Circadian Responses to Fragmented Light: Research Synopsis in Humans

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Light is the chief signal used by the human circadian pacemaker to maintain precise biological timekeeping. Though it has been historically assumed that light resets the pacemaker’s rhythm in a dose-dependent fashion, a number of studies report enhanced circadian photosensitivity to the initial moments of light exposure, such that there are quickly diminishing returns on phase-shifting the longer the light is shown. In the current review, we summarize findings from a family of experiments conducted over two decades in the research wing of the Brigham and Women’s Hospital that examined the human pacemaker’s responses to standardized changes in light patterns generated from an overhead fluorescent ballast. Across several hundred days of laboratory recording, the research group observed phase-shifts in the body temperature and melatonin rhythms that scaled with illuminance. However, as suspected, phase resetting was optimized when exposure occurred as a series of minute-long episodes separated by periods of intervening darkness. These observations set the stage for a more recent program of study at Stanford University that evaluated whether the human pacemaker was capable of integrating fragmented bursts of light in much the same way it perceived steady luminance. The results here suggest that ultra-short durations of light—lasting just 1-2 seconds in total—can elicit pacemaker responses rivaling those created by continuous hour-long stimulation if those few seconds of light are evenly distributed across the hour as discreet 2-millisecond pulses. We conclude our review with a brief discussion of these findings and their potential application in future phototherapy techniques.

INTRODUCTION

The circadian pacemaker is a biologically conserved timekeeping system that evolved to anticipate daily recurring changes in the environment produced by the solar light-dark cycle [1]. This anticipation grants organisms, from unicellular prokaryotic cyanobacteria to multicellular eukaryotes, the ability to optimize timing of activities critical for reproductive success and survival [2]. In mammals, the light and dark signals that serve to synchronize the central pacemaker are processed through the retinohypothalamic tract [3-5]. Photoreceptors in the retina provide the sole input into this pathway, which sends information about light transitions in the environment (i.e., dawn and dusk) and photoperiod length directly to the central pacemaker neurons seated within the supra-

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†Abbreviations: SCN, suprachiasmatic nucleus; CBT_min, core body temperature minimum; DLMO, dim-light melatonin onset; h, hour; LED, light-emitting diode; PRC, phase response curve.

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THE PHASE RESPONSE CURVE TO LIGHT

The pacemaker’s timekeeping adjustments to natural light are often modeled by a sinusoidal-like phase response curve (PRC) that has been documented in many species to date through the use of electric light sources (Figure 1a) [15]. Electric light administered in the first half of the evening invariably triggers a phase delay in one’s subjective schedule of physiology and behavior, while later administration in the second half causes a phase advance (by convention, the magnitude of the delay and advance shifts mobilized by light are plotted with negative and positive numbers, respectively; Figure 1a) [16,17]. The shape of the PRC, which can be adapted to different seasonal photoperiods, likely reflects an entrainment logic designed to maintain the temporal niche of an animal [18]. If light in the early evening is perceived as an extension of the sunset—and that seen in the latter half the leading edge of a sunrise—then such phase regulation would maximize the contact that diurnal animals have with the sun and minimize the exposure of nocturnal animals. The amplitude characteristics of the PRC, though different in its particulars from one species to the next, bear out this suggestion.

The most sensitive parts of the delay and advance zones can be found several hours after dusk (Figure 1b) and several hours before dawn (Figure 1c), respectively. If electric light presented in these areas affects the circadian system the same as sunlight, then this would alter the pacemaker’s interpretation of the solar cycle, forcing it to manufacture large 2-4-h phase jumps in order to realign the timing of its internally created day and night to the newly perceived twilight zones. This is, in fact, what happens after light stimulation in commonly used animal models such as Drosophila, rodents, as well as humans [16,19-23]. When electric light exposure occurs within the less sensitive parts of the PRC in the 1-2 h of the night bookending dusk (lights-off) or dawn (lights-on), smaller phase shifts commensurate in magnitude with the difference in timing between the photic stimulation and the offsets/onsets of the light schedule are also routinely observed (Figure 1b-c). The picture that emerges with the PRC is a simple one: with the addition of photoreceptor input, the central pacemaker has become an exquisitely sensitive sunrise and sunset detector. To withstand natural selection, nearly every surface dwelling animal on the planet was obliged to use the same exact data stream of twilight information to measure lapses in time, whether their species started with the necessary nervous system hardware (or clock molecules) to do so or not. Because of this, circadian pacemakers are considered an exemplar of convergent evolution [24].

ATTEMPTS TO EXPLAIN CIRCADIAN RESPONSES TO LIGHT THROUGH RECIPROCITY: ANIMAL STUDIES

Despite the progress that has been made in elucidating the nature of the molecular machinery responsible for circadian oscillations in cells [25-27], relatively little is known about how the pacemaker, as a “systems unit,” samples from the naturally occurring light and darkness it receives, and then factors in differences in irradiance, spectral quality, and probability of exposure to deduce geophysical time. One strategy that many investigators have used to begin to deconstruct this logic is to assess what parameters guide the pacemaker’s short-term reaction to nighttime photic stimulation with incandescent or cool fluorescent lamps [28]. Construction of electric light PRCs across a variety of animal models has continued to reaffirm that the single most important variable driving the magnitude and direction of a phase shift is the precise timing of light administration [15,29]. However, within the same timeframe of the subjective night, other photic variables have also emerged that further influence phase-shifting. These include the spectral weighting of a light pulse and its brightness or length [30,31], along with the light level or duration of lighting under which a person or animal is housed (i.e., dim light histories in either case amplify phase-shifting responses to sub-saturating light stimuli) [32,33]. Based on knowledge regarding the biophysical properties of ocular photoreceptors, recent studies have expanded the parameter space in sophisticated ways by showing for instance that different sequences of monochromatic light designed to activate—and then re-sensitize—pacemaker targeting photoreceptors can augment phase resetting by maximizing information flow to the pacemaker [34,35].

Recent observations notwithstanding, the historical dogma that has organized much of the study on the pacemaker’s relationship with light and darkness is summarized by the reciprocity hypothesis. It postulates that any shift made in response to light at a fixed point in the subjective night results solely from the number of photons
Figure 1. A canonical phase response curve to light. (a) Phase-shifts of physiological/behavioral rhythms that result from electric light exposure at night are typically charted with the magnitude of the shifts shown on the y-axis against the timing of light administration on the x-axis. Light’s ability to trigger a circadian response is gated throughout the night, with particular pockets of sensitivity occurring several hours after dusk (b) or several hours before dawn (c). The pattern of these responses suggests that the pacemaker’s photic sensitivity is scaled so that light exposure produces shifts that will always align the pacemaker’s daily rhythm to the transitions of the light-dark cycle under which a person or animal is kept. Please note that not all human PRCs have a “dead zone” of photic insensitivity occurring during the subjective day. Depending on the stimulation protocol, some may take highly linear shapes, with a constant negative slope traveling between the advance and delay regions [73]. Figure adapted from [70].
that are registered by the pacemaker: the brighter or longer the pulse, the greater the resulting phase jump one should see up to some pseudosaturation level [36]. The implications of the reciprocity hypothesis for how the pacemaker uses sunlight in the real world to orient circadian timekeeping have never been directly articulated. Perhaps a safe assumption would be that the mere presence of light is enough to convince the pacemaker that the timing of dawn and dusk has migrated and that the size of this “bet” scales with irradiance and perceived illumination. Albeit simplistic, the general principle of reciprocity appears to hold when conventional electric lighting is shown to animals for periods exceeding 5 minutes up to about an hour. That is, the pacemaker will integrate broadband input the same way over this span such that different trains of non-saturating pulses from ~5-60 min will elicit the same final phase shift as long as the overall photon flux is conserved [36-39]. Once a saturation point is hit, whether it be from a short, bright pulse or a long, dim one, the rate at which the phase-shifting response increases with further stimulation approaches zero.

**RECIROCITY IN HUMANS**

The proposition of testing the reciprocity hypothesis in humans is complicated by the logistics of maintaining people under free-running conditions and tracking bio-markers closely associated with the endogenous phase of the pacemaker (most commonly body temperature or dim-light melatonin onset, DLMO; [40,41]). For these reasons, investigators ordinarily don’t have the opportunity to assess light-induced circadian responses at hourly intervals of the subjective night, but must depend on more general assessments of the delay and advance areas of the PRC using blunter, less precise light regimens (for exceptions, see [42,43]). Often but not always, there is less description of the exact PRC transition where the mode of circadian resetting changes from delay-to-advance. Previous data suggest that this crossover does tend to coincide with a person’s core body temperature minimum (CBT$_{min}$) [44-46], which in most individuals occurs near the end of nighttime sleep around 4:00-5:00 in the morning (marked by a fulcrum in Figure 2a).

Within this framework, Charles Czeisler and colleagues from the human chronophysiolog lab at Brigham and Women’s Hospital have conducted what is arguably the longest-running series of experiments to quantify the effects of light and darkness on the human circadian system [22,23,47-50] (light protocols are illustrated in Figure 2, panels b-g; the results from these protocols are enumerated in the Table). During the late biological night, the research team exposed cohorts of healthy volunteers to 5 h of white fluorescent light centered 1.5 h after the CBT$_{min}$ for 3 consecutive days. The magnitude of pacemaker advances to different light levels ranging from 12-9500 lux in these 5-h blocks was measured (Figure 2b-d). Conversely, in the several hours of the early subjective night preceding the CBT$_{min}$, other cohorts were exposed to 6.5-h dim or bright light (0-9100 lux), or to different durations of 10,000 lux light, to evaluate how increasing photon count with brighter—as opposed to longer—pulses impacted delay resetting (Figure 2e-g). The results of these experiments spoke to several broad themes. First, in line with the reciprocity hypothesis, subjects that were exposed to higher illumination in the early or late biological night showed greater resetting [22,23,47] (Figure 2b, c, and e; Table 1, rows 1-8 and 12-13). Both half-maximal phase-delays and advancing responses were achieved with approximately 100 lux, with logistic curves best describing the relationship between light level and phase shift of the melatonin rhythm [22,23]. Second, longer (as opposed to briefer) pulses of the same illumination were more effective phase-shifting agents in the early evening before CBT$_{min}$. Subjects shown 12 min of bright light exhibited significant 1-h delays in melatonin secretion, whereas those shown longer pulses (exceeding 2 h) had delays that were two-and-a-half times larger [48] (Figure 2f; Table 1, rows 14-17). The duration-response relationship for light delivered in the early evening, like the illuminance-response relationship, could be modeled with high accuracy by a multi-parameter logistic function [22,48]. It is worth noting that the reciprocity trends documented by the Czeisler group are visible in the overall shape of the human PRC to broad spectrum fluorescent light that has been charted by several research teams. As the length of a 5,000-12,000-lux probe stimulus increases from 1 to 7 h, so does the peak-to-trough amplitude of the PRC (i.e., summed height of the advance and delay regions) expand from about 2.2 h to 5.5 h [20,43-46].

**RECIPROCITY IS NOT THE WHOLE STORY: THE EFFECTS OF LIGHT FRACTIONATION**

A third feature of photic resetting that fell out of the Czeisler experimental series was the observation that, after a particular threshold of exposure, further introduction of light produced diminishing returns on phase movement (i.e., the more the light was shown, the less the photic information got translated into the magnitude of the phase shift; Figure 3, insert) [47,48,51]. This meant that shorter durations of light, or dimmer pulses, had significantly greater resetting capacity per min of exposure or per lux invested than longer/brighter pulses. It also meant that the human pacemaker was most impressionable to information available at the beginning of a light stimulus (just as it was in non-human animals; [39]). The nonlinear duration-response function to light prompted Richard Kronauer, a colleague of Czeisler’s, to develop a mathemat-
Figure 2. Minireview of human reciprocity experiments. (a) Schematic for the boundaries of the delay and advance zones in the human PRC to light. Coordinates revolve around the timing of a person’s CBT_{min} (represented by black triangles; [44-46]). Evening light exposure prior to CBT_{min} delays the phase of the pacemaker rhythm; exposure after CBT_{min} phase advances it. Though human phase-shift experiments are ordinarily done under free-running conditions, for perspective, the delay and advance regions here are illustrated with respect to where they would occur under entrainment to the solar light-dark cycle. (b-d) Diagrams of phase-shifting experiments conducted in the pacemaker’s advance zone. Light exposure is marked with rectangles (red = dim light; orange = medium amount of light; yellow = bright light; yellow/black = intermittent bright light protocol). Illuminance-response relationships for light-induced resetting of endogenous rhythms of body temperature or melatonin were standardized against a protocol where volunteers received a 5-h pulse centered 1.5 h after CBT_{min} on 3 consecutive days [23,47]. The effects of fragmenting the 5-h pulse into briefer episodes of intermittent light sandwiched between periods of complete darkness were examined for the brightest light condition (i.e., 9500 lux, [49]). (e-g) Diagrams of phase-shifting experiments conducted in the pacemaker’s delay zone. Illuminance and duration-response relationships for light-induced resetting of melatonin rhythms were standardized against a protocol where volunteers received a onetime 6.5-h pulse terminating about half-an-hour before CBT_{min} [22,48]. The effects of fragmenting the 6.5-h pulse into briefer episodes of light separated by darkness were examined for the brightest light condition (i.e., 10,000 lux, [50]).
Table 1. Data from human reciprocity experiments.

| Reference | Stimulation Protocol | Light Exposure | Physiology |
|-----------|----------------------|----------------|------------|
| 1 Boivin et al., 1996 | Uniform stimulation, once per day for 3 consecutive days | 180 lux, 5.0 hours | 69.6 min |
| 2 Boivin et al., 1996 | Uniform stimulation, once per day for 3 consecutive days | 1260 lux, 5.0 hours | 161.4 min |
| 3 Boivin et al., 1996 | Uniform stimulation, once per day for 3 consecutive days | 9500 lux, 5.0 hours | 269.4 min |
| 4 Zeitzer et al., 2005 | Uniform stimulation, once per day for 3 consecutive days | 12 lux, 5.0 hours | -41.4 min |
| 5 Zeitzer et al., 2005 | Uniform stimulation, once per day for 3 consecutive days | 180 lux, 5.0 hours | 108.0 min |
| 6 Zeitzer et al., 2005 | Uniform stimulation, once per day for 3 consecutive days | 600 lux, 5.0 hours | 225.0 min |
| 7 Zeitzer et al., 2005 | Uniform stimulation, once per day for 3 consecutive days | 1260 lux, 5.0 hours | 166.8 min |
| 8 Zeitzer et al., 2005 | Uniform stimulation, once per day for 3 consecutive days | 9500 lux, 5.0 hours | 259.8 min |
| 9 Rimmer et al., 2000 | 5-min stimulation alt. with 20-min of rest for 5 hours (once per day for 3 consecutive days) | 9500 lux, 1.6 hours | 172.2 min |
| 10 Rimmer et al., 2000 | 46-min stimulation alt. with 44-min of rest for 5 hours (once per day for 3 consecutive days) | 9500 lux, 3.2 hours | 234.0 min |
| 11 Rimmer et al., 2000 | Uniform stimulation, once per day for 3 consecutive days | 9500 lux, 5.0 hours | 271.2 min |
| 12 Zeitzer et al., 2000 | Uniform stimulation (one day of exposure) | 106 lux, 6.5 hours | -108.0 min |
| 13 Zeitzer et al., 2000 | Uniform stimulation (one day of exposure) | 9100 lux, 6.5 hours | -192.0 min |
| 14 Chang et al., 2012 | Uniform stimulation (one day of exposure) | 10,000 lux, 0.2 hours | -64.2 min |
| 15 Chang et al., 2012 | Uniform stimulation (one day of exposure) | 10,000 lux, 1.0 hour | -93.0 min |
| 16 Chang et al., 2012 | Uniform stimulation (one day of exposure) | 10,000 lux, 2.5 hours | -137.4 min |
| 17 Chang et al., 2012 | Uniform stimulation (one day of exposure) | 10,000 lux, 4.0 hours | -159.0 min |
| 18 Gronfier et al., 2004 | 15-min stimulation alt. with 60-min of rest for 6.5 hours (one day of exposure) | 10,000 lux, 1.5 hours | -140.4 min |
| 19 Gronfier et al., 2004 | Uniform stimulation (one day of exposure) | 10,000 lux, 6.5 hours | -181.8 min |

*Delays are listed with negative numbers, advances with positive numbers. See Figure 2, for an illustration of each protocol's timing.*
ical model for how pacemaker photosensitivity changes throughout continued light exposure [52]. Broadly con-
strued, the Kronauer model proposes that light stimula-
tion always prompts an initial response by the pacemaker
that persists in decaying fashion for a period of time after
the stimulation has stopped (like the initial pedals of a
bicycle wheel). In order to optimize phase-shifting drive,
the onsets of the pulse must be long enough to reach full
phase-shifting strength and then be balanced with periods
of darkness so that steady activation of the pacemaker
can occur without triggering competing processes that
curb photosensitivity [52,53]. Implicit in this model is
the general hypothesis that continuous light exposure can
never reach the phase-shifting efficiency achieved with
interrupted pulses when the pattern of intermittent ex-
posure has been specifically tailored to match the rates
with which the pacemaker build-ups and exhausts the
responses set in motion by light. If true, the proposition
would overturn the notion that reciprocity is the chief
mechanism used by the pacemaker to compute the size
of a phase shift.

The Czeisler group tested these assumptions by
adapting their late and early subjective-night light ad-
ministration protocols [49,50]. In the advance region,
volunteers were given 3 consecutive days of 5-h white
fluorescent light that was broken up into 5-min ramping
pulses with 20 min of intervening darkness (31 percent
duty cycle), or 46-min ramping pulses with 44 min of
intervening darkness (63 percent duty cycle; Figure 2d;
Table 1, rows 9-10) [49]. In the delay region, a separate
cohort was given a 6.5-h regimen where 15-min blocks
of light alternated with 60 min of darkness (amounting
to a 23 percent duty cycle; Figure 2g; Table 1, row 18)
[50]. Ostensibly, intermittent light exposure was nearly
as effective a regimen as constant light in eliciting circadi-
ann responses. A 63 percent duty cycle during the late
biological night preserved almost 90 percent of median
resetting (Table 1, rows 9-11), while a 23 percent duty
cycle during the early biological night preserved about
80 percent of it (Table 1, rows 18-19) [49,50]. Howev-
er, a closer look at these numbers would suggest more
modest gains. The investigators, possibly owing to the
novelty of their study, did not consider the possibility that
the total light exposure accumulated over fragmented
stimulation was already closing in on levels that would
saturate phase-shifting. The insert in Figure 3 plots the
phase-shifting drive achieved in the delay and advance
zones with the intermittent versus continuous protocols
as a function of total light exposure (expressed in units
of time-integrated illuminance or luminous exposure,
lux \_ hour). Consistent with previous animal and human
data, the reset efficacy of both types of protocols decayed
exponentially with increasing amounts of light. Against
this backdrop, one might expect that intermittent photic
stimulation would counter the trend and achieve a reset
efficacy per quanta higher-than-expected for the amount
of light that was delivered. This was the case for the 23
percent duty cycle administered over the early biological
night (i.e., in the delay zone; Figure 3 insert, left panel,
grey data point), but was not obvious for either of the
fractionation strategies used in the late evening (i.e., in
the advance zone; Figure 3 insert, right panel, grey data
points). The larger takeaway from the Czeisler group’s
exploration of the Kronauer model is a positive one: in
principle, light fractionation is a strategy that can be used
to communicate more efficiently with the pacemaker.
Nevertheless, the length of the pulses (e.g., 15 min vs.
shorter blocks) and the breaks between them require fin-
er calibration to balance the rate constants that Kronauer
described.

Other characterizations of intermittent light and its
potential to treat circadian disorder have been spearhead-
ed by Rush University’s Biological Rhythms Research
Laboratory in another series of studies led by Charmane
Eastman and Helen Burgess (elegant applications can be
found in [42,54-56]). The Rush group’s studies have not
shown the same continuity as those reported by Czeis-
ler et al., who have historically used the same lighting
equipment to evaluate the circadian responses generated
by several interrelated stimulation protocols. However,
the Rush group’s varied approach has allowed them to
explore other aspects of intermittent light administration,
including constructing the first PRC to patterned expo-
sure to blue LED light (three 30-min pulses delivered
over 2 hours at \_185 lux, once a day for 3 days; [42]) and
the first adolescent PRC to bright fluorescent (5000 K)
light intermixed with periods of ambient room lighting
(4100 K) [55]. These experiments have been pivotal to-
dwards demonstrating the real-world applicability of light
fractionation from a commercially available LED unit as
a countermeasure to sleep-circadian disruption and have
provided some insight into how phototherapy might be
practiced in a population naturally vulnerable to circadian
misalignment (i.e., teenagers, whose delayed chronotype
is at odds with early school start times).

Buoyed by insights that were first made in wild bats
and lab rodents [57-61], light fractionation in circadian
phase-shifting is now being surveyed in humans at several
finer temporal resolutions, from milliseconds to seconds
[51,62-67]. As might be predicted from Kronauer-esque
drive models, the circadian effects of intermittent flashes
delivered in the millisecond range do not taper off with
repeated application and possess significantly more reset-
ting capacity than uninterrupted stretches of light that last
an hour, a minute, or just a few seconds [61,67-69]. How
much more capacity? The phase-shifting drive created by
millisecond photic exposure is sufficiently powerful that
it can’t be visualized properly on the same reset efficacy
Figure 3. Pacemaker responses to intermittent versus continuous light. (Insert) The efficiency with which continuous or intermittent light phase-shifts the human body temperature or melatonin rhythm is plotted as a function of total light exposure. Reset efficacy in the delay and advance zones is calculated by dividing the size of the shift observed (in minutes) by the amount of light used to produce it (in units of lux-hours × 100). Data in the insert are derived from the mean or median phase-shift values reported in [47-50] and summarized in Table 1, rows 1-3, 9-11, and 14-19. (Larger graph) The delay zone data in the insert is rescaled within the larger graph to accommodate the scales-of-magnitude difference in reset efficacy between hour-long and millisecond stimuli. Millisecond data, plotted with blue triangles, are derived from the average phase-shift values reported for individuals exposed to 2-msec flashes of xenon light (473-2995 lux) delivered once every 30-60s for an hour. Subjects were administered these flash sequences while awake [65] or asleep [66]. These regimens produced 30-45 min shifts of the melatonin rhythm.
body entrained to sunlight. In short, the pacemaker is trying to locate dawn and dusk, twilight sequences where light levels steadily (and then precipitously) increase or decrease and where the progression of these illumination changes is pegged to changes in the spectrum of incident light. When this concept is merged with the empirical data collected from light fractionation experiments, the two together suggest that there are multiple layers of temporal integration (threads) that go into pacemaker’s time-of-day estimates and that in this dynamic reading frame the pacemaker orients to combined fluctuations in light intensity, spectrum, duration, and probability of overlapping exposures. To track the movement of the sun, the pacemaker can resort to photon detection (irradiance); however, the precision of this tracking in nature is dependent on the inclusion of light characteristics such as color and on discrete episodes of light that are expected to follow the incremental flow of twilight at dawn or dusk [8]. What this perspective also raises is the prospect of artificially inserting information into the pacemaker’s reading frame for phototherapeutic application. If the photic information that is guiding the pacemaker’s phase response is naturally synthesized one syllable at a time before a coherent phase jump is triggered, then it is theoretically possible to modify each of those syllables along several parameters to refine the response so that it suits a particular individual or condition. With the commercialization of LEDs, devices which emit narrowband light with highly precise temporal control, future phototherapy protocols will be able to change stimulus parameters from one syllable to the next in a way that could not have been previously envisioned. Across milliseconds, light regimens can be assembled that specify particular sequences of flash exposure that morph in series, modifying parameters such as illuminance (e.g., 0–100 lx), pulse shape (e.g., patterns of intensity modulation within a pulse), width (e.g., milliseconds to seconds), color (visible spectrum, ~380-760 nm), UVA content (non-toxic part of UVA radiation spectrum, 365 nm), or interstimulus interval (temporal distance between syllables). When properly considered, the breadth of this parameter space is actually quite large. Nevertheless, if recent work is any indication,
we will—eventually—have to grapple with this complexity. By just modifying the interstimulus interval between the millisecond flashes in their hour-long light protocols (incorporating as few as 30 flashes), Najjar and Zeitzer showed that fractionation can not only increase the reset efficacy of light exposure per quanta but—in an absolute sense—produce larger delay shifts in people than those produced with 1 h of continuous stimulation with equiluminous light (this, despite the dramatic 3800× difference in exposure; see Figure 4 for a reproduction of data from 29 individuals [67], with reset efficacy plotted according to total illuminance). This finding poses a serious challenge to our current understanding of light’s effects on the human circadian system and opens a new field of inquiry regarding how high-speed administration of LED pulses can be deployed in the clinic to treat disorders. We will lose a valuable opportunity to help more patients if we don’t begin to consider the novel biological effects that might be afforded by newer precision light emission technologies such as LED lighting.

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