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Light as a circadian stimulus for architectural lighting

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Consideration is being given to the adoption of a new metric characterizing light as it affects the human circadian system. Much has been learned over the past couple of decades about light as a stimulus for circadian system regulation, so it is appropriate that these discussions take place. The present paper develops an argument for adopting circadian stimulus as a metric for quantifying light in architectural spaces. The circadian stimulus metric (a) was developed from several lines of biophysical research, including those from basic retinal neurophysiology; (b) has been validated in several controlled experiments; and (c) has been used successfully in a number of real-world applications. Any discussions of new metrics should take each of these foundational points into consideration.

1. Introduction

Much has been learned over the past 20 years about the impact of retinal light exposure on the regulation and the disruption of circadian rhythms in humans.[1–18] It seems appropriate at this time to begin to develop metrics that might be used for engineering electric lighting systems and daylight control systems as they might systematically and reliably affect those rhythms.[19] Toward that end, it is first necessary to consider the scientific evidence supporting the development of metrics to characterize light as it may affect outputs of the circadian system. Then, an informed decision can be made about the adoption of these metrics for practical purposes in architectural lighting.[20]

2. Modelling the circadian stimulus

Much of what we know about human circadian phototransduction (how the retina converts light into neural signals for the circadian system) comes from studies of the impact of light at night on the synthesis of the hormone melatonin. Melatonin circulates in the blood stream and is easily absorbed by individual cells, serving as a coordinating time-of-day (specifically, nighttime) signal throughout the body. At night, light absorbed by retinal photopigments signal the biological clock in the suprachiasmatic nuclei (SCN) of the hypothalamus to suppress nocturnal melatonin production by the pineal gland. While nocturnal melatonin suppression (measured in percent reduction from levels in the dark) is not the only output measure of the circadian system that is affected by retinal light exposure, data are available for developing insight into the spectral and absolute sensitivities of the human circadian system.

Empirically, nocturnal melatonin suppression in humans was shown in two independent studies[6,7] to have a peak spectral sensitivity at 460 nm and a 110-nm wide absorption band at half-maximum sensitivity. These two biophysical studies were conducted in much the same way using a

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constant-criterion method. Briefly, constant-criterion methods are employed to deal with possible non-linear response characteristics of a biological system to variable amounts of a physical stimulus. A fixed response criterion is selected and the amount of a variable stimulus needed to reach that criterion is recorded. For these two studies, a fixed half-saturation criterion of nocturnal melatonin suppression (≈35%) was selected because this response was above threshold and below saturation; the corneal irradiance at each test wavelength needed to reach that criterion was recorded. The reciprocal of the amount of irradiance at each wavelength needed to reach the constant criterion defined the spectral sensitivity of nocturnal melatonin suppression to narrowband light stimuli in both studies. Although both experiments are in good agreement over much of the wavelength range of sensitivity, there is an obvious discontinuity in the data sets at about 505 nm, not coincidently, the wavelength that appears to the visual system as unique green (Figure 1). A light that appears unique green is neither 'blue' nor 'yellow' because the spectrally opponent blue versus yellow (b-y) colour mechanism is physiologically unresponsive to that light. An analysis of the nocturnal melatonin suppression data, consistent with retinal neurophysiology, suggests that the b-y colour channel defines the obvious discontinuity, and thus plays an important role in human circadian phototransduction.

No single retinal photopigment exhibits the properties illustrated in Figure 1, including the photopigment melanopsin[2] found in the intrinsically photosensitive retinal ganglion cells (ipRGCs).[11] Direct measurements of the action spectrum for melanopsin in human ipRGCs show a peak spectral sensitivity at approximately 479 nm[22] and like all other known retinal photopigments has a half-maximum sensitivity bandwidth of about 95 nm; with pre-retinal filtering, primarily from the crystalline lens, the peak sensitivity

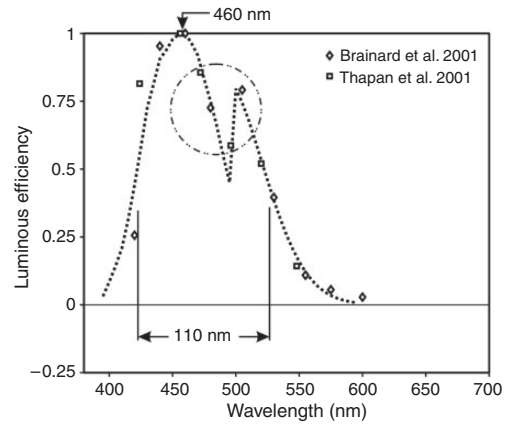

Figure 1 Spectral sensitivity of nocturnal melatonin suppression for different narrowband spectra. Results are shown from two studies[6,7] both using a constant criterion method for characterizing the spectral sensitivity of the human circadian system. Except for the circled spectral region, wavelengths near unique green (close to 505 nm), the results show good agreement with peak sensitivity at approximately 460 nm and a half-maximum sensitivity of about 110 nm. The dotted line represents predictions from the model of circadian phototransduction by Rea *et al.*[10,21] described later in the text.

shifts to approximately 484 nm and the half-maximum sensitivity bandwidth is about 90 nm (Figure 2). *Prima facie* then, the empirical data rule out any simple spectral sensitivity model for nocturnal melatonin suppression based upon a single photopigment, including melanopsin. Logically too then, in an intact retina, no single photoreceptor, specifically ipRGCs, can be responsible for circadian phototransduction. Indeed, results from several electrophysiological experiments with vertebrates[3,23,24] show that, while ipRGC efferent axons are the main conduit of light signals to the master clock, ipRGC afferent dendrites receive indirect input from the more distal rods and cones. Therefore, a completely successful model of human circadian phototransduction must take into account all retinal photoreceptors and their supporting neural mechanisms and must be constrained by the known anatomy and physiology of the retina and brain[25]

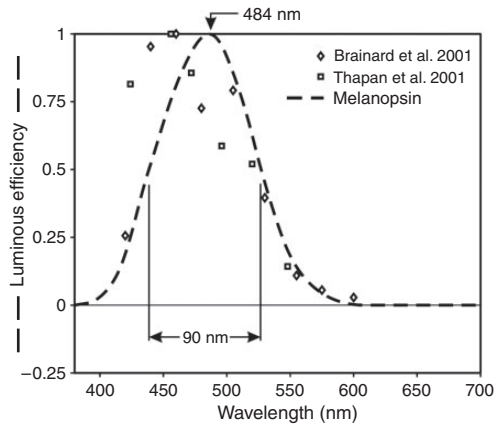

Figure 2 The action spectrum of the photopigment melanopsin after pre-retinal screening by the human crystalline lens. The spectral sensitivity peaks at about 484 nm with a half-maximum sensitivity of about 90 nm.

including neural interactions under a wide range of operating conditions.[10,26]

Practical metrics developed from a complete model must also consider the amount of light incident on the retina as it affects the response of the circadian system from threshold, the first measurable response to light, to saturation, the maximum output of the system. Those metrics must also reflect, if required, changes to the spectral sensitivity of the system with the amount of light incident on the retina. For example, a brightness metric would have to reflect the fact that the human visual system becomes relatively more sensitive to short-wavelengths as light level increases.[27] Regarding the circadian system, once its spectral sensitivity is defined, it is then possible to generate a stimulus-response function using the empirical data from Brainard *et al.*[6] and Thapan *et al.*[7] Again, similar to our many visual responses to light, a single photopigment model cannot possibly represent the dynamic operating characteristics of the neural mechanisms underlying circadian phototransduction.

$$CL_A = \begin{cases} 1548\left[\int Mc_\lambda E_\lambda d\lambda + \left(a_{b-y}\left(\int \frac{S_\lambda}{mp_\lambda}E_\lambda \mathrm{d}\lambda - k\int \frac{V_\lambda}{mp_\lambda}E_\lambda \mathrm{d}\lambda\right) - a_{rod}\left(1 - e^{\frac{-\int V'_\lambda E_\lambda \mathrm{d}\lambda}{RodSat}}\right)\right)\right] \\ \qquad \text{if } \int \frac{S_\lambda}{mp_\lambda}E_\lambda \mathrm{d}\lambda - k\int \frac{V_\lambda}{mp_\lambda}E_\lambda \mathrm{d}\lambda > 0 \\ 1548\int Mc_\lambda E_\lambda \mathrm{d}\lambda \quad \text{if } \int \frac{S_\lambda}{mp_\lambda}E_\lambda \mathrm{d}\lambda - k\int \frac{V_\lambda}{mp_\lambda}E_\lambda \mathrm{d}\lambda \le 0 \end{cases} \tag{1}$$

CL_A: circadian light. The constant, 1548, sets the normalization of CL_A so that 2856 K blackbody radiation at 1000 lux has a CL_A value of 1000.

E_λ: light source spectral irradiance distribution

Mc_λ: melanopsin (corrected for crystalline lens transmittance)

S_λ: S-cone fundamental

mp_λ: macular pigment transmittance

V_λ: photopic luminous efficiency function

V'_λ: scotopic luminous efficiency function

RodSat: half-saturation constant for bleaching rods $= 6.5\,W/m^2$

$k = 0.2616$

$a_{b\text{-}y} = 0.7000$

$a_{rod} = 3.3000$

Equation (1) mathematically defines the modelled spectral sensitivity of the human circadian system. Circadian light (CL_A) from Rea *et al.*[10,21] is measured in units of spectrally weighted (equation (1)) flux per unit area. More details of the model can be found in other publications[10,21] but briefly, all known photopigments contribute to the spectral sensitivity of the circadian system. Rod bleaching controls the absolute sensitivity of cone contributions to the modelled circadian system $\left[a_{rod}\left(1-e^{\frac{-\int V'_\lambda E_\lambda d\lambda}{RodSat}}\right)\right]$. Above threshold cone responses to light are processed through a spectrally opponent blue versus yellow (b-y) mechanism $\left[a_{b-y}\left(\int \frac{S_\lambda}{mp_\lambda}E_\lambda d\lambda - k\int \frac{V_\lambda}{mp_\lambda}E_\lambda d\lambda\right)\right]$. The b-y mechanism is represented by the difference between S-cone response and the sum of the L- and M-cone response (or V(λ)). Output from the b-y mechanism can add to the response of the ipRGCs $\left[\int Mc_\lambda E_\lambda d\lambda\right]$ if the spectral power distribution of the light source causes it to signal 'blue' (i.e., b-y > 0), but no response is added to that of the ipRGC if it signals 'yellow' (i.e., b-y < 0) or generates no response (i.e., unique green, where b-y = 0). For these later two conditions, the spectral sensitivity of the system is defined by melanopsin only $\left[\int Mc_\lambda E_\lambda d\lambda\right]$. Figure 3(a) shows the modelled spectral sensitivity of the human circadian system for narrowband, 'cool' (b-y > 0) and 'warm' (b-y < 0) light sources at a specific scotopic corneal illuminance (300 scotopic lux); Figure 3(b) shows the modelled spectral sensitivity of the system at a much higher level (300,000 scotopic lux). These plots illustrate the spectral sensitivity of the modelled system at different scotopic

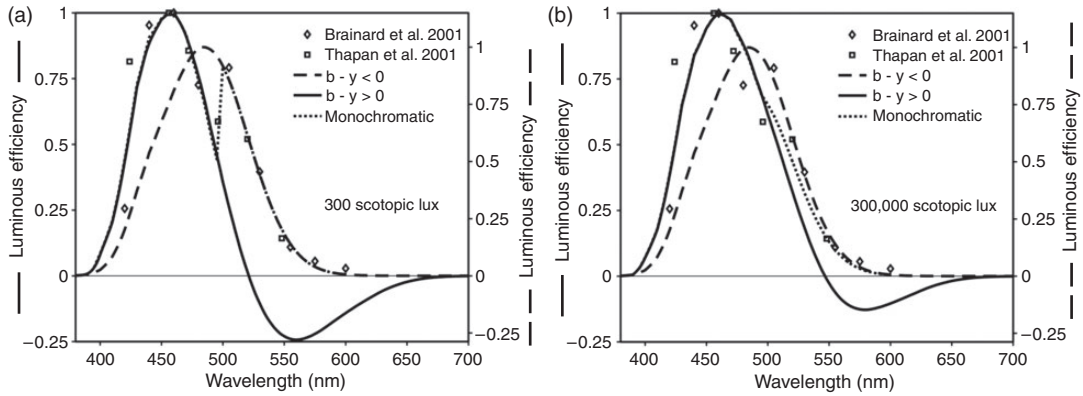

Figure 3 The spectral sensitivity of the human circadian system at two light levels, (a) 300 scotopic lux at the cornea (left) and (b) 300,000 scotopic lux (right). For 'cool' sources where the b-y mechanisms signals 'blue' (b-y > 0), the spectral sensitivity of the system is defined by the solid line, including a subadditive region of the spectrum where the spectral power from a light source subtracts from the overall response of the system. For 'warm' sources where the b-y mechanism signals 'yellow' (b-y < 0), the spectral sensitivity is defined by the dashed line, the pre-retinal filtered melanopsin (Figure 2). It is worth pointing out that the measured spectral sensitivities in Figure 3(a) are well described by the two-state model (equation (1)) at 300 scotopic lux (also shown in Figure 1) but much less so in Figure 3(b) at 300,000 scotopic lux (dotted lines in both graphs). At 300 scotopic lux, the circadian system is operating midway between threshold and saturation, consistent with the operating range of the circadian system where a constant criterion method can be successfully applied for determining spectral sensitivity. At 300,000 scotopic lux, the system is operating at or near saturation where a constant criterion method cannot be used to determine spectral sensitivity. Thus, the modelled response in Figure 3(b) (dotted line) poorly fits the empirical data collected at a much lower light level.

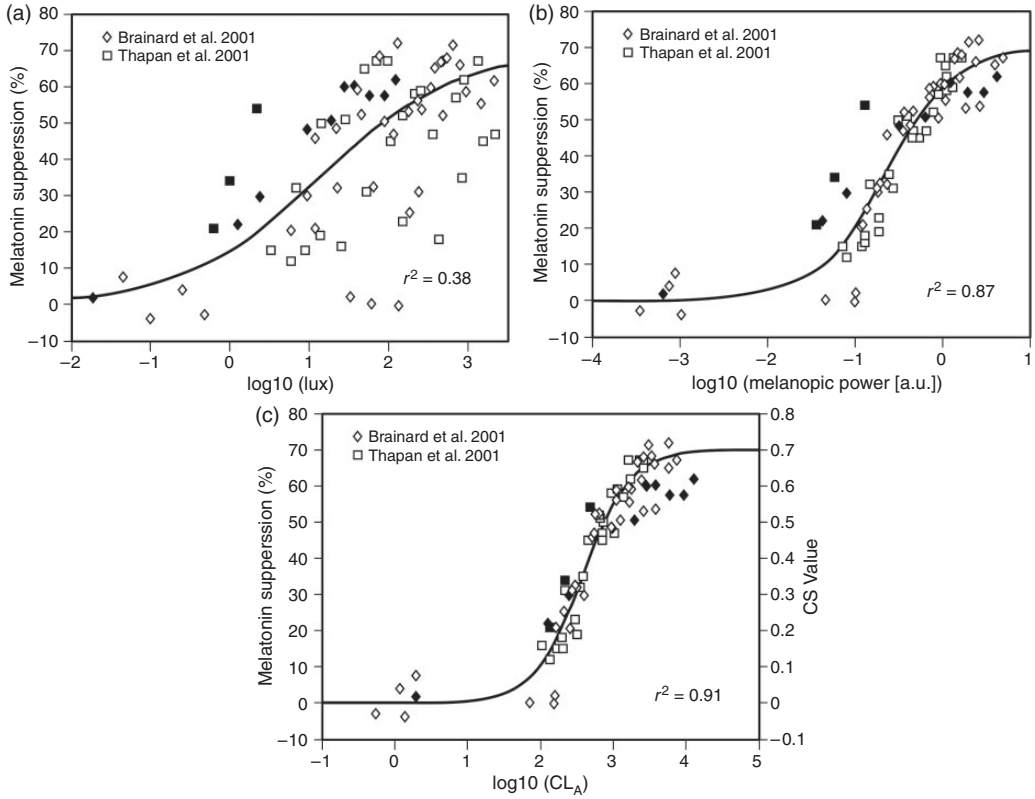

Figure 4 Nocturnal melatonin suppression for different narrowband spectra plotted as a function of $\log_{10}$ photopic illuminance, $\log_{10}$ 'melanopic lux' (in arbitrary units, a.u.), and $\log_{10}$ CL_A (equation (1)). The data from Brainard *et al.*[6] and Thapan *et al.*[7] were used to estimate the spectral sensitivity of nocturnal melatonin suppression in Figure 1. The filled diamonds represent the 440-nm data from Brainard *et al.*[6] and the filled squares represent the 424 nm data from Thapan *et al.*[7]; see text for an explanation of their significance.

illuminance levels because rods control absolute sensitivity of the cones. As can be seen from Figure 3, the modelled spectral sensitivity changes with the level of rod saturation; at higher light levels, rods exhibit greater bleaching resulting in a change in the overall spectral sensitivity.

Figure 4 shows the nocturnal melatonin suppression values from Brainard *et al.*[6] and Thapan *et al.*[7] for the different narrowband spectra as a function of $\log_{10}$ photopic illuminance (Figure 4(a)), $\log_{10}$ melanopsin-weighted irradiance (arbitrary units) (Figure 4(b)), and $\log_{10}$ CL_A (Figure 4(c)). Among these three possible characterizations of light for the human circadian system, CL_A best characterizes the spectral sensitivity of nocturnal melatonin suppression data. Although the overall residual error is incrementally smaller with CL_A ($r^2 = 0.91$) than with melanopsin-weighted irradiance ($r^2 = 0.87$), it is important to note that the regression errors in Figure 4(b) are systematically larger than those in Figure 4(c) for the short-wavelength data (solid symbols for 424 and 440 nm in Figure 4). Thus, the functional impact of light sources with a high proportion of short-wavelength radiation (i.e., 'cool' sources) will be systematically underestimated using a metric based upon melanopsin-

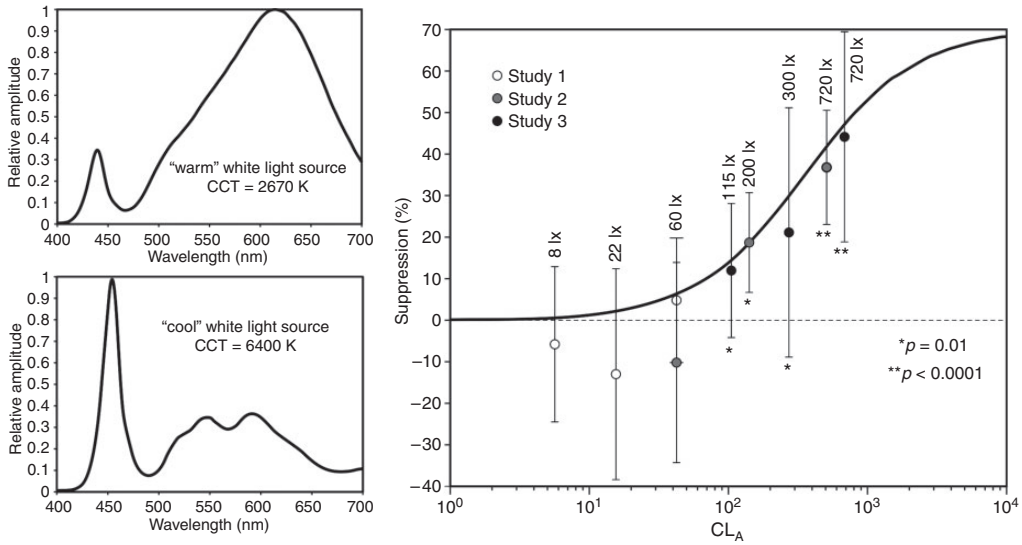

Figure 5 Nocturnal melatonin suppression from 1-hour exposures to two polychromatic sources (left panels) at different corneal photopic illuminance levels as a function of CL_A. Means and standard deviations are plotted from three independent studies conducted at different times. Studies 1 and 2[28] used the 'warm' white light source and study 3 used the 'cool' white light source. It should be noted that the data from study 3 are unpublished; however, they were collected using apparatus, methods and procedures identical to those used in studies 1 and 2.

weighted irradiance. Equation (2) provides the functional relationship between circadian stimulus (CS; right ordinate of Figure 4(c)), which is proportional to percent nocturnal melatonin suppression (left ordinate of Figure 4(c)), and $\log_{10}$ CL_A.

$$CS = 0.7 - \frac{0.7}{1 + \left(\frac{CL_A}{355.7}\right)^{1.1026}} \qquad (2)$$

Figure 5 shows empirically measured nocturnal melatonin suppression for commercially available 'warm-white' and 'cool-white' polychromatic light sources. It should be noted that the model predictions (solid line) were not fitted to these suppression data for the 'white' light sources. Rather, Figure 5 serves to illustrate the validity of the circadian phototransduction model (equations (1) and (2)) for predicting nocturnal melatonin suppression from polychromatic sources that might be engineered for architectural lighting.

Figure 6 shows predictions of nocturnal melatonin suppression for different polychromatic sources at one photopic illuminance level using two different spectral sensitivity functions, one based upon a melanopsin-only spectral weighting of irradiance and one using CL_A (equation (1)). The sharp transition for the blackbody radiator at about 3500 K for the CL_A predictions reflects the change in the modelled overall spectral sensitivity based upon whether the spectrally opponent b-y channel signals 'blue' or 'yellow'. It should be noted that CCT is not an accurate characterization of a light source. One must use the complete spectral power distribution of the source to determine whether the b-y channel signals 'blue' or 'yellow'.

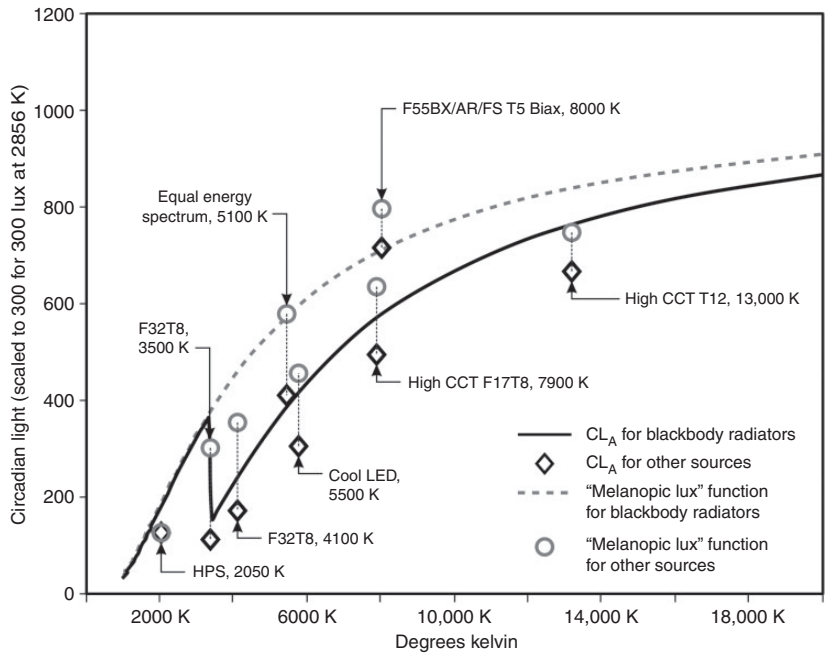

Figure 6 Circadian light using two spectral weighting functions, CL_A and melanopsin (a.u.), for different light sources, scaled to have equal illuminance (300 photopic lux) at the cornea. The continuous solid and dashed lines are circadian light values for a continuous range of ideal blackbody radiators. The discrete points are for selected light sources.

3. Adopting metrics

One alternative to adopting metrics for specifying circadian-effective light for architectural lighting is simply to avoid the problem altogether, thereby avoiding any possible errors of commission (do no harm). As previously articulated,[29] however, avoiding the problem also avoids any errors of omission (do not be negligent). Our knowledge of the basic science and, as described below, our validation of the science in several applications suggests we know enough about the impact of light on circadian rhythms that avoiding the problem is much more likely to lead to errors of omission than errors of commission. Assuming that avoiding the problem is not a viable alternative, two basic approaches are considered.

The first approach would be to develop lighting recommendations based upon the conventional photopic luminous efficiency function [V(λ)]. Currently, luminous efficacy requirements for light sources are defined in terms of photopic lumens per unit electrical power (lm/W) and illuminance requirements for architectural spaces are defined in terms of photopic lumens per unit area (lm/m^2). For recommendations of circadian-effective light, one would need to apply 'corrections' to light source spectral data and to existing illuminance levels to account for the long-wavelength bias of V(λ). This approach would necessitate the adoption of a luminous efficiency function that represents the spectral sensitivity of the human circadian system better than V(λ). Once that function was adopted, 'correction ratios' would be applied to photometric quantities based upon V(λ) in an attempt to more accurately represent the relative impact of the lamp spectrum and light level on stimulating the human circadian system.

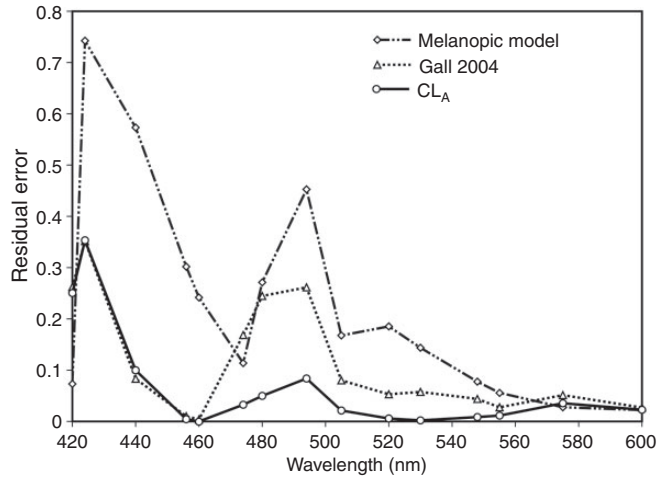

Figure 7 Residual errors from three models of the spectral sensitivity of nocturnal melatonin suppression obtained by Brainard *et al.*[6] and Thapan *et al.*[7] (adapted from Rea *et al.*[21]).

Gall and Bieske,[30] for example, using the empirical spectral sensitivity results from Figure 1, generated a 'connect-the-dots' luminous efficiency function. That is, Gall and Bieske[30] developed this function without any consideration to the neuroanatomy, neurophysiology, or the operating characteristics of the circadian system. Some have proposed using melanopsin as a luminous efficiency function.[31] As discussed earlier, a luminous efficiency function based upon a single photopigment for calculation and measurement methods would have no scientific grounding in the neuroanatomy, neurophysiology and operation characteristics of the circadian system. And this approach would actually be worse than one based upon the Gall and Bieske[30] 'connect-the-dots' function because it would more seriously misrepresent the spectral sensitivity of the circadian system (Figure 7). As already noted, the effectiveness of 'cool' light sources, narrowband or polychromatic, that might be preferentially selected for stimulating the human circadian system would not be properly characterized at any light level. Perhaps more insidiously, 'corrections' based upon melanopsin, unlike one based upon the strictly empirical Gall and Bieske[30] function, could mislead application engineers and designers into believing there was a scientific foundation for applying 'melanopic lux' to lighting.

More fundamentally, however, any 'correction ratios' applied to V(λ) based upon a single luminous efficiency function would necessarily assume that human circadian phototransduction is additive. If adopted, this assumption should be made explicit, thereby consciously dismissing the evidence for subadditive spectral sensitivity, illustrated in Figure 3.[26,32]

As another consideration, a unit convention would also need to be established if a luminous efficiency function was selected as characterizing the spectral sensitivity of the human circadian system. Conventional photometry includes a constant such that the luminous efficiency function is set, by definition, equal to 683 lm/W at 555 nm. This procedure can be readily applied to an additive luminous efficiency function,[19] but because of the resulting units, the photometric quantities generated with a luminous efficiency function based upon Gall and Bieske[30]

or upon melanopsin would be much larger than comparable quantities based upon the photopic luminous efficiency function. This is simply an artefact of the very low efficiencies at 555 nm for both the Gall and Bieske[30] and melanopsin functions. This disparity between photopic and circadian light quantities is not a major hurdle, but users would need to get used to these new quantities, analogous to users becoming familiar with temperature measurements in Celsius rather than Fahrenheit.

In addition to considerations given to representation of the spectral sensitivity of the human circadian system, the response of the system to different amounts of light also needs to be considered. Once 'correction ratios' for V(λ) were adopted to account for different lamp spectra, so would *post hoc* 'corrections' need to be adopted and applied to recommended photopic illuminance levels. This approach could become quite cumbersome, but would be necessary to prescribe, and thereby achieve, a particular design goal. One might, as did Rea and Figueiro,[28] want to estimate a threshold light level for melatonin suppression such that lighting in homes would not stimulate the circadian system. Rea and Figueiro used a CS value of 0.05 (equivalent to 5% melatonin suppression) as a target threshold. This CS threshold criterion translates into 50 photopic lux at the cornea (not on the horizontal work surface) of the 'warm white' light source in Figure 5 but only 26 photopic lux of the 'cool white' light source. If such recommendations were developed, which seems very important, both the spectrum and the amount of light would need to be 'corrected'.

The second approach is to embrace the CS metric. It is the most accurate, the most complete, and the only validated approach for characterizing a light stimulus as it affects human melatonin suppression and perhaps the human circadian system more broadly. Importantly, it is also constrained by the neuroanatomy, neurophysiology and the operating characteristics of the circadian system. Although the CS metric is based upon a non-linear calculation method, users need not be bothered with a hand calculator or slide rule to compute quantities. A spreadsheet is publicly available (http://www.lrc.rpi.edu/programs/lightHealth/index.asp) and CS can be readily calculated for any light source at any photopic light level. Portable spectroradiometers and software make measurement of CS as easy as measuring photopic illuminance. Also, a convention for CL_A units has already been developed (equation (1)) so that these quantities are quite similar to those one would obtain for the same amount of photopic illuminance.

More importantly, the CS metric has been successfully applied to quantify light interventions in several laboratory studies, including three studies of self-luminous displays,[9,33,34] and in several field studies, including nuclear submarines,[35] senior facilities for persons with Alzheimer's disease[15,36] and offices.[18,37] In the laboratory studies, light stimuli from self-luminous displays were measured using the Daysimeter,[38,39] a calibrated device placed near the cornea during the experiment that was used to calculate CS exposure. For example, Wood *et al.*[9] showed that melatonin suppression from self-luminous displays after subjects were using the devices for one hour was, on average, 3%. The actual average measured melatonin suppression was also 3%. In the field applications, it was hypothesized that a CS value of at least 0.3 would be enough to promote entrainment in various populations, and as a result of this increased entrainment, sleep, mood and behaviour would be improved. In three published studies, Figueiro *et al.*[15,36,40] showed that exposing Alzheimer's disease patients to a CS of 0.3 or higher from waking to 6:00 pm improved sleep quality, increased sleep duration, and reduced symptoms of depression and agitation. Similarly,

Young *et al.*[35] showed that exposing submariners to a lighting installation delivering a CS of at least 0.3, increased measures of circadian entrainment (increased total melatonin in first morning urine void), increased sleep efficiency, as measured by actigraphy and reduced self-reports of sleepiness during the shift. Finally, recent data from Figueiro and Rea[37] showed that office workers in summer months had greater sleep duration and efficiency than in winter months. During the summer months, the average CS values were close to 0.3 while the average CS exposure in winter months was less than 0.2. Unpublished data (manuscript in preparation) using close to 200 subjects working in five different federal buildings show that, compared to those receiving a CS of 0.15 or less in the morning hours (between 8:00 am and noon), office workers who receive a CS greater than 0.3 in the morning hours fell asleep earlier, reported having better sleep quality, less disturbed sleep, and feeling less depressed.

While no studies to date have used the melanopic lux metric in the field, Revell *et al.*[8] in a laboratory study showed that subjective alertness was significantly lower under a 479-nm narrowband light source than under a 437-nm and a 532-nm narrowband light source, as well as under two polychromatic white light sources (4000 K and 17000 K) that were photon matched to similarly stimulate the melanopsin photopigment. In another study, Hommes and Gimenez[41] performed a re-evaluation of existing published data where the Karolinska Sleepiness Scale (KSS) was used to assess subjective alertness under various lighting conditions. When delta KSS values were plotted against a melanopic illuminance function, the fit was better than when compared to the photopic illuminance function. These results were as would be expected, confirming that the circadian system is sensitive to short-wavelengths. In terms of modelling, Bellia and Seraceni[32] showed predictions from a simplified, additive luminous efficiency function (Gall[42]) were well correlated with the non-linear predictions from CL_A. However, none of these studies offers insights into the spectral or absolute sensitivities of the phototransduction mechanisms. Importantly, none of these studies contradict the quantitative predictions from equations (1) and (2).

In summary, the CS metric has been shown in laboratory studies to be predictive of nocturnal melatonin suppression, but perhaps more importantly, in the application studies, the CS metric has been shown to be predictive of clinically relevant outcomes, such as alignment of melatonin rhythms with sleep opportunities,[35] reductions in depression and agitation among persons with Alzheimer's disease,[15,36,40] and better sleep at night for people working in offices during the day.[18,37]

4. The central question

We recommend that open discussions among manufacturing, research and application stakeholders about characterizing light for the circadian system in various applications begin to take place. The model of human circadian phototransduction[10] may not be easy to understand for some metrologists and practitioners[19,43] but difficulty in understanding should not be a reason to adopt an alternate approach that is knowingly cumbersome and without validation. Importantly, the CS metric developed from the model has been shown to be predictive of acute melatonin suppression in laboratory studies and of clinical and sleep outcomes in field studies. Even if an approach where *post hoc* 'corrections' to conventional photometric quantities was taken,[19] it would still be necessary to have those 'corrections' guided by data where the spectral and absolute sensitivities were obtained (e.g., Figures 3 and 5).[32,43] Therefore, it would be difficult to ignore the science when developing 'correction' metrics

for engineering and applying circadian-effective light.

Not everything is known about circadian phototransduction, but the science is mature enough now for lighting stakeholders to collectively make a conscious, informed, and documented decision as to which approach to take. Moving forward, the discussion will boil down to one question:

> Should we adopt an approach based upon conventional photometry employing *post hoc* 'correction' factors guided by scientific evidence or should we embrace a completely new approach to computing and measuring light grounded in that scientific evidence?

5. Important caveats

We recognize, as with all areas of research, that our knowledge about the human circadian system is incomplete.[32] There is some evidence, for example, that neural signals from the retina reach the SCN without conduction through the ipRGC axons, but these connections are limited.[44] We also recognize that the CS metric is incomplete. Other lighting characteristics that affect the circadian system (timing, duration, distribution and photic history) are not considered in the CS metric. Moreover, no consideration is given to the spectral and the absolute sensitivity of the non-visual alerting effects of light exposure. This is an important outcome measure because 40 lux to 200 lux at the eye of a 640-nm (red) light has been shown to enhance brain activity during the day and night without affecting melatonin concentrations.[45,46] From a practical perspective, the spectral reflectances of architectural materials must also be considered in the CS calculations[20] as well, of course, as the integration of non-visual and visual effects from architectural lighting. Finally, the ideas expressed here are based upon the authors' experience and insights as both scientists and application engineers. These ideas, however, have not been vetted among the entire community of lighting stakeholders. Hopefully, the present paper provides important points for that collective discussion.

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