Light Therapy

Michael Terman and Jiuan Su Terman

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Chapter 149

Abstract

The susceptibility of the circadian system to selective phase shifting by timed light exposure has broad implications for the treatment of sleep phase and depressive disorders. Light therapies have been developed to normalize the patterns of delayed sleep phase disorder (through circadian phase advances) and advanced sleep phase disorder (through circadian phase delays). Physicians and patients need to be cognizant of the daily intervals when light exposure—and darkness—can facilitate or hamper adjustment to the desired circadian phase. The critical intervals lie at the edges of the subjective night, which coincide with the tails of the nocturnal melatonin cycle, but can be inferred clinically through a morningness–eveningness questionnaire or habitual sleep timing. The schedule of light exposure might have to be continually adjusted during treatment as the subjective night shifts gradually in the desired direction.

The treatment strategy for seasonal and nonseasonal depression is similar. In winter depression, the size of circadian rhythm phase advances correlates with the degree of mood improvement, and the optimal timing of light therapy must be specified relative to the patient's circadian clock rather than solar time. Apart from its use as a monotherapy, light therapy in outpatient and inpatient trials indicates that it accelerates remission of nonseasonal depression in conjunction with antidepressant and mood-stabilizing medication.

Exposure of the eyes to light of appropriate intensity and duration, at an appropriate time of day, can have marked effects on the timing and duration of sleep and on the affective and physical symptoms of depressive illness.[1] Here we review and evaluate delivery systems and the application of light therapy for circadian rhythm sleep disorders including delayed sleep phase disorder (DSPD), advanced sleep phase disorder (ASPD), and free running (or non–24-hour) sleep disorder. The most extensive clinical trials have focused on wintertime recurrence of major depression (seasonal affective disorder) [SAD]. We also cover light therapy for nonseasonal depressions (recurrent, chronic, bipolar), including combination treatment with sleep deprivation (wake therapy) and antidepressant medication. Light therapy holds promise in treating several clinical disorders.[1a-1f]

LIGHT DELIVERY

Apparatus

Light Boxes

Many of the early research studies used a standard 60-cm by 120-cm (2-foot by 4-foot) fluorescent ceiling unit, with a plastic prismatic diffusion screen, placed vertically on a table about 1 meter (3 feet) from the user. A bank of fluorescent lamps—full spectrum or cool white—provided approximately 2500-lux illuminance. Smaller, more lightweight units have become commercially available; however, specific design features of marketed light boxes have most often not been clinically tested.

Factors include lamp type (output intensity and spectrum), filter, ballast frequency (for fluorescent lamps), size and positioning of radiating surface, heat emission, and so on. One clinically tested model (Fig. 149-1) illustrates modifications in second-generation apparatus, including smaller size, portability, raised and downward-tilted placement of the radiating surface to reduce glare, height adjustment relative to the patient's head, a smooth polycarbonate diffusion screen with maximal ultraviolet (UV) filtering, and high-output fluorescent lamps (non-glaring 4000-Kelvin color temperature) driven by high-frequency solid-state ballasts. The combination of elements in this configuration yields a maximum illuminance of approximately 10,000 lux with the patient seated in a position with the eyes about 30 cm (1 foot) from the screen.

With the direction of gaze downward toward the work surface, such a configuration provides pleasant illumination suitable for reading and, despite illuminance far higher than in normal home lighting, is generally well tolerated (see Adverse Effects of Bright Light Exposure, later). The presentation of light from above eye level is supported by a study showing enhancement of melatonin suppression with directional illumination of the lower retina.[2] As the apparatus becomes miniaturized, however, the field of illumination narrows, and even small changes in head position can substantially reduce the intensity of light that reaches the eyes.

Clinicians should seek documentation of the safety and effectiveness of any apparatus under consideration. Home construction of such an apparatus is discouraged because of the danger of excessive irradiation affecting the eyes and eyelids.

Claims for the specific efficacy of any particular lamp type or spectral distribution, although commonly made, need to be questioned. Unfortunately, systems have been marketed that provide excessive visual glare, exposure of naked bulbs, direct intense illumination from below the eyes ("ski slope" effect), and intentionally augmented short-wavelength blue and UV radiation. The earliest light therapy trials used full-spectrum white fluorescent light at high color temperature with increased blue and near-UV radiation, in an attempt to approximate the spectral distribution of skylight relative to standard fluorescent sources. UV, however, was shown to be unnecessary for the antidepressant effect,[3] leading to the use of alternative light sources and light boxes with UV filters to reduce exposure hazard to the eyes and skin. Claims that blue light is selectively therapeutically active[4] have not yet been substantiated. Full-spectrum lighting is tolerable at 2500 lux, but aversive glare is excessive at 10,000 lux, the dose designed

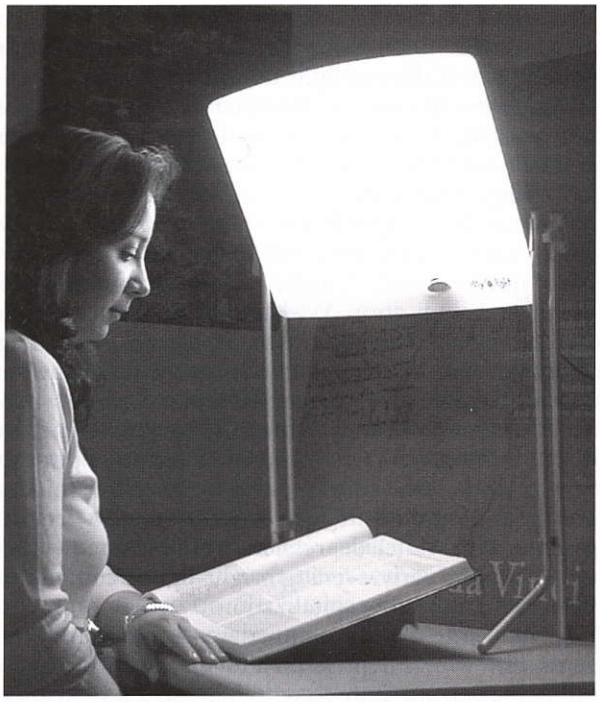

Figure 149-1 Table-mounted, tilted, height-adjustable, 10,000-lux, UV-filtered, diffused 4000-Kelvin fluorescent light system. (Photograph courtesy of the Center for Environmental Therapeutics, http://www.cet.org.)

to reduce average session duration from 2 hours to 30 minutes.[5] Softer white light with lower color temperature (3000 to 4000 Kelvin) works as well. Both the clinician and consumer must be vigilant in the selection of apparatus. Criteria are reviewed on the therapy Web page of the nonprofit Center for Environmental Therapeutics, http://www.cet.org.

Dawn Simulators

Dawn simulation methodology provides a major contrast to bright light therapy. A microprocessor-controlled lighting device delivers a mimic of gradual twilight transitions found outdoors in the spring or summer. A laboratory study of healthy young adults demonstrated that the addition of simulated, naturalistic dawn exposures blocked the delay drift of circadian rhythms under dim light–dark cycles, when subjects were awakened to view the signal.[6] For clinical use, relatively dim dawn signals are presented to the patient while asleep, when the eyes are adapted to the dark and the circadian system is most susceptible to phase advances (see Timing of Morning Light Exposure, later). The initial open-label studies of dawn simulation found an antidepressant response; normalization of hypersomnic, phase-shifted, and fractionated sleep patterns; phase advances of the melatonin cycle; and truncation of melatonin production after simulated sunrise.[7-9]

Avery and colleagues[10] conducted a 6-week controlled clinical trial of sigmoid light-onset ramps (which differ from the curvilinear acceleration of naturalistic dawns) rising to 250 lux between 4:30 AM and 6:00 AM, which were significantly more antidepressant than dim red control signals rising to 0.5 lux. The treatment was superior to postawakening light therapy at 10,000 lux administered between 6:00 AM and 6:30 AM (which may have been too early for these phase-delayed patients). Terman's group[11] conducted a controlled trial of naturalistic dawn simulation ending at individually selected wake up times and found similar rates of improvement to postawakening bright light therapy, and both methods were superior to low-density negative air ions, a nonphotic placebo. The average posttreatment phase advance of dim light melatonin onset was 0.58 hours, in contrast to a small delay shift of 0.19 hours under placebo (2-tailed *t*-test, $P = .001$).[11a]

The effectiveness of dawn simulation may depend on the presentation of diffuse, broad-field illumination that reaches the sleeper in varying postures. Such efficacy has not been demonstrated for commercial "light alarm clocks," which have small directional fields. Adequate apparatus remains an active focus of industrial research and development.

Safety of Bright Light for the Eyes

Ophthalmologic evaluations of unmedicated patients with normal oculoretinal status have thus far shown no obvious acute light-induced pathology or long-term sequelae.[12] Although the intensity of bright light treatment falls well within the low outdoor daylight range, the exposure conditions differ from those outdoors, and prolonged use entails far greater cumulative light exposure than is normally experienced by urban dwellers and workers.[13,14]

There remains a risk of direct retinal photoreceptor and pigment epithelium damage, drug photosensitization, and acceleration of age-related macular degeneration to visible spectral components above the UV range up to about 500 nm (blue light).[15-17] Although wavelengths from ~450 to 500 nm preferentially suppress nocturnal melatonin production[18] and enhance circadian phase shifting,[19] the selective therapeutic benefit of such light has yet to be ascertained. Nevertheless, manufacturers have marketed a variety of blue-light devices. Because the blue-light hazard is magnified at wavelengths below 450 nm, ideally these should be completely filtered out, although commercial fluorescent apparatus has yet to do so. Clinical studies of phase shifting[20] and antidepressant effect[21] with blue light thus far have shown no advantage over broad-spectrum white light, which remains the standard. White light, of course, includes a blue component, though in lower relative proportion than from narrow-band or blue-supplemented sources. The potential adverse effects of concentrated short-wavelength radiation suggest that clinical implementation for long-term treatment is problematic.[22]

At the opposite end of the light spectrum, ocular exposure to infrared illumination, which makes up about 90% of the output of incandescent lamps, poses risk of damage to the lens, cornea, retina, and pigment epithelium.[23] Thus, despite being marketed for light therapy, incandescent lamps are contraindicated.

Light-box diffusion filters vary widely in short-wavelength transmission (for examples, see reference 24). Transmission curves should be demanded of manufacturers and compared with published standards. Normal clouding of the lens and ocular media that begins in middle

age, as well as formation of cataracts, serves to exacerbate perceptual glare that can make high-intensity light exposure quite uncomfortable.[24]

Furthermore, both UV and short-wavelength blue light can interact with photosensitizing medications—including many standard antidepressant, antipsychotic, and antiarrhythmic agents, as well as common medications such as tetracycline—to promote or accelerate retinal pathology, whether acute, or slow and cumulative.[23] In one reported case, a patient received combination treatment with clomipramine and full-spectrum fluorescent light. After 5 days, the patient had reduced contrast sensitivity, foveal sensitivity, and visual acuity and central scotomas and lesions, fortunately with only minor residual aftereffects in contrast sensitivity and scotoma 1 year after discontinuation.[25]

Filtered glasses are available (see Resources, later) that eliminate transmission of short-wavelength blue light while maximizing exposure above the range of primary circadian photoreception (>535 nm), reducing glare, enhancing visual acuity and subjective brightness,[26] and minimizing the risk of drug photosensitization. Such protection is clinically useful to forestall circadian rhythm phase delays and melatonin suppression[27] when patients are exposed to ambient light, especially before sleep, with increased risk of initial insomnia.

Although there are no definite contraindications for bright light treatment other than the retinopathies, research studies have routinely excluded patients with glaucoma or cataract. Some of these patients have used light therapy effectively in open treatment; this should be done, however, only with ophthalmologic monitoring. A simple eye checkup is advised for all new patients, for which a structured examination chart has been designed (see Resources, later).[28] The examination has occasionally revealed preexisting ocular conditions that should be distinguished from potential consequences of bright light treatment.

Adverse Effects of Bright-Light Exposure

If evening light is timed too late, the patient can develop insomnia and hyperactivity. If morning light is timed too early, the patient can awaken prematurely, well before light onset, and be unable to resume sleep. These problems are responsive to timing and dose (duration and intensity) adjustments during treatment of circadian sleep phase disorders and mood disorders.

The emergence of side effects relates in part to the parameters of light exposure, including intensity, duration, spectral content, and method of exposure (diffuse or focused; direct or indirect; forward, upward, or downward angle of incidence relative to the eyes). Thus far, side effects have been assessed primarily in patients with seasonal and nonseasonal mood disorders, and information is lacking for sleep disorders without mood disturbance.

The earliest clinical trials of 2500-lux full-spectrum fluorescent light therapy for winter depression noted infrequent side effects of hypomania, irritability, headache, and nausea.[29,30] Such symptoms often subside after several days of treatment. If persistent, they can be reduced or eliminated by decreasing the dose. Rarely have patients discontinued treatment due to side effects.

Two cases of induced manic episodes have been reported in drug-refractory nonseasonal unipolar depressives beginning after 4 to 5 days of light treatment.[31] A few cases of light-induced agitation and hypomania have been noted, also in patients with nonseasonal depression.[32] A patient with seasonally recurrent brief depressions developed rapid mood swings after light overexposure (far exceeding 30 minutes per day at 10,000 lux),[33] and a patient who had unipolar winter depression and similar exposure showed his first manic episode[34]; both patients required discontinuation and medication. We had one bipolar patient with winter depression who became manic after the use of light and was given lithium as an effective countermeasure; almost all patients using mood stabilizers have responded to light therapy without mania. However, some have switched into mixed states with early morning light exposure, which was resolved by moving treatment to midday.[35] Three cases of suicide attempt or ideation, also occurring in patients with winter depression, were reported within 1 week of standard early-evening bright-light treatment, and the patients required hospitalization.[36]

A 42-item side-effect inventory was administered to 30 patients with winter depression after treatment with unfiltered full-spectrum fluorescent light at 2500 lux for 2 hours daily.[37] Other than for one case of hypomania, there were no clinically significant side effects. Patients given evening light (the timing relative to bedtime was unspecified) reported initial insomnia. Mild visual complaints included blurred vision, eyestrain, and photophobia.

Of specific interest is the side-effect profile for patients using a downward-tilted fluorescent light box protected by a smooth diffusion screen (see Fig. 149-1), with 30-minute daily exposures at 10,000 lux, because this method has had widespread application. A study of 83 patients with winter depression who were evaluated for 88 potential side effects[38] identified a small number of emergent symptoms at a frequency of 6% to 16%, including nausea, headache, jumpiness or jitteriness, and eye irritation.[39] These results must be weighed against the improvement of other patients who showed similar symptoms at baseline but became asymptomatic after light treatment: All symptoms, except nausea, showed greater improvement than exacerbation, which forces attention to the risk-to-benefit ratio. Indeed, emergence of symptoms might have reflected the natural course of the depression in nonresponders to light, rather than a specific response to light exposure.

CASE MANAGEMENT, TIMING, AND DOSING

Monitoring of Patients

Light treatment is typically self-administered at home on a schedule recommended by the clinician. To the extent that the timing of light exposure is important to obtain a therapeutic effect, patient adherence is a sine qua non. The patient and clinician need to work together to find the optimum dosing–timing combination. At the Columbia clinic, the two talk by telephone after the first 3 days of treatment, although the patient is expected to report any side effects the same day they are experienced. Patients maintain a daily sleep, mood, and energy log (download-

able in hard copy or PC formats),[40] which they are asked to email or fax before subsequent scheduled contacts. This information is essential for tracking sleep-phase changes in response to the treatment, and adjusting the dose or timing. Patients with depression also complete a hard copy or online symptom rating scale (see Resources, later). With the personalized feedback presented online, this exercise also teaches patients to track their status independently, in preparation for self-management of light therapy after the initial period of guided treatment.

In contrast with structured research studies, the motivation and adherence of patients in open treatment can be problematic. Despite an agreement to awaken for light treatment at a specific hour, patients might ignore the alarm, considering additional sleep to be the priority of the moment, and might delay or skip treatment. The initial evaluation should explicitly cover the patient's weekly schedule of major activities, early morning and late evening commitments, dinner times, regularity of work hours, coordination with partner's sleep schedule, pattern of late nights out, and so on. Such factors can adversely interact with the treatment regimen, and it may be important to reset priorities. Patients often attempt to test whether improvement can be achieved without rigid compliance, and they might quit if treatment is managed too rigidly. Adolescents often promise adherence without following through, yielding to social peer pressure and norms. Indeed, the behavioral investment in a maintenance regimen of light therapy is considerable, far exceeding that of pharmacotherapy (which, of course, has its own adherence issues).

For hypersomnic or DSPD patients who are unable to wake when instructed, light exposure initially should be scheduled at the time of habitual awakening and then edged earlier across days toward the target interval. Some depressed hypersomnic patients compensate for earlier wake up times with earlier bedtimes or napping, but others are comfortable with less sleep as the antidepressant effect sets in. Clinical experience suggests that most such patients could not sustain earlier awakening without the use of light to phase-advance their circadian rhythms.

Variability in the sleep pattern, if it occurs, can yield important information for determining the course of treatment. Continual adjustments in scheduling, although labor intensive for the clinician, often succeeds. Our strategy has been to encourage the adherence to a recommended light exposure schedule but to consider the obtained sleep pattern as a dependent measure that often reflects changes in mood state, sleep need, and circadian rhythm phase. Rarely do we set a patient's bedtime (rise time is linked to the light schedule); rather, we ask the patients to go to bed once they are sleepy. In this way, we have a better handle on circadian clock phase as it changes in response to treatment.

Timing of Morning Light Exposure

The thrust of recent clinical trials (see Seasonal Affective Disorder, later) leads to the recommendation that patients with winter depression initially be given morning light shortly after awakening. The dose of 10,000 lux for 30 minutes[5,41] appears to be most efficient. A similar strategy applies to patients with DSPD, with evening light for ASPD, but longer exposure duration up to 60 minutes may been needed. Although intensities as low as 2500 lux can also be effective, they require longer exposure,[42,43] and to accommodate such treatment in the morning most patients would have to wake far earlier than their habitual wake up time, with a risk of counterproductive circadian phase delays.

The advantage of morning light appears to lie in circadian rhythm phase advances, which can be measured as shifts in the time of nocturnal melatonin onset.[44] The magnitude of the antidepressant response varies with the magnitude of phase advances. In a protocol with 10,000-lux treatment for 30 minutes on habitual awakening, the magnitude of antidepressant response was negatively correlated with the interval between dim-light melatonin onset (DLMO) and treatment time ($r = -0.38$, $P = .01$).[45] The greatest improvement was seen with light therapy 7.5 hours after DLMO, which produced a 2.7-hour phase advance over 3 weeks. Overall, light therapy given 7.5 to 9.5 hours after melatonin onset yielded twice the remission rate (80% versus 38%) of light given 9.5 to 11.0 hours after DLMO.[46] Clock time per se should not be used to schedule morning light administration, because the baseline DLMO—our metric for circadian time—spans a 7-hour range.

Unfortunately, a phase diagnostic based on melatonin assays is not readily available in clinical practice. The melatonin assay remains primarily a research tool, with high cost and slow turnaround time, although user-friendly clinical kits are becoming available. To provide the clinician a basis to readily specify treatment time, a quick, approximate solution lies in the relation between melatonin onset and the Horne-Östberg Morningness–Eveningness Questionnaire (MEQ)[47] score, which for unmedicated winter depression patients are strongly correlated ($r = -0.73$; Fig. 149-2). Healthy subjects without depression show a similar relationship.[48]

One thus can schedule morning light exposure at individually determined circadian times by estimating the time of melatonin onset from the MEQ score, a strategy that

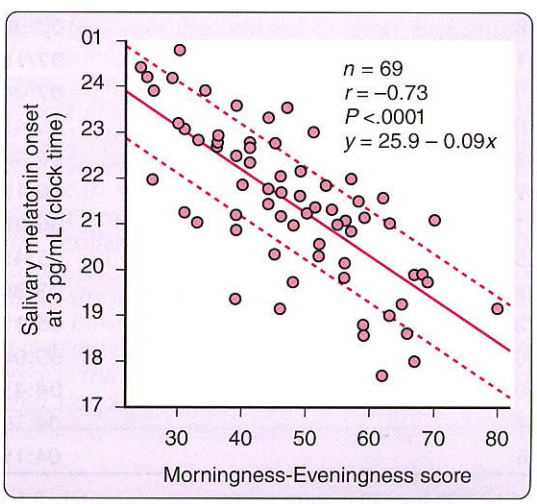

Figure 149-2 Correlation of Morningness–Eveningness (MEQ) score and dim-light melatonin onset (DLMO) in a group of 69 patients with winter depression. A 10.2-point difference in MEQ score corresponds to a 1-hour difference in DLMO. *Solid line,* linear regression; *dashed lines,* DLMO within 1 hour of the MEQ predictor.

facilitates circadian rhythm phase advances and the antidepressant response. Given the spread of DLMOs around the regression line (see Fig. 149-2), there is a risk that light will be scheduled too early, which might lead to premature awakening or counterproductive phase delays. The MEQ-based recommendation maximizes the chance that the patient will receive light in the window of 7.5 to 9.5 hours after DLMO. With a target time of 8.5 hours after DLMO, most patients (44/69, or 64% in our sample) will be captured in this window (± 1 hour) and 66/69 (96%) will receive light within ± 2 hours.

A list of recommended initial light exposure times, derived from the regression of the MEQ score on melatonin onset for 8.5 hours after DLMO, is shown in Table 149-1 (An online version of the MEQ,[49] which has been used successfully for more than 6 years, automatically returns the recommended light exposure interval to the user.) This schedule is a best-guess solution, and prompt timing adjustments may be needed. For example, if the circadian phase estimate were too early, a patient might report an uncontrollable urge to resume sleep for several hours after treatment (unintended phase delay), or sleep-onset insomnia beyond habitual bedtime, in which case light exposure should be moved later to bring the session into the 7.5 to 9.5 hour post-DLMO range. Similarly, if the patient suddenly starts waking up hours earlier than expected, the session should be moved later. On the other hand, with light exposure later than 9.5 hours after DLMO, there might be inadequate response. If 5 days of treatment shows no sign of improvement, the light should be moved earlier.

Table 149-1 Timing of Morning Light Therapy* Based on Morningness–Eveningness Score

MEQ SCORE	START TIME
16-18	08:45
19-22	08:30
23-26	**08:15**
27-30	**08:00**
31-34	**07:45**
35-38	**07:30**
39-41	**07:15**
42-45	**07:00**
46-49	**06:45**
50-53	**06:30**
54-57	**06:15**
58-61	**06:00**
62-65	**05:45**
66-68	**05:30**
69-72	**05:15**
73-76	**05:00**
77-80	**04:45**
81-84	04:30
85-86	04:15

*Start of 10,000-lux, 30-minute session, approximately 8.5 hours after estimated melatonin onset. **Bold** indicates the range confirmed in clinical trials.

MEQ, Horne-Östberg Morningness–Eveningness Questionnaire.

Data from Terman M, Terman JS. Light therapy for seasonal and nonseasonal depression: efficacy, protocol, safety, and side effects. CNS Spectr 2005;10:647-663.

The MEQ solution is not diagnostic of circadian phase.[50] For example, as shown in Fig. 149-2, it is possible for individual evening types (score below 42) and morning types (score above 58) to share the same DLMO.[51] Another source of error, of course, lies in the DLMO measure itself.[48] The MEQ algorithm offers the clinician an intelligent starting point that varies substantially from patient to patient, minimizing the problem of inappropriate treatment times, which would otherwise often be too early or too late.

According to this algorithm, treatment often begins earlier than habitual wake up time, depending on the patient's sleep duration. (Beyond the DLMO, the MEQ score is significantly correlated with sleep onset and offset derived from sleep logs of 1 week or longer.) For example, a short sleeper, whose bedtime is at midnight and who awakens at 6 AM, would start treatment on habitual awakening. In contrast, a longer sleeper, with onset at 11:30 PM and awakening at 7:30 AM, would need to wake up 1 hour earlier, at 6:30 AM. For every half hour of sleep beyond 6 hours, awakening for light treatment is 15 minutes earlier than habitual wake up time—up to 1.5 hours earlier for a sleep duration of 9 hours.

Although the algorithm is based on winter depression data, it has been applied successfully to patients with nonseasonal unipolar and bipolar depression[52] and moderate delayed sleep phase. However, for DSPD patients going to sleep after 2 AM, night shift workers, or the retired elderly, several questions on the MEQ will be difficult or impossible to answer, and timing decisions should be based on a pretreatment sleep log instead. Additionally, the MEQ score may be distorted by masking effects of hypnotic medication, compromising its usefulness as a circadian phase estimator.

INDICATIONS FOR TREATMENT

Circadian Rhythm Sleep Disorders

Delayed Sleep Phase Disorder

Patients with DSPD have difficulty initiating sleep before 2 to 7 AM, with commensurate difficulty awakening in the morning (for a review and discussion of the relationship with circadian rhythm phase, see reference 53). The problem is often exacerbated by light exposure during the night, even at normal room light levels, which can induce and maintain circadian rhythm phase delays. Similarly, forced early awakening and exposure to light can induce phase delays rather than advances. Once awake on their delayed schedule, most patients exhibit normal alertness and energy, but others report difficulties for several hours after awakening and spurts of energy after midnight. Often patients with DSPD show comorbid mood and personality disorders.

Morning light therapy can directly normalize the timing of the sleep episode without the need for progressive daily delays into the desired phase position,[54] an arduous procedure now rarely used. In one study, patients with DSPD were given 2 hours of early-morning light treatment at 2500 lux and light restriction after 4:00 PM.[55] Both the

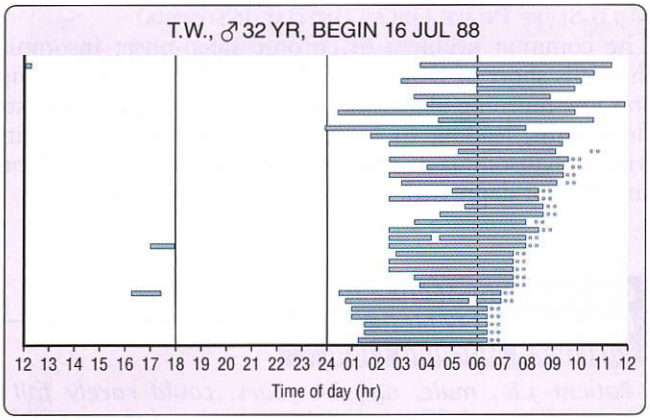

Figure 149-3 Self-report sleep and light therapy record for Patient T.W. (delayed sleep phase disorder). *Green bars*, sleep episodes on successive days (top to bottom); *sun symbols*, 15-minute intervals of 10,000-lux light exposure. Pretreatment baseline, 11 days (see Case 1 in the text).

body temperature rhythm and daily cycle of sleep-onset latencies showed phase advances, with an increase in morning alertness within 1 week. These effects were not obtained with the use of a dim light control.

The cases presented here, accompanied by sleep records, relate the experiences of three patients using morning light alone, morning light following progressive phase delays, or morning light in conjunction with evening low-dose melatonin.

Case 1: DSPD Phase Advances with Postsleep Light

Patient T.W. (Fig. 149-3) was a male, age 32 years, a freelance writer. He reported a lifelong history of DSPD with variable sleep onset averaging 5:00 AM and occasional hypersomnic episodes lasting 11 to 12 hours. Although not depressed when he sought help, he also reported experiencing subsyndromal symptoms of winter depression. The treatment consisted of gradually shifting light exposure earlier across days, beginning at 10:30 AM, a time of typical spontaneous awakening.

The patient monitored his level of sleepiness and time of awakening to determine the rate of shift. He was able to achieve successively earlier wake-up times over a period of 2 weeks while light-treatment sessions were advanced from 10:30 AM to 7:30 AM, but even at that point he could not fall asleep before 2:30 AM. However, when the treatment session was further advanced to 7 AM—an unprecedented time of awakening for this patient—sleep onset abruptly jumped approximately 2 hours earlier. The sleep episode stabilized at about 1:15 AM to 6:30 AM with light treatment on awakening.

After several months, the patient reported having increased light duration from 30 minutes (at 10,000 lux) to 45 or 60 minutes to enhance daytime energy. He also reported a relapse when discontinuing treatment twice within the next year. He self-managed his readjustments and reported the remission of depressed mood in the winter.

When morning light treatment fails to induce and maintain the desired phase advance, the patient may undergo a schedule of successive delays in the sleep episode until the desired target phase is achieved, usually within a week. The patient then attempts to keep sleep–wake timing consistent. As delay chronotherapy was originally applied,[54] the shifting sleep episodes were couched in darkness. It followed that the timing of light exposure changed during and after the phase adjustment. By the end of the procedure, the patient begins to receive a normalized pattern of daily light exposure that serves to maintain the target phase. Early morning light therapy can forestall further drifting toward the original delayed sleep phase, which is a large risk. Additionally, presleep light exposure can be used to expedite the daily phase delays, with a switch to postsleep light exposure during maintenance treatment.

Case 2: DSPD Progressive Delays with Presleep Light Followed by Stabilization with Morning Light

Patient S.P., male, age 47 years, experienced a delayed sleep pattern that had been present since childhood. Although he was groggy on awakening, afternoon and evening energy levels were high, and he could work productively at those times. On nights when he used a benzodiazepine hypnotic, he could sometimes advance sleep onset by a few hours, which he considered trivial.

An attempt was then made to phase advance the sleep episode with the use of 10,000-lux, 30- to 45-minute light exposures on awakening. Despite intense effort over a 1-week trial, the patient could not be awakened before 12:30 PM, making successively earlier treatment sessions impossible. An alternative course of chronotherapy was then attempted.

The patient was instructed to delay successive sleep episodes by 2 hours, in conjunction with 1-hour light treatment sessions ending 2 hours before bedtime (a procedure intended to facilitate successive phase delays). However, he refused to enter bed until ready to fall asleep, resulting in daily delays that varied between 1 and 5 hours. After 6 days, the presleep light was discontinued, with the instruction to substitute light exposure for 2 hours at 6 AM in an attempt to halt the delay drift. In the next weeks, the patient was able to maintain sleep onset between 11 PM and midnight and to awaken by 7:30 AM or earlier.

The resilience of the adjustment was tested on the occasion of two late-night parties after which the desired sleep pattern was easily recaptured. Subsequently, however, the patient discontinued treatment and resumed his former schedule, citing family stresses that he preferred to escape by sleeping during the day.

With the identification of phase-shifting properties of exogenous melatonin in humans,[56] we have the prospect of an enhanced protocol for treatment of DSPS. Melatonin can be used to phase-advance the cycle with a dosing regimen[57] distinct from its use close to bedtime

as a soporific agent (which remains controversial).[58,59] The phase response curve to melatonin is approximately opposite to that for light. Thus, morning light and evening melatonin both serve to promote phase advances. In the present application, melatonin is administered in low, physiologic doses (0.1 to 0.3 mg) several hours before bedtime (earlier than endogenous pineal melatonin onset). Because it is not soporific, the patient can continue normal evening activities. However, once the melatonin is ingested, the patient must avoid inappropriate, countervailing ambient light until bedtime, which could induce phase delays and inhibit the onset of pineal melatonin production. Additionally, melatonin is a suspected retinal photosensitizer,[23] which mandates cautionary eye protection. An alternative to dark goggles,[60] which would constrain evening activities, is blue-blockers (see Resources, later) that maintain clear visibility while blunting retinal circadian photoreception.

Melatonin has been used as monotherapy to halt the free-running cycle of totally blind people,[61] in which case there is no interaction with light exposure. Clinically, we have found melatonin especially useful for DSPS in conjunction with postsleep light therapy. Although light monotherapy can be effective for DSPS (e.g., Case 1), the advancing schedule is especially difficult for patients who are not falling asleep until early morning. Adding melatonin 4 to 6 hours before current sleep onset and moving light exposure and melatonin ingestion gradually earlier—as an ensemble—can expedite progress. Once sleep has stabilized, melatonin might maintain the pattern after discontinuation of light therapy, with spontaneous exposure to early morning postsleep light.

Case 3: DSPD Phase Advances with Evening Melatonin and Blue Blockers and Postsleep Light

Patient J.M. (Fig.149-4) is male, age 28 years, and an accomplished scholar. He had experienced extreme delayed sleep phase disorder since adolescence. It was aggravated in recent years, with sleep onset around 7 AM and waking around 3 PM. The distinct downside for him was the inability to collaborate with colleagues during the normal workday. He resisted hypnotic medication but intermittently used alcohol to fall asleep a few hours earlier.

The phase-advancing strategy combined three chronotherapeutic methods: 0.2 mg controlled-release melatonin[62] in the evening, blue-blocking (400 to 535 nm) glasses until sleep onset, and 1 hour of light therapy at 10,000 lux upon awakening. Melatonin was initially taken too early (about 7 hours before sleep onset) because the patient insisted he was ready to fall asleep earlier; after stabilization, the delay to sleep was about 4 to 5 hours. He followed a 1- to 3-day schedule of 30-minute advances in wakeup time and light therapy, based on his level of confidence that he would not oversleep. Sleep onset remained spontaneous throughout. He achieved the goal of sleep from 11:30 PM to 7 AM in about 2 weeks and expressed incredulity that this could happen.

Mild Sleep Phase Delay (Initial Insomnia)

The common problem of chronic sleep-onset insomnia that falls short of DSPD but also entails difficulty arising and low morning alertness, is readily treatable with postsleep light, leading to rapid adjustment. Many patients with such insomnia do not respond to hypnotic medication and are not depressed.

Case 4: Mild Sleep-Phase Delay Counteracted by Postsleep Morning Light

Patient J.B., male, age 34 years, could rarely fall asleep before 1:30 AM or wake up in time for a normal workday. Although he was allowed to work from midmorning into the evening, he was handicapped by low alertness until midafternoon and headaches at a computer terminal during the late afternoon.

Light treatment began with 10,000-lux exposures at 8 AM for 30 minutes, with no effect in advancing sleep onset for several days. When the session was advanced to 7:30 AM, sleep onset immediately advanced by about 1 hour. However, several days of terminal insomnia followed, with awakenings before 6 AM, signaling an overdose. Reducing the treatment duration to 15 minutes at 7:30 AM alleviated this problem, with sleep onset maintained around midnight. This regimen was continued, with effortless awakening accompanied by improved morning alertness and complete remission of the headache.

Advanced Sleep Phase Disorder

Advanced sleep phase disorder, in which sleep onset occurs in the evening with awakening well before dawn, would seem to provide a counterpart to DSPD, treatable with late evening light, but such treatment has not been extensively investigated. Light presented in the first part of the subjective night is known to elicit phase delays in the onset of nocturnal melatonin secretion[44] and the decline of body temperature,[63] which might promote later sleep onset.

Case 5: ASPD Counteracted by Presleep Evening Light

The experience of a 38-year-old woman with a lifetime history of ASPD[64] illustrates the potential use—and limitations—of evening light treatment. Patient K.W. was a mildly hypomanic high achiever, without seasonal pattern, who typically fell asleep at about 9:00 PM and woke up between 2 AM and 4 AM, a pattern that led to marital stress. She could remain awake for occasional late-evening engagements by compensating with delayed time of arising at 5 AM to 6 AM. At baseline, she exhibited an early melatonin onset, at about 7:45 PM. Light exposure at 2500 lux for up to 2 hours beginning at 8 PM barely affected sleep phase or melatonin onset, whereas light exposure beginning at 9 PM succeeded in maintaining sleep onset at about 11 PM and wakeup time between 4 AM and 5 AM, which was accompanied by a 1-hour delay in melatonin onset.

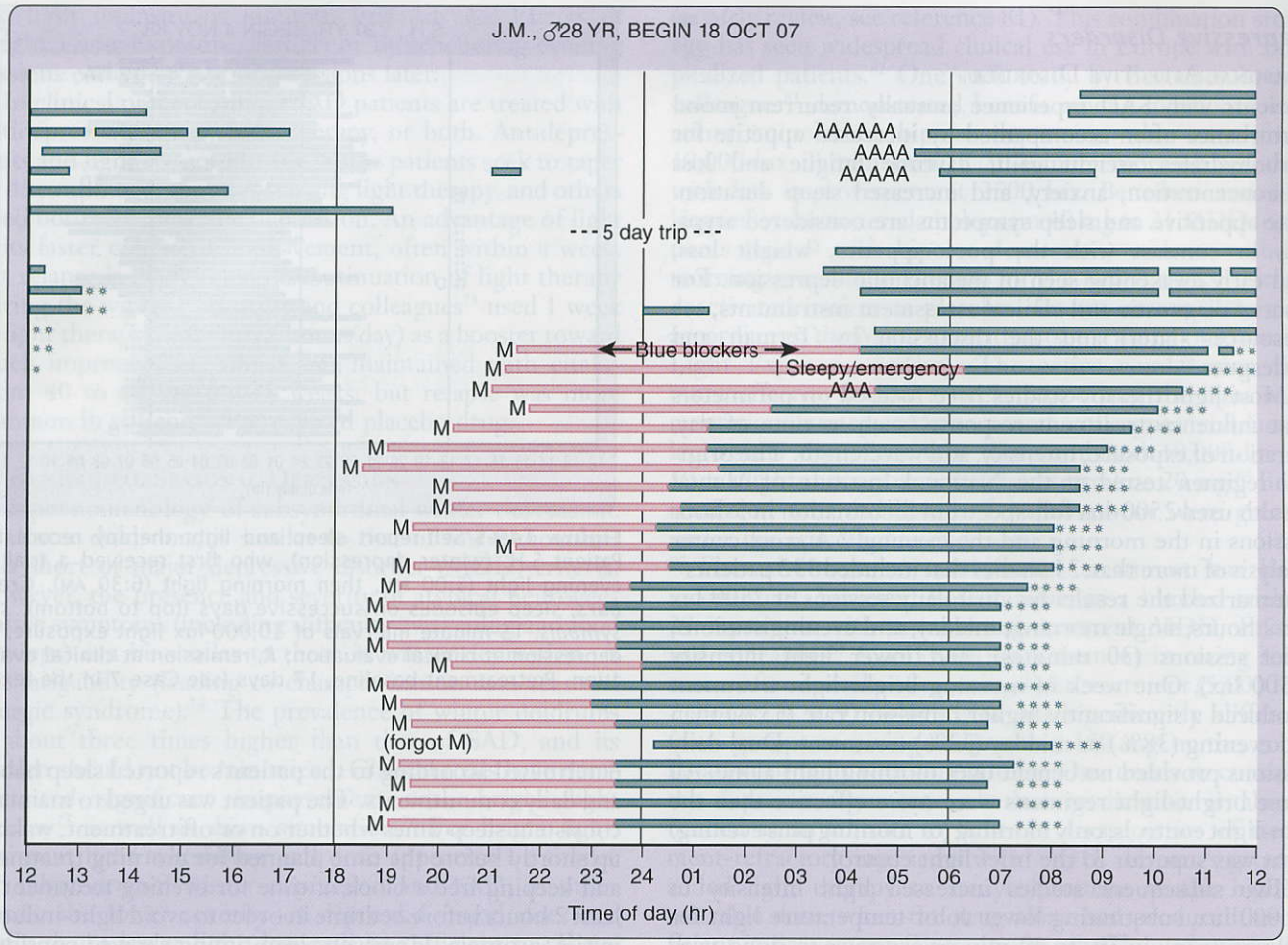

Figure 149-4 Self-report sleep and light therapy record Patient J.M. (delayed sleep phase disorder). *Green bars*, sleep episodes on successive days (top to bottom); *sun symbols*, 15-minute intervals of 10,000-lux light exposure; *red bars*, blue-blocking glasses; *A*, alcohol; *M*, 0.2 mg controlled-release melatonin; *dashes*, gap in record. Pretreatment baseline, 13 days (see Case 3 in the text).

Although ASPD is not strictly age-related, it is more prevalent among the elderly, whose early rise time is a common cause of concern. Campbell and colleagues[65] compared the effects of evening bright-light exposure (>4000 lux for 2 hours) with a dim red-light control in elderly subjects with histories of sleep maintenance insomnia. The bright light group showed improved sleep efficiency; after 12 days of treatment, nighttime wakefulness decreased by about 1 hour. Despite this benefit, most subjects declined to continue treatment, given the long exposure sessions and glare discomfort. These drawbacks might be corrected with shorter exposures to higher intensity light in a glare-free configuration (see Apparatus, earlier).

Free-Running Sleep Disorder

When sleep phase does not stabilize but continually shifts later relative to clock time, the pattern resembles the free run seen in normal subjects under conditions of temporal isolation without day and night cues. However, in free-running sleep disorder, despite the presence of such cues, a failure of entrainment is evidenced by a hypernychthemeral[66] sleep pattern. Some patients with DSPD break into transient hypernychthemeral patterns, which suggests that free-running sleep disorder and DSPD are associated disorders of varying severity.[67] To halt the delay drift, light therapy is introduced when the subjective and objective nights coincide.

Case 6: Free-Running Sleep Halted by Morning Light

Patient B.C., male, age 31 years, showed a sleep–wake cycle length averaging 25 hours over approximately 13 years preceding treatment.[68] He was unemployed and socially withdrawn and refused to attempt to sleep when alert. Treatment began when sleep onset had drifted to midnight. The patient was exposed to light of 4000 to 8000 lux for about 2.5 hours on awakening. The free run immediately decelerated, and the sleep interval was maintained at approximately 1:30 AM to 8:15 AM for several weeks. In the long run, however, the sleep pattern continued to drift at a period of about 24.08 hours, a problem that might have been corrected with increased light dose, adjunctive evening melatonin treatment, or both.

Depressive Disorders

Seasonal Affective Disorder

Patients with SAD experience annually recurrent mood disturbance often accompanied by increased appetite for carbohydrates, weight gain, daytime fatigue and loss of concentration, anxiety, and increased sleep duration. The appetitive and sleep symptoms are considered atypical, in contrast with the poor appetite, weight loss, and early awakening seen in melancholic depression. For a set of diagnostic and clinical assessment instruments, see Resources (later) and the discussion by Terman and colleagues.[69]

Most light therapy studies have focused on parameters that influence treatment response, such as time of day, duration of exposure, intensity, and wavelength. The original regimen tested at the National Institute of Mental Health used 2500-lux full-spectrum illumination in 3-hour sessions in the morning and the evening.[29] A cross-center analysis of more than 25 studies that included 332 patients[70] summarized the results for dual daily sessions at 2500 lux for 2 hours; single morning, midday, and evening sessions; brief sessions (30 minutes); and lower light intensity (<500 lux). One week of morning bright-light treatment produced a significantly higher remission rate (53%) than did evening (38%) or midday (32%) treatment. Dual daily sessions provided no benefit over morning light alone. All three bright-light regimens were more effective than the dim-light controls; only morning (or morning plus evening) light was superior to the brief light control.

Two subsequent studies increased light intensity to 10,000 lux, substituting lower color-temperature light for full-spectrum, in 30- to 40-minute exposure sessions, with remission rates of approximately 75%, matching the most successful 2500-lux, 2-hour studies.[5,71] At these short durations, both dim light (400 lux) and lower-level bright light (3000 lux) were significantly less effective.

Three large controlled clinical trials have clarified the specific action of antidepressant light relative to placebo and the influence of exposure time of day. Eastman's group[42] administered light in the morning or evening, and an inert placebo (an inactive negative-ion generator), to parallel groups. Although all groups showed progressive improvement over 4 weeks, patients administered morning light were most likely to show remissions, exceeding the placebo rate. Lewy's group[43] conducted a crossover study of morning and evening light. Although there was no placebo control, morning light proved to be more effective than evening light. Terman's group[41] performed both crossover and balanced parallel-group comparisons, which included nonphotic control groups that received negative air ions at a low or high concentration. Morning light produced a higher remission rate than evening light and the putative placebo, low-density ions. However, the response to evening light also exceeded that for placebo. Indeed, in the trials of Lewy's group[43] and Terman's group,[41] a minority of patients responded preferentially to evening light.

Figure 149-5 compares morning and evening light therapy and its relation to the sleep cycle for a representative patient with SAD who received 10,000-lux light treatment in 30-minute sessions. Treatment timing was determined according to the patient's reported sleep habits and daily commitments. The patient was urged to maintain consistent sleep times whether on or off treatment, waking up shortly before the time planned for morning treatment and keeping free a block of time for evening treatment at least 2 hours before bedtime in order to avoid light-induced initial insomnia. However, sleep timing showed considerable variability depending in part on the time of treatment and treatment response.

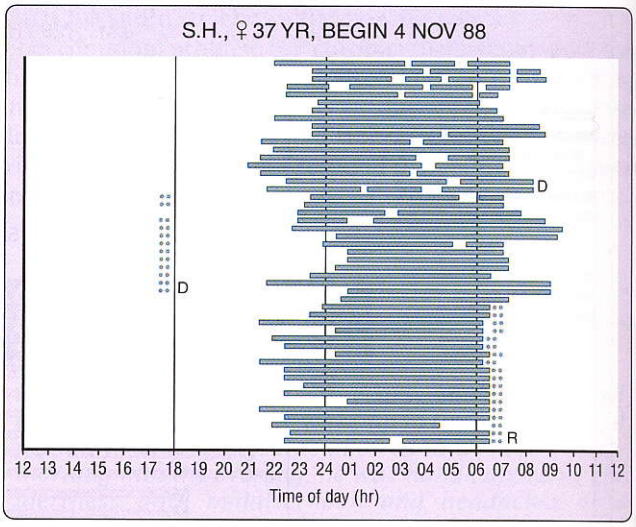

Figure 149-5 Self-report sleep and light therapy record for Patient S.H. (winter depression), who first received a trial of evening light (5:00 PM), then morning light (6:30 AM). *Green bars*, sleep episodes on successive days (top to bottom); *sun symbols*, 15-minute intervals of 10,000-lux light exposure; *D*, depression at clinical evaluation; *R*, remission at clinical evaluation. Pretreatment baseline, 17 days (see Case 7 in the text).

Case 7: Differential Antidepressant Response to Evening and Morning Light Therapy for Winter Depression

Patient S.H. (see Fig. 149-5), female, age 37 years, became depressed every winter, with middle insomnia and oversleeping on weekends. During the course of early-evening light treatment at 5 PM, sleep onset was gradually delayed, with reduced nighttime awakening. She remained depressed. In contrast, under morning light treatment at 6:30 AM, sleep onset returned to the baseline pattern, sleep interruptions were largely eliminated, and the depression remitted.

The generally poor response to evening light appears to be correlated with delayed sleep onset, time of arising relative to baseline, or both. By contrast, morning light usually serves to truncate morning sleep. Some patients conserve sleep duration by going to bed earlier, but many do not and feel fine with sleep shortened by about 30 minutes. Although the common symptom of interrupted sleep is often eliminated under effective treatment, initial, middle, or terminal insomnia sometimes emerges. The insomnia could signal light overdose that can be dealt with by reduc-

ing light intensity or duration (see Adverse Effects of Bright Light Exposure, earlier) or by scheduling evening sessions earlier or morning sessions later.

In clinical practice, many SAD patients are treated with antidepressant drugs, light therapy, or both. Antidepressants and light are compatible. Some patients seek to taper or discontinue drugs after starting light therapy, and others need both to relieve the depression. An advantage of light is its faster course of improvement, often within a week. Yet relapse is likely upon discontinuation of light therapy during the winter.[72] Martiny and colleagues[73] used 1 week of light therapy (5000 lux, 2 hours/day) as a booster toward quick improvement, which was maintained with citalopram 40 to 60 mg for 15 weeks, but relapse was more common in patients who received placebo drug.

Subsyndromal Seasonal Depression

The phenomenology of subsyndromal winter depression, or winter doldrums, is similar to that of SAD, though it falls short of major depressive disorder (MDD) criteria. However, the presence and severity of atypical neurovegetative symptoms (including difficulty awakening and food cravings) can be similar to those in winter depression, as can fatigability (leading to characterization as a seasonal anergic syndrome).[74] The prevalence of winter doldrums is about three times higher than that of SAD, and its burden should not be minimized. Clinical trials have demonstrated significant improvement with bright light therapy[75] as well as dawn simulation.[76] For bright light, optimum light scheduling and dose appear to be similar for subsyndromal winter depression and SAD; in other words, the lower severity of depressed mood does not imply that a lower light dose will be sufficient to relieve symptoms.

Nonseasonal Unipolar Depression

Monotherapy with Light

Beyond its established application for winter depression, light therapy for nonseasonal depression appears both safe and effective. Kripke[77] compared several controlled trials in terms of the relative benefit of light versus placebo and found light therapy for as little as 1 week produced improvement within the range of classic antidepressant drug studies of 4 to 16 weeks. For example, Yamada and colleagues[78] gave 7 days of light therapy to 27 inpatients with nonseasonal major depression and obtained a benefit of 24% over a dim-light placebo. However, morning or evening exposure times showed no difference, nor did phase shifts of body temperature relate to clinical improvement. Wirz-Justice and colleagues[79] used a ceiling-light installation at 3000 to 4000 lux in a 10-day open-label trial with 28 unmedicated hospitalized patients, with improvement greater than 50% in half the cases. Goel and colleagues[80] gave 5 weeks of morning light therapy (10,000 lux, 60 minutes) to outpatients with chronic major depression. The remission rate was 50%, and a control group given low-density negative-air ionization as placebo showed negligible improvement.

Combination Treatment

Several investigators have combined light with drugs and found accelerated improvement relative to drugs alone (for an early review, see reference 81). This combination strategy has seen widespread clinical use in Europe with hospitalized patients.[82] One such study by Beauchemin and colleagues[83] demonstrated benefit among inpatients with either unipolar or bipolar depression who were given 10,000-lux illumination in 30-minute morning sessions, with less improvement at 2500 lux. Benedetti and colleagues[52] administered citalopram 40 mg to 21 MDD inpatients (and 9 with bipolar depression) with or without morning green-light therapy for the first 2 weeks, using a deactivated ionizer as placebo. Treatment was scheduled according to the MEQ algorithm (see Timing of Morning Light Exposure, earlier). The active combination was more effective within 1 week, supporting the light augmentation strategy. Martiny and colleagues conducted large-scale outpatient trial that combined 10,000-lux or 50-lux light therapy with standard sertraline 20 mg.[84] Both remission rate and speed of improvement were greater under the active light condition.

The advantage of combination treatment has been inconsistent, however. For example, in a blinded trial of 29 inpatients with nonseasonal recurrent MDD, Prasko and colleagues[85] found 64% improvement in rating scale scores after 3 weeks of morning light treatment (5,000 lux, 2 hours; $n = 9$), which was not significantly different from groups receiving imipramine 150 mg or the combination of light with imipramine. One needs to consider the flip-side argument—whether medication is always needed as an adjunct to light. In a case series of treatment-refractory inpatients with MDD, Terman's group[86] added light therapy to tranylcypromine at the highest tolerable dose, when the drug alone had produced insufficient improvement. Two patients who showed remission under this combination, and continued with home treatment after discharge, experienced relapse within 2 days of skipping light therapy sessions, yet recovered within 2 to 4 days of resumption. The necessity of drug treatment could be questioned.

A Cochrane meta-analysis[87] has confirmed the therapeutic utility of bright light boxes (but not other exposure methods) for nonseasonal unipolar depression, noting that results were strongest in combination with drugs. With more selective screening of high-quality studies, Even and colleagues[88] have endorsed light therapy for nonseasonal depression, but only as an adjuvant. They urged refinement of homogeneous MDD subgroups that might respond to light monotherapy on the basis of circadian rhythm characteristics.

Going a step further, Neumeister and colleagues[89] combined light with drugs and a single session of late-night sleep deprivation[90] (wake therapy) at the start of treatment and achieved marked improvement in 1 day and benefit over a dim light control within 1 week. Combined light and wake therapy can be self-administered at home to MDD patients who are able to self-manage it. Loving and colleagues[91] used this method in a group for whom standard antidepressants and psychotherapy were inadequate, and achieved a remission rate of 43%.

Nonseasonal Bipolar Depression

The light therapy strategy for bipolar depression is similar to that for MDD, although clinicians must be vigilant

about emergence of manic or mixed states, even with mood stabilizers.

Colombo and colleagues[92] administered three cycles of wake therapy over 1 week in patients who were either stabilized on lithium or were medication free. Subgroups also received 30-minute morning light therapy at 2500-lux white or 150-lux red or normal ambient room illumination at 80-lux. Patients receiving bright-light therapy with or without lithium showed sustained, incremental improvement without mood reversals following sleep deprivation. The lithium-alone group showed similar improvement, but without lithium or bright light there was minimal overall improvement, with distinct reversals after sleep-deprivation nights. This study underscores the advantage of light therapy in sustaining and enhancing the transient benefit of wake therapy.

In a follow-up study, Benedetti and colleagues[93] combined wake therapy, morning light therapy, lithium, and antidepressants for inpatients with bipolar depression. Of 27 patients with histories of drug resistance, 44% responded during 1 week of acute intervention, and nonresistant patients showed 70% success. Patients were followed for 9 months at home on their usual medications (without light treatment), and the relapse rate differed significantly for drug-resistant (83%) and nonresistant (43%) subgroups. Although this study does not isolate light therapy as the critical intervention, it underscores the effectiveness of the chronotherapeutic combination with wake therapy in acutely reversing the depressive episode even in resistant cases. A case series of drug-resistant bipolar patients who continued light therapy at home with sustained benefit[86] points to the need for and utility of long-term maintenance treatment.

In an instructive pilot study of light therapy during depressed phases of rapid-cycling bipolar disorder, Leibenluft and colleagues[94] were struck by increasingly labile mood when 10,000-lux light was administered in the morning for up to 60 minutes, whereas midday presentation was effective. Sit and colleagues[35] studied nine women with longstanding nonseasonal bipolar I or II disorder in which mood stabilizers controlled manic phases, but antidepressants did not relieve depressed phases. Light duration was flexibly dosed between 15 and 45 minutes to maximize improvement while minimizing side effects. Three of four patients beginning 7000-lux morning bright-light therapy experienced disruptive mixed states forcing discontinuation, but the fourth showed a full, sustained response. The next five patients used midday light without such disruption, with success in three cases. One midday nonresponder who switched to morning light then responded. Clearly, despite the impressive initial results, the dosing and timing of light therapy for bipolar depression needs further scrutiny.

Though dawn simulation has not yet been tested in bipolar depression, it is a milder intervention that might especially suit this population. In a retrospective chart review of 187 inpatients with bipolar depression,[95] those who occupied east-facing rooms with direct natural dawn illumination were discharged 3.7 days earlier than those facing west. No such benefit was found for patients with MDD, however.

Other Indications

Practice parameters or meta-analytic endorsement of light therapy for circadian sleep phase disorders[96,97] and seasonal[98] and nonseasonal depression[87,88] are now in place. Other indications for light therapy might emerge in the future. See reviews[46,86] of clinical trials of light therapy for antepartum depression, premenstrual dysphoric disorder, geriatric depression,[98a] bulimia nervosa, adult attention-deficit/hyperactivity disorder, dementia, and Parkinson's disease.

TOWARD AN INTEGRATED CHRONOTHERAPEUTICS

Three main applications of light therapy have emerged: light therapy alone, combination of light therapy with antidepressant and mood-stabilizing drugs, combination of light therapy with other chronotherapeutic methods to accelerate and maintain treatment response, and combination of all three types of therapy (light, medication, and chronotherapy).

The range of chronotherapeutic methods beyond light therapy includes sleep deprivation, or wake therapy; wake therapy followed by sleep phase-advance therapy, in which bedtime is moved earlier, with maintained wakefulness at the end of the night; low-dose melatonin administered several hours before scheduled bedtime, to facilitate circadian rhythm phase advances; and blue-light restriction in the evening (alone or in conjunction with melatonin) to forestall phase delays or in the morning to promote phase delays. Blue-light restriction and melatonin administration in the evening derive from laboratory studies[27,99] and clinical experience, with clinical trials still pending.

Light monotherapy is a potent tool for treating the circadian rhythm sleep disorders, though patients with extreme delayed sleep phase appear to respond most rapidly to a combination of postsleep light in coordination with physiological-dose melatonin 4 to 6 hours before sleep onset, with light restriction (in the short-wavelength range below ~535 nm) until bedtime. For patients already using hypnotic drugs, the chronotherapeutic regimen should be introduced before attempting to taper and discontinue the drug. Although tapering can require weeks to months, it can begin as soon as the patient reports onset of sleepiness before taking the sleeping pill.

Light monotherapy for depression has been best demonstrated for treatment of seasonal affective disorder, for which complete clinical remission with normalization of hypersomnia is often obtained within a week. Clearly, light alone should be the first course of treatment for patients with SAD. For those already using antidepressants, light can be added and the drug subsequently tapered, even to discontinuation. Combining light with drugs may be more effective for treating nonseasonal depression, even though light monotherapy works in individual cases, notably in patients with drug resistance whose main issue is circadian phase maladjustment.

For hospitalized patients with depression, a chronotherapeutic combination in conjunction with ongoing medication appears very promising, with the prospect of unprecedented rapid, maintained remission and shorter hospital stay. The protocol combines multiple nights

of wake therapy with interspersed recovery sleep,[93,100] earlier bedtime and rise time for recovery sleep (sleep phase advance),[101] and initiation of daily light therapy after the first night of sleep deprivation. Timing of all the chronotherapeutic procedures—not only light—is anchored individually to the patient's assumed circadian phase position, as inferred from the MEQ, habitual sleep schedule, or both.[102]

RESOURCES

The nonprofit Center for Environmental Therapeutics (http://www.cet.org) offers the following materials for clinicians and patients:

- A 10,000-lux light box: tilted, height-adjustable, 4000-Kelvin fluorescent with polycarbonate filter diffuser
- Blue-blockers: short-wavelength filtered fitover or nonfitover glasses
- Clinical instruments: the Columbia Clinical Assessment Tools packet, including rating scales and structured eye examination chart
- Self-rating instruments: downloadable or automated assessments of morningness–eveningness (MEQ-revised with American idiom and categorical scoring),[49] seasonality and depression, with personalized feedback.

The Society for Light Treatment and Biological Rhythms (http://www.sltbr.org) offers a continuing medical education course associated with its annual scientific meeting and hosts a lively listserv for members.

❖ Clinical Pearl

Appropriately timed artificial light exposure can correct sleep-phase maladjustment and counteract seasonal and nonseasonal depression. The clinician's tasks are to determine the interval of the individual patient's subjective night and to schedule light at its end for phase advances or at its beginning for phase delays.

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