the 10% uncertainty level for assaying melatonin) after one hour of exposure to the warmer 5200 K “cool-white” LED, the MH or the HPS sources. For both practical scenarios, some melatonin is expected to be

suppressed for the cooler 6900 K “cool-white” LED source: 12% for scenario 1 and 15% for scenario 2. It should be noted that people older than 20 years of age will have lower retinal irradiances due to senile miosis and denser crystalline lenses (Rea and Ouellette 1991), so melatonin suppression would be less, on average, for older individuals for all four sources under the reference condition and under the two more realistic scenarios. Further, anyone exposed to that same light level for durations shorter than one hour will also likely exhibit less melatonin suppression. Finally, it is important to note that there are only limited data available in the literature on the characteristics of the circadian system response near threshold activation, so a precise estimate of melatonin suppression near threshold cannot be made. Figueiro et al. (2006b) suggest, for example, that light-induced nocturnal melatonin suppression levels must be greater than 15% to be measured reliably; a more conservative 10% criterion is used for the present assessment.

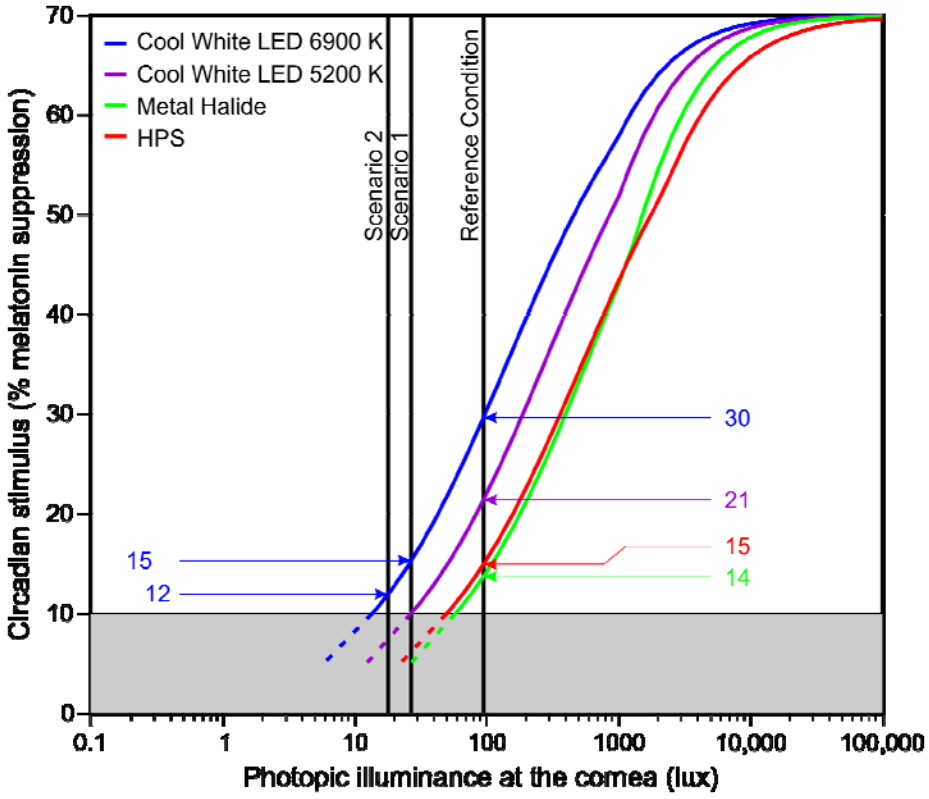

Fig. 3. Melatonin suppression (%) by the human circadian system in response to two “cool-white” LEDs, metal halide (MH), and high-pressure sodium (HPS) sources plotted for a wide range of corneal photopic illuminance levels. The calculations (Rea et al. 2010, 2012) are based upon age-dependent pupil area (Berman et al. 1992) for a one-hour exposure according to the model by Rea et al. (2005, 2012). The vertical lines indicate the photopic illuminance at the cornea for a reference laboratory condition and for two practical street lighting scenarios explained in the text. The shaded gray area reflects the absolute level of uncertainty for assaying melatonin from saliva or blood plasma.

Limitations

Although the information presented represents a state-of-the-art analysis of light-induced nocturnal melatonin suppression, there are several limitations to this analysis due to the uncertain causal relationship between retinal light exposure at night and human health. First, this analysis depends fundamentally upon the assumption that nocturnal melatonin suppression is in fact directly related to human health. This causal relationship has not been firmly established yet in humans, although there does appear to be indirect evidence for that link from animal studies. Light at night can also delay the timing of the circadian system, and thereby may potentially disrupt a regular 24-hour biological rhythm, much like jet lag or shift work. Light can also affect hormones other than melatonin (e.g., cortisol) and enzymes (e.g., alpha amylase) that are important markers of circadian regulation in various biological systems. Melatonin is not synthesized at a constant rate at night but, rather, exhibits a pulsatile nature (Arendt 1994). Light may affect this pulsatile behavior with unknown implications for communicating circadian timing to other biological systems as they might affect human health. The calculations reported here assumed that the model by Berman and colleagues (1992) can be used to scale the spectral irradiance distributions at the cornea to characterize the effective retinal stimulus for the circadian system, but there are of course wide individual differences in crystalline lens transmission and pupil response to light that would directly affect the amount of light actually reaching the retina. Individuals with inherently high concentrations of melatonin may be less susceptible to diseases, such as cancer, than those with inherently low concentrations, regardless of the impact of light at night on circulating melatonin. A person's light history also affects the degree to which light can suppress melatonin (Hébert et al. 2002, Smith et al. 2004). A person working outdoors during the day will have a higher threshold to light-induced nocturnal melatonin suppression than those who spend the day in dimly illuminated interiors. So a fixed level of light may have differential consequences on people with different lifestyles. As already noted, there is great uncertainty in the threshold response to light at night. Whether a small but constant suprathreshold amount of suppression has a cumulative effect on human health is also unknown. In general then, we are coming closer to a quantitative understanding of how light affects the circadian system, but we still do not fully understand if or how light at night might affect human health through the circadian system.

Conclusions

Based upon the model predictions, as illustrated in Figure 3 and as pointed out by Figueiro et al. (2006b), a reasonable and conservative working threshold for suppressing nocturnal melatonin by light at night following a 30-minute exposure would be about 30 lx at the eye for a "white" light source. This working threshold is based upon the determination of a reliable degree of light-induced nocturnal melatonin suppression of 15% or greater. As suggested by Figure 3, any given threshold value (10% for this analysis) will show that different light sources, depending

upon their spectral irradiance distributions, will require different photopic illuminance levels to be considered above or below that threshold value. With regard to narrowband light, the threshold would be either higher for long-wavelength (red) light or lower for short-wavelength (blue) light.

It is important to stress yet again that this analysis is not specific to a determination of the risk to human health and well-being from outdoor lighting. The analysis is limited to estimating the stimulating effects of light sources used from outdoor lighting on the human circadian system as measured in terms of nocturnal melatonin suppression. Nevertheless, the correct characterization of the light stimulus *must* be made before any inferences can be drawn about the potential health effects of exposure to outdoor lighting. Indeed, providing a *complete* quantitative estimate of the impact that light exposure at night has on the human circadian system is the necessary first step in responsibly discussing the potential impact of outdoor lighting on human health and well-being. Based upon this analysis then, it would appear that under the practical application scenarios examined here, three of the four sources examined would not meaningfully stimulate the human circadian system after one hour of exposure. The cooler of the two "cool-white" LEDs is predicted to have a small stimulating effect on the human circadian system after one hour exposure (corresponding to 12 – 15% nocturnal melatonin suppression).

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