BRIEF REPORTS

Dawn and Dusk Simulation as a Therapeutic Intervention

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Introduction

Daily alternations of light and darkness entrain circadian rhythms of body temperature, rest–activity, and melatonin and other neuroendocrine secretions (cf., Moore-Ede et al. 1982). Annual cycles of changing light–dark patterns underlie seasonal metabolic and behavioral cycles and, in humans living distant from the equator, a syndrome of winter depression with prominent atypical neurovegetative symptoms of fatigue, hypersomnia, carbohydrate craving, and weight gain (Rosenthal et al. 1984). Affected individuals suffer annual cycles of spontaneous relapse and remission, generally in autumn and spring, respectively.

Early-morning artificial bright light (>2000 lux) provides effective treatment in this variant of depression (Rosenthal et al. 1985; Lewy and Sack 1986; Terman 1988), while also inducing immediate suppression of nocturnal melatonin secretion (Lewy et al. 1980), and within several days, circadian phase shifts (Lewy et al. 1985; Terman et al. 1988). In humans, normal indoor illumination levels (<800 lux) have proven markedly less effective in all of the above respects, though there is a dose–response relationship (Brainard et al. 1989) and some individuals show a "supersensitive" response to such low levels of light (Lewy et al. 1981).

Such findings come from laboratory studies and therapeutic manipulations of light timing and duration that have relied upon sudden off/on switching of lights. In contrast, light signals in nature are embedded in dynamic light–dark profiles, spanning an approximate 8-log unit range of illuminance from starlight to midday maxima (Figure 1). Seasonal progression determines both timing and shape of this illuminance profile. Daily and monthly changes occur in momentary rate of change and absolute level of illuminance at a given time of day and also in the integrated sum and distribution of light exposure preceding a given illuminance level. Seasonal differences in these parameters increase as a function of distance from the equator and are most pronounced, at any given latitude, during twilight transitions—dawn and dusk. Specific behavioral sensitivity to one or more of these twilight variables has been suggested in animal experiments using natural outdoor lighting (e.g., Daan and Aschoff 1974) and indoor simulated twilight (e.g., Kavanau 1968; Terman and Schlager 1989).

Conventions in human light therapy and analyses of light–circadian system interactions may then be considered to be "nonphysiological" in their failure to account for biological sensitivity to such parameters of dim light around dawn

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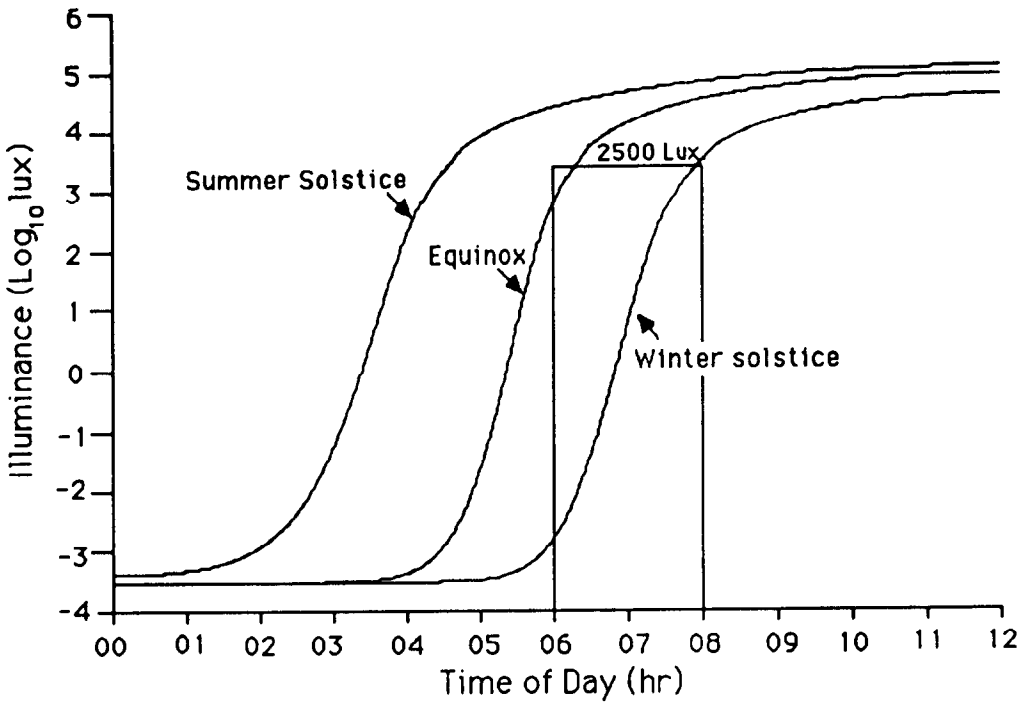

Figure 1. Algorithmic representation, at 45° north latitude, of dawn twilight illuminance patterns at summer solstice, autumnal equinox, and winter solstice. The standard bright light therapy signal of 2500 lux administered for 2 hr upon awakening at 6 AM is superimposed. Note differences in shape, as well as position, among the different curves.

and dusk, or to the continuing changes of brighter, midday light.

This article reports the development of a lighting system that precisely simulates natural outdoor light–dark cycles, including twilight transitions. Clinical and physiological sensitivity to the twilight signal in human seasonal depression is explored.

Methods

The system consists of a computer algorithm that specifies instantaneous illumination levels—from starlight, skylight, and sunlight sources—and controls a light attenuation apparatus attached to a high-intensity fluorescent light source (Terman et al. 1989). A photosensor feedback serves to fine-adjust light levels for close fit to the model, which has been validated in outdoor tests.

The system is capable of delivering dynamic twilight signals over 6 log units of illuminance between starlight levels and approximately 1000 lux, the latter achieved around sunrise and sunset. The twilight profile can be specified to any particular day of the year at any latitude on earth and can be delivered while a subject sleeps and awakens spontaneously.

Three subjects having major affective disorder, depressed, seasonal pattern (winter) by DSM-IIIR criteria were selected for a clear history of responsiveness to conventional bright light therapy. Baseline Hamilton Rating Scale for Depression (HAM-D) score was >12 to enter. The subjects were exposed to simulated twilight signals of dawn-only or dusk-plus-dawn, delivered at the bedside during 7–14-day home trials. The therapeutic dawn-only profiles were that of May 5, halfway between the equinox and summer solstice, corresponding to a date well

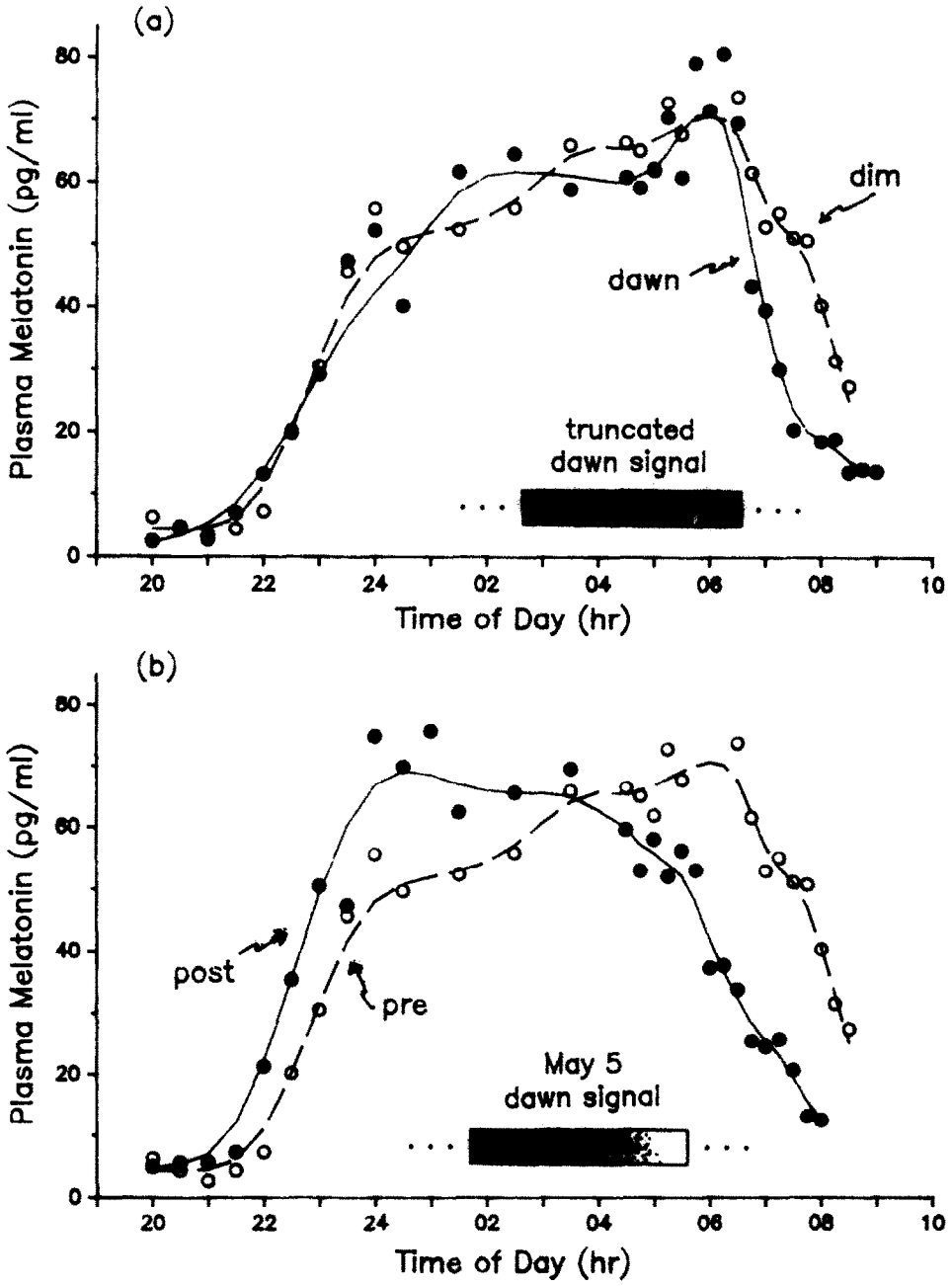

Figure 2. (a) Early morning suppression of nocturnal melatonin secretion in a winter depressive patient in the presence of simulated equinox dawn signal, truncated suddenly to darkness, as compared to baseline dim light (<200 lux) conditions on 2 consecutive nights. Curve fits are based on an iterated running median. (b) Phase advance of nocturnal plasma melatonin in a winter depressive patient, measured under dim light conditions before and after 8-day home exposure to a May 5 dawn signal. Dawns progressed beyond sunrise to a maximum, steady signal of approximately 1000 lux by 5:00 AM.

past spontaneous natural remission for most winter depressives. The dusk-plus-dawn profile was of June 22 (summer solstice) and all were of 45° north latitude. May 5 dawn light onset was at 2:59 AM, progressing to sunrise (approximately 800 lux) at 4:53 AM, and to a maximum of 1000 lux achieved about 1 min later and maintained until the patient arose.

Overnight plasma melatonin patterns were measured in our Biological Studies Unit before, during, and after twilight exposure using radioimmunoassay technique (Terman et al. 1988). Clinical improvement was judged in psychiatric interviews utilizing the SIGH-SAD subscales of HAM-D and atypical symptoms (Williams et al. 1988). Sleep data were collected with daily, self-rating sleep logs.

Results

Baseline nocturnal melatonin secretion patterns under dim light conditions (<200 lux) were altered in the presence of the dawn signal, where high nocturnal levels persisted until sunrise, then showed an exponential suppression toward the daytime minimum (Figure 2a). This phenomenon of melatonin suppression in the presence of the dawn twilight was seen in both subjects tested, at both baseline and posttreatment conditions, regardless of whether the signal progressed beyond sunrise or was abruptly truncated to darkness just prior to sunrise. Circadian phase, as indicated by melatonin secretion pattern measured under dim light conditions without twilight, clearly advanced after a week's treatment with dawn twilights (Figure 2b).

Clinical evaluation indicated full remission in one patient (HAM-D/atypical scores from 14/18 to 5/3) and significant reduction of depressive symptoms in another (13/10 to 7/3). A third subject reported sustained improvement in energy upon awakening under the dawn twilight, but showed no significant improvement in mood or other symptoms (10/20 to 13/18). It should be noted that this clinically unresponsive patient exhibited both circadian phase advance and immediate melatonin suppression during the dawn exposure trials.

Initial wakings occurred well before sunrise in the dawn twilight period, though sleep resumed easily, with final spontaneous awakening and arising within minutes of sunrise. Patients also reported a pleasant hypnotic sensation with summer solstice dusk twilights presented at bedtime, and consistently fell asleep within 30 min of simulated sunset. Dawn and dusk together

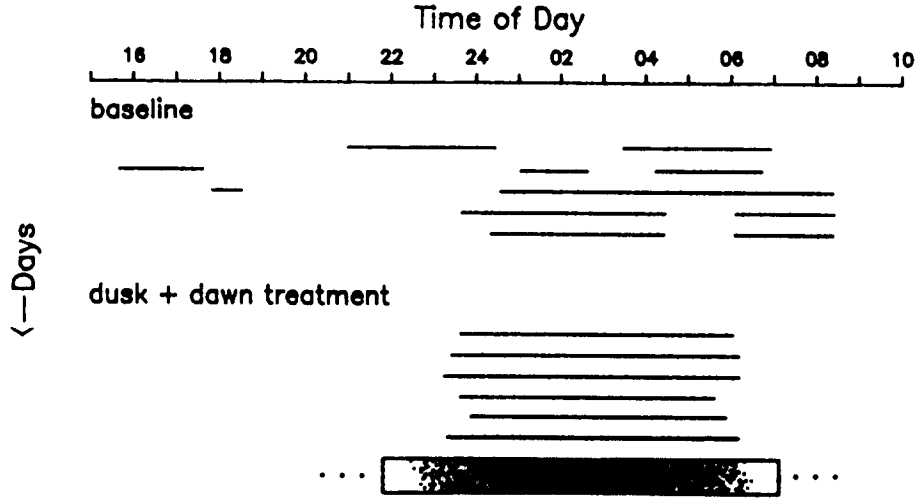

Figure 3. Pretreatment disrupted sleep pattern and adjustment to dusk and dawn signals. Horizontal lines portray sleep episodes.

were most effective in eliminating poor sleep patterns (e.g., Figure 3).

Discussion

Twilight exposure appears able to promote circadian phase adjustments, morning melatonin suppression, regularized sleep patterns, and antidepressant responses. This represents the first indication in humans of physiological and/or behavioral sensitivity to such light signals.

Larger, controlled studies will be needed to determine the active components of each of these twilight effects, their relation to one another, and the range of subjects exhibiting such sensitivity. Nevertheless, we hypothesize the nonmodulated bright light constitutes a supernormal stimulus. The eyes may be primed at twilight hours for reception of changing intensities of low-level light. Alternatively, seasonal variations in dawn twilight exposure may prime photosensitive systems in the retina or brain for differential responses to postawakening bright light exposure. A role for such graded exposures in treatment of winter depression, sleep disturbance, and other circadian disorders should be explored.

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