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CHAPTER 7

Can light make us bright? Effects of light on cognition sleep

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Abstract: Light elicits robust nonvisual effects on numerous physiological and behavioral variables, such as the human sleep–wake cycle and cognitive performance. Light effects crucially rely on properties such as dose, duration, timing, and wavelength. Recently, the use of methods such as fMRI to assess light effects on nonvisual brain responses has revealed how light can optimize brain function during specific cognitive tasks, especially in tasks of sustained attention. In this chapter, we address two main issues: how light impinges on cognition via consolidation of human sleep–wake cycles; and how light directly impacts on sleep and cognition, in particular, in tasks of sustained attention. A thorough understanding of how light affects sleep and cognitive performance may help to improve light settings at home and at the workplace in order to improve well-being.

Keywords: light; cognition; sleep; circadian clock; human.

Introduction

The 24-h reoccurrence of light and darkness represents the most systematic time cue on earth. Thus, it is not surprising that all living organisms integrated the light–dark cycle in their physiology and optimally adapted their anatomy and behavior to anticipate dawn and dusk. In humans, light is intuitively linked with an alert or wakeful state, whereas in nocturnal animals, the light phase comprises the rest phase, thus representing a different temporal niche for sleep. The human visual system is designed to accommodate the needs of a diurnal species from a visual perspective. However, also the nonvisual responses to light point to the role of light as the mediator of inducing daytime physiology in humans. Hormonal secretion, heart rate, body temperature, sleep propensity, alertness, pupillary constriction, and gene expression are all immediately influenced by light—or even hours after light exposure ended—in order to pursue optimal adaptation to

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the imposed light–dark cycle (Berson, 2003; Bromundt et al., 2010; Cajochen et al., 2005; Hatori and Panda, 2010; Lavoie et al., 2003; Muñoz et al., 2005).

Recently, these long-term and acute effects of light on physiology have been described as belonging to the non-image forming (NIF) system, given that these responses are not associated with the classical involvement of rod and cone photopigments (Guler et al., 2008). This has opened a new area of research, which, apart from the role of light in regulating circadian rhythms, focuses on the physiological and anatomical underpinnings of light in modulating sleep and cognition.

Thus, in this chapter, we address two issues: (1) how light impinges on cognition via consolidation of human sleep–wake cycles and (2) how light directly impacts on sleep and cognition, in particular, in tasks of sustained attention.

Effects of light on the circadian timing system and sleep–wake cycles

The response of the central circadian pacemaker located in the suprachiasmatic nuclei (SCN) to light pulses plays a crucial role in the synchronization to the environmental light–dark cycles. Light pulses presented during the subjective day rapidly induce expression of the immediate early gene *c-fos* (Rusak et al., 1990) and the clock gene *Per1* within the SCN (Albrecht et al., 2001; Edelstein et al., 2003), resulting in phase shifts of behavioral circadian rhythms. Thus, the mammalian *Per* genes are not only light-responsive components of the circadian oscillator but also are involved in resetting of the circadian clock (for a review, see Oster et al., 2002). In humans, exposure to light late in the biological day (dusk) leads to a delay in human sleep onset, while exposure to light early in the biological day (dawn) advances activity onset (for a review, see Czeisler and Gooley, 2007). This phase-shifting property of light denotes a clear NIF effect of light, which relies on circadian phase and substantially impacts on the temporal organization of sleep and wakefulness.

Thus, light as the major "Zeitgeber" (i.e., time giver) is a prerequisite for ideal synchronization between the temporal organization of sleep and wakefulness and the external light–dark cycle. As a consequence, attenuated Zeitgeber strength (i.e., not enough light, or light of an inappropriate wavelength, i.e., light at longer wavelengths, >600 nm) and light at inappropriate times (i.e., during the biological night) can lead to improper entrainment between internal (i.e., circadian) and external (i.e., 24-h earth rotation) time, which often occurs in people working on rotating shifts and/or older individuals as well as visually impaired people. There is ample evidence that timed light exposure is a successful countermeasure of circadian misalignment in shift work (Burgess et al., 2002). Exposure to bright light did not only stabilize sleep–wake rhythms in demented older people, but significantly attenuated the decline in mental capabilities over the investigated time span of 4.5 years (Riemersma-van der Lek et al., 2008). Similarly, bright light promoted circadian alignment and prevented the detrimental effects of night work on sustained attention, as measured in an increased response speed on the psychomotor vigilance test (Santhi et al., 2008). The beneficial effects of light on circadian entrainment clearly carry on to cognitive performance. Thus, in this way, it is rather the stabilization of sleep–wake rhythms that leads to better brain function, than the exposure to bright light *per se*. Strong evidence for this stems from the fact that, if sleep and wakefulness occur out of phase with internal biological time, this impairs several cognitive functions such as learning in humans (Wright et al., 2006). Further, fragmentation of the rest-activity rhythm correlates with age-related cognitive deficits (Oosterman et al., 2009). Thus, stable and consolidated circadian sleep–wake rhythms are an essential requirement for proper cognitive functioning in health and disease, and light is only a means to an end. However, there is recent evidence that light *per se* may

directly impinge on sleep, alertness, cognitive performance, and even mood levels probably even without its action via the central circadian pacemaker.

Light directly impacts on sleep and cognition

In nocturnal animals, there is recent evidence that the acute light-induction of sleep is mediated by melanopsin-based photoreception (Lupi et al., 2008; Tsai et al., 2009), potentially reflecting an additional clock-independent photic input to sleep. To our knowledge, a similar wakefulness-inducing effect of light during sleep has not been substantiated as such in the "diurnal" humans, as it is difficult to apply light while sleeping, and testing the effects of short-wavelength light during sleep is impossible because of the filtering properties of the eyelids (Moseley et al., 1988). Therefore, most of the evidence of acute light effects on sleep in humans comes from studies applying light shortly before or after sleep. Bright light in the morning has been shown to shorten sleep duration (Dijk et al., 1989) and advance circadian rhythms (i.e., melatonin profile), without effects on nonrapid eye movement (NREM) sleep homeostasis (Carrier and Dumont 1995; Dijk et al., 1989). An artificial dawn in the morning during the last 30 min of sleep caused more superficial sleep along with a faster decline in skin temperatures and less sleepiness after waking up (Werken et al., 2010). Despite positive effects on alertness in the morning, the use of the same artificial dawn during 2 weeks did not induce a significant change in circadian phase (Giménez et al., 2010). Bright light in the evening may lead to an increase in sleep latency to NREM sleep stage 2 (Cajochen et al., 1992; Carrier and Dumont 1995) and changes in the temporal dynamics of electroencephalographic (EEG) slow-wave activity (SWA), such that SWA is lower during the first and higher during the fourth NREM–REM sleep cycle as compared to dim light condition (Cajochen et al., 1992). Exposure to bright polychromatic light (2500 lux) in the morning (06:00 h–09:00 h) or in the evening (18:00 h–21:00 h) for 3 consecutive days can result in earlier sleep termination following morning light than after evening light (Gordijn et al., 1999). Interestingly, the duration of the first REM sleep episode was longer after morning light than after evening light, most likely due to a phase advance of the circadian influence on REM sleep production. This is supported by the observed advance of the circadian rhythm of melatonin. Thus, both sleep termination and REM sleep duration can be manipulated by light exposure. All these effects have been interpreted as reflecting a carry-over effect of light's alerting action into sleep (i.e., longer sleep latencies, reduced SWA in the first cycle with an intrasleep rebound of SWA in the last cycle), reflecting the repercussion of the immediate induction of a circadian phase advance or delay on the following sleep episode, and showing the positive effects of artificial dawn on the dissipation of sleep inertia after awakening, which could not be explained by circadian mechanisms.

Circadian and homeostatic influences on the alert state

Wakefulness is a construct associated with high levels of environmental awareness, which can be tracked down by a wide array of responses, such as subjective perception, and behavior, subcortical and cortical activity (Buysse et al., 2003). Basically, it comprises self-reported low levels of fatigue or sleepiness, fast and more accurate responses in tasks of sustained attention, low power densities in the EEG theta frequency range (4–8 Hz), and high power densities in the EEG beta frequency range (12–30 Hz; Badia et al., 1991; Daurat et al., 2000). Subjective perception of wakefulness crucially relies on a time-of-day dependency, to the extent that diurnal fluctuations of alertness follow similar dynamics of core body temperature (CBT), with

its maximum in the evening and nadir in the early morning (Kleitman, 1987).

Two important protocols have been developed in order to dissect out the relative contributions of circadian and sleep–wake homeostatic processes in humans: the constant routine and the forced desynchrony protocols (Duffy and Dijk, 2002). In the first, the amplitude and phase of many circadian rhythms can be elucidated without the masking effects of food, posture, light, and so forth. In the second protocol, subjects live on artificially very long or very short days so that the circadian system is no longer entrained to the imposed sleep–wake cycle. The desynchronized/synchronized subjects sleep at different circadian phases of the entire 24-h cycle, which enables to differentiate the contribution of the sleep homeostatic process or the circadian system to a given variable (Dijk et al., 1997). Usually—but not always—both factors contribute substantially to measures as alertness, mood, and neurobehavioral performance (Cajochen et al., 1999a, b; Koorengevel et al., 2003). Accordingly, some forced desynchrony studies have revealed that the deleterious effects of prior wakefulness on alertness were strongest during the minimum of the endogenous CBT rhythm. This strongly indicates that optimal levels of alertness can be achieved when the phase relationship between the endogenous circadian timing system and the sleep–wake cycle is such that the former opposes the wake-dependent deterioration of alertness and performance, as conceptualized in the "two-process" model (Borbély 1982; Daan et al., 1984). The most effective means of obtaining this occurs when the waking day starts ~2 h after the endogenous circadian minimum of CBT rhythm, which corresponds to ~2 h after the circadian maximum of the plasma melatonin rhythm. Taken together, this implies that the circadian process plays a wake-promoting role that counteracts the accumulating homeostatic drive for sleep during wakefulness. The result of the interaction between these two fundamental properties emanating from the central nervous system is that humans are able to maintain alert wakefulness for ~15–17 h (Czeisler et al., 1994; Dijk and Czeisler 1994). Considering the temporal dynamics of these processes on alertness, one can hypothesize that light exerts its alerting effects most strongly when the circadian drive for sleep is at its maximum (i.e., in the early morning at the CBT minimum), under high homeostatic sleep pressure (i.e., after more than 16 h of wakefulness).

The impact of light on sleep and wakefulness, however, does not happen in a homogenous manner for all types of light exposure, but intimately depends on intensity, timing, duration, and wavelength. Thus, in the next sections, we describe how these aspects of light exposure can play a crucial role on wakefulness.

Do light effects impact on sleep and wakefulness irrespective of timing?

While applying light during sleep is difficult in humans, measuring light effects during wakefulness is easy, but its interpretation rather multifaceted, since factors such as the duration of prior wakefulness, endogenous circadian phase, and prior environmental light exposure all interact with each other.

Most studies on the effects of light in humans are conducted at night (Badia et al., 1991; Campbell and Dawson 1990; Foret et al., 1996; Lockley et al., 2006). Considering how light acts on the circadian and homeostatic systems (as described in the previous section), one does in fact expect that, even during the biological night in extended wakefulness (when sleep pressure is rising), light can dramatically enhance subjective alertness and reduce objective markers of sleepiness (e.g., slow eye movements). From a circadian perspective, there is a considerable body of evidence that suggests that, at night, the strong melatonin suppression caused by exposure to high-intensity light may be one of the underlying mechanisms (Cajochen et al., 1998; Sack et al., 1992). Although not consistently shown, it has

been hypothesized that melatonin attenuates SCN-dependent mechanisms responsible for promoting and maintaining cortical and behavioral arousal at particular times in the circadian cycle (Cajochen et al., 1999a,b; Dijk and Czeisler 1995; Sack et al., 1997; Wright, 1997).

However, it is very likely that there are additional mechanisms that mediate the alerting effects of light. Light exposure at night on the nasal part of the retina does induce suppression of melatonin, without effects on alertness (Ruger et al., 2006). Also during the biological day, when melatonin is at minimal levels, light does impact on alertness. A study in which participants were exposed to either bright light (5000 lux) or dim light <10 lux (control condition) either between 12:00 and 16:00 h or between 00:00 and 04:00 h showed that bright light had a time-dependent effect on heart rate and CBT, such that bright-light exposure at night, but not in daytime, increased heart rate and increased CBT. However, the effects of bright light on the psychological variables was time independent, since both nighttime and daytime bright light reduced sleepiness and fatigue significantly and similarly (Fig. 1a and b; Ruger et al., 2006).

Further evidence in support of daytime effects of light effects on alertness was found in an "in-lab" study, with daytime exposure to short (21 min) white light at >7000 lux; cortical activity was enhanced during an oddball task and subjective alertness improved in a dynamic manner (Vandewalle et al., 2006). This suggests that light may modulate activity of subcortical structures involved in alertness, thereby promoting cortical activity in networks involved in ongoing nonvisual cognitive processes.

It remains inconclusive and controversial whether the timing of light exposure does have an impact on human sleep. For instance, in an ultrashort sleep–wake schedule (Kubota et al., 2002), exposure to evening bright light (5000 lux) delayed the diurnal fluctuation of sleep propensity, which suggests that light may have the potential to phase-advance or -delay sleep phase in a similar manner as to the phase-response curve (PRC) derived from CBT—or melatonin—rhythm. Melatonin has been hypothesized to act as a mediator to convey the output of the circadian pacemaker to the sleep–wake system (Lavie, 1997). Nocturnal bouts of sleep propensity observed in an ultrashort sleep–wake schedule seem to occur in parallel with an increase in melatonin secretion near to habitual bedtime (Kubota et al., 2002). However, it is also likely that the magnitude of the phase change in melatonin secretion after bright-light exposure may not correlate with that in sleep propensity rhythm. An alternative hypothesis suggests that an indirect effect of melatonin on sleep–wake cycle mediated via temperature may be essential in considering coupling mechanisms between the circadian pacemaker and sleep, since melatonin acts strongly on the temperature rhythm (Van Someren, 2000).

However, timing *per se* is not the only factor one should bear in mind when considering how light impacts on alertness. Since the aforementioned studies used polychromatic light above 1000 lux, it may be that the intensity of the light exposure is, in fact, responsible for such alerting effects, which appear to be irrespective of time-related dependency. This leads to the next question: What is the threshold of light intensity that can keep us awake?

Dose–response effects of light: Is there a saturation point?

Light is a powerful synchronizer which resets the endogenous circadian pacemaker to the 24-h day in an intensity-dependent manner. Although it is clearly recognized that bright light (1000 lux or more) is an effective synchronizer in humans, one might believe that the human circadian pacemaker is insensitive to lower levels of light illumination (i.e., <100 lux). However, the relationship between the resetting effect of light and its intensity follows a compressive nonlinear function,

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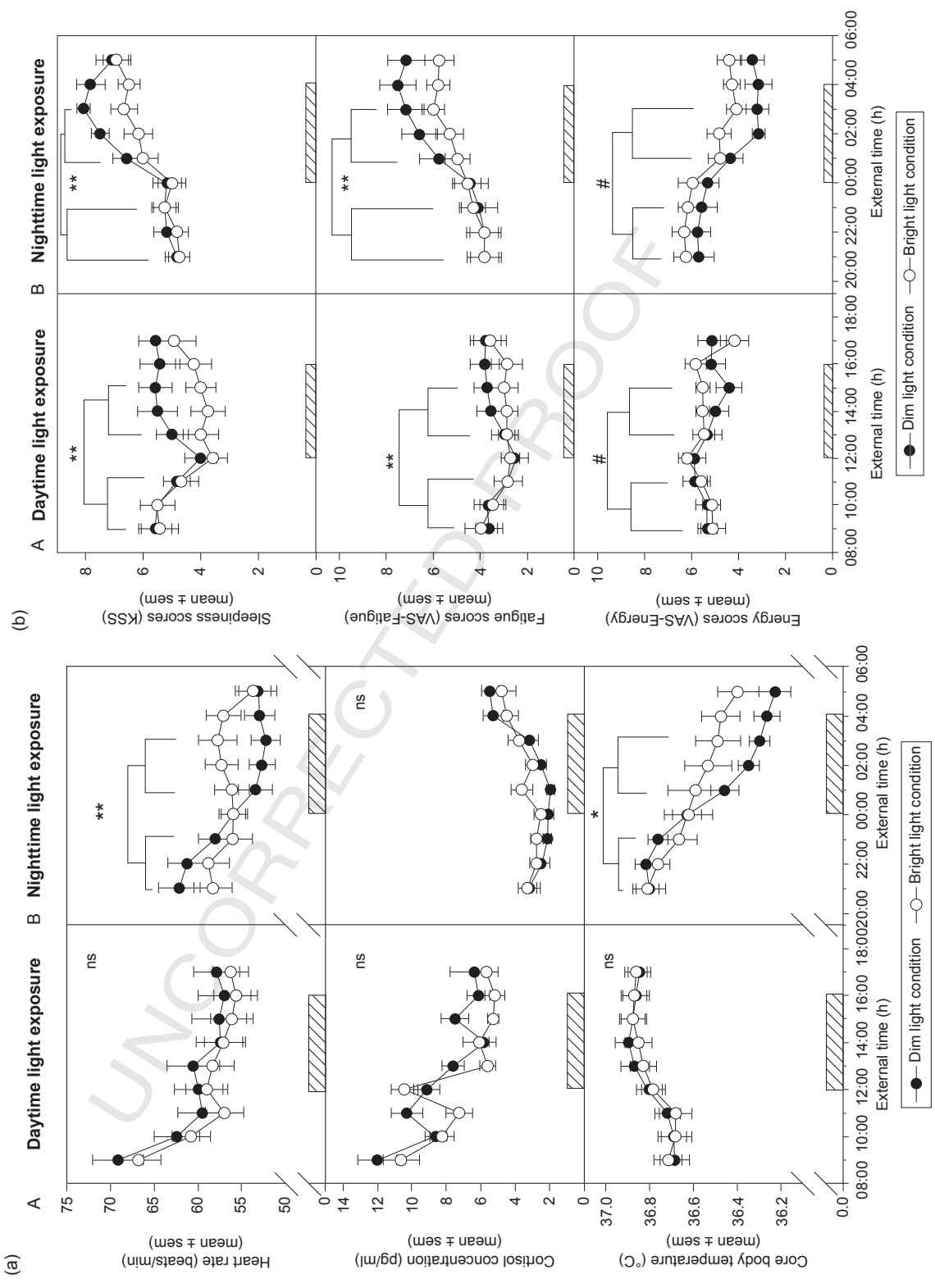

Fig. 1. (a) Time course of heart rate, cortisol concentration, and core body temperature for the two experiments, before and during bright-light exposure versus dim light. Hatched bars, period of light exposure (daytime experiment: noon until 4:00 p.m.; nighttime experiment: midnight until 4:00 a.m.; Ruger et al., 2006). (b) Time course of subjective sleepiness, fatigue, and energy for the two experiments, before and during bright-light exposure versus dim light. Hatched bars, period of light exposure (daytime experiment: noon until 4:00 p.m.; nighttime experiment: midnight until 4:00 a.m.; (Ruger et al., 2006).

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such that exposure to lower illuminances still elicits a strong effect (Boivin et al., 1996). Indeed, the dose–response function to a single episode of light prior to CBT nadir is such that 50% of the maximal resetting response to bright light at 9100 lux can be obtained with dim room light (100 lux; Cajochen et al., 2000; Zeitzer et al., 2000; Fig. 2). Further, humans have the capacity to keep stable entrainment to a 24-h cycle even when ambient light levels are around 1.5 lux, which suggests that also low-lit environments can induce small shifts in the circadian system (Duffy and Wright 2005).

The phase-shifting dose–response function to light is fairly similar to the dose–response function for the alertness enhancing effects of light (Zeitzer et al., 2000). In other words, nighttime exposure to typical room light can exert an alerting effect in humans, as indexed by lower subjective ratings of sleepiness, less slow eye movements, and less theta and alpha waking EEG activity. Taken together, this implies that the answer for the title of this section is yes—there is a saturation point for light impacts on alertness, and this high sensitivity may explain why, sometimes, a direct effect of light has not been observed, given that these light effects were compared to dim light sufficient to elicit near maximal effects (Dollins et al., 1993; Myers and Badia 1993).

If even low-light intensities can keep us awake, could it be that the duration of light exposure and/or our prior light history also matter and not only the intensity of light? In the next section, we will delineate some possible answers.

Duration of light and prior light exposure

A recent systematic evaluation of the duration dependence of the circadian resetting responses of a single dose of light in mice shows that light

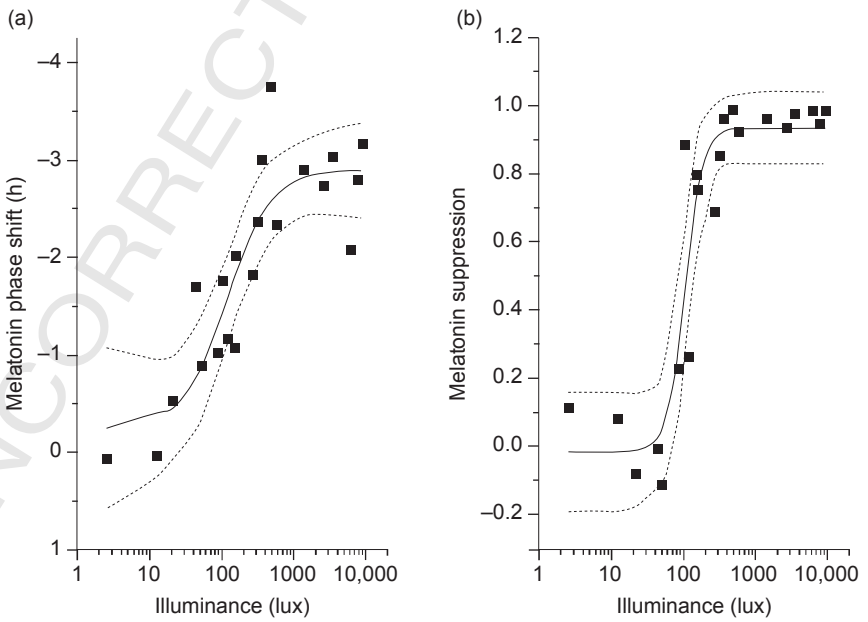

Fig. 2. Illuminance–response curve of the human circadian pacemaker. (a) illustrates the phase shift of melatonin rhythm, as assessed on the day following exposure to a 6.5 h experimental light stimulus, while (b) illustrates the magnitude of melatonin suppression during the light exposure (Zeitzer et al., 2000).

pulse duration affects both amplitude and shape of the PRC (Comas et al., 2006). Using a model in which only phase shifts but no period or amplitude changes to light pulses were included, the authors concluded that phase-shifting effects are largest in the first hour of the light pulse, and reduce to a factor 0.22 during all hours after the first hour. Similar conclusions were drawn from an analysis of available data in humans (Beersma et al., 2009). Analyses of human PRC indicate that during exposure to 6.5 h of bright white light (~10,000 lux), phase delays occurred when light was centered before the critical phase at CBT minimum, while phase advances occurred when light fell after the critical phase (Khalsa et al., 2003). Exposure to intermittent light also seems to be highly effective at resetting the human circadian system. The phase-resetting effects of 5 h of continuous bright white light (~10,000 lux) are comparable to a 5-intermittent exposure of six cycles of 15 min of bright light (~10,000 lux; Gronfier et al., 2004). Thus, a single sequence of intermittent bright-light pulses has a greater resetting efficacy on a per-minute basis than does continuous light exposure. This has been explained by a response saturation process, which is fundamental to proper functioning of the circadian pacemaker in a natural environment (Beersma et al., 2009). In a subsequent study, exposure to two 45-min pulses of bright light in the early subjective evening entrained the circadian system to a non-24-h day, again indicating that intermittent pulses are highly efficient at resetting human circadian rhythms (Gronfier et al., 2007).

Response saturation plays a role in phase resetting to long light pulses, but also adaptation to light clearly affects phase-shifting responses to light. Prior exposure to high levels of light during periods varying from 3 days to 1 week attenuated suppression of the melatonin concentration at night in response to 200–500 lux of light, as compared to prior exposure to 3 days to 1 week of low levels or dim light (Smith et al., 2004). However, if preexposure to room light can desensitize circadian phase resetting or attenuates the alerting effects of light remains an open question.

Short-wavelength effects: Conventional visual photoreception is not the key mediator

The relationship between the wavelength of light and its alerting response seems to indicate a predominance of short-wavelength light (470 nm and lower) in comparison to other wavelengths (Lockley et al., 2006; Münch et al., 2006; Revell et al., 2006). Exposure to 460-nm (blue) monochromatic light for 6.5 h during the biological night (maximum levels of melatonin secretion) can substantially decrease both subjective sleepiness, improve cognitive performance in tasks of sustained attention (i.e., psychomotor vigilance task), and decrease waking EEG power density in the delta–theta frequency range (Fig. 3), when compared to light of equal photon density of 555-nm (green) monochromatic light (Lockley et al., 2006). The magnitude of greater responses following exposure to an equal number of photons of 460-nm light, as compared with 555-nm light, strongly suggests that the photoreceptors mediating these acute effects of light are blue shifted with respect to the visual photopic system. Similarly, a 2-h evening exposure to monochromatic light of two different wavelengths (460 and 550 nm) at very low intensities resulted in more alertness during exposure at 460 nm, which further suggests a blue-shift response to light (Revell et al., 2006). However, these responses do not confine only to effects on wakefulness, but have been observed on a wide array of physiological variables, such as melatonin suppression (Lewy et al., 1980; Zeitzer et al., 2000), circadian phase shifting (Cajochen et al., 1992), nocturnal decline in EEG SWA (Münch et al., 2006; Fig. 4), and circadian gene expression (PER2) in oral mucosa (Cajochen et al., 2006). Nevertheless, novel evidence supports that cone photoreceptors may also contribute to NIF responses at the

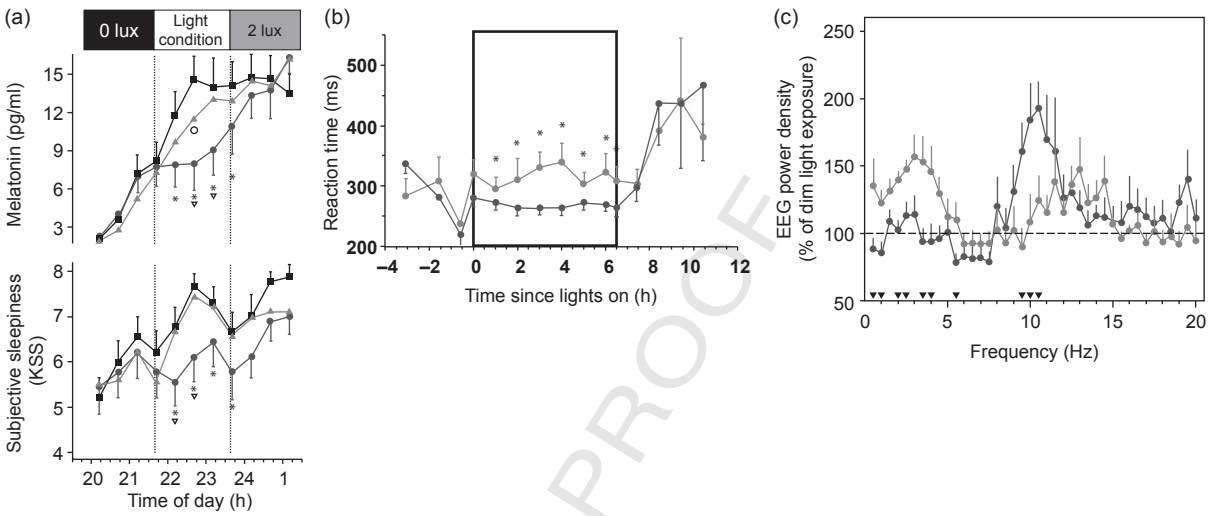

Fig. 3. Effect of light exposures on melatonin secretion, subjective sleepiness, performance, and the electroencephalogram in young healthy volunteers. (a) Effects of a 2-h darkness period (black line) and of 2-h monochromatic light exposures at 460 nm (blue line) and 550 nm (green line) in the evening on salivary melatonin levels and subjective sleepiness (Cajochen et al., 2006); (b) continuous 6.5 h nighttime exposure to blue monochromatic light (significantly improved auditory reaction times to a simple vigilance task when compared to green monochromatic light; Lockley et al., 2006); (c) EEG power density during continuous 6.5 h nighttime exposure to monochromatic blue light and monochromatic green light (Lockley et al., 2006). (For interpretation of the references to color in this figure legend, the reader is referred to the Web version of this chapter.)

beginning of a light exposure and at low irradiances, while melanopsin can be the primary circadian photopigment in response to long-duration light exposure and at high irradiances (Gooley et al., 2010).

The neurophysiology underpinnings that account for light-induced responses are still not fully comprehended. It is known that intrinsically photosensitive retinal ganglion cells (ipRGCs) project to a range of targets, including the SCN, subparaventricular zone, and the pretectal area that are implicated in mediating NIF responses (Hattar et al., 2002). Further, these cells also project directly to the ventrolateral preoptic area, a hypothalamic nucleus lateral to the optic chiasm and rostral to the SCN that also receive secondary afferents from the SCN, subparaventricular zone, and dorsomedial hypothalamus (Hattar et al., 2002). Ventrolateral preoptic area innervates all of the major nuclei of the ascending monoaminergic and, in particular, the histaminergic pathways, which are thought to play a key role in wakefulness and EEG arousal (Aston-Jones et al., 1999; Lin et al., 1996). Direct photic input to this nucleus may therefore alter ventrolateral preoptic area activity. The locus coeruleus (LC) located in dorsal tegmentum of the pons is also involved in the regulation of the sleep–wake cycle (Saper et al., 2005) regulating the amplitude of the sleep–wake circadian rhythm set by the SCN by increasing wakefulness during the active period (Gonzalez and Aston-Jones 2006).

Alternatively, the acute alerting effects of light may happen through acute melatonin suppression, given that increased melatonin suppression can be associated with greater arousal and/or attenuation of the endogenous circadian drive for alertness (Lockley et al., 2006). In other words, if short-wavelength light can reduce circulating melatonin levels and/or high-frequency

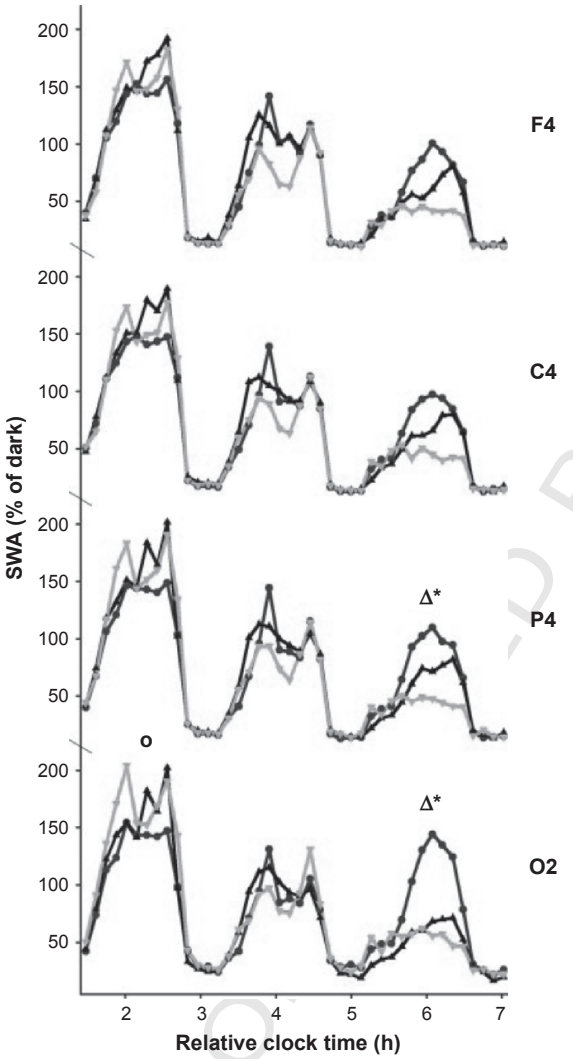

Fig. 4. Dynamics of SWA per NREM–REM sleep cycles 1–3 after sleep onset for EEG derivations F4, C4, P4, and O2. Values are expressed as percentages of the dark condition and are plotted against relative clock time ($n=8$) for blue light (460 nm, blue circle), green light (550 nm, green triangle, down), and dark condition (0 lux, black triangle). $^{*}P<0.05$; $^{\circ}P<0.1$, green light versus blue light; $^{\Delta}P < 0.05$, blue light versus dark condition (Münch et al., 2006). (For interpretation of the references to color in this figure legend, the reader is referred to the Web version of this chapter.)

alpha activity (9.25–12.0 Hz; a marker of the endogenous circadian drive for alertness), it can enhance subjective and objective arousal states. This assumption seems to hold true, given a number of studies which show a reduction of either or both under blue-light exposure (Lockley et al., 2006; Münch et al., 2006). Nevertheless, it is also quite likely that, while melatonin could have a direct role in mediating alertness during the biological night, there might be alternative pathways. For instance, in contrast to Lockley et al. (2006), Phipps-Nelson et al. (2009) did not find blue-light exposure effects on EEG alpha activity, subjective sleepiness, or salivary melatonin, while they observed differential response on the delta/theta activity. These discrepancies may be explained by the fact that in this study, the intensity of blue light was of 2.1 $\mu W/cm^2$, while the other study used a much higher intensity (12.1 $\mu W/cm^2$). Exposure to 460 nm light at 3.1 $\mu W/cm^2$ resulted in significant suppression of plasma melatonin (Brainard et al., 2001), whereas exposure to the same wavelength at ≤ 2.3 $\mu W/cm^2$ did not result in significant suppression. Thus, one might speculate that if the irradiance of the blue-light (460 nm) stimulus is below the threshold of irradiance required for melatonin suppression, other systems may, in turn, undergo light effects, such as the homeostatic rather than circadian mechanisms (Lockley and Gooley 2006).

In the next section, we now turn to what are the cerebral correlates of light that can play a pivotal role on cognitive performance.

Cerebral correlates of light impacts' on cognitive performance

The effect of light on cognitive performance impinges on subcortical and cortical regions in a differential way. Light modulations on cortical activity during auditory cognitive tasks affect alertness-related subcortical structures, such

as the brainstem (LC—compatible region; Vandewalle et al., 2007a,b); the hypothalamus, in a location encompassing the SCN (Perrin et al., 2004), and dorsal and posterior parts of thalamus (Vandewalle et al., 2006, 2007), in long-term memory and emotion-related areas, such as the hippocampus (Vandewalle et al., 2006) and amygdala (Vandewalle et al., 2007a,b). As can be observed, these responses point to a wide-range of subcortical and cortical regions that are differentially activated by the nonvisual effects of light, during specific cognitive tasks. Further, fMRI assessed brain responses undergo a wavelength dependency for higher executive task (two-back task), such that blue light enhances modulations in the brainstem (in an LC-compatible location), in the thalamus and insula, in relation to green (550 nm) and violet exposures (430 nm). In this case, the effects of blue light occur 1 min after the start of the exposure (Vandewalle et al., 2007a,b) and lasted for ~20 min (Vandewalle et al., 2007a,b). Nonetheless, the degree, temporal dynamics, and regional brain distribution of nonvisual effects of light crucially relies on light properties, such as dose, duration, and intensity. Contrary to subcortical regions, which are activated faster and show short-lasting responses to light, long-lasting and widespread task-related responses occur only when light exposure has a longer duration and at a higher intensity (Perrin et al., 2004). For instance, exposure to 20 min of bright white light has been shown to induce thalamic and cortical modulations that steadily declined after light exposure despite the lasting effects (responses were observed after several minutes of the end of the light exposure; Vandewalle et al., 2006). However, when light exposure duration was less than a minute, the majority of effects were elicited for subcortical structures, such as the dorso-posterior thalamus and the brainstem (LC-compatible area; Vandewalle et al., 2007a,b).

The overarching significance of LC-related areas in this case is due to its projections to numerous cortical sites, which favors its role as a mediator for light changes in alertness and cognitive performance (Gonzalez and Aston-Jones 2006). The thalamus, in particular, its dorsal and posterior nuclei (i.e., pulvinar), is a key structure involved in the interaction between alertness and cognition (Portas et al., 1998), and light-related changes in its activity can be directly implicated in enhanced alertness during light exposure. Bearing in mind that the thalamus plays a critical role in the relay of information to the cortex, effects of light on the thalamus may result in widespread cortical effects.

Interestingly, the effects of monochromatic blue light on the hippocampus and amygdala followed a dissimilar pattern (Vandewalle et al., 2007a,b), as responses in these limbic structures happened almost immediately after light onset. These swift limbic light-induced responses might have occurred due to the anatomical connectivity of these structures. The amygdala receives direct inputs from ipRGCs and indirect retinal inputs through the superior colliculus and thalamus. In turn, the amygdala has direct projections to the hippocampus, which receives activating inputs from the brainstem (Vandewalle et al., 2009). The functional relevance of these limbic responses remains uncertain, but raises the question if blue light might support an early affective and mnemonic arousal, which enables a prompt behavioral adaptation to the environment. Similarly, the light-induced modulation of amygdala activity may correspond to one of the underlying reasons for the therapeutical property of light in mood disorders. Light therapy is the treatment of choice of seasonal affective disorder (SAD), and the relative contribution of blue light to overall natural light exposure is smaller during the winter than during the summer (Thorne et al., 2009). However, it is still uncertain as to whether the long-term effects of repeated exposures on mood as used in light therapy are related to the acute modulation of brain activity to tasks that do not involve emotion.

Summary

Light exerts dominant nonvisual effects on numerous physiological variables, such as the human sleep–wake cycle and cognitive performance, primarily through properties such as dose, duration, timing, and wavelength. The use of reliable and sensitive methods such as fMRI to evaluate light-induced nonvisual brain responses have increased our understanding on how light can optimize brain function during specific cognitive tasks. These stimulating discoveries will certainly help to unravel how retinal and suprachiasmatic networks modulate the complex interplay of circadian rhythms, sleep–wake homeostasis, and cognition.

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