MESSAGE FROM THE OUTGOING PRESIDENT

From what I can gather, the Fourth Annual Meeting of our society, held at NIH in Bethesda, Maryland, was regarded as a great success. I should mention that, as organizer, I heard mainly good things about it and it is possible that people were just being polite. But I doubt it. Having organized the first annual meeting at the NIH some four years ago, I found our members not to be unduly inhibited about expressing their dissatisfaction with the size of the room, the air conditioning (or lack of it), the poor organization, etc., etc., and they were right. Back then, in our salad days, we were cramped and hot and somewhat disorganized. That made it all the more satisfying to be able to sit back in the comfortable auditorium at Lister Hill and listen to the presentations. This time the organization was efficient and streamlined, thanks largely to our SLTBR secretary, Marty McCullough (recently promoted to executive director), as well as to our support staff at the NIMH, in particular Diana Miller. So we had reason to celebrate even before hearing any of the presentations or seeing any of the posters. The evaluations we received were generally very positive. We appreciate the constructive suggestions offered by several participants and will take them into account when developing future meetings.

I was intrigued and delighted at the scope of the topics covered by the presenters. Epidemiology, clinical descriptions and treatment studies were all represented. I will not attempt to summarize or even highlight the major findings presented as I know that plans are underway to feature those in the next issue of LTBR. One area that might have been better covered was psychobiology. Despite our advances in other areas, I was left with a renewed respect for how difficult it is to come to grips with mechanisms, both to explain the pathophysiology of a psychiatric illness and the mode of action of effective treatments. Clearly these will be important directions for future studies.

We appointed new officers for the organization. Anna Wirz-Justice was appointed president-elect and Thomas Wehr vice president. David Avery was appointed to chair the Federal-Industrial Liaison Committee, succeeding George Brainard who now assumes responsibilities from Charmane Eastman as chair of the Membership Committee. We thank both George and Charmane (who remains on the Board of Directors) for their past and ongoing efforts on behalf of SLTBR.

Our corporate membership increased this past year and ten members displayed their wares at the meeting. Many of the corporate members met separately on two occasions and established their own group, “Circadian Lighting
Association", under which rubric they plan to work cooperatively in areas of interest to themselves, to us and to our patients. Of primary importance is the delivery of safe and effective devices and accuracy of advertising. In providing these services efficiently and at competitive prices, our colleagues play a valuable role in ensuring that patients have ready access to appropriate treatment. It is to everyone's advantage that they operate effectively and responsibly in this regard. While self-monitoring of these services is highly desirable, there is a necessary role for outside monitoring by the FDA in the United States and corresponding organizations in other countries.

These issues were discussed in a panel by Dr. Frederick Goodwin, Director of NIMH; Drs. Michael Terman and George Brainard, who represented SLTBR; John Stigi and Ronald Parr, representing the FDA; and Kirk Renaud and Neal Owens, representing our corporate members. The issues involved in gaining FDA approval for existing fixtures are complex and would be best outlined elsewhere, but the panel discussion represented an important step towards defining them.

The business meeting was surprisingly lively. There was general consensus that the SLTBR meeting should be held annually, rather than biennially. Many people felt that early May is suboptimal for the meeting as it does not give SAD researchers sufficient time to analyze their winter data. There was discussion about possibly scheduling our

SLTBR BOARD MEMBERS AND OFFICERS gathered for a group photo between meetings at Amelia Island, FL. Pictured are President Michael Terman, Norman Rosenthal, President-elect Anna Wirz-Justice, Alfred Lewy, Charmaine Eastman, Treasurer Robert Sack, and Vice President Thomas Wehr.
next meeting in conjunction with the APSS meeting in Los Angeles in June 1993, but that has yet to be decided. There was strong support for a meeting in Europe, perhaps in two years' time, when Anna Wirz-Justice is due to be SLTBR president. There was also agreement that we want to encourage new members to join and, to this end, that we will tell potentially interested colleagues about the society.

As outgoing president, I was gratified by the level of the presentations, the enthusiasm and the collegial atmosphere that prevailed. I feel privileged to have been able to serve as president and look forward to continued participation in the society and to watching its continued success.

Norman E. Rosenthal, M.D.

"CHRONOARCHEOLOGY"

Another route for Hufeland’s interest in 24-hr rhythms?

Christoph Wilhelm Hufeland (1762-1836) was probably the most prominent German physician at the time of Goethe. He went to school at Weimar where his father was a general practitioner. When only 14 years old, Hufeland joined a company of dilettante actors organized by Goethe. He was much influenced by an uncle who made him read the Letters of Lord Chesterfield to His Son. Fifty-six years later, when writing his autobiography, he remembered the advice from it: "Grace, my dear son, grace is the main thing if you wish to make your fortune in the world" (Brunn, 1937). At the age of 18, Hufeland began studying medicine at the University of Jena. At that time, Jena was "beyond all bounds crude, slovenly, and rollicking." Hence, encouraged by his father, Hufeland decided to move to the University of Göttingen. There he found not only more assiduous and decent students, but also more eminent and stimulating teachers. Among them was the physicist Georg Christoff Lichtenberg (1742-1799). As will become clear later on, it is because of Lichtenberg that I have written this report.

In 1793, Hufeland became Professor of Medicine at the University of Jena, where he lectured on macrobiotics and pathogeny, a subject not least incited to him by his reading of Bacon’s Historia Vitae et Mortis. The book originating from this lecture first came out in 1797, entitled Die Kunst das menschliche Leben zu verlängern [The Art of Prolonging Human Life]. The heading Macrobiotics was not added until the 3rd edition. The 5th edition of 1823 which I have at hand carries a flowery dedication to Frederick William II, King of Prussia. This is by no means surprising because at that time Hufeland was already living in Berlin, where he was a professor at the leading hospital, the Charite, and "Physician in Ordinary" to the king.

In 1797, when the first edition of his bestseller was published, Hufeland was still Professor in Jena, and a dedication to Duke Karl August (1757-1828) would have been appropriate. Hufeland, however, decided for his "teacher and friend," Lichtenberg, well known not only as a physicist for his investigations on electricity, but also as a brilliant satirist and humorist. He wrote on almost all aspects of life. As all chronobiologists know, Hufeland, in his Macrobiotics, pointed out that "the 24-hr periodicity is the natural unit of our chronometry." It is likely that he got this idea early in his career as a physician in Weimar when, in his little spare time, he was studying the sleep-movements of leaves in the "moving plant," Hedyasarum gyrans (Aschoff, 1991).

However, it could also be that he was first confronted with the problem when attending the lectures of Lichtenberg. Indeed, there is a hint that Lichtenberg was aware of the 24-hr periodicity as an important feature of life. In a widely read literary magazine of that time, the Göttinger Taschen-Kalender, Lichtenberg once published an article entitled, "Hupazoli und Cornaro, oder: Thue es Ihnen nach wer kann" [...]Emulate them, whoever can do so. In this short essay, Lichtenberg tells the story of people who lived longer than 100 years, describes their lifestyle and diet (especially their modesty in eating), and emphasizes their punctuality. His main conclusion is, "Die sogenannten Leute nach der Uhr werden gewöhnlich alt. Das Handeln nach der Uhr aber setzt inneure uhrmässige Anlangen voraus..." [People who live by the clock usually live to be old. But to act according to the clock presupposes an internal clock-like disposition]. It seems that Lichtenberg had already envisioned the existence of a biological clock in humans in 1793, two decades prior to Burdach (1811), who meditated on shaping (in analogy to Linne's flower clock) a "Menschen-Uhr" (human clock), and prior to Virey (1814), who coined the term "l'horloge vivante" (living clock).

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REFERENCES
AURORA AUSTRALIS

SAD, rhythm disorders and light therapy in Australia

Does seasonal affective disorder (SAD) exist "Down Under" in Australia? What bright light research and treatment is taking place in this area of the southern hemisphere? These questions will be addressed in this review of work in the major southeastern Australian cities of — going from east to west — Sydney, Melbourne and Adelaide.

Philip Boyce, now at the University of Sydney, could be said to be Australia's pioneer SAD researcher and clinician. His interest in seasonality of depression developed when he discovered abnormal melatonin rhythms in melancholia (Boyce, 1985) and investigated the suppression of nocturnal plasma melatonin levels with varying light intensities (Boyce and Kennaway, 1987). In a survey study he identified the existence of SAD in the southern hemisphere associated with autumn/winter which, here, are the months from April through August (Boyce and Parker, 1988). He has played an educative role in the Australian medical community about SAD and light therapy (Boyce, 1987) and continues to have good clinical success in treating winter SAD patients with morning bright light produced by a locally manufactured light apparatus.

Moving southwest around the coast from Sydney to Melbourne finds an increasing number of SAD and rhythm researchers, perhaps because Melbourne winters are darker and gloomier. In Melbourne, investigations into the effects of bright light began in 1987 with an interest in effects on plasma melatonin which diversified into the therapeutic effects of light in SAD, PMS and sleep disorders. The work was carried out by Stuart Armstrong of the Psychology Department at La Trobe University in collaboration with Iain McIntyre, Trevor Norman and Graham Burrows of the Department of Psychiatry, Melbourne University at the Austin Hospital.

The initial work showed that human plasma melatonin suppression by a one hour light pulse given at midnight is intensity dependent from 3,000 lux to 200 lux, with even 200 lux having a suppressive, although non-significant, effect (McIntyre et al., 1989b). This finding was followed by investigations of a) the effects of one hour light pulses given early in the rising limb of the melatonin curve (2100 hr) to look for a rebound phenomenon (not found) and also on the descending limb at 0400 hrs (McIntyre et al., 1989a) and b) an attempt to look at the relationship between duration and intensity at low light levels (200, 400 and 600 lux) over a three hour period. Each light intensity produced its own individual maximum suppression by the end of the first hour of exposure. Increasing the duration of exposure a further two hours had no further suppressive influence (McIntyre et al., 1989c). In addition to concentrating on plasma melatonin levels, the effects of light exposure sufficient to suppress melatonin levels were examined in other hormones. Cortisol and prolactin levels did not alter in response to light exposure and melatonin suppression, thereby arguing against the proposal in the literature that there is a reciprocal balance between melatonin and cortisol.

Some preliminary work carried out in 1988 on light treatment of SAD replicated the then already established therapeutic effect of light reported overseas which was not generally accepted at the time by psychiatrists in Australia (McIntyre et al., 1989). In order to disseminate the work on bright light, circadian rhythms and SAD, an "Agenda" article was written for Today's Life Sciences. This was successful in spreading the gospel to a wide audience in the Australian biomedical sciences (Armstrong and McIntyre, 1989). Some preliminary work on changes in sensitivity of the melatonin rhythms to light before bright light therapy and after successful light therapy in a single SAD case was also carried out (McIntyre et al., 1990a). More recently, given the success of bright light treatment in SAD and the reports of a positive effect in PMS sufferers in North America, an initial investigation has been established in a collaborative project between Lorraine Dennerstein and Helen Ferguson of the Key Centre for Women's Health in Society, Melbourne University and Stuart Armstrong. Preliminary results should be available later in 1992. Finally, since there has been no clinical expertise available in Melbourne to treat SAD outside research projects, Peter Marriott of the Melbourne Clinic, in collaboration with Stuart Armstrong, now gives a professional diagnosis and advice on light therapy procedures for SAD sufferers.
The incidence of SAD in Australia has been looked at twice by the Melbourne Group, but not yet been published. One survey, conducted in 1989, was restricted to Melbourne and utilized the Swiss extension of the Seasonal Pattern Assessment Questionnaire. Results showed a much higher sensitivity to seasonal changes than reported by North American researchers. The whole seasonality curve was shifted to the left, with very few people (n=5 out of 247 completed questionnaires) reporting no sensitivity to mood changes with the seasons. In terms of prevalence rates, while that of SAD appeared to be similar to previous North American reports, the incidence of sub-SAD was far greater than expected. Since these findings appeared extreme, it was decided that there must have been problems with the sampling procedure or questionnaire reliability. However, a more recent survey, Australia-wide, carried out in collaboration with David Hay and Greg Murray of La Trobe University as well as the Key Centre for Women's Health, appears to be confirming the earlier findings.

It would appear therefore, that although several well known Australian psychiatrists, based upon their clinical experience, denied the existence of SAD in the Australian population, this is not the case. Not only is SAD prevalent in Australia but the sensitivity of the population as a whole to seasonal changes may be far more extensive than was hitherto suspected.

At the Psychology Department at Monash University in Melbourne Jenny Redman and her collaborators are carrying out both animal and human studies of biological rhythms. Paula Mitchell, in a doctorate program, is investigating the effects of caffeine on the circadian system by measuring time of day effects of caffeine on cognitive and motor performance and on hormone levels. A second graduate student, Helen Jarvis, is examining the effects on the circadian system of withdrawal from benzodiazepines. Two joint projects with researchers from La Trobe University are currently in progress. One, with Grahame Coleman, is designed to investigate the effects of ambient temperature cycles on circadian rhythms of activity and body temperature in laboratory rats and an Australian marsupial. In a second series of experiments with Stuart Armstrong, the effects of chemical synchronization, including both natural hormones and synthetic analogues, on the rat circadian system are being studied. In this latter program, there is particular emphasis on the role of melatonin and the pineal gland in the aging rodent circadian system.

Murray Johns, Director of the Sleep Disorders Unit at the Epworth Hospital in Melbourne, has used morning light therapy for patients with delayed sleep phase syndrome (DSPS). His experience is that generally some initial phase advance is produced by this treatment but that the effects do not last long (a few days to weeks).

If we now leave the Melbourne gloom and take a giant 750 km step northwest along the coast, we arrive in Adelaide, South Australia where circadian rhythm and bright light research has been in progress since 1986. Leon Lack's sleep laboratory at Flinders University has been studying the interaction between sleep and circadian rhythms since 1982. Abnormalities of the body temperature rhythm were found in various types of insomnia (Lack et al., 1985; Lack et al., 1988). In the same laboratory Drew Dawson, for his doctoral research program, carried out some basic experimental work on the circadian rhythm effects of bright light stimulation. He showed that a single 4-hour light pulse of 10,000 lux in the evening could delay the core temperature rhythm (Dawson et al., 1989a) and a single 4-hour light pulse in the morning could advance the core temperature rhythm (Dawson et al., 1989c). It was also apparent that the amount of phase-shift was dependent upon the relative phase difference between the time of light stimulation and time of body temperature minimum which defined a phase response curve (Dawson et al., in press). Other studies showed that the phase changes resulting from air travel or shift work are probably mostly or entirely due to the change of light environment rather than the change of the sleep/wake timing by itself (Dawson and Lack, 1988; Dawson et al., 1989b; Dawson and Campbell, 1991).

Dawson extended his research with bright light effects into more applied areas with Scott Campbell at Boston's Institute of Circadian Physiology and then at Cornell's Institute of Chronobiology. One technical report from this collaboration dramatized how rapidly the effective light intensity declines as distance increases or gaze changes from an intended light source (Dawson and Campbell, 1990). This reinforces the need in the research or clinical setting for high compliance of looking behavior or alternatively, the need to develop a tolerable and effective ambulatory light source which will ensure continuously effective light stimulation regardless of patient movements (McIntyre et al., 1990a). The applied studies showed the benefits of evening light therapy for night shift workers (Dawson and Campbell, 1991) and sleep maintenance insomniacs (Campbell et al., submitted).

Now Dawson is back at the University of Adelaide's Department of Obstetrics and Gynecology, Queen Elizabeth Hospital where they are studying the effects of
of both light and melatonin administration on the circadian system. In particular, they are looking at how light and melatonin can be used alone or in combination to treat circadian dysfunction. These studies are focused on two separate clinical populations, shiftworkers and geriatric insomniacs. They are also conducting a series of studies on theoretical aspects of light therapy. In these studies, they are examining differences in the effect of different wavelengths on the circadian system. Specifically, interest is in the role melatonin plays in mediating the circadian effects of light.

Meanwhile, back across town, work is continuing at Flinders University with Leon Lack, Helen Wright, Gill Zimmermann, Jeremy Mercer and crew with the use of light therapy for the treatment of sleep onset insomnia (mild to severe DSPS) and evening light therapy for early morning awakening insomnia (ASPS). Earlier, we established that sleep onset insomniacs are effectively mild DSPS individuals (Morris et al., 1990). Recently we have successfully used morning light therapy to phase advance and reduce the sleep onset latencies of sleep onset insomniacs. In clinical practice we have had fairly consistent success in thus treating mild DSPS/sleep onset insomniac high school students. Morning bright light therapy (2,500 lux) for one hour every morning upon awakening for 10 days (weekend as well) is reinforced with counseling, sleep hygiene advice and parental cooperation. The students have benefitted with earlier sleep onsets, longer sleep, and generally improved daytime functioning.

We do share some qualifications with Murray Johns in Melbourne about the use of morning light therapy, particularly in older DSPS patients. We find that compliance can be a problem for an adult population. This is perhaps not surprising given the motivations and time constraints of these individuals. Some of them are either unemployed or have occupations which allow them to sleep late and simply do not value a phase advance highly enough to justify the aversiveness of morning light therapy. Other DSPS patients may have inflexible early morning job commitments and find it difficult to sacrifice another hour of sleep, at least in the first few nights of therapy, by the necessity of arising even earlier to get in sufficient light therapy before work. It would seem that for many DSPS patients, morning light therapy, to be effective, needs to be supported with individually tailored behavioral management programs aimed at increasing compliance and decreasing sleep onset latency at the beginning of the sleep period. The Flinders group has also completed two studies with the use of evening light therapy for early morning awakening insomnia, the first of which is submitted for publication. It found with only two nights (2000 - 2400 hours) of light exposure (2,500 lux) a delay of temperature and melatonin rhythms, a delay of final wake-up time and increase of more than an hour of total sleep time. In summary, there are active research programs in Adelaide on the use of light therapy for insomnia and shiftwork. "Aurora Australis" is spreading forth in the great Southland!

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SAD AND LIGHT THERAPY RESEARCH IN HUNGARY

In Budapest, we have been investigating patients with Seasonal Affective Disorder (SAD) and light therapy since 1987. This brief report summarizes our own clinical experience and the results of our biochemical investigations.

Thirty-one patients with winter depression (22 females and 9 males) were first studied. SAD patients assessed in our clinic have similar clinical features to those previously described, although we found more unipolar (61%) than bipolar II (39%) patients and later age of onset of illness (28.9 y). Two of us (A. Németh, M. Arató) studied winter depression in Hamilton, Ontario, Canada in 1990/91 with R. Guscott, D. MacCrimmon and B. Saxena. There was a similar unipolar/bipolar ratio (54% vs 46%) and age of onset (28 y) in this group as well. These results were reported at the SLTBR meeting in Toronto (Németh et al., 1991). Light treatment used incandescent light (2500 lux at eye level) for 1 hr/day in the morning. After 7 days, 71% of the patients with SAD (n=31) improved significantly based on strict criteria. In contrast, the same light regimen in non-SAD patients yielded only one responder (7%, n=14).

Platelet $^{3}H$-imipramine binding ($B_{max}$ and $K_{d}$ values), serotonin content, MAO activity and plasma prolactin, T3, T4, TSH, cortisol levels, and dopamine-beta-hydroxylase (DBH) activity were studied in 22 SAD patients, 8 non-SAD patients and 22 healthy controls. Mean platelet $B_{max}$ imipramine binding, which is thought to be associated with presynaptic serotonin uptake mechanisms, was significantly lower in both patient groups than in the controls. After 7 days of light therapy the mean $B_{max}$ significantly increased in SAD patients (from 685.3 ± 32.2 to 998.2 ± 61.2 fmol/mg protein), reaching the same values as in healthy controls. The mean $B_{max}$ values of the patients with non-SAD, and of the controls, showed no significant change from baseline values after light therapy. This result suggests that the low $B_{max}$ value of imipramine binding is a state-dependent biological marker, at least in winter depression, and in these cases the artificial bright light exposure increases imipramine binding simultaneously with the clinical improvement. The decreased $B_{max}$ in SAD patients may relate to a dysregulation of the serotonergic system (Szádóczyk et al. 1991).

We found no significant difference in pretreatment plasma prolactin levels between the three groups [Depue et al. (1990) found significantly lower prolactin in SAD patients]. However, prolactin decreased significantly in SAD patients after 7 days of light therapy (from 7.6 ± 5.7 to 5.3 ± 3.0 ng/ml p<0.05). This result is difficult to interpret owing to the complexity of the prolactin regulatory system. Prolactin secretion is regulated or influenced by a number of factors that are of interest in affective disorders (such as dopamine, norepinephrine, serotonin, thyroid hormones, corticosteroids and calcium).

There was no difference in baseline cortisol levels between SAD patients and controls, but cortisol level was significantly higher in non-SAD patients. The non-SAD patients were older and more severely depressed (measured by SIGH-SAD) than the SAD patients. These factors may explain this result. However, it was a bit surprising that the high cortisol level decreased significantly after light therapy in non-SAD patients, in spite of the fact that only one of them improved markedly.

There were no significant differences in any other parameters (thyroid hormones, DBH, platelet serotonin content, MAO activity) between the three groups before and after light therapy (Németh et al., 1991).

Serotonergic mechanisms have also been implicated in the regulation of auditory evoked potentials (AEP). We have studied the distribution, amplitude and latency of the peak value (P300 of AEP). Our preliminary data, based on the investigation of 7 SAD patients, suggest the responder SAD patients show a "goggle-shape" posterior structure in P300 distribution before light therapy, and that light therapy induced a shift to the right in these patients. A predominantly serotonergic drug, clomipramine, has
induced similar asymmetry in quantitative EEG (MacCrimmon and Arató, 1991). In accord with this finding, Hegerl et al. (1991) have described the simultaneous change of blood serotonin and AEP under fluvoxamine treatment and light therapy. The amplitude of visual evoked potential increased after light therapy without change in the distribution of the peak values (Arató et al., unpublished data).

Our results indicate that serotonergic dysregulation plays a role in the pathophysiology of winter depression but further studies are needed to confirm these findings.

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LIGHT THERAPY IN CZECHOSLOVAKIA

Bright light therapy of depressive inpatients started in Czechoslovakia in 1985 at the Ostrov Psychiatric Department. Now light is used in five psychiatric centers: Prague Psychiatric Center, Charles University Clinic in Plzeň, Purkyně University Clinic in Brno, Sádská Psychiatric Department and Charles University Clinic in Prague. It is also being introduced to Psychiatric Hospitals Havlíčkuv Brod and Horní Bečkovice. Unlike other countries, the accent is on experimental treatment of endogenous depressed inpatients without marked seasonal pattern. Usually light therapy (LT) is combined with tricyclic antidepressants (TCA). There is also the ongoing and well known theoretical research on the influence of light on circadian rhythms in hamsters by Helena Illnerová and her colleagues.

1. Hastened onset of the effect of antidepressive drugs when using intensive white light (Práško et al., 1987) compared two groups of endogenous depressed inpatients at Ostrov Psychiatric Department. Twelve patients on combined LT (5000 lux, 5-6 a.m., 6 days) and TCA improved more rapidly (by days 3-6 HAM-D ratings, ANOVA p < 0.001) than nine patients on TCA alone.

2. Light therapy in endogenous depression — the beginning of therapy with and without TCA (Práško et al., 1988a) compared endogenous depressive inpatients (3 day washout) randomly assigned to TCA alone (n=10), TCA with LT as above (n=12) or LT for 3 days followed by combined LT and TCA (n=11). Both LT groups improved more than the group on TCA only (ANOVA p < 0.001).

3. Hastened onset of the effect of antidepressive drugs when using three types of timing of intensive white light (Práško et al., 1988b) involved three groups of endogenous depressive inpatients who were compared with respect to timing of LT: TCA was combined with morning light, as above (n=11) midday light (1-2 p.m., n=9) or evening light (9-10 p.m., n=9), and compared with a fourth group receiving TCA only. Onset of improvement was hastened by the combination therapy. It was most marked after morning light and most stable after midday light. The results of evening light were problematic.

4. Case studies — the effect of LT in patients with masked depression (Práško et al., 1989a) described four successfully treated inpatients with masked depression.
5. LT in patients with schizoaffective disorder (Praško, 1991b). In this pilot study at Prague Psychiatric Center 10 inpatients with depression in schizoaffective disorders were treated by a combination of neuroleptics and LT (5000 lux, 6 days, 5-6 a.m.). Significant improvement occurred after 3 and 6 days of therapy.

6. Holan and Vohlídková (1989) replicated the study of Praško et al. (1987) at Charles University Clinic, Plzeň. The group of 17 endogenous depressed inpatients treated by a combination of TCA and LT (5000 lux, 6 days, 5-6 a.m.) improved more than the group of 15 patients with TCA alone.

7. Štepánková (1991) at the Sadska Psychiatric Department compared two groups of patients suffering from endogenous major depression. The group (n = 12) treated with TCA and LT (5000 lux, 5 days, 5-6 a.m.) improved (Beck) more than the group (n = 12) with TCA alone. This improvement was also shown in psychological tests (Burdon, Numerical Quadrat).

8. Synek (1991) at Purkyně University Clinic, Brno treated 31 inpatient women suffering from major depressive disorder with LT alone (2500-5000 lux, 5 days, 5-7 a.m.) after 2-5 days washout period in an open study. 50% of the patients responded (HAM-D and Serejiski scales).

9. Praško (1991a) treated 10 inpatients with SAD at Prague Psychiatric Center in an open study. The effect of LT (5000 lux, 6 days, 5-6 a.m.) without TCA was very rapid according to changes of scores in MADRS and HAM-D (T-test, 3rd day of treatment p < 0.001).

10. Praško (1991a), Prague Psychiatric Center, compared the effect of LT (5000 lux, 3 days, 5-6 a.m.) without TCA in 10 patients with SAD and in 19 patients with Major Depression. The effect of LT was much greater in SAD patients (ANOVA p < 0.01 in MADRS and HAM-D).

11. Praško (1991a), Prague Psychiatric Center, studied the effects of LT on the circadian rhythms of melatonin, cortisol and prolactin in one patient with SAD. The 24-hr levels of hormones were measured before and after 9 days of LT (5000 lux, 5-6 a.m.). All three hormones were pathologically high in the morning and afternoon hours; LT lowered and phase advanced all rhythms.

12. Praško et al. (1989b), (1991) and Praško (1992) studied 47 inpatients with Major Depression in three centers (Prague Psychiatric Center Charles University Clinic, Plzeň; and Charles University Clinic, Prague) in 1992. Patients improved after three days of LT (5000 lux, 5-6 a.m.) (MADRS scores, t-test, p < 0.01).

13. Praško and colleagues at Psychiatric Center Prague (Praško et al., 1990) and Praško (1992) studied circadian rhythms of body temperature before and after LT (5000 lux, 6 days, 3 groups with different timing: 5-6 a.m., 1-2 p.m., 9-10 p.m.) in 32 inpatients with unipolar recurrent depression, depressive phase of bipolar disorder and depressive phase of schizoaffective disorder. There was no difference between the timing of LT and between diagnostic groups in the effect of light therapy on clinical state and on 24-hr temperature curves. The most marked effect of LT is to raise mean body temperature (t-test, p < 0.01). Phase shifts were inconsistent.

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REFERENCES


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SLTBR PUBLICATIONS UPDATE
The SLTBR executive office is in the process of updating the society’s publication list and order form. Due to the increasing volume of information produced since the inception of SLTBR, we no longer can publish a Complete Works of manageable size. Consequently, this volume will be discontinued and its components (LTBR, meeting abstracts, insurance endorsement information and SAD Assessment Tools) will be available as individual compendiums. A revised publications list will be included in the next issue of LTBR.

Abstracts of the 1992 annual meeting (Volume 4) are available from the executive office at a postpaid cost of $15.00 for members, $20.00 for non-members.

As participants of the annual meeting are aware, the proceedings at Lister Hill Auditorium were audio taped. While recording levels were not consistent throughout the meeting, tapes of the 1 May open forum and tutorial are of reasonable quality. In order to consider the advisability of reproducing these portions of the meeting, we need to assess the demand for these tapes. If you would like to purchase tapes of one or both of these sessions, please contact the SLTBR office, P.O. Box 478, Wilsonville, OR 97070 to express your interest.

The April 1992 issue of LTBR reported the availability of a revised edition of the SAD Assessment Tools Packet, including the SIGH-SAD, SIGH-SAD-SR and HIGH-SAD instruments. A revised edition of these instruments will should be available by the end of July. Orders for the packet will be held until the revised version is published.

BIOLOGICAL EFFECTS OF LIGHT SYMPOSIUM
The Light Symposium Foundation, headquartered in Atlanta, GA USA, will sponsor the third Symposium on the Biological Effects of Light from 3-5 June 1993 in Basel, Switzerland. The symposium will cover the following topics:

- Effects of light on skin, eyes and other systems
  UV-inflammation, carcinogenesis, photorepair, circadian rhythms, immune system, vitamin D₃.
- External and internal influences
  Ozone layer depletion, artificial light protection, cancer risk depletion, new light sources and devices.
- New developments in phototherapy and light therapy
  Photodynamic therapy, IR-therapy of hypertension, UVA-therapy of SLE, light therapy of winter depression.

On the occasion of the symposium, the fourth Arnold Rikli Prize will be awarded for research in the field of photobiology in relation to human beings. Further information can be obtained from Light Symposium Foundation, Bahnhofstrasse 47a, CH-4132 Muttenz, Switzerland; fax (41) 61-610051.
LIGHT AT THE TIMES
Public interest in light treatment and biological rhythms — and the work of SLTBR members — is strong, to judge by four articles that appeared in The New York Times this spring. "Harnessing the Power of Light", by Susan Gilbert, was featured in the 26 April Good Health Magazine. The National Institute of Mental Health was reported as having spent $15.5 million last year on light therapy research. R. Curtis Graeber of Boeing described futuristic lighting applications for combating jet lag. Charles A. Czeisler of Harvard University described applications of light for shift work and sleep disorders, with circadian phase shifts of up to 12 hours in as little as 2-3 days of treatment. Margaret L. Moline of Cornell University sounded a cautionary note, stating that the generality of such findings remains undetermined. George Brainard of Jefferson Medical College spoke of architectural lighting design as a biological manipulation, and described the light visor as a means to make the therapeutic intervention less intrusive. Michael Terman of Columbia University described high success rates using bright light therapy for SAD, and the development of a naturalistic twilight simulator that might provide a simple bedroom treatment. Hugh McGrath Jr. of Louisiana State University described a new UVA-I lighting technique for treatment of lupus symptoms including joint pain and fatigue. Daniel F. Kripke of University of California, San Diego described the finding that nighttime exposure to dim light can regularize abnormally long menstrual cycles. Scott S. Campbell of Cornell described success using light treatment in elderly people with insomnia.

On 12 May, the Science Times reported on Raymond W. Lam's studies at the University of British Columbia, suggesting retinal subsensitivity in SAD patients in comparison to normal controls. Norman E. Rosenthal indicated that the finding has been independently replicated. A National News feature on 16 May concentrated on night shift work, in which Timothy H. Monk of Pittsburgh pointed out increasing difficulties as workers age. Donald I. Tepas of the University of Connecticut noted the proliferation of round-the-clock operations, which is adversely affecting family life, including higher divorce rates. He emphasized that many night workers suffer chronic sleep deprivation. David Liskowsky of the Office of Technology Assessment, U.S. Congress, described the recently published task force report, Biological Rhythms: Implications for the Worker, previously reviewed in these pages by Torbjörn G. Akerstedt [LTBR (1992) 4: 25-26]. On 26 May, the Science Watch column reviewed the work of Joan Blom and Randy Nelson of Johns Hopkins, who reported increased sensitivity to a carcinogen administered to mice under long photoperiods than under short periods, which may be related to more sustained nocturnal melatonin production given winter-like lighting conditions.