Light Treatment for Sleep Disorders: Consensus Report.

III. Alerting and Activating Effects

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Abstract In addition to the well-established phase-shifting properties of timed exposure to bright light, some investigators have reported an acute alerting, or activating, effect of bright light exposure. To the extent that bright light interventions for sleep disturbance may cause subjective and/or central nervous system activation, such a property may adversely affect the efficacy of treatment. Data obtained from patient samples and from healthy subjects generally support the notion that exposure to bright light may be associated with enhanced subjective alertness, and there is limited evidence of objective changes (EEG, skin conductance levels) that are consistent with true physiological arousal. Such activation appears to be quite transient, and there is little evidence to suggest that bright light-induced activation interferes with subsequent sleep onset. Some depressed patients, however, have experienced insomnia and hypomanic activation following bright-light exposure.

Key words sleep, light, arousal, alertness, phototherapy, EEG, shift work

INTRODUCTION

Bright light exposure has been used as a treatment for a number of sleep difficulties, most of them associated with alterations in the circadian timing system (i.e., advanced and delayed sleep phase disturbance, jet lag, and shift work). For some of these applications, usually in which a phase delay of circadian rhythms is indicated (e.g., advanced sleep phase syndrome), the optimal time for light exposure is in the evening, often immediately prior to bedtime. Although such timing of light exposure is desirable from a circadian rhythms perspective, a potential drawback exists to such light

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exposure schedules as well. This is due to a property of bright light exposure that has received relatively less attention than the phase-shifting and entraining effects—that of an acute alerting, or activating, effect. The present report focuses on the issue of alerting/activating effects of bright light exposure as they may affect negatively the use of light treatment for insomnia, and only briefly addresses effects on waking performance. A more detailed discussion of the potential benefits of such alerting/activating effects of bright light exposure, in terms of performance enhancement, is beyond the scope of this report.

EVIDENCE FOR ALERTING/ ACTIVATING EFFECTS

Patient Samples

The possibility that exposure to bright light may result in subjective and/or central nervous system (CNS) activation has been recognized since the first studies of light therapy for seasonal affective disorder (SAD). Rosenthal and coworkers (Rosenthal et al., 1984) reported "hypomanic irritability and hyperactivity in a few cases" of bright light treatment, which subsided when treatment was discontinued. Subsequent studies of light therapy of patients with SAD have documented similar behavioral responses following bright light exposure, including decreased sleep and insomnia. It should be pointed out that these responses in patients with SAD may not be related directly to light exposure. Rather, they may result from a decrease in the symptom of hypersomnia associated with recovery, or an increase in more typical depressive symptoms (i.e., insomnia). For example, in a study of side effects associated with three different light treatment regimens (morning, evening, and daily alternation of morning and evening exposure), five of eight subjects who received evening exposure reported difficulty getting to sleep (Labbate et al., 1993). Other studies have reported relatively lower incidence of "feeling wired" and hypomanic activation (Wirz-Justice et al., 1986; Oren et al., 1991; Levitt et al., 1993). In addition, two studies have reported side effects of hypomania and mania in a small proportion of nonseasonal depressives treated with light (Kripke et al., 1983; Schwitzer et al., 1990).

With regard to other patient samples, a group of pathologically sleepy subjects (obstructive sleep apnea) exhibited reduced sleep tendency on Multiple Sleep Latency Tests (MSLTs) that were immediately preceded by 2 h of exposure to bright light (10,000 lux). Moreover, in a "drowsy" subset, this alerting effect of bright light exposure carried over to a subsequent MSLT before which no bright light was administered (Finley et al., 1992). This was not the case, however, for a group of narcoleptic subjects who were exposed to bright light (approx. 5000 lux) from 0700 h to 0900 h and 1800 h to 2000 h for 10 days (Hajek et al., 1989). In that study, there were no changes in sleep latency measures on the MSLT; this despite the fact that the MSLTs included one challenge (0900 h) immediately following 2 h of light exposure. In addition, there were no changes in activity levels and no changes in self-rated tiredness following light exposure.

Normal subjects: light exposure prior to day sleep. Bright-light-induced alertness/activation has been reported in healthy subjects by a number of investigators, though not all (Dollins et al., 1993), using protocols involving all-night exposure (Campbell and Dawson, 1990; Czeisler et al., 1990; Hannon et al., 1992), exposure during parts of the night (Badia et al., 1991; Dawson and Campbell, 1991), or during the early morning (Clodore et al., 1990). None of these studies, however, has reported deleterious effects on subjects' ability to initiate subsequent daytime sleep episodes as a consequence of bright light exposure. This result is perhaps not surprising, since the sleep deprivation associated with remaining awake throughout the night may effectively override any potential alerting effects of light exposure the following morning.

Normal subjects: light exposure prior to nocturnal sleep. It might be expected that any alerting effects of light on subsequent sleep would become more evident in protocols that do not involve significant sleep deprivation. Yet Drennan et al. (1989) observed no significant lengthening of nocturnal sleep latency (bedtime at approx. 2300 h) immediately following one night's exposure to bright light between 1800 h and 2100 h. The same result was reported by Bunnell and coworkers (1992) in subjects exposed to 2 h of bright light (> 2500 lux) immediately prior to nocturnal sleep. Similarly, the ability of healthy young subjects to initiate a daytime nap was not affected by 2 h of exposure to bright light (5000 lux) immediately prior to the nap attempt (Murphy et al., 1991). In contrast to these findings, Dijk and coworkers (Dijk et al., 1991) observed significantly increased latencies to sleep onset in subjects exposed to bright light (approx. 2500 lux)

for 3 h immediately prior to nocturnal sleep. The conflicting results may be due to the fact that this study differed from the others in terms of timing (Drennan et al., 1989) and duration (Bunnell et al., 1992) of the light exposure period.

WHAT IS THE MECHANISM?

Although the findings of Dijk and coworkers are consistent with a general activating effect of light, the authors suggest that the increased difficulty initiating sleep may have been a result of light's delaying effect on the pacemaker driving body temperature rhythm, instead. In this regard, there were no significant changes in waking brain electrical activity associated with bright light exposure, though there was a trend for reduced activity in the theta band compared to the dim light control condition (Cajochen et al., 1992).

Yet two studies by Badia and coworkers seem to support the notion that true physiological arousal may be induced by exposure to bright ambient light. In a preliminary report, Badia and coworkers (1990) observed significantly higher tonic skin conductance levels in a bright light condition versus the dim light control, indicating increased levels of arousal. In a subsequent study (Badia et al., 1991), these authors also reported increased EEG beta activity when subjects were exposed to bright light. In a group exposed to alternating intervals of bright and dim light (every 90 min), log power density of the beta band increased and decreased with changes in illumination. Moreover, the dominant frequency within the beta band was significantly higher during bright light exposure (19 Hz) than during dim light exposure (16 Hz). Such changes in the EEG were considered by the investigators to reflect an arousing influence of bright light exposure.

Based on the putative somnogenic properties of melatonin, and the demonstrated capacity of bright light to suppress nighttime plasma melatonin levels (see, e.g., Lewy, 1983), it has been suggested that alerting/activating effects of bright light exposure may be mediated through the action of melatonin, perhaps linked to thermoregulation (Badia et al., 1991; Sack et al., 1992). It should be noted, however, that a recent study by Dollins et al. (1993) found no effects of bright light (1500 and 3000 lux) on performance or subjective alertness, though the light was effective in suppressing melatonin.

Nevertheless, a number of studies have shown that bright light exposure transiently increases body temperature and that enhanced alertness and performance are associated with such increases. Moreover, exogenous melatonin administration counteracts bright light effects on body temperature, and this is associated with reductions in alertness (Badia et al., 1991; Sack et al., 1992). The finding that bright light exposure does not alter daytime sleep latencies (when melatonin secretion is low or absent), but does affect nighttime measures of sleep tendency, has been offered, as well, in support of the role of melatonin in the alerting/activating effects of bright light exposure (Murphy et al., 1991). Indeed, Murphy et al. (1991) go on to say that such evidence suggests that bright light exposure does not have "intrinsic energizing effects."

Whether bright light provides an intrinsic activating effect, or whether such an effect is mediated through another, or multiple, systems, it seems clear that increased alertness/activation is associated with bright light exposure. That such activation does little, at least in healthy subjects, to interfere with subsequent "sleepability" suggests that the effects are relatively short lived. That is, following removal of the light stimulus, activation effects decline precipitously, even though the effects on body temperature may last for several hours (Dijk et al., 1991). This notion is supported by the findings that body temperature, melatonin levels, and beta activity all vary in close association with changes in light exposure intensity. It is unlikely, therefore, that acute effects of bright light exposure (as opposed to phase-shifting properties), even immediately prior to bedtime, would induce, or exacerbate, insomnia, at least in healthy young adults. Such a conclusion is less certain for patients with SAD as well as nonseasonal depression, for which bright-light exposure can induce hypomanic activation and sleep-onset insomnia in some cases. Not surprisingly, the likelihood of the latter side effect is greater for those receiving evening light.

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