

Light Treatment and Biological Rhythms

Bulletin of the Society for Light Treatment and Biological Rhythms



Volume 6, Number 2

December 1993

EDITORIAL

SLTBR COMMUNICATIONS

When Anna Wirz-Justice asked if I would be a guest editor of *LTBR*, I had some initial qualms that I would be set adrift trying to maintain the standard of excellence that she and Michael Terman achieved. I needn't have worried that I would be left on my own, however, because Anna looked over my shoulder and offered helpful hints and suggestions at every step of the process. How was she able to do this, considering that she is in Basel, Switzerland, while I am half-way across the world in Vancouver, Canada? The answer is in the magic of electronic mail, or e-mail, to the initiated. We have been corresponding regularly for over 3 years via Internet, a world-wide system of computer networks that has started a small revolution in information retrieval and telecommunications. The Internet allows easy and immediate communication between anyone in the world with a computer and a modem. For example, Carl Hagfors' manuscript (from Finland), Michael Terman's suggestions (from New York), and Marty McCullough's memos (from Oregon) were all sent to me via e-mail at nominal cost. The Internet was once only accessible to academic institutions and computer wizards. Now many commercial and local computer bulletin boards (e.g., MCI Mail, Compuserve, Genie) offer complete access to the Internet. Once connected, an Internet user can participate in a wealth of services including e-mail, discussion groups (with topics as diverse as *Star Trek* episode reviews to quantum mechanics), and enormous databases. Many of you are no doubt well acquainted with the Internet; for those of you who wish to start, there are some excellent

introductory texts such as *The Internet Companion: A Beginner's Guide to Global Networking* by Tracy LaQuey and Jeanne Ryer (Addison-Wesley Publishing, Reading MA, 1993, US\$10.95 in paperback).

The Internet is definitely the way of the (very near) future for information exchange. I have discussed informally with Michael and Anna the idea of starting a Usenet (one of the networks included in the Internet) newsgroup on SAD and light therapy. Such a group could include discussion about clinical and research issues of interest to members. Some of these discussions may prove interesting and relevant enough to publish in *LTBR* for the general readership. If you might be interested in such a

In This Issue

<i>Raymond Lam</i> Editorial	15
<i>Norman Rosenthal</i> on the first decade of SAD	16
<i>Norman Rosenthal and Michael Terman</i> on DSM-IV update	18
<i>David Avery</i> on Federal-Industrial relations	18
<i>Scott Campbell</i> on alerting effects of light	19
<i>Carl Hagfors</i> on "light sensitivity" and the SPAQ	22
<i>Raymond Lam</i> on Hagfors' "light sensitivity"	24
<i>Maria Corral</i> on N. Rosenthal's <i>Winter Blues</i>	25
Bulletin Board	26

MARK YOUR CALENDARS!

SLTBR 1994 ANNUAL MEETING

23-24 June 1994

**NIH Campus • Lister Hill Auditorium
Bethesda, Maryland**

Cosponsored by The National Institute of Mental Health

Light Treatment and Biological Rhythms
Bulletin of the Society for Light Treatment and Biological Rhythms



Editor
 Anna Wirz-Justice, Ph.D.

Managing Editor
 Marty McCullough

Editorial Board

Charmaine I. Eastman, Ph.D.
Alfred J. Lewy, M.D., Ph.D.
Norman E. Rosenthal, M.D.
Michael Terman, Ph.D.

Unsolicited manuscripts, letters to the editor, and Bulletin Board announcements should be submitted to Dr. Anna Wirz-Justice, Editor, Psychiatric University Clinic, Wilhelm-Klein-Strasse 27, CH-4025 Basel, Switzerland. Please submit one double-spaced hard copy and a diskette file (Macintosh: MS-Word, MacWrite; IBM: WordPerfect as Textfile; BITNET: WIRZ@URZ.UNIBAS.CH. Manuscripts and Diskettes will not be returned. We reserve the right to edit and condense letters to the editor.

This publication is provided to SLTBR members as an entitlement for part of their dues. Annual subscription rate for non-members is \$15.00. Single copies are available at \$2.50 for members; \$3.50 for non-members. For subscription and membership information, please write to SLTBR at the address below.

Copyright © 1993, Society for Light Treatment and Biological Rhythms, Inc., P.O. Box 478, Wilsonville, OR 97070 USA. All rights reserved.

group, send me a message at my Internet address (rlam@unixg.ubc.ca). I'll let you know if there is sufficient interest to go ahead.

In the meantime, I hope you enjoy this issue. The new year will soon be upon us, and it sometimes seems amazing that 1994 will mark ten years since the *Archives of General Psychiatry* published a paper by Dr. Norman E. Rosenthal and colleagues identifying a "new" type of depression that they termed Seasonal Affective Disorder. Spurred on by (or perhaps, despite) the popular acronym of SAD, an explosion of research and clinical activity on SAD and light therapy has occurred during the past decade. In commemoration of that seminal paper, I asked Norman to pen an editorial giving us his perspective of this eventful period. He sent it to me via fax, not e-mail, but I am sure you will agree that his editorial is a most appropriate way to start the next decade of SAD research.

Raymond W. Lam, M.D., F.R.C.P. (C), Department of Psychiatry, University of British Columbia, 2255 Wesbrook Mall, Vancouver, B.C. V6T 2A1, Canada. Tel 604-822-7325; fax 604-822-7756; e-mail: rlam@unixg.ubc.ca.

THE FIRST DECADE OF SAD: A SUBJECTIVE PERSPECTIVE

My subjective impression of the first decade of SAD is that it flew by very quickly. That may be due in part to the unforgettable events that marked the beginning of the decade and the singular characters involved at that time. By the beginning of the decade, the seminal study of Al Lewy and colleagues, in which they showed for the first time that light was capable of suppressing human melatonin, and the well known study of the effects of light on Herb Kern's depressions were behind us, but their influence lingered on. Al Lewy was by then in Oregon, but memories of the time we shared in an office the size of a meat locker were still fresh in my mind. And as for Herb, who could forget those weeks when he turned one of the NIMH ward rooms into an office and pored through heaps of diaries, counting the number of pages and graphing their seasonal distribution?

It is really hard to know how to date the beginning of the first decade of SAD, but the recruitment of the first cohort of SAD patients is as good a place to start as any. That was an exciting time for several reasons. A *Washington Post* article designed to find what we believed would be a small number of patients suffering from a rare disease provoked, instead, a blizzard of responses. There was a striking uniformity in the patients' complaints and these became the basis of the syndrome of SAD. There was an air of suspense as we watched the first group of patients, recruited in the summertime when they were well, enter the fall season. Would they develop the symptoms they had reportedly suffered winter after winter when they were observed prospectively or turn out to be highly suggestible individuals and malingerers as some of our less kindly colleagues surmised? And finally, would the light work?

There was an enormous amount of good fellowship among the doctors who followed the patients: Tom Wehr, Dave Sack, Chris Gillin and myself. Operating in cramped and inconveniently arranged rooms, we followed and rated the patients. I remember all those initial patients, details about their lives and, in some cases, even how they responded to light. After the first handful of patients had been treated, I would have bet good money that we were dealing with an active biological principle and not merely a placebo. As we know, however, it took many controlled studies by different groups before that conclusion was corroborated scientifically and even now, a decade later, some might take issue with the contention that light therapy has been convincingly demonstrated to be more than just a placebo.

As the decade progressed, research and treatment of SAD was taken up by many other centers all over the world. It has been rewarding to see this creative process at work as different researchers have formulated and addressed different questions pertaining to SAD and light therapy and have come up, in some instances, with surprising results. Such diverse involvement in the field has been extremely important for the development of the science and, ultimately, for the millions of patients who stand to benefit from it.

I think we have learned a great deal about SAD in the last decade though, as a scientist, I feel more curiosity and frustration about what we have yet to learn than complacency about what we already know. We have found out that SAD is common, that it affects people of all ages, that women appear more susceptible than men, that its prevalence increases with latitude, and that it occurs in many different ethnic groups and in both Northern and Southern Hemispheres. We have learned that within a certain range, more light is better, that light does not have to be full-spectrum to work, that light treatment can work well (at least in some people) regardless of when it is administered in the course of the day, that simulating the dawn is useful, and that SAD can be treated effectively with some medications.

On the other hand, there are many things about light therapy and SAD that we do not understand. For example, we do not know why women are more susceptible to SAD than men or whether different ethnic groups are more or less vulnerable to the condition. Although there is evidence for a genetic basis for seasonality in general and a greater prevalence of SAD in the families of our patients, we have no knowledge about the putative genetic underpinnings of the condition. We don't understand why light therapy works better in some than in others and why it is often less effective than summertime in reversing the symptoms of SAD.

An understanding of the pathophysiology of SAD and the mechanism of action of light has lagged behind other areas of research. One theory of mechanism, the melatonin hypothesis, appeared unpromising after a series of studies in the early years of the decade but has refused to die. Another theory, the phase-shift hypothesis that has dominated much of the intellectual focus on mechanism throughout the decade, is supported by some studies but not by others. It has survived the decade, in part by undergoing mutation, but seems less robust now than when it was first articulated.

Biological findings have, to some degree, come and gone though a few have withstood attempts at replication. Replicated biological abnormalities in SAD include delayed circadian rhythms, low plasma prolactin levels, reduced electro-oculogram ratios and aberrant behavioral responses to the serotonin agonist m-CPP. Offhand, I can think of no others. In the rush of excitement following the emergence of the syndrome and the success of light therapy, I personally approached the issue of pathophysiology and treatment mechanisms with naive and excessive optimism. After all, why should definitive answers come easily to us when they have eluded investigators of other major psychiatric disorders for decades? I believe that for some time to come we will need to ask less of our hypotheses, to content ourselves with partial answers and to accept that a grand unification theory of SAD and light therapy is probably a long way off.

As our understanding of SAD and light therapy has matured, so has our sense of collegiality and community, assisted to a great degree by the formation and persistence of our society. The SLTBR has succeeded in disseminating information, promoting communication internationally and fostering a sense of shared endeavor. To a marked degree, in my view, unhealthy competition has been replaced by healthy collaboration in the many areas where our interests converge. At one point, for example, it seemed to me that there was an excessively critical spirit abroad in our review of one another's manuscripts. This fostered a sense that we SAD researchers couldn't agree among ourselves on basic issues, which was quite disadvantageous to our shared goals. In my view, things have settled down and in the review process as in other areas, I think we all recognize the need to deal with one another in a fair and collegial manner.

In summary, the first decade of SAD has flown by. Its accomplishments were real but the questions left unanswered beckon to us no less seductively than those of days gone by. I am confident that we will answer some of these questions in the next decade though we will need the help of creative and energetic young investigators to do so. I am hopeful that we will find such younger investigators or, rather, that they will find us — perhaps because the amazing effects of light therapy never cease to intrigue. Even nowadays, ten years after the event, I am approached at meetings every now and then by some colleague from my training years, who informs me — usually in a lowered tone, as one might confide a secret — "That light therapy

of yours. I tried it on one of my patients and, what do you know! It actually works!"

Norman E. Rosenthal, M.D., Director of Outpatient Services, Clinical Psychobiology Branch, National Institute of Mental Health, Bldg. 10/42-239, 9000 Rockville Pike, Bethesda, MD 20892. Tel 301-496-2141; fax 301-496-5439.

DSM-IV DEBATE CONTINUES

Subsequent to our previous reports in *LTBR* (1991, 4: 3; 1992, 4: 21; 1993, 5: 49), the DSM-IV draft criteria for Seasonal Pattern have been further modified in anticipation of publication of the new manual early in 1994. Drs. Norman E. Rosenthal and Michael Terman have responded to this draft in the letter reprinted below. The DSM-IV committee will be able to consider final changes only within the next few weeks. Colleagues who feel strongly about the matter should write promptly to Dr. Allen Frances (fax 919-681-8092), with cc. to Dr. John Rush (fax 214-648-4278).

10 November 1993

We are concerned to see that the latest draft criteria for "seasonal pattern" still have a few problems that we had noted when we were first asked to peruse them.

Most serious of these problems is the requirement outlined in Criterion C, namely that:

In the last two years, two episodes have occurred that demonstrate the temporal seasonal relationships defined in A and B, and that no non-seasonal episodes have occurred during that same period.

To understand why this criterion presents such a problem for those of us who treat and study patients with a seasonal pattern, let us consider a thirty-five-year-old woman with a fifteen year history of winter depressions but no summer depressions. Last winter, however, was an exception. She was sent on a work assignment to Bermuda between September and March and as a result did not suffer from her winter depression. Now it is October and she is back from Bermuda and beginning to feel all her familiar winter symptoms.

Such an obvious case of "seasonal pattern" would fail to meet your new criteria simply because the pattern in the last two years had been aberrant, albeit for an obvious reason.

The same would apply had she been free of depression this past winter because she had been on Prozac. This year, perhaps, she doesn't want to go back onto Prozac. She would rather try light therapy — but her insurance says she can't be reimbursed for it because she doesn't meet criteria for "seasonal pattern".

The solution is simple. Criterion C should be removed. In any event, it constitutes an addition to the criteria of DSM-III-R without having any new data to support it and thus violates one of the stated principles on which (as we understand it) DSM-IV is supposed to be based.

The second problem with the new criteria is the reintroduction of *No seasonally varying psychosocial stressors* as an exclusionary criterion. This criterion was removed from DSM-III-R on the advice of researchers and clinicians who pointed out how infrequently regularly occurring seasonal depressions could plausibly be attributed to such stressors. Even in the presence of such stressors, such as the regular loss of a job in the winter, it is often unclear whether the job loss precedes the depression or vice versa. The reintroduction of this criterion once again represents a change in DSM-III-R criteria without any data to support it. We recommend that this criterion be removed.

Finally, we understand that there is a plan to remove all qualifiers, including "seasonal pattern", from the classification listing. Since many clinicians refer to this listing and not the full manual, we are concerned that all these modifiers, seasonal pattern included, would not be readily available even if the patient fits the criteria. Once again, this would constitute a change without data from DSM-III-R and, from our perspective, a retrogressive one.

We look forward to your response to our concerns.

N.E.R. and M.T.

UPDATE: FEDERAL-INDUSTRIAL RELATIONS

U.S. Food and Drug Administration activity

The Circadian Lighting Association has hired legal counsel specializing in FDA-related problems. The group anticipates filing a reclassification petition within the next several months. The petition will propose reclassifying bright light boxes from Class III to Class II (see *LTBR*, 1992, 5: 1-8).

A manufacturer of bright light boxes received a letter from the FDA last summer asking the company to remove any reference in its advertising to bright light therapy "altering any bodily function". This letter appears to be a broader restriction than previous FDA statements which prohibited any mention of using the light boxes for treatment of medical disorders.

Light box regulation in Germany

Henner Giedke, M.D. (Department of Psychiatry, University of Tübingen) has filed the following report concerning light box regulation and advertising in Germany:

In Germany, medico-technical devices are grouped into four classes:

Class 1 Twenty-five specified energy-driven apparatus such as defibrillators, heart-lung machines, surgical lasers.

Class 2 Energy-driven implants.

Class 3 All energy-driven equipment not belonging to Classes 1 and 2.

Class 4 All other devices.

Light boxes belong to Class 3. By federal order, manufacturers are obliged to construct all devices according to "generally accepted technical rules, work protection and safety precautions". Though not obligatory, producers generally apply for the quality approval of the regional Technical Inspection Commission (TOV). In addition, manufacturers have to provide instructions for use. In the case of light boxes, the latter generally include the advice to consult a physician before starting treatment. Some firms require a physician's prescription. In principle, however, a patient can buy a light box on his own responsibility (prices: US\$800-2200).

Proof of a device's medical effectiveness is not required. In the case of obvious or suspected quackery, local or federal offices (Factory Inspection, Federal Department for Public Health) can become active, spontaneously or upon request.

Light boxes are not yet included in the list of remedies approved by the federal organization of insurance companies. Reimbursement for devices which are registered in this list are totally or partially covered by these companies, provided there is a medical prescription. Thus, up to now, each patient has had to apply for a refund individually — which has been, and will be, necessary in the case of regional companies. Currently

there are considerable differences among insurance companies regarding refunding practice, but, in most cases, the cost of purchasing a light box is reimbursed.

Insofar as light boxes are used to treat mental disorders, advertising is only allowed within the professional community. There is a law regulating what *must* be and what *may not* be stated in the advertisement. These restrictions are not valid for instances in which the law does not consider a condition to be a disease. Possibly subsyndromal seasonal affective disorder (SSAD) can be considered to be such a state. Here, however, great caution and timely legal advice is needed.

H.G.

David H. Avery, M.D., Harborview Medical Center ZA-99, 325 9th Ave., Seattle, WA 98104. Tel 206-223-3425; fax 206-287-8615.

ALERTING/ACTIVATING EFFECTS OF LIGHT

Based on a draft report to the ASDA/SLTBR Task Force on The Use of Light Therapy for Sleep Disorders

Bright light exposure has been employed as a treatment for a number of sleep difficulties, most of them associated with alterations in the circadian timing system (i.e. advanced and delayed sleep phase disturbance, jet lag, shift work). For some of these applications, usually in which a phase delay of circadian rhythms is indicated (e.g., advanced sleep phase syndrome), the optimal time for light exposure is in the evening, often immediately prior to bedtime. Although such timing of light exposure is desirable from a circadian rhythms perspective, a potential drawback exists to such light exposure schedules, as well. This is due to a property of bright light exposure that has received relatively less attention than the phase-shifting and entraining properties — that of an acute alerting, or activating, effect. The following paper focuses on the issue of alerting/activating effects of bright light exposure as they may affect negatively the use of light therapy for insomnia, and only briefly addresses effects on waking performance. A detailed discussion of the potential *benefits* of such alerting/activating effects of bright light exposure, in terms of performance enhancement, is beyond the scope of this paper.

Evidence for Alerting/Activating Effects

Patient samples. The possibility that exposure to bright light may result in subjective and/or CNS activation has been recognized since the first studies of light treatment for seasonal affective disorder. Rosenthal and co-workers (1984) reported "hypomanic irritability and hyperactivity in a few cases" of bright light treatment, which subsided when light therapy was discontinued. Subsequent studies of light therapy of SAD patients have documented similar behavioral responses following bright light exposure, including decreased sleep and insomnia.

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

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

Normal subjects: Light exposure prior to nocturnal sleep. It might be expected that any alerting effects of light on subsequent sleep would become more evident in protocols that do not involve significant sleep deprivation. Yet,

Drennan et al. (1989) observed no significant lengthening of nocturnal sleep latency (bedtime: ~ 2300h) immediately following one night's exposure to bright light between 1800h and 2100h. The same result was reported by Bunnell and co-workers (1992) in subjects exposed to two hours of bright light (> 2500 lux) immediately prior to nocturnal sleep. Similarly, the ability of healthy young subjects to initiate a daytime nap was not affected by two hours of exposure to bright light (5000 lux) immediately prior to the nap attempt (Murphy, et al., 1991). In contrast to these findings, Dijk and coworkers (Dijk et al., 1991) observed significantly increased latencies to sleep onset in subjects exposed to bright light (~ 2500 lux) for three hours immediately prior to nocturnal sleep. The conflicting results may be due to the fact that this study differed from the others in terms of timing (Drennan et al.) and duration (Bunnell et al.) of the exposure period.

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

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

What is the Mechanism?

Based on the putative somnogenic properties of melatonin, and the demonstrated capacity of bright light to suppress nighttime plasma melatonin levels (e.g., Lewy, 1983), it

has been suggested that alerting/activating effects of bright light exposure may be mediated through the action of melatonin, perhaps linked to thermoregulation (Badia et al., 1991; Sack et al., 1992). It should be noted, however, that a recent study by Dollins et al. (1993) found no effects of bright light (1500 and 3000 lux) on performance or subjective alertness, though the light was effective in suppressing melatonin.

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

Whether bright light provides an "intrinsic" activating effect, or whether such an effect is mediated through another, or multiple, systems, it seems clear that increased alertness/activation is associated with bright light exposure. That such activation does little, at least in healthy subjects, to interfere with subsequent "sleepability" suggests that the effects are quite short-lived. That is, following removal of the light stimulus, activation effects decline precipitously. This notion is supported by the findings that body temperature, melatonin levels, and beta activity all vary in close association with changes in light exposure intensity. It is unlikely, therefore, that acute effects of bright light exposure (as opposed to phase-shifting properties), even immediately prior to bedtime, would induce, or exacerbate, insomnia.

Scott S. Campbell, Ph.D., *Institute of Chronobiology, New York Hospital, 21 Bloomingdale Rd., White Plains, NY 10605. Tel 914-997-5924; fax 914-682-1536.*

REFERENCES

- Badia, P., J. Culpepper, B. Myers, M. Boecker, and J. Harsh (1990) Psychophysiological and behavioral effects of bright and dim light. *Sleep Research* 19: 387.

- Badia, P., B. Myers, M. Boecker, and J. Harsh (1991) Bright light effects on body temperature, alertness, EEG and behavior. *Physiol. and Behav.* 50: 583-588.
- Bunnell, D.E., S.P. Treiber, N.H. Phillips and R.J. Berger (1992) Effects of evening bright light exposure on melatonin, body temperature and sleep. *J. Sleep Research* 1: 17-23.
- Cajochen, C., D.J. Dijk and A.A. Borbély (1992) Dynamics of EEG slow-wave activity and core temperature in human sleep after exposure to bright light. *Sleep* 15: 337-343.
- Campbell, S.S. and D. Dawson (1990) Enhancement of nighttime alertness and performance with bright ambient light. *Physiol. and Behav.* 48: 317-320.
- Clodure, M., J. Foret, O. Benoit, Y. Touitou, A. Aguirre, G. Bouard and C. Touitou (1990) Psychophysiological effects of early morning bright light exposure in young adults. *Psychoneuroendocrinology* 15(3): 193-205.
- Czeisler, C.A., M.P. Johnson, J.F. Duffy, E.N. Brown, J.M. Ronda and R.E. Kronauer (1990) Exposure to bright light and darkness to treat physiologic maladaptation to night work [see comments]. *N. Eng. J. Med.* 322: 1253-1259.
- Dawson, D. and S. Campbell (1991) Timed exposure to bright light improves sleep and alertness during simulated night shifts. *Sleep* 14: 511-516.
- Dijk, D.J., C. Cajochen and A.A. Borbély (1991) Effect of a single 3-hour exposure to bright light on core body temperature and sleep in humans. *Neurosci. Letter* 121: 59-62.
- Dollins, A.B., H.J. Lynch, R.J. Wurtman, M.H. Deng and H.R. Lieberman (1993) Effects of illumination on human nocturnal melatonin levels and performance. *Physiol. Behav.* 53: 153-160.
- Drennan, M., D.F. Kripke, and J.C. Gillin (1989) Bright light can delay human temperature rhythm independent of sleep. *Am. J. Physiol.* 1: 2.
- Finley, W.W., R.A. Haring and M.L. Eisenstadt (1992) Effect of bright light exposure prior to napping on the multiple sleep latency test for obstructive sleep apnea patients. *Sleep Research* 21: 375.
- Hajek, M., E.K. Meier, J.A. Wirz, I. Tobler, J. Arendt, H. Dick and G. Fink (1989) Bright white light does not improve narcoleptic symptoms. *Eur. Arch. Psychiat. Neurol. Sci.* 238: 203-207.
- Hannon, P., G. Brainard, W. Gibson, J. French, D. Arnall, L. Brugh, C. Crank, S. Fleming, J. Hanifin and M. Rollag (1992) Bright light suppresses melatonin and improves cognitive performance during nighttime hours in humans. *Sleep Research* 21: 377.
- Lewy, A.J. (1983) Effects of light on human melatonin production and the human circadian system. *Prog. Neuropsychopharmacol. Biol. Psychiat.* 7: 551-556.
- Murphy, P., B. Myers, P. Badia, and J. Harsh (1991) The effects of bright light on daytime sleep latencies. *Sleep Research* 20: 465.
- Rosenthal, N.E., D.A. Sack, J.C. Gillin, A.J. Lewy, F.K. Goodwin, Y. Davenport, P.S. Mueller, D.A. Newsome and T.A. Wehr (1984) Seasonal Affective Disorder: A description of the syndrome and preliminary findings with light therapy. *Arch. Gen. Psychiat.* 41: 72-80.
- Sack, R.L., M.L. Blood, G.M. Ormerod, G.B. Rich and A.J. Lewy (1992) Oral melatonin reverses the alerting effects of nocturnal bright light exposure in humans. *Sleep Research* 21: 49.

"LIGHT SENSITIVITY"

A Real or Expressiveness Subscale on the *SPAQ*?

In an epidemiological study of SAD-prevalence using the *Seasonal Pattern Assessment Questionnaire (SPAQ)*, we conducted over 2000 telephone interviews on a statistically representative sample in Finland. We derived a Light Sensitivity Scale (LSS) from the *SPAQ* by adding two additional items to the question, "How do weather changes make you feel?". To the original 10 items we added: "bright snowy days" and "dark slushy days". The scale values for responses to each item vary from -3 = "In very low spirits or markedly slowed down" to +3 = "Markedly improves your mood or energy level". A principal component factor analysis of these 12 items revealed three factors. A varimax rotation of the data is given in Table 1. The weighting of each item to each of the three factors is given. This weighting reflects the effect of that item on mood state. The larger the number (either positive or negative), indicates greater weighting of that item to that factor. For example, the items that weight on Factor II include cold weather, hot weather and sunny days.

We interpreted the three factors as a darkness/lightness factor (I), a temperature factor (II) and a humidity factor (III). We derived a new scale consisting of 7 items having high loadings on the darkness/lightness factor (I). [Item 8 (high pollen count) was rejected for better content validity but it also could have been included as an expressiveness item.] We decided to call it the Light Sensitivity Scale (LSS) because it reflects a high emotional reactivity both for bright and dark days. In an item analysis for the new scale, intercorrelations varied between 0.482 and 0.651. Total reliability for the LSS was calculated with an alpha-coefficient of 0.824.

We then looked at correlations between the LSS and other variables on the *SPAQ*. The 20-item CES-D scale, a self-rating scale for depression (Radloff, 1977), was also included in the study. We found modest correlations to different variables, including the Global Seasonality Score (GSS) and the degree of seasonal problem (Table 2). Because women in all age groups had slightly higher mean values for the GSS, we also calculated partial correlations controlling for sex, but this had only minimal effect on the correlations. The highest correlation was between the LSS and GSS, but there was still only a 19% common variance.

Table 1. Varimax rotation for mood and weather changes.

Item:	Factors:	I	II	III	Common Variance
1. Cold Weather		.299	-.547	.301	.481
2. Hot weather		.087	.810	-.109	.677
3. Humid weather		.087	-.029	.845	.720
*4. Sunny days		-.526	.556	-.123	.602 (r)
5. Dark days		-.130	.269	-.164	.467
*6. Gray cloudy days		.542	-.150	.269	.404
*7. Long days		-.617	.408	-.052	.550 (r)
8. High pollen count		.504	.226	.019	.305
*9. Foggy smoggy days		.619	-.071	.462	.602
*10. Short days		.685	-.053	.194	.512
*11. Bright snowy days		-.623	.307	.145	.504 (r)
*12. Dark slushy days		.727	-.153	.227	.602
N=1983		35%	10%	9%	

(*)-marked items were included in the LSS.

(r)-marked items were scored reversed, in the same direction as darkness items, i.e. high sensitivity for bright and dark days were scored in the same (darkness) direction.

Table 2. Product moment correlations from LSS to other variables.

	LSS	LSS (controlled for sex)
GSS	.435	.419
Degree of seasonal problem	.204	.194
CES-D score	.118	.106

We thought that perhaps the LSS would give useful additional information, e.g., that individuals with a high LSS may be particularly good candidates for light treatment.

We then examined LSS distribution as a function of seasonality group. Seasonality groups (SAD, subsyndromal SAD, and normal) were defined as per criteria in Kasper et al. (1992):

- 1) seasonal affective disorder (SAD): GSS = 10 or greater and problem = moderate or more;
- 2) subsyndromal SAD (S-SAD): GSS = 10 or greater and problem = none or mild, or GSS = 8 or 9 and problem = mild or greater.

The distribution of scores showed that 5% of subjects scored 20 or 21 on the seven-item scale (maximum score = 21) while 10% of all subjects had scores of 19 to 21. Cross tabulation with seasonality groups revealed that

17.9% of SAD subjects belong to the top 5% LSS group while 37.3% belong to the top 10% LSS group (Table 3). Because the top scorers on the LSS endorse extreme values on almost all items (not all relating to seasonality), it is possible that this reflects a certain individual expressive style, that is, using "capital letters" to describe how weather changes affects the person's mood. The same tendency to use extreme responses might also be partly reflected in the GSS and degree of problem.

This possibility suggests that we should take a closer look at these subjects scoring high on the LSS and classified in the SAD group. The LSS items are clearly biased towards a consistent seasonal pattern (e.g., "dark, slushy days" only occur in winter, "high pollen count" only in summer), which might partly explain the correlation to seasonality groupings. It should be noted, however, that "bright snowy days" is inconsistent with a winter SAD pattern while "cloudy days" could occur in winter or summer. Therefore, high scoring LSS subjects are not only affected by season, but also react strongly to daily weather conditions. Some of these subjects might really have SAD (and perhaps be very sensitive to light treatment) and some others might simply use extreme responses and thus be wrongly classified as SAD instead of S-SAD or normal. If this interpretation is correct, the LSS could perhaps be used as a covariate to correct *SPAQ* data in epidemiological studies of SAD prevalence, or in screening of subjects for studies. This question can be answered empirically by looking at the light treatment results for different subjects and comparing their pre-treatment *SPAQ* and LSS data.

Table 3. Cross tabulation of LSS and SAD classifications.

	N	Normal (%)	S-SAD (%)	SAD (%)*
LSS: Top 5%	103	63 (3.7)	28 (11.1)	12 (17.9)
95%	1912	1632 (96.3)	225 (88.9)	55 (82.1)
Total	2015	1695	253	67
LSS: Top 10%	204	131 (7.7)	48 (19.0)	25 (37.3)
90%	1811	1564 (92.3)	205 (81.0)	42 (62.7)
Total	2015	1695	253	67

*P < 0.00001

The *SPAQ* assumes that subjects are responding honestly to the questions but there is no way to determine if subjects understand or interpret the questions correctly. Perhaps some MMPI-type empirical scale could be considered as a more reliable method for preliminary screening of SAD subjects for light treatment trials because this assumption may not be correct. For example, cultural differences in interpreting questions on the *SPAQ* may explain some of the differences in epidemiologic SAD studies between different countries. While the GSS frequencies in Finland are only slightly lower than those in Maryland (Kasper et al., 1989), seasonal changes were a problem for only 15.7% of our subjects compared to 27% of subjects in the U.S. study. It is possible that Americans use the word "problem" less rigorously than Europeans, and therefore more of them would be diagnosed as SAD and S-SAD. This may also partly explain the surprisingly low seasonality observed in Iceland and emigrated Icelandic descendants in Canada (Magnusson et al., *in press*), if the U.S. data have been biased towards too high SAD prevalence and/or the European data biased to too low SAD prevalence.

Carl Hagfors, Ph.D., Department of Psychology, University of Jyväskylä, P.O. Box 35, SF-40351 Jyväskylä, Finland. Tel (358)-41-602 851; fax (358)-41-602 841; e-mail: hagfors@jyilk.jyu.fi.

REFERENCES

- Hagfors, C., K. Koskela and J. Tikkanen, Seasonal affective disorder (SAD) in Finland: An epidemiological study. (*manuscript in preparation*).
- Kasper, S., T.A. Wehr, J.J. Bartko, P.A. Gaist and N.E. Rosenthal (1989) Epidemiological findings of seasonal changes in mood and behavior: A telephone survey of Montgomery County, Maryland. *Arch. Gen. Psychiat.* 46: 823-833.
- Radloff, L.S. (1977) The CES-D scale: A self-report depression scale for research in the general population. *Applied Psychological Measurement* 1: 385-401.
- Magnusson A. and J. Axelsson. The prevalence of seasonal affective disorder is low amongst descendants of Icelandic emigrants in Canada. *Arch. Gen. Psychiat.* (*in press*).

EDITOR'S COMMENTS

This preliminary communication by Dr. Hagfors raises interesting questions about SAD and subjective light sensitivity. Few studies have examined the effects of weather, independent of season, on SAD (the only study I

am aware of — Albert et al., 1991 — looked only at energy). The clinicians in my clinic have often commented about how some patients relate their problems to daily environmental illumination rather than to season, i.e., they complain of depression during cloudy days in summer and do not feel depressed during sunny days of winter. These patients seem to have a very reactive mood state, similar to Kasper's notion of recurrent brief depression (Kasper et al., 1992), and often will not meet DSM-III-R criteria for major depression, which requires at least two weeks of feeling depressed most days. Are these patients a different subgroup than SAD patients who do not have this light sensitivity or mood reactivity? Are they more likely to respond to light therapy, or are they more likely to have a placebo response to light? Unfortunately, we have not gathered these *SPAQ* data for the patients who have undergone light therapy studies, but I imagine that other researchers do have these data. I would encourage those researchers to look carefully at this question. Incidentally, our studies of retinal light sensitivity, using electrophysiological tests of retinal function, do not address the issue of subjective sensitivity to light. I am unaware of any studies that have actually examined the relationship between subjective and objective measures of light sensitivity.

The question of validity of seasonal diagnoses based on data from the *SPAQ* is also a good one. The *SPAQ* has been useful, but any self-rating questionnaire is limited when it comes to making clinical diagnoses. Certainly we see a number of people with high GSS and degree of problem scores who do not have clinical depressions and would not be diagnosed with SAD by DSM-III-R criteria. The next phase of epidemiologic studies of SAD will need to use clinical interviews. The Toronto group is completing a telephone survey of metropolitan Toronto using a structured interview that will allow DSM-III-R diagnoses to be made (A.J. Levitt, personal communication). This study will generate interesting SAD prevalence data that will allow comparison to other epidemiologic surveys.

R.L.

REFERENCES

- Albert, P.S., L.N. Rosen, J.R. Alexander, Jr., and N.E. Rosenthal (1991) Effect of daily variation in weather and sleep on seasonal affective disorder. *Psychiat. Res.* 36: 51-63.
- Kasper, S., S. Ruhrmann T. Haase and H.J. Moller (1992) Recurrent brief depression and its relationship to seasonal affective disorder. *Eur. Arch. Psychiat. Clin. Neurosci.* 242: 20-26.

BOOK REVIEW

Winter Blues: Seasonal Affective Disorder — What It is and How to Overcome It

By Norman E. Rosenthal, M.D. (New York, NY: The Guilford Press, 1993, 325 pp., US\$14.95).

Despite my initial dismay at the change of title from the eloquent and apropos *Seasons of the Mind*, this revised edition does not disappoint. Like the original edition, this book is directed at the general reader or patient. The author conveys an unabashed enthusiasm about his clinical and research work in seasonal syndromes throughout this book. He unapologetically draws upon his personal and professional experiences to thoroughly cover what has come to be a rapidly growing field of scientific inquiry.

Dr. Rosenthal begins with an overview of SAD and light therapy, helping the reader discover that SAD is a common and eminently treatable disorder. The next chapters are devoted to a thorough presentation of signs and symptoms enlivened by frequent clinical vignettes. The inclusion of the *Seasonal Pattern Assessment Questionnaire (SPAQ)* allows readers to discover the extent of their seasonality and to define the severity of their symptoms. The attention he pays to the symptoms of seasonality in children and adolescents emphasizes the importance of early recognition. The fourth chapter has a rather unfortunate title — "SAD: An Owner's and Parent's Manual" — that sounds like an automobile repair book. Despite this, and some repetition in this chapter, the reader is introduced to the use of the bio-psychosocial model of understanding psychiatric illness. He goes on to use the model to explain why only select people get SAD despite the ubiquity of seasonality in humans. There is, however, an emphasis on light deprivation as a trigger for SAD that may not be shared by all researchers [*Editor's note: See Hagfors' article in this issue*].

A unifying technique is Dr. Rosenthal's use of references to colleagues' work throughout the book. This conveys the sense of community which exists among SAD researchers. More importantly, the reader becomes aware that SAD is a worldwide phenomenon and not simply the latest North American fad. This point is reinforced by the inclusion of a greatly expanded list of researchers and clinicians in the resource section at the end of the book. Dr. Rosenthal also capitalizes on the growing awareness of SAD and seasonality to educate readers about depression in a more general sense.

In the second part of the book, Dr. Rosenthal gives an up-to-date and well-balanced presentation of available treatments for SAD. He emphasizes the need for a more open minded and collaborative approach by physicians in the successful treatment of SAD. He rightly cautions his readers against adherence to a particular theory to explain everything.

The coverage of light therapy for SAD is both complete and practical. He addresses commonly asked questions about light therapy and does not neglect controversial issues in treatment, such as the unresolved question of the need for ophthalmologic examination prior to initiating light therapy. The amount of attention paid to dietary advice seems excessive, even though readers do invariably ask questions about diet. The coverage of antidepressant medications in the treatment of SAD is comprehensive and balanced, including the controversial issue of suicidality and fluoxetine. Dr. Rosenthal also includes a comprehensive chapter on research in SAD which summarizes well the past, present and future of research in this field.

The advice he gives to family and friends of affected individuals seems reasonable, for the most part. However, while SAD patients clearly need their family's support and understanding, it is unlikely they will all appreciate company while sitting in front of the lights, as the author advises family to do. They would do well to ask first.

The third part of the book is entitled "Celebrating the Seasons". I enjoyed this fascinating historical perspective of seasonality, with intriguing titles such as "A brief history of seasonal time", "Polar tales" and "Creating with the seasons". In these chapters, Dr. Rosenthal is at his most entertaining and illuminating. In Chapter 15, the author attempts to answer the question of how the importance of the physical environment was lost over time. In the process, he seems to go a bit far in suggesting that Freud's influence "obscured our consideration of alternative hypotheses" (p. 227). My concern is that the author's views seem to contribute to the schism between "biological" and "non-biological" psychiatry.

This last caveat aside, however, Dr. Rosenthal has written this book with equanimity, knowledge and empathy for his readers. By allowing the reader a glimpse into how he conducts his day-to-day practice, he is able to reach them in a very personal way. His style of presentation and examination, without imposing his own views, will likely encourage patients to take responsibility for making informed decisions about their treatment. As a result, he

succeeds in achieving his stated goal of educating his readers about seasonality and seasonal mood disorders. This is a book that clinicians can recommend to their patients without reservation.

Maria R. Corral, M.D., F.R.C.P.(C), Department of Psychiatry, University of British Columbia, #8-1864 W. First Ave., Vancouver, B.C. V6J 1G5, Canada. Tel 604-732-3321.

BULLETIN BOARD

WELCOME TO NEW MEMBERS

We welcome new members who have joined SLTBR since publication of the September 1993 issue:

Associate Members

Leslie Demytruk
Christine A. Gibaud
Matthew M. Keats
Wendy Morray-Jones
Kimberly G. Peters

Sheryl L. Felty
Phyllis E. Heinemann
Kenneth W. Macek
Peter J. Muise
Mary Ann Wehlar

Student Members

Julia Struyck
Eus J.W. van Someren

MEMBERSHIP RENEWALS DUE

Included with this issue of *LTBR* is an invitation to renew your SLTBR membership for 1994. The membership renewal form includes space for you to update address and communication information for our files and membership directory as well as the opportunity for clinician and research members to provide data on practice interests. Please note the renewal deadline of 20 January 1994. Renewals received after 30 January will be assessed a \$10.00 late penalty. Clinician referral information and corporate membership listings will be revised after the renewal deadline to reflect current members. Please note the prerequisites explained in your renewal cover letter if you wish to be included in these listings. SLTBR does not endorse either clinical services provided or products manufactured by its members.

SLTBR ANNUAL MEETING UPDATE

Watch for your copy of registration and abstract materials for the SLTBR 1994 annual meeting scheduled for 23-24 June at Lister Hill Auditorium on the campus of the National Institutes of Health in Bethesda, MD. Meeting information packets will be mailed in late January 1994.

Our meeting precedes the 19th Congress of the Collegium Internationale Neuro-Psychopharmacologicum (CINP) which will be held in Washington, D.C., 27 June - 1 July, 1994. SLTBR members who wish to attend this conference may obtain information and registration materials by writing *CINP '94 Meeting Registration*, 42 Music Square West, Suite 103, Nashville, TN 37203-3234.

SLTBR PUBLIC INFORMATION BROCHURE REVISED

The recently updated *Questions and Answers About Light Therapy*, the Society's public information brochure, is now available for purchase (single copies or bulk quantities) through the SLTBR publications program. The brochure is also included in the information packet provided at a cost of \$7.00 (member cost \$5.00) to those who inquire about SAD and light treatment. Members wishing to have a complimentary copy included in their annual meeting information mailing may send a written request to the SLTBR office, P.O. Box 478, Wilsonville, OR 97070.

1994 NATIONAL AND INTERNATIONAL MEETINGS

Annual Meeting of the Society for Research on Biological Rhythms, Amelia Island Plantation, FL, 4-8 May. Contact: Dr. Robert Y. Moore, Center for Neuroscience, W1656, Biomedical Sciences Tower, University of Pittsburgh, Pittsburgh, PA 15216. Fax 412-648-8376.

Annual Meeting of the Society for Biological Psychiatry, Philadelphia, PA, 18-22 May. Contact Joel E. Kleinman, M.D., Ph.D., Clinical Brain Disorders Branch, NIMH Neuroscience Center at St. Elizabeths, William A. White Bldg., 2700 Martin Luther King, Jr. Ave. S.E., Washington, DC 20032. Tel 202-373-6227; fax 202-373-6214.

Annual Meeting of the American Psychiatric Association, Philadelphia, PA, 21-26 May. Contact APA, 1400 K Street, N.W., Washington, DC 20005. Tel 202-682-6000; fax 202-682-6345.

Annual Meeting of the Association of Professional Sleep Societies, Boston, MA, 4-9 June. Contact APSS, 1610 14th St. N.W., Suite 300, Rochester, MN 55901. Tel 507-287-6006; fax 507-287-6008.