

Light Treatment and Biological Rhythms

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ANNUAL MEETING REVIEW

Mechanisms of Action of Light Therapy

There are three major theories regarding the underlying mechanisms of action of light in seasonal affective disorder: the melatonin hypothesis, the phase-shift hypothesis and the amplitude hypothesis. Several presentations at the June SLTBR meeting shed new light on these hypotheses.

Melatonin Hypothesis

In seasonal species, such as the hamster and sheep, effects of changing photoperiod are mediated by alterations in the secretory pattern of melatonin. Since bright light is capable of suppressing melatonin in the human, it has been postulated that melatonin mediates the effects of shortening days on the winter symptoms of SAD. Studies using atenolol to suppress nighttime melatonin and of skeleton photoperiod effects on the melatonin secretory pattern have not provided supporting evidence of a role of melatonin in SAD. Such studies, however, have failed to take into account the circadian rhythm in melatonin sensitivity. This rhythm, which has been demonstrated in experimental animals, is itself regulated by the photoperiod as well as by other factors. The relationship of melatonin secretion to the sensitivity rhythm and other rhythms may therefore be important.

Avery and co-workers reported on a study of core temperature under a constant routine used to eliminate such masking effects. Under these conditions, a phase-delay in the temperature acrophase was observed in SAD subjects and there was a

statistical trend for phase-advance of the temperature acrophase by light therapy. The thyrotropin and cortisol rhythms were also phase-delayed in SAD patients.

Depue examined the circadian rhythms of activity and of core temperature. In SAD subjects, a phase-delay of the temperature rhythm was seen with dim light treatment, delivered in morning and evening, and there was a significant phase-advance with bright light treatment. Bright light also produced changes in the activity pattern in SAD subjects which were not seen in controls.

These studies emphasize the importance of examining markers of circadian rhythms other than melatonin. They also suggest that the melatonin hypothesis should not be rejected prematurely. The possibility cannot be excluded that the pattern of

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melatonin secretion in relation to the pattern of other rhythms—some of which are presumably linked to melatonin sensitivity—is a key factor in the pathogenesis of SAD.

Phase-Shift Hypothesis

According to Lewy et al. [*Science* (1987) 235: 352-354], patients with SAD show a phase delay of melatonin secretion and light therapy produces an antidepressant effect by phase-advancing these patients. In keeping with this theory, morning light has been shown to be more effective in treating SAD than midday or evening light. In fact, according to phase response curve theory, evening light should be ineffective or even produce increased symptomatology as it produces further phase-delay. The finding that evening light is efficacious (further supported by a study by Doghramji et al. reported at the SLTBR meeting), is now accounted for by Lewy et al. (1987) by a second hypothesis, that light has a secondary, "energizing effect." Lewy

et al. have also reported that patients with SAD show a phase-delay in dim light melatonin onset with a significant phase-advance of this onset following morning light treatment. The latter finding has been supported by some, but not all, investigators.

The question of SAD patients' sensitivity to light was addressed by several investigators at the meeting. This issue has a direct bearing on the phase-shift hypothesis, as one mechanism by which a phase-shift might occur would be an altered sensitivity to light. Oren and co-workers hypothesized that patients with SAD would prove to be *less* sensitive to dim light, and hence would have phase-delayed rhythms. They examined depressed SAD patients and age- and sex-matched controls using a modified Goldmann-Weekers adaptometer with Bekesy tracking. Using this measure, an average *supersensitive* response was found in SAD patients. It remains to be determined whether the measure, which involves conscious perception of light, is appropriate. If melatonin alteration is seen as a key factor, it may be essential to measure melatonin itself.

Partonen and co-workers presented a paper which provides support for the phase-delay finding. SAD patients in Helsinki showed a 1-to-2 hr phase delay in the melatonin rhythm; this rhythm was advanced significantly by morning light treatment administered for 1 hr over 5 days. All patients reported feeling better after light therapy.

Schlager and co-workers successfully treated winter depressives with dawn twilight therapy using precisely-controlled naturalistic illumination with light intensity < 500 lux. In support of the phase-shift hypothesis, overnight plasma melatonin showed dawn-induced suppression and a circadian phase-advance. The studies by Avery and Depue, described above, also provide support for the phase-shift hypothesis.

Gaddy and co-workers examined light-induced melatonin suppression in depressed SAD patients.

Melatonin suppression sensitivity was compared with that of conscious perception of light, as tested with an Octopus 2000 Ganzfeld stimulator. In this study, perception did not differ from clinical norms; however, patients exhibited a 52% suppression of melatonin when exposed to an hour of 500 lux of light. The investigators suggest that this provides evidence for supersensitivity to the neuroendocrine effects of light. Unfortunately no matched controls were examined in this study.

The question of the sensitivity for suppression in normal humans was examined by Armstrong and co-workers. A midnight light pulse of 1 hr duration caused significant suppression of melatonin over a range of 3000 lux down to 350 lux. Suppression of melatonin with 500 lux and below in normal humans is contrary to that reported in the literature and further illustrates the need for comparison of patient and control groups under identical conditions.

Taken together, these studies suggest that there is no evidence of subsensitivity to light in SAD, but that there may be supersensitivity. These findings suggest that subsensitivity to light is not the mechanism underlying phase-delay. Lewy and co-workers have proposed that SAD patients have a longer underlying free-running period (τ) than normals, as one explanation of phase-delay. Lack of subsensitivity makes this explanation more likely. If supersensitivity is established in future research, it will be difficult to reconcile with the phase-shift hypothesis.

Amplitude Hypothesis

A third theory regarding the effects of exposure to bright light holds that light therapy may act by increasing abnormally low circadian amplitude in patients with SAD [Czeisler *et al.* (1987) *Psych. Clin. North America* 10: 687-709]. This theory was based on an unanticipated finding that exposure to bright light could either markedly enhance or suppress endogenous circadian amplitude of

temperature, depending on the circadian phase of the light administration. Bright light in either morning or evening has an amplitude-enhancing effect. In contrast, effects of bright light on phase are differential, with exposure in the morning or evening inducing a phase-advance or phase-delay, respectively.

Levendosky and co-workers presented a study which found that circadian rhythm of core body temperature did not differ in SAD patients and controls in either winter or summer. In the winter, however, nocturnal temperature was decreased by light treatment, so that there is an increased amplitude of the temperature rhythm without any effect on phase. The design of the study was such that masking could create an artifact in both phase and amplitude of the temperature rhythm.

As noted above, Avery and co-workers examined core temperature rhythms using a constant routine to eliminate masking effects. No differences in amplitude were found between patients and controls with or without treatment.

The three major theories of the biological effects of bright light treatment, therefore, all received some support at the annual meeting. These three theories, which are not mutually exclusive, have been useful in helping to focus research directions. However, even if one or more of these theories were proven to be correct, our understanding would still be incomplete, as it is essential that we know the internal biological processes that mediate these effects.

Photon-Counting Hypothesis

A further hypothesis, only touched upon briefly, was the photon-counting hypothesis. Many workers find differences between SAD patients and controls or between SAD patients in winter and summer. Furthermore, even evening light has been reported as effective. Thus, the intensity of light (photon counts) may be important.

Role of Ultraviolet Light

Another aspect of light that received attention at the meeting was the question of whether ultraviolet radiation (UV) is critical for the antidepressant response. This issue is important, as we are confronted with manufacturers who are advertising full spectrum fluorescent light as a cure for "light malnutrition."

Lam and co-workers investigated patients exposed to full spectrum fluorescent light for two hours in the morning for one week while wearing specially prepared eye glasses that block 99% of UV transmission without affecting the intensity. UV and UV-blocked light were both effective on atypical symptoms, but UV was essential for significant changes in Hamilton Depression Scale scores. One problem with this study is that the photometers used to measure light intensity are relatively insensitive in the UV region and therefore may underestimate its contribution to the intensity.

Yet another study by Lebeque and co-workers compared full spectrum to cool white light and found the two treatments equally effective in reducing and/or eliminating SAD symptoms. Oren and co-workers examined different light wavelengths in treatment of SAD. SAD patients were assigned to red or green light treatments for one week in a cross-over design. Green light had efficacy equivalent to full spectrum light, while red resembled placebo effects in other studies. The green light was filtered through an ultraviolet filter, thus eliminating all UV.

In general, these studies suggest that although UV may have some clinical effects, it is not essential for the treatment of SAD.

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BOOK REVIEW

Seasons of the Mind: Why You Get the Winter Blues & What You Can Do About It

The study of seasonal depression has reached the point where we can expect to see a number of resource manuals for the SAD sufferer in the near future. Although such books can play an important role in educating the general public—within the coming year, at least seven will be on the shelves—I must admit to viewing this trend with some reservations: it is already difficult to find SAD research participants who are naive observers of their symptoms.

Three books for the lay reader are already available. *The Hibernation Response*, by P. Whybrow and R. Bahr [reviewed in Vol. 1, No. 2] and *Light Up Your Blues*, by R.N. Moreines and P.L. McGuire [reviewed in Vol. 1, No. 5], are both quick and light introductions to the topic of fall-winter depression. The authors appear to have some personal familiarity with light therapy, but they have not been intensively involved in the study of SAD. Now, *Seasons of the Mind*, by N.E. Rosenthal (Bantam Books, 1989), represents the first comprehensive discussion of SAD written for the lay public by a leading innovator in the field.

Whatever else may be said about the book, *Seasons of the Mind* is the most thorough description available of the clinical aspects of fall-winter depression. The book is rich in case material as well as historical reference. It begins with anecdotes concerning the first identified cases of seasonal variations in mood (including those of the author), and then moves on to discuss the typical symptom picture of the SAD sufferer. This is followed by a chapter on diagnosing SAD, including a revised version of the *Seasonal Pattern Assessment Questionnaire (SPAQ)*. This sort of material is always problematic to a self-help book: does it encourage the reader to attempt self-treatment, and does it create a risk of misdiagnosis? Rosenthal takes

what I think is a reasonable position about self-diagnosis. He suggests several times that the reader should seek clinical assistance if there is any evidence of an intrusive seasonal pattern, and the reader is cautioned that the cut-off scores provided should not be considered exact. Still, once information is quantified it takes on an illusion of truth that can override more measured statements. Furthermore, validation studies of the SPAQ are still pending.

The second section of the book focuses on treatment and research. The writing benefits from Rosenthal's extensive experience with SAD patients; in fact, a number of clinical insights are included almost as "throw-away" points. Among the points I found particularly interesting were his comments on insurance reimbursement, characteristic responses to medication, and phenomenological aspects of the response to light. The first chapter focuses on light treatment, and includes a thorough description of recommended light regimens. Again, the question must be asked—although I confess I don't have the answer—whether providing this type of information encourages self-treatment, and if so whether such encouragement is appropriate. Light therapy represents a unique problem in psychiatry with regard to this question. Neither psychotherapy nor chemotherapy is generally possible without the involvement of a professional, but light boxes can be acquired easily without supervision. Although the regimens described are reasonable, I was surprised to find that Rosenthal recommends both morning and evening light to start, despite the meta-analytic evidence that evening light does not produce a significant increase in improvement above morning light alone.

I was impressed by Rosenthal's discussion of the biochemistry of SAD. It is as current as one would expect for a book of this type, appropriately limited in its claims given our state of knowledge, and it concludes on a cautionary note.

Seasons of the Mind is not meant solely for the lay public (indeed, its style may be too academic for some readers). The book should be required reading for any professional who works with SAD patients. I have already noted the wealth of clinical data included. The resource materials provided at the back of the book are particularly helpful. In addition to mention of several distributors of light boxes, a lengthy list of service providers is included.

I should note some additional reservations about the book, though I consider them to be relatively minor. There is a tendency at times to present tentative research data as conclusive, although this problem is probably pervasive in the self-help literature (and is even more evident in *The Hibernation Response* and *Light Up Your Blues*). I found the diet information in the back of the book curious. The extensive recommendations concerning diet suggest a clear understanding of the relationship between SAD and nutrition, which is not supported by research—a problem also noted by M.L. Moline in her review of *The Hibernation Response*. I think the limited comments in the body of the text are more appropriate for the SAD sufferer, given our present state of knowledge.

Despite these peccadillos, *Seasons of the Mind* is a book I would recommend without reservation to any professional who wants to learn more about seasonal depression and light therapy. I would also recommend it highly for patients who want to learn about SAD, with the one caveat that the reading may be heavy-going for the unsophisticated patient. However, since most SAD patients are self-referred, they tend to be psychiatrically knowledgeable and intellectually inquisitive. For this important group, *Seasons of the Mind* provides a valuable introduction to SAD.

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LETTER TO THE EDITOR

Assessment of Light Damage

I read Brian Rafferty's report of the SLTBR work session on safety standards and guidelines [Vol. 2, No. 1] with great interest, and would like to raise a few additional points.

The ANSI [*American National Standard for the Safe Use of Lasers, ANSI Z136.1, New York, ANSI, 1986*] standards obviously were developed for lasers and thus cover only one type of photochemical injury. According to functional and structural evaluation criteria, two classes of damage are recognized in the retina. "Ham-type," or blue-light damage refers to lesions induced by high energy monochromatic or narrow-band radiation of short exposure durations in the visible or UV ranges. "Noell-type" damage shows a visual pigment-related action spectrum after relatively low intensity long-term white light exposure. The latter conditions are common in modern lighting environments and are not covered by present safety standard guidelines. The two classes of damage differ in action spectrum, threshold irradiant dose, reciprocity of irradiance level and exposure duration, and perhaps also in mediating pigments and structures primarily affected. One can infer from animal studies that both classes occur in the human retina—even in the same individual—depending on exposure conditions. Which safety standards, then, should be applied to define potential risk factors in light therapy?

There are several criteria for evaluating photochemical injury to the retina with inherently different results. Perhaps the most subtle—albeit unfeasible for human diagnostics—is electron microscopy. Light or UV-induced photoreceptor membrane alterations can be distinguished in specific domains of single cells, thus lowering the detectable damage threshold in comparison to more superficial indices. Biochemical tests of retinal or photoreceptor membrane alterations provide a different mode of damage analysis in the laboratory context. By contrast, there are non-invasive techniques that do not, however, detect early (and re-

versible) signs of damage. The electroretinogram, for example, is a "mass response" which would not make obvious the loss or functional impairment of a small cell population. Observation of the ocular fundus by ophthalmoscopy can reveal acute edema or long-term scar formation, which often reflect distinct alterations in the outer retina not easily quantified and dependent on individual ocular factors. In densitometry, the bleaching and regeneration of visual pigment can be measured in defined fundus areas, with defects taken to index damage.

Which damage evaluation criteria should be applied for safety guidelines in light therapy? In this context, it is worth noting that the transmission of a young human eye may vary by 25% from the average standard curve in the near-UV and blue spectrum. Another difficulty in defining "safe" light exposure is the high prevalence of cumulative subthreshold lesions in humans and animals. Photoreceptor renewal systems effectively compensate for many of these lesions, until the cellular repair capacity is gradually exhausted—as, for example, in the aging retina. Existing safety standards do not take this into account. How can exposures causing additive, subthreshold lesions be classified? Gradual accumulation of minor lesions over many years is a widespread phenomenon.

Finally, our knowledge about the interaction of UV and visible light with photosensitizing drugs in the retina is limited. To what extent are photochemical lesions exacerbated by such drugs? This is particularly relevant for mental health clinicians, because several psychoactive drugs are known or suspected photosensitizers in the skin or the lens of the eye.

Clearly, safety standards for light therapy must be refined, taking into account the major differences among light sources and the consequent variation in types of damage.

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MEETING REVIEW

APSS 1989 Bursts With Light

Following last June's SLTBR meeting, several investigators presented results of their research on light at the meeting of the Association of Professional Sleep Societies in Washington, D.C. Studies presented at APSS, but not at SLTBR, are summarized below. For abstracts, see *Sleep Research* (1989), Vol. 18.

Two investigations explored human phase-response curves (PRCs) to light. Czeisler et al. [see also *Science*, 244: 1328-1333] conducted 45 laboratory light exposure trials in 14 subjects, using three cycles of bright light (7000-12,000 lux, 5 hr), ordinary indoor light (150 lux, 16 hr) and darkness (< 2 lux, 8 hr) per trial. Exposures during the subjective night, centered near the minimum of core body temperature ($T_{\min}$), produced the largest phase shifts (up to 12 hr delays or advances), indicative of Type 0 resetting. Exposures during the subjective day produced the smallest responses. Exposures early in the subjective night produced phase delays, while those later in the subjective night produced phase advances. Rhythms of core temperature, plasma cortisol and urine production all shifted in parallel, suggesting that light affected a master pacemaker controlling these rhythms. The timing of room light exposure relative to bright light also affected the phase shifts. The authors concluded that the human phase-response to light is similar to that of lower organisms.

Dawson et al. produced a partial PRC to a single 4-hr pulse of bright light (12,000 lux) in eight subjects. The light was presented immediately before the subject's normal bedtime. The circadian phase of the light pulse relative to $T_{\min}$ was varied by choosing subjects whose $T_{\min}$ fell at different times during sleep. The results were qualitatively similar to those of Czeisler et al. in that light exposure before $T_{\min}$ produced phase delays, with the largest delays occurring with exposures near $T_{\min}$. However, quantitative aspects of the results differed, perhaps because Dawson et al. studied some sub-

jects whose baseline $T_{\min}$ was in an abnormal phase position relative to sleep.

In another study, Dawson et al. compared the phase-shifting effects of delaying sleep onset by 4 hr to delaying dusk by 4 hr, in a counterbalanced crossover design. Subjects were kept in the dark prior to sleep for the sleep delay and in bright light (12,000 lux) prior to sleep for the dusk delay. Delaying dusk delayed the circadian phase of $T_{\min}$ in all four subjects (mean delay, 4 hr 45 min), while delaying sleep produced negligible changes in three subjects, and a statistically questionable delay of 1 hr 45 min in the fourth. The authors concluded that light is a stronger zeitgeber than sleep, and that apparent zeitgeber effects of sleep may be due to the gating effect of sleep on light.

Kronauer et al. performed mathematical analyses of the data of Czeisler et al. discussed above. They concluded that circadian pacemaker sensitivity to light appeared to vary by as much as 4.6:1 over the course of each cycle. Maximum sensitivity occurred near $T_{\min}$, minimum sensitivity near $T_{\max}$. The authors hypothesized that this variation could be due to circadian changes in the responsiveness of the pacemaker itself, or to changes in the sensitivity of the visual system.

Three studies suggested that bright light was effective in treating sleep phase disorders. Singer and Lewy treated a woman suffering from advanced sleep phase syndrome with 2500 lux evening bright light. Two weeks of treatment from 2000 to 2200 hr delayed dim light melatonin onset (DLMO) from 1950 to 2010 hr, but did not affect mean wake-up time (0334 hr at baseline, 0328 hr with treatment). An additional two weeks of treatment from 2100 hr to 2300 hr delayed DLMO to 2040 hr, and mean wake up time to 0501 hr. The authors note that the superior effect of the later treatment is consistent with the hypothesized human phase-response curve, but caution that a treatment order effect cannot be ruled out. Vanderpool et al. compared the effects of 2500 lux to 300 lux morning light (2 hr) on 20 patients with delayed sleep phase

syndrome (DSPS), in a randomized crossover design. The 2500 lux treatment was supplemented by dark goggles worn from 1600 hr until dusk, the 300 lux treatment with clear goggles for the same period. The bright light condition was greatly preferred by the subjects. Bright light, but not dim light, lengthened morning sleep latency and advanced the phase of body temperature during sleep. Dawson et al. reported that two DSPS patients showed a greater phase advance of core body temperature in response to a single pulse of 4 hr of morning light (12,000 lux) than did four normal controls (mean advance 4 hr 12 min vs. 0 hr 38.5 min).

Three new studies on SAD were presented at the APSS meeting. Avery et al. compared morning (0600-0800 hr) to evening (1900-2100 hr) bright light treatment in 19 SAD patients in a randomized crossover design. Both treatments significantly reduced Hamilton depression scores, but morning light produced significantly greater improvement. However, morning light was superior to evening light only in subjects with hypersomnia ($n = 13$). Two of four subjects with insomnia plus early morning awakening responded much better to evening light. The authors concluded that their results are consistent with the phase-shift hypothesis of Lewy. Wirz-Justice et al. reported combined data on several dozen SAD patients treated with bright light in a variety of protocols. Their findings included no difference between morning (0600 hr or 0700 hr) and evening (2200 hr) treatment times, a dose-response effect of morning light on remission rate, and a positive correlation between strength of response to light in winter and degree of spontaneous improvement in summer. Both light and summer selectively reduced carbohydrate intake. Rosenthal et al. presented three studies of sleep architecture in 41 SAD patients. They reported that untreated patients had greater total sleep time in winter than in summer, but spent less time in delta sleep. Untreated patients' winter delta sleep was shorter than that of light-treated patients or age-matched normal controls. Untreated patients also had lower sleep efficiency and higher REM density than light-treated patients. Both treated and un-

treated patients had a higher percentage of REM sleep than normal controls.

Based on a 14-day questionnaire study of 40 subjects, Violani et al. reported that the spring change to daylight savings time caused sleep onset and offset to shift to slightly later clock times (onset, 6.6 min later; offset, 11.4 min later), while the fall change back to standard time caused a larger shift in the opposite direction (onset, 18 min earlier; offset, 45 min earlier). These effects were especially evident on the weekend following the time change.

Moline et al. compared the effects of 4 hr of daily bright light (2500 lux) on sleep in five subjects with no treatment in five control subjects during simulated jet lag (6 hr advance of the sleep/wake cycle in the laboratory). Light exposure began at pre-shift mid-sleep time on the first post-shift day, and immediately after waking on the next three days. Effects on sleep efficiency and slow-wave sleep were mixed, while percent REM sleep appeared to return more quickly to baseline in the bright light group. The authors reported that bright light appeared to flatten the core temperature rhythm and, if anything, to delay its adjustment to the new schedule.

Two studies explored the effects of bright light on actual jet lag. Sasaki et al. studied four subjects for five days after a flight from Tokyo to San Francisco (time change +8 hr). Two received bright light (>3000 lux), two room light (<500 lux) from 1100 to 1400 hr local time. Bright light appeared to improve sleep efficiency, reduce wake after sleep onset and hasten adjustment of the core body temperature rhythm to local time. Cole and Kripke studied 19 subjects after trans-Pacific flights to San Diego (time changes +6.5 to +10 hr). Subjects were randomly assigned to self-administer 2 to 3 hr of either bright white light (2500 lux, $n = 11$) or dim red light (<50 lux, $n = 8$) upon awakening for three days. In the white light group, treatments early on the first post-flight day were associated with greater consolidation of self-reported sleep into nighttime

hours over the next three days, whereas later treatments were associated with more fragmented sleep. No such effect occurred in the red-light group. Wrist-activity data were consistent with these findings.

Eastman used 2 hr bright light (4000 lux), supplemented by dark goggles and/or staying indoors at selected times of day, to help subjects adapt to gradually advancing or delaying sleep schedules at home. Light upon awakening was used to help advance schedules; light before sleep was used to help delay them. The light-dark schedule appeared to help shift the temperature rhythm in a pilot study of three subjects. However, study of eight additional subjects on gradually delaying schedules showed that shifts in the temperature rhythm were incomplete, lagging several hours behind shifts in the light and sleep schedules, without greatly affecting sleep length or mood.

Sack et al. reported that melatonin and cortisol rhythms in four free-running blind people had identical periods (mean, 24.6 hr). With the masking effect of sleep averaged out, the cortisol nadir occurred at the time of melatonin onset, and the cortisol peak at the time of melatonin offset. The authors hypothesized that the two rhythms are generated by a common oscillator.

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BOOK REVIEW

Day Light Robbery and The Light Fantastic

Review of the book Day Light Robbery, by Dr. Damien Downing (Arrow Books, London, 1988), and the brochure The Light Fantastic, with an introduction by Dr. Downing (issued by the Aurora Partnership, P.O. Box 229, York YO1 2DQ, England). Dr. Downing practices nutritional

medicine in York and is a founder of the British Society for Nutritional Medicine.

In *Day Light Robbery*, a wide variety of subjects passes before the footlights, such as the relationship between the exposure to sunlight or full-spectrum light (including UV) and SAD, hormone and vitamin D production, dental caries, cancer, invisible bioradiations, white blood cells, and cardiovascular performance. Further topics include the germicidal effect of UV light and UV irradiation of blood to increase oxygen-carrying capacity. This variety of topics is the interesting aspect of the book.

Dr. Downing succeeds in explaining complex matters in easy, readable language. The illustrations that accompany the text are clear and well-chosen to support his anecdotes and publications. Studies done in Russia and in the 30's, 40's and 50's are reported. The author makes a good point in questioning the supposed risk of sunlight exposure in developing melanomas, and offers a helpful schedule for safe suntanning, with maximum sunlight exposure times taking into account month of the year, skin type, and latitude. Particular attention is paid to experiments that contrast exposure to artificial light with and without UV frequencies.

In spite of these positive remarks, there are also some serious criticisms. Quite frequently Dr. Downing tends to conclude more than can be concluded. For example, in discussing the relationship between cancer and sunlight exposure, he mentions a 1924 study that finds a correlation between cancer and latitude ("the further a city is away from the equator, the greater number of cancers"); a 1936 report suggesting an inverse relationship between skin cancer and other cancers among Navy personnel; a 1940 study that finds inverse relationships between cancer mortality and time spent outdoors and hours of sunlight in that area; and a 1985 study, the results of which "support the suggestion that vitamin D and calcium may reduce the risk of colorectal cancer. . . but cannot show conclusively its protection. . ."

Dr. Downing, though, goes a step further, writing, "Vitamin D levels appeared to give significant protection. . . ." And later, after discussing how light might exert a curative effect and citing some reports of John Ott on curative effects of sunlight on cancer, he takes yet another step, stating: "Take all of this evidence together and a pattern does emerge. It seems clear that we can modify our lifestyle in one simple way that will decrease our risk of developing cancer. . . ."

With respect to SAD, Dr. Downing draws conclusions where researchers raise questions. Based on the work of these same researchers, he attributes a major role to melatonin in the pathogenesis of SAD, the regulation of the sleep/wake cycles and the synchronization of bodily (circadian) rhythms. This is evidenced in such statements as: "Melatonin appears. . . to be a major factor in seasonal affective disorder" (p. 125); "SAD sufferers need a minimum intensity of 2500 lux to suppress melatonin production. . . ." (p. 149); "Melatonin production. . . sends us to sleep" (p. 28); "The only way of synchronizing bodily rhythms to a new time zone is by controlling melatonin levels—either by taking it orally or by adjusting the pineal's rhythm of output" (p. 31).

A possible feedback between melatonin levels and the central oscillator(s) in the SCN—melatonin being an internal zeitgeber—is currently still under investigation. Furthermore, there are recent indications that light of low intensities (not necessarily including UV) can also have suppressing effects (Armstrong, Brainard, Terman) and phase-shifting propensities, depending on timing (Lewy, Czeisler).

Dr. Downing overlooks the fact that one can sleep while melatonin levels are minimal or when sleep-wake rhythm and melatonin rhythm are desynchronized, as in the bunker studies. In a healthy subject, the rhythms are correlated, but a causal relationship and the nature of such a relationship is still in question. As it is doubtful whether suppression of melatonin is important in SAD or in sleep

disorders, Dr. Downing's emphasis on the necessity of full-spectrum light (FSL) lacks a firm basis. FSL would be better in "controlling melatonin, and thus our wake/sleep rhythm, because it produces UV and a higher intensity in the 450-550 Hz range than ordinary indoor lighting."

Referring to Margot Dietzel et al. [*7th European Sleep Congress Abstracts*], Dr. Downing writes that several studies show that a dose of bright light for a period of one hour can produce a significant but small improvement in mood as well as sleep in all types of depressed patients. Certainly, interesting results are found in treating nonseasonal depressions with artificial light (Kripke, 1989), but as further testing is needed, it is premature to promote light therapy for depression-in-general. Dr. Downing seems to take this position, as he states, "If you think it might help you, try it; there's nothing to lose."

In the brochure, *The Light Fantastic*, which seems to be based on the book, this issue returns. We read: "Fact: FSL can play a major role in the treatment of depression," followed by this comment: "There are more than one million tranquilizer addicts in the U.K." More facts are considered in the brochure, such as "FSL reduces hyperactivity in children;" "Agitated, fretful children are a major source of stress. FSL is calming and reassuring to both the child and his harassed parent;" or "FSL reduces the incidence of minor illness in children;" followed by, "When both parents work, a sick child can mean reorganization and lost income. A recent *Lancet* article reported that the inclusion of FSL in schools reduced sick days by 1/3."

The scientific support of these and most other "facts" in the brochure is meager, to put it mildly. The *Lancet* article is a letter to the editor reporting an (interesting) study, not blind, performed in one school. The book mentions three studies dating from 1942 and 1945 concerning reduction in infection rate using UV light. Concerning hyperactivity in school children, only one study is re-

ported, by John Ott, in his book *Health and Light* (1973). It could be that Dr. Downing is familiar with other confirming literature, but he does not mention it.

The brochure introduces the Aurora Full Spectrum System, an easily-installed lighting unit, UV-transmitting spectacles, a Psychodynamic Color Planning Service, and the book. It includes an order form for the lighting units.

Why, in *LTBR*, should one be critical of a sympathetic book which advocates the application of a therapy available to all of us, and a brochure issued by a company which sells products that are beneficial? It is because of a different understanding of what is "obvious."

Dr. Downing ends his book confronting the reader with the choice to take it or leave it. "We can modify our lifestyle, change our indoor lighting and put pressure on our employers to do the same, or we can continue to fungate in the dark." A dilemma, apparently, to be solved with a leap of faith. On the following page, the addendum "What to Do" opens with: "If we believe that sunlight is good for us, then. . . ." Why, though? One swallow does not a summer make.

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Bulletin Board

1990 Dues Notice

All members who joined SLTBR before August 15, 1989 will receive a dues invoice with this issue. In order to avoid a penalty for late payment, and to receiving *LTBR* without interruption, dues must be received before January 15, 1990. Membership for those who joined after August 15 will be automatically extended through 1990.

MARK YOUR CALENDAR: Annual Meeting 1990

SLTBR will hold its second annual meeting on May 13-14 at Columbia University's Health Sciences campus in New York City. The meeting will be sandwiched between the Biological Psychiatry and American Psychiatric Association meetings, also in New York. Please plan to make your housing arrangements through those groups, or individually. More details, and forms for preregistra-

tion and abstract submission, will follow in future issues of *LTBR*.

Application for Membership

The SLTBR Membership Application will no longer be included in the *Bulletin*. Individuals or corporations wishing to apply for membership should write to the following address for information and an application form: *SLTBR Membership, 722 West 168th Street, Box 50, New York NY 10032.*

SIGH-SAD Notes

A new self-rating version of the *Structured Interview Guide for the Hamilton Depression Rating Scale-Seasonal Affective Disorder* (SIGH-SAD-SR), developed by J.B.W. Williams and colleagues, is now available for researchers to use on a trial basis in comparison with the clinician-administered SIGH-SAD. Currently, several research centers

are administering both versions in a collaborative effort to determine the reliability and utility of the self-rating version for clinical purposes.

Both the self-rating and clinician-administered versions are administered to patients at at least three points in a protocol: while depressed, just prior to treatment; at the end of the first treatment regimen, regardless of response (minimum one week evaluation interval); and during a follow-up while remitted in spring or summer. Only experienced clinical raters may administer the SIGH-SAD, and only SAD patients meeting DSM-III-R criteria for Major Depression, seasonal (winter) type, may be included. Data will be pooled, and comparability of the two versions assessed. A cross-center, interrater reliability study of the SIGH-SAD, including videotaped interviews for training new raters, has also been proposed to NIMH but is not yet funded.

SLTBR Publications offers a complete packet of SIGH-SAD + HIGH-SAD assessment instruments (see order form, attached).

Investigators interested in participating in the cross-center investigation may contact *Martha Link*, *New York State Psychiatric Institute*, 722 West 168th Street, Box 50, New York NY 10032, Tel (212) 960-2469.

Welcome, New Members!

The Board of Directors is pleased to welcome the following new SLTBR members who joined since publication of the last issue of *LTBR*.

Regular Members

Iain McIntyre, John Nurnberger, Daniel Wagner.

Associate Members

Sharon Bahu, Bruce Baker, Eugene Carpenter, Len Ceder, Jonna Duff, Elizabeth Harrison, Franklin Hilf, Paul Keck, John McAuliffe, Cajetan Odunze, Virginia Peden, John Raasoch, Steven Resnick, Enrique Rubio, Gary Sachs, Mae Sperber, Paul Sullivan, Maryellen Visconti, Gary Warstadt.

Student Members

Donna McMillan, Meryl Rosofsky.

Corporate Members

Lighting Specialties Inc., SML - Licht - U. Bestrahlungstechnik.

Membership Directory in Production

The SLTBR Membership Directory will be published in early January, 1990. Directory entries will include area of specialization, treatment and research information, and status as a lighting apparatus supplier.

Members will automatically receive a copy of the Membership Directory (there is no need to order this item on the Publications Order Form).

Publication Price Change

SLTBR membership continues to grow, along with our list of colleagues and contacts, and our mailing list now includes nearly 800 names and addresses. To cover this significant expansion, and to defray printing and production costs, the price of the SLTBR mailing list has been increased to \$250 for the first set (the price of additional sets remains at \$50). This change has been noted on the Publications Order Form, attached.

MCI Mail ↔ BITNET Transfers

Many SLTBR members now communicate through MCI Mail or BITNET [see Vol. 1, No. 2]. BITNET is a university-based system which has previously been inaccessible to commercial services. Now users of either system can reach each other, which should further facilitate interactions. Other commercial and governmental systems (e.g., Compu-Serve) are also accessible. Oppenheimer Software [79th Street Basin, Box 39, New York NY 10024, Tel (212) 724-9785] can arrange for you to receive MCI Mail service and explain transfers among the various systems.