

Light Treatment and Biological Rhythms

Bulletin of the Society for Light Treatment and Biological Rhythms



Volume 5, Number 4

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ANNUAL MEETING SCHEDULE

The SLTBR Program Committee has finalized the schedule for the Fifth Annual Meeting on Light Treatment and Biological Rhythms cosponsored by the Society and the University of California, San Diego, School of Medicine. The two-day meeting will convene at 8:30 a.m. on 19 June 1993 in Liebow Auditorium in the Medical School's Basic Sciences Building. The schedule features oral research presentations, poster presentation sessions and corporate exhibits. In addition, an afternoon education program on 20 June will include an overview of light treatment for a variety of chronobiologic disorders. SLTBR has applied for CME credit for the educational portions of the meeting. Social events include a reception in the poster/exhibit area of the meeting site and a poolside banquet at the host hotel, the Radisson La Jolla.

Late registration is available until 10 June with payment of a \$10.00 late fee. Meeting registrants who have not reserved banquet seating may do so prior to 10 June with payment of \$30.00. Please use the registration form mailed to each member in March 1993 and return completed information with appropriate payment to the SLTBR Executive Office, P.O. Box 478, Wilsonville, OR 97070. You may, instead, call/fax your request (VISA/Mastercard payment only, with card number and expiration date included) to the SLTBR office: 503-694-2404. Current members may register for the meeting with payment of \$95.00 (includes late fee); non-members \$135.00 (includes Associate Member dues). Banquet reservation is an additional \$30.00. On site meeting registration will include a \$15.00 late fee.

ANNUAL MEETING SCHEDULE

Saturday, 19 June 1993

8:00 - 8:30 Registration, exhibits, refreshments
8:30 - 12:00 Oral Scientific Presentations I
10:20 - 11:00 Break: Posters, exhibits, refreshments
12:00 - 13:20 Lunch

13:20 - 15:00 Oral Scientific Presentations II
15:00 - 15:10 Break
15:10 - 16:10 SLTBR Business Meeting
16:10 - 17:30 Reception: Posters, Exhibits
18:30 - Banquet

Sunday, 20 June 1993

8:20 - 12:00 Oral Scientific Presentations II
10:00 - 10:40 Break: Posters, exhibits, refreshments
12:00 - 13:20 Lunch
13:20 - 17:05 Education course

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Light Treatment and Biological Rhythms

Bulletin of the Society for Light Treatment and Biological Rhythms

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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.

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ORAL SCIENTIFIC PRESENTATIONS I

Biological Mechanisms of SAD and its Treatment

T. Wehr, Chair

Avery *et al.*: Is PSI_{M-E} increased in winter depression?

Eastman *et al.*: Searching for clues to SAD in the circadian rhythm of temperature.

Endo *et al.*: Changes in circadian rhythms and sleep structure of seasonal affective disorder by morning bright light.

Gorman *et al.*: Ophthalmological profile of 71 SAD patients: A significant correlation between myopia and SAD.

Kripke *et al.*: San Diego adults vary over 100-fold in photon exposure.

Lam *et al.*: The effects of light therapy on retinal electrophysiologic tests in winter depression.

Lewy *et al.*: Circadian phase-shifting effects of "dim" green light.

Mate-Kole *et al.*: Neuropsychological function in seasonal affective disorder and effects of light therapy.

ORAL SCIENTIFIC PRESENTATIONS II

Biological Mechanisms of SAD and its Treatment

R. Sack, Chair

Parry *et al.*: Neuroendocrine effects of light therapy in premenstrual dysphoric disorder.

Putilov *et al.*: Diurnal and seasonal variations of melatonin, cortisol, prolactin, thyrotropin and thyroid hormones in SAD.

Rosenthal *et al.*: Abnormal behavior and hormonal responses to m-CPP in SAD.

Schwartz *et al.*: Core vs. peripheral thermoregulation and serotonin in SAD: Results from an m-CPP infusion study.

Wirz-Justice *et al.*: Endogenous circadian rhythms in SAD: Light treatment improves performance without affecting alertness.

ORAL PRESENTATIONS III

Clinical Aspects of Seasonal and Circadian Rhythm Disorders

S. Ancoli-Israel, Chair

SAD: Clinical Features

Arbisi *et al.*: Self-report and direct measurement of carbohydrate consumption in SAD.

Brown *et al.*: SAD: The NIMH follow-up study.

Carskadon *et al.*: Reports of seasonal mood in couples: Role of circadian phase preference and latitude.

Terman, M. *et al.*: Is seasonal affective disorder a variant of atypical depression? II. Diagnostic similarities.

SAD: Non-photoc Treatment of SAD

Köhler *et al.*: The treatment of SAD by periodic physical activity.

Schlager *et al.*: Early-morning, short-acting beta-blockers for treatment of winter depression.

Disorders of Circadian Rhythms and Sleep: Light Treatment

Campbell *et al.*: Alleviation of sleep maintenance insomnia with timed exposure to bright light.

Lack *et al.*: Bright light treatment for insomnia.

Seasonal Influences on Other Syndromes

Marriott *et al.*: Seasonality in panic disorder.

POSTER PRESENTATIONS

All posters will be mounted for viewing and discussion during both days of the meeting. Presenters will be available for discussion at their posters during morning breaks and the Saturday afternoon reception.

Armstrong et al.: Overnight human plasma melatonin, prolactin and cortisol under conditions of normal sleep, sleep deprivation and sleep recovery.

Brown, F. et al.: Critique of 17 models of shift-work effects.

Brunner et al.: Sleep parameters in SAD: Effects of midday light, season and sleep deprivation.

Christ et al.: Phase shifts in core body temperature and slow wave sleep distribution in extended sleep.

Cole et al.: Seasonal variation in illumination exposure in humans at two different latitudes.

Dumont et al.: MSLT after bright light exposure.

Eder et al.: Sleep architecture in symptomatic SAD and changes following dawn simulation: A naturalistic study.

Elliott et al.: Phase relation of evening and morning oscillator regulates amplitude of hamster PRC to light.

Juarez et al.: SAD symptoms and daily illumination exposures.

Kräuchi et al.: Light treatment at midday decreases heart rate in SAD at every circadian phase and during sleep.

Lee et al.: Inpatient light exposure.

Lovell et al.: The effect of bright light on agitation: Case reports.

Lubkin et al.: Is miotic medication in glaucoma patients associated with depressive symptoms?

Meesters et al.: 10,000 lux bright light therapy in various temporal schemes.

Moffit et al.: Bright light treatment of late-life depression.

Murray et al.: Seasonal affective variation in Australia: Disorder or preference?

Norden et al.: Correlations of heat and light with alcohol consumption and violence: Possible implications for serotonin function.

Oren et al.: Ambient light exposure in winter seasonal affective disorder.

Ozaki et al.: Studies of platelet serotonergic systems in winter seasonal affective disorder.

Pearl et al.: The effects of light treatment on the symptoms of fibromyalgia.

Rex et al.: Light treatment of long menstrual cycles.

Rosen et al.: Bedroom window effects on sleep patterns.

Terman, J.S. et al.: Relapse rate following light treatment of winter depression.

Turner et al.: Seasonal hot flushes responsive to phototherapy in a perimenopausal SAD patient.

Veith et al.: The influence of the menstrual cycle on the female circadian system.

EDUCATION COURSE

A pragmatic overview of diagnosis and treatment of a variety of chronobiologic disorders. Presentation format will be twenty-minute presentations with discussion time between presentation sets.

Raymond Lam, M.D.

University of British Columbia, Vancouver, B.C.

Diagnosis of winter depression

Practical aspects of light therapy

Dan Oren, M.D.

National Institute of Mental Health, Bethesda, MD

Light therapy devices

David Avery, M.D.

University of Washington, Seattle, WA

Dawn simulation for winter depression

Robert Sack, M.D.

Oregon Health Sciences University, Portland, OR

Light therapy for sleep disorders

Daniel Kripke, M.D.

University of California, San Diego, CA

Light therapy for nonseasonal depression

Barbara Parry, M.D.

University of California, San Diego, CA

Light therapy for PMS

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POST-MEETING OPEN HOUSE

Immediately following the Sunday program, SLTBR meeting attendees and their guests are invited to an open house at Dan Kripke's new Circadian Pacemaker Laboratory. Dan and his staff encourage you to join them — get some bright light in the isolation rooms and share some light refreshment.

PRESS INVITED TO ANNUAL MEETING

Members of the press are cordially invited to attend SLTBR oral presentations, poster presentations, exhibits and the education course at no charge. Please request a press badge in advance by writing on letterhead to Marty McCullough, Executive Director, SLTBR, P.O. Box 478, Wilsonville, OR 97070.

DSM-IV DRAFT CRITERIA FOR SEASONAL PATTERN

The DSM-IV criteria are currently in draft form, including new criteria for Seasonal Pattern. The DSM-III-R and DSM-IV criteria are reprinted in this issue of *LTBR* for comparison. Recall that the DSM-IV Task Force asked the Work Groups to take a conservative approach to revisions: to make no changes unless empirical data dictated change, or unless criteria were found to be unworkable.

With this conservative approach in mind, several proposed changes can be highlighted:

1. The course modifier can be applied to Bipolar I, Bipolar II, or Recurrent Major Depressive Disorder (application to the Not Otherwise Specified [NOS] diagnoses has been dropped).
2. The exclusionary criteria in DSM-III-R for seasonally varying psychosocial stressors has been dropped.
3. The 60-day window in DSM-III-R for at least three episodes in at least two consecutive years has been dropped. The pseudoprecision of 3:1, seasonal: non-seasonal, episode ratio has also been dropped. These have been replaced by a simpler requirement for two "clean" seasonal years (i.e., two seasonal episodes and no nonseasonal episodes), plus a predominant seasonal pattern over the individual's lifetime.

This is a good time for feedback to the Work Group, since these changes are not yet written in stone. For instance, there may be new data evaluating the comparability of recurrent major mood disorder patients with NOS patients. This could provide an empirical rationale for applying the modifier to the ill-defined NOS disorders, as well as to well-established recurrent major mood disorders.

As another example, replacing the 3:1 requirement with a shorter duration, though cleaner, seasonal pattern requirement may increase the diagnostic "catch." On the other hand, requiring a lifetime pattern in which seasonal episodes "substantially outnumber any nonseasonal episodes" would restrict the diagnosis to those with a longstanding pattern. If there are recently available data on the temporal stability of the diagnosis, or on the effect of requiring long-term vs. short-term seasonal pattern on sample homogeneity, response to light treatment, etc., they may be helpful in simplifying these requirements.

Expert opinion from SLTBR members may be helpful in formulating the final revision. Empirical data-sets published or analyzed since the last call for SLTBR input would, of course, be the most powerful input to the Work Group. Please forward input to *A. John Rush, M.D., Chair, DSM-IV Mood Disorders Work Group, University of Texas, Southwestern Medical School, Suite 600, 5959 Harry Hines Blvd., Dallas, TX 75235. Fax 214-688-4278.*

Mark S. Bauer, M.D., Dept. of Veterans Affairs Medical Center; Dept. of Psychiatry and Human Behavior, Brown University, Providence, RI 02908-4799. Tel 401-457-3083; fax 401-457-3370.

DSM-III-R: Diagnostic criteria for seasonal pattern

- A. There has been a regular temporal relationship between the onset of an episode of Bipolar Disorder (including Bipolar Disorder NOS) or Recurrent Major Depression (including Depressive Disorder NOS) and a particular 60-day period of the year (e.g., regular appearance of depression between the beginning of October and the end of November).

Note: Do not include cases in which there is an obvious effect of seasonally related psychosocial stressors, e.g., regularly being unemployed every winter.

- B. Full remissions (or a change from depression to mania or hypomania) also occurred within a particular 60-day period of the year (e.g., depression disappears from mid-February to mid-April).
- C. There have been at least three episodes of mood disturbance in three separate years that demonstrated the temporal seasonal relationship defined in A and B; at least two of the years were consecutive.
- D. Seasonal episodes of mood disturbance, as described above, outnumbered any nonseasonal episodes of such disturbance that may have occurred by more than three to one.

DSM-IV: Draft

Specify if: With Seasonal Pattern (can be applied to Bipolar I Disorder, Bipolar II Disorder, and Major Depressive Disorder, Recurrent):

- A. There has been a regular temporal relationship between the onset of an episode of Bipolar I or Bipolar II Disorder or Major Depressive Disorder, Recurrent, and a particular time of the year (e.g., regular appearance of depression in the fall or winter).
- B. Full remissions (or a change from depression to mania or hypomania) also occur at a characteristic time of the year (e.g., depression disappears in the spring).
- C. In the last two years, two episodes have occurred that demonstrate the temporal seasonal relationship defined in A and B, and no nonseasonal episodes have occurred during that same period.
- D. Seasonal episodes of mood disturbance, as described above, substantially outnumber any nonseasonal episodes of such disturbance that may have occurred over the individual's lifetime.

SLTBR CONTINUES WITH DSM-IV INPUTS

In response to publication of the DSM-IV draft criteria in March 1993, Drs. Norman Rosenthal (Past President) and Michael Terman (President) sent the following letter to Dr. Allen Frances (Chair, DSM-IV Task Force) and members of the DSM-IV Mood Disorders Work Group.

21 April 1993

We gather that the DSM-IV committee is interested in hearing from clinicians and researchers with areas of special expertise in relation to the recently circulated draft of DSM-IV. In that spirit we'd like to provide some feedback about the proposed criteria for "seasonal pattern" as a modifier of recurrent mood disorders. In general we feel that the proposed criteria are an improvement over those in DSM-III-R. The committee has dropped several criteria that were hard to implement and were of dubious benefit. With respect to the following two points, however, the new criteria seem less useful than their predecessors:

1. The new criteria require that "In the last two years, two episodes have occurred that demonstrate the temporal seasonal relationship defined in A and B, and no nonseasonal episodes have occurred during the same period." This choice of the last two years seems rather

arbitrary and we can easily envisage that clinicians who apply the criteria strictly would miss many legitimate cases. After all, what is so special about the last two years? During that period a winter depression could easily have been missed for a definable reason, such as a long trip south, or as a matter of chance. It is our common experience that a cycle of depression can be mild in any given year for reasons that are not always clear. We would therefore urge you to return to a criterion of "two consecutive years" rather than "the last two years" in order to avoid missing these cases. This "two consecutive years" criterion has been around since seasonal affective disorder was first defined. It has served researchers and clinicians well and we see no reason to change it to a new, problematic and untested criterion.

2. As it stands in the current draft, "seasonal pattern" can be used to modify only Bipolar I and II Disorder and Major Depressive Disorder, Recurrent. We would ask you to consider adding to the list of categories any other form of recurrent mood disorder to be included in DSM-IV (such as "not otherwise specified" or "recurrent minor depressive disorder"). Evidence suggests that light therapy works for these individuals as well as for the more severe and classical recurrent depressives and a fair number of studies have included such individuals among their numbers. Second, insofar as "seasonal pattern" describes a temporal relationship

and is not *per se* a severity criterion, we think these variants should be included.

3. Finally, it would be helpful if the season showing depressed mood were specified, e.g., "with seasonal pattern, winter type" in order to help define meaningful subgroups of this pattern.

Dr. Terman then met with Dr. Frances and Dr. Michael First (DSM-IV Editor), at New York State Psychiatric Institute, for further discussion. In summary:

- On point 1, it was mentioned that the "last two years" was used to ensure a "current" diagnosis. Terman emphasized that the definition of seasonally recurrent mood disorders necessarily involves consideration of a long-term pattern. Recent onset of a seasonal disorder might be provisionally detected based on two years' information, but the longer course is far more definitive for most patients. He recommended retaining the "two consecutive years" criterion against a baseline of a past "regular relationship" to the seasons, as stated in criterion A. This would serve to cover both cases: recent onset of the disorder, or longstanding history of the disorder in which there might have been a transient aberration in the last two years.
- On point 2, it was mentioned that the intent in DSM-IV is to disallow the use of course specifiers for NOS disorders, which are "unofficial". However, Dr. First mentioned that a strong argument for retaining the seasonal course specifier for NOS/minor depressive disorder is that it was used in DSM-III-R, and there are no data to support its elimination now. Terman pointed out that the logical cohesiveness of the diagnostic structure is reduced if a disorder which clearly can occur with seasonal pattern may not be described as such.
- On point 3, Dr. First suggested that the season in which the depression is *predominant* be specified. This is not necessarily the season of episode onset (e.g., December 19 would be listed as "winter", not "fall").

N.E.R. and M.T.

AGENCY FOR HEALTH CARE ACKNOWLEDGES SAD AND LIGHT THERAPY OPTION

After more than two years of study, the Depression Guidelines Panel of the USPHS Agency for Health Care Policy and Research (A. John Rush, Chair) has published its clinical guidelines for primary care practitioners. Although the use of antidepressant drugs and psychotherapy are its primary foci, the report offers a detailed description of SAD and endorses the use light therapy under the guidance of a clinician trained in the technology.

The *Patient's Guide*, for example, gives the following advice: "Light therapy may help people who have mild or moderate seasonal depression. This treatment should only be given by a specialist until it has been studied more thoroughly."

Volume 1 of the clinician's text, *Detection and Diagnosis*, lists the following subgroups of Major Depressive Disorder: Psychotic, Melancholic, Atypical, Seasonal, and Postpartum psychosis/depression. The efficacy of medications and psychotherapy for SAD are listed as unknown, while light therapy is considered "an option". The strength of evidence for the seasonal subgroup is listed as "A" ("good research-based evidence, with some panel opinion, to support the guideline statement"). As for bipolar patients,

A seasonal pattern has been found in a subset of patients who have bipolar II disorder (and in some, but fewer, cases of bipolar I disorder). . . . Perhaps 10 percent of patients with bipolar II disorder experience such seasonal episodes. It is unclear whether lithium is differentially effective in these patients and, indeed, whether the disorder becomes nonseasonal over time. .

Volume 2, *Treatment of Major Depression*, provides the following guideline: "Light therapy is a treatment consideration only for well-documented mild to moderate seasonal, nonpsychotic, winter depressive episodes in patients with recurrent major depressive or bipolar II disorders or milder seasonal episodes." The strength of evidence is "B" ("fair research-based evidence, with substantial panel opinion, to support the guideline statement"). In more detail:

- Light therapy is a logical consideration only for well documented seasonal, nonpsychotic, winter depressive episodes in patients with recurrent major depressive or bipolar II disorders or milder seasonal episodes.

- It should be administered by a professional with experience and training in its use who deems it suitable for the particular patient.
- It may be a second-line treatment option after the patient has failed to respond to an adequate medication trial.
- It may be a first-line treatment for these patients if they are not severely suicidal and if there are medical reasons to avoid antidepressants, if the patient has a history of a positive response to light therapy and no negative effects, if the patient requests it, or if an experienced practitioner deems it indicated.

Further:

Light therapy should not be used as an adjunct to medication until either one alone has been optimally used. Light therapy can be useful to augment the response (if partial) to antidepressant medication and vice versa. As with any treatment, the patient's response should be closely monitored. Respond to light therapy can be rapid (4 to 7 days), but for some, response may be delayed to 2 weeks. However, the placebo response rate may be significant as well. Therefore, one or several 'extended evaluation' visits may be useful in identifying those in whom symptoms persist. Caution is urged in the use of light therapy with patients with specific ophthalmologic or other conditions. . . . Since safety and efficacy have not been fully established beyond 2 weeks, consultation with a specialist may be helpful in determination specific risks and benefits for particular patients.

Free copies of the AHCPR materials can be obtained by contacting the *AHCPR Publications Clearing House* (800-358-9295; outside U.S., 301-495-3452 or write *AHCPR Publication Clearing House, P.O. Box 8547, Silver Springs, MD 20907-8547*). The relevant publications are:

- *Depression is a Treatable Illness: A Patient's Guide.* #93-0553.
- *Depression in Primary Care: Detection, Diagnosis, and Treatment (Quick Reference Guide for Clinicians).* #93-0552.
- *Depression in Primary Care: Vol. 1. Detection and Diagnosis.* #93-0550.
- *Depression in Primary Care: Vol. 2. Treatment of Major Depression.* #93-0551.

Forthcoming publications will include detailed evidence tables, etc. Single copies of the advisory report to the panel are available from the authors by request on

professional letterhead: Terman, M., and J.S. Terman (1990) *Light therapy for seasonal affective disorder: Report to the Depression Guidelines Panel, USPHS Agency for Health Care Policy and Research*. New York State Psychiatric Institute, New York (722 West 168th Street, New York, NY 10032).

The following sentence may now be a helpful addition to insurance reimbursement endorsement letters sent by clinicians: "These procedures conform to 1993 USPHS AHCPR guidelines for management of this disorder."

M.T.

BRIGHT LIGHT TREATMENT FOR JET LAG

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

Rapid transmeridian travel across multiple time zones elicits a range of symptoms that include difficulty in initiating or maintaining sleep, daytime sleepiness and decrements in subjective alertness and performance, gastrointestinal distress, and other psychosomatic manifestations (Comperatore and Krueger, 1990; Winget et al., 1984). Collectively known as jet lag, these symptoms are attributable to three major concomitants of long-distance air travel: flight-related stress and fatigue, sleep loss, and altered temporal relations between the circadian timing system and the new local day-night cycle, as well as between different rhythmic functions within the individual traveler. Of these, the disruptions in circadian phase relations are unique to east-west flights (Hautey and Adams, 1966c; Gerritzen et al., 1969; Sasaki et al., 1985; Buck et al., 1989) and, due to the gradual nature of circadian reentrainment, their effects can last considerably longer than the one or two days required to recover from flight-induced fatigue and sleep loss.

The following sections will examine the course and duration of reentrainment of circadian rhythms following time zone changes, the nature of the sleep disturbances resulting from such changes as determined by polysomnography, and, finally, recent attempts to accelerate the reentrainment process and alleviate the symptoms of jet lag using timed exposure to bright light.

Circadian reentrainment following time zone changes

The time required for circadian rhythms to resynchronize to a shifted day-night cycle depends not only on the size of

the shift (the number of time zones crossed) but also on its direction, the rate of readjustment being generally faster after westward than after eastward travel. For flights across 5-11 time zones, mean reentrainment rates of 92 min/day (westward) and 57 min/day (eastward) were derived from one set of studies (Aschoff et al., 1975), and rates of 88 min/day (westward) and 56 min/day (eastward) from another (Klein and Wegmann, 1980).

This directional asymmetry is not dependent on whether the flights are outbound or homebound, nor on whether they are day or night flights (Klein et al., 1970; Klein et al., 1972a,b; Wegmann et al., 1970). Rather, it is generally attributed to the fact that, following westward travel, circadian rhythms reentrain to the delayed day-night cycle by gradual phase delays, i.e., by temporarily assuming periods longer than 24 h, while after eastward travel, circadian rhythms usually reentrain by gradual phase advances, i.e., by temporarily assuming periods shorter than 24 h. Since the natural periods of human circadian rhythms, measured in the absence of daily time cues, tend to be longer than 24 h, it follows that reentrainment will be faster after westward than after eastward flights (Aschoff et al., 1975).

This explanation also accounts for the occasional occurrence of reentrainment by phase delay following phase advances of the day-night cycle. Such an antidromic phase response (Klein and Wegmann, 1980) has been observed in laboratory studies after 6-h phase advances (Wever, 1980; Moline et al., 1992) as well as in field studies after eastward flights across 8-9 time zones (Colquhoun, 1979; Klein and Wegmann, 1980; Gander et al., 1989). In some of these cases, one rhythmic function reentrained antidromically while another did so in the usual direction, by phase advance, a phenomenon termed reentrainment by partition (Aschoff, 1978).

The reentrainment rates listed above are average figures across the entire reentrainment period, and across different rhythmic functions, different subjects, and different conditions. As such, they hide a number of complexities. First, the rate of reentrainment is rarely constant over time. Rather, the size of the daily phase adjustment tends to be proportional to the phase angle difference between the circadian timing system and the shifted Zeitgeber (Klein and Wegmann, 1980). Thus, reentrainment rate is generally highest immediately after the shift, decreasing progressively thereafter (Aschoff et al., 1975).

Different rhythmic functions often reentrain at different rates (Klein and Wegmann, 1980; Fèvre-Montange et al.,

1981; Halberg et al., 1977; Elliott et al., 1972; Hauty and Adams, 1966 a,b), leading to a transient internal dissociation within the individual (Aschoff, 1978). Klein and Wegmann (1979, 1980) have pointed out that the more slowly adapting functions tend to show more robust circadian oscillations under normal entrainment conditions than rapidly adapting functions, suggesting that the rate of adaptation of a given function may reflect the relative contributions of endogenous circadian timing and exogenous masking effects in determining its daily pattern. For some functions, assessment of reentrainment rate is further complicated by distortions of the daily waveform, with different phase references — daily maxima and minima, for example — resynchronizing at different rates (Désir et al., 1981; Wegmann et al., 1970; Sasaki, 1964). Reductions in the range of oscillation and changes in mean daily level have also been reported (Klein and Wegmann, 1979; 1980). Recent computer simulations (Daan and Beersma, 1992) indicate that masking effects, waveform distortions, and the specific curve-fitting procedures used for phase assessment can all lead to unequal reentrainment rates for different overt rhythms, even when these rhythms are generated by the same circadian pacemaker.

The conditions travelers are exposed to and the activities they engage in after transmeridian flights can markedly affect the duration of readaptation. Klein and Wegmann (1974) found that reentrainment of psychomotor performance rhythms following 6-h time zone shifts was 50% faster in subjects allowed outdoor activities every other day than in subjects confined to their hotel rooms. The authors attributed this result to differences in the strength of social time cues experienced by the two groups. However, recent demonstrations that human circadian rhythms can be reset by bright light suggest that the faster adaptation seen in the outdoor group may have been due, at least in part, to their being exposed to daylight. Exposure to strong social and light cues may also account for the unusually rapid initial adjustment reported in military units following eastward airlifts (Adam et al., 1972; Colquhoun, 1979; Graeber, 1980; Graeber et al., 1981), as military populations frequently engage in both group and outdoor activity.

There are also marked individual differences in the rate of adjustment after time zone transitions (e.g., Klein et al., 1977). One of the factors underlying these differences is age. Following flights between Oklahoma City and Tokyo, 40-48 year-old subjects required longer to reentrain their body temperature rhythms and reported greater fatigue than 19-23 year-old subjects (Hauty and Adams, 1965). In a laboratory study simulating a 6-h eastward (advance) transition, middle-aged subjects showed more sleep

disruption than young subjects and reported larger decrements in daytime alertness and a greater increase in sleepiness (Moline et al., 1992). Although the mean rate of reentrainment of body temperature rhythms did not differ between the two groups, two of the eight older subjects showed an antidromic (delaying) response, whereas all six young subjects reentrained by phase advance. Greater sleep disturbances in older travelers were also observed after flights between London and San Francisco (Evans, 1970; Evans et al., 1972). Flight crews operating long-haul routes showed no age-related differences in sleep alterations during the first layover after a transmeridian flight (Nicholson et al., 1986a), but older airline pilots showed greater cumulative sleep loss over the course of a 15-day operational tour with multiple layovers (Preston, 1973). The higher susceptibility of older persons to jet lag has been attributed to greater difficulty in sleeping at abnormal circadian phases (Klein et al., 1980; Graeber, 1982; Moline et al., 1992).

Other factors contributing to interindividual variations include such rhythm characteristics as phase angle of entrainment (Colquhoun, 1979) and, possibly, rhythm amplitude and stability (Comperatore and Krueger, 1990; Klein and Wegmann, 1980; Graeber, 1982; Winget et al., 1984). Personality variables, among them introversion/extraversion and neuroticism, may also play a role (Colquhoun and Folkard, 1978).

Overt daily rhythms are the product of endogenous circadian timing as well as exogenous influences from the environment and from one's own behavior, particularly activity, sleep, and meal patterns. Most transmeridian travelers immediately shift their daily habits to conform with the new local time, thereby influencing the apparent rate of reentrainment of their rhythmic functions. In simulation studies, when masking influences are minimized through the use of constant routines (Mills et al., 1978), or when their estimated effects are eliminated mathematically (Folkard et al., 1991), reentrainment rate is consistently slower than when it is assessed using standard procedures.

Sleep disturbances

Westbound transmeridian flights are typically scheduled as day flights. Passengers boarding an airplane in San Francisco at 1200h would, after an 11-h flight, reach Tokyo at 1600h local time. Due to the 7-h time difference, this would correspond to 2300h San Francisco time, roughly the time at which they would normally retire for the night. By immediately adopting local time schedules, however, they would delay their sleep for several hours,

eventually retiring at about 2300h Tokyo time, or 0600h San Francisco time.

Eastbound flights, on the other hand, are typically night flights. Passengers departing San Francisco at 1700h would arrive in London at 1100h the next morning, local time, having crossed 8 time zones in the course of a 10-h flight. By postponing sleep until 2300h London time, they would be going to bed at 1500h San Francisco time. Thus, in both cases, the travelers would be going to sleep at abnormal circadian phases after being awake considerably longer than usual. Sleep characteristics under these conditions have been documented by polysomnography in passengers (Evans, 1970; Evans et al., 1972; Klein et al., 1976; Sasaki and Endo, 1977; Endo et al., 1981; Sasaki et al., 1985; Désir et al., 1981; Nicholson et al., 1986b) as well as in aircrew during the first layover after transmeridian flights (Dement et al., 1986; Sasaki et al., 1986; Nicholson et al., 1986a; Wegmann et al., 1986). The results are generally consistent with current understanding of circadian and homeostatic regulation of sleep, itself derived largely from laboratory studies of sleep displacement and deprivation.

Sleep latency is usually reduced on the first night after both eastward and westward travel, mainly as a result of prior sleep deprivation. On subsequent nights, sleep latency returns to normal after westward flights, in some cases gradually, while after eastward flights sleep latency may return to or exceed normal values.

Total sleep time is often unchanged following flights in either direction, although decreases after the first night have been reported following westward flights. Early awakening, sleep interruptions in the later part of the night, and, occasionally, a reduction in sleep efficiency, are also observed after westward flights.

An increase in slow wave sleep (SWS) is generally reported on the first night after both eastward and westward travel, as well as after north-south flights, and is attributable to prior sleep loss. Two or more days may be required before SWS levels return to baseline.

Rapid eye movement sleep (REMS) is frequently reduced on the first night, particularly after eastward travel. This effect is due to the advance in sleep onset time after eastward flights and, possibly, to competition from SWS which may have a higher priority after prolonged wakefulness. The decrease in REMS can persist for two nights or more after eastward flights, but REMS levels return to or exceed baseline levels following westward flights. Westward travel is also followed by a decrease in

REMS latency, with occasional sleep onset REMS episodes, and a shift in REMS to an earlier time of the night.

As with other functions, the time required for all sleep parameters to return to baseline levels tends to be longer after eastward than after westward travel (Nicholson et al., 1986b; Sasaki et al., 1985; Gander et al., 1989).

Sleep disturbances are of particular concern in airline personnel on transmeridian routes. Such disturbances are consistently reported by flight crews (Raboutet et al., 1958; Lavernhe et al., 1965; Cameron, 1969; Smolensky et al., 1982; Sasaki et al., 1985), with night wake and difficulty falling asleep as the prime complaints (Sasaki et al., 1985). In a survey of French air crew on transatlantic routes, 25-35% of the respondents reported needing three nights or more before achieving normal sleep (Lavernhe et al., 1965). Sleep deficits may also be incurred on long-haul routes with multiple layovers (Preston and Bateman, 1970; Preston, 1973; Samel and Wegmann, 1988), although many crewmembers are able to obtain normal amounts of sleep during such routes (Nicholson, 1970; Preston, 1973; Spencer et al., 1991). However, in a study of military transport aircrew on transmeridian missions lasting several days, crew members reported sleeping slightly longer than before departure (7.3 h versus 6.8 h), yet slept for 9.9 h, 9.2 h, and 8.9 h on the first three nights after the missions (Hartman, 1971). These results suggest that changes in sleep quality, resulting from sleep displacement and fragmentation, can contribute to the accrual of a significant sleep debt even when sleep quantity is unchanged.

Bright light treatment for jet lag

Recent demonstrations of entrainment and resetting of the human circadian system with light (Czeisler et al., 1981; 1986; 1989; Wever et al., 1983; Honma and Honma, 1988; Minors et al., 1991) hold the promise for the development of similar procedures to alleviate the effects of jet lag by accelerating reentrainment to new time zones. That promise, however, remains largely unfulfilled, as only a handful of field studies, all preliminary, has been performed to date, and even laboratory simulations have thus far yielded mixed results.

The first field attempt relied on scheduled exposure to natural daylight following eastward flights across 9 time zones (Daan and Lewy, 1984). Two subjects were studied, one of whom was asked to expose himself to daylight for 3 h starting at 0700h local time (2200h in the original time zone), the other starting at 1000h (0100h original time).

The schedule was followed for seven days, starting on the first post-flight day. The timing of light exposure was derived from animal phase response curves (PRCs) for light, as human PRCs had not yet been published. Accordingly, the transition between light-induced delays and advances was assumed to lie midway between dusk and dawn. Thus, daylight at 0700h was intended to induce a phase delay, and daylight at 1000h a phase advance. Based on sleep logs and oral temperature readings during waking hours, the subject exposed to light at 1000h advanced to the new phase in about six days, whereas the subject exposed to light at 0700h appeared to reentrain antidromically, by phase delay, and was still not fully adapted after 13 days. These results, however, are difficult to reconcile with the more recent human PRCs (Czeisler et al., 1989; Honma and Honma, 1988; Minors et al., 1991), as both exposure times fall within the delay region of these PRCs. Indeed, light exposure would be expected to cause a larger phase delay at 1000h than at 0700h.

The phase-resetting protocol developed by Czeisler et al. (1986) has proven highly effective in a laboratory setting. This protocol was applied to a single subject upon his return from Tokyo to Boston (Czeisler and Allan, 1987). The phase of his body temperature rhythm was first assessed during constant routine, and the subject was then exposed to bright artificial light (7000-12000 lux) for several hours on three consecutive days starting in early afternoon. Reassessment of temperature phase during a second constant routine five days after the flight indicated a large delay of 11.25 h.

Wever (1985) compared reentrainment rates of two subjects following two phase delays of an artificial light-dark (LD) cycle in a laboratory study simulating westward time shifts. The first shift was performed under normal indoor light intensities (<1500 lux), the second under bright light (2000-5000 lux in most parts of the experimental room). Mean reentrainment rates of body temperature over the first three post-shift days were 1.0 h/day under normal illumination and 1.4 h/day under bright light.

Other simulation studies have been less successful. Moline et al. (1989; 1990) exposed subjects to 2500 lux for 4 h following a 6-h phase advance of a dim, indoor level, LD cycle (300-500 lux). The light treatment began at the pre-shift mid-sleep time on the first post-shift day, and immediately after waking on the next three days. During the first five days after the shift, the temperature rhythms of the bright light subjects advanced at the same rate as those of dim light controls, but then appeared to delay

again on the following two days. Some of the bright light subjects also showed long-lasting reductions in circadian amplitude. Polygraphic sleep recordings revealed no substantial differences between the two groups. More recently, Samel et al. (1992) exposed subjects to bright light for 4 h following a similar 6-h advance shift. The treatment was scheduled on two consecutive days, starting either at 0400h or at 1300h. Reentrainment rates of body temperature rhythms did not differ between the two light exposure times, except on the seventh day when the phase shift of the morning light group exceeded that of the afternoon group by 1.16 h.

Two additional field studies have examined the effects of morning bright light treatment on post-flight sleep patterns. In the first (Cole and Kripke, 1989), 19 travelers returning to California from trips to the Orient or South Pacific (advance shifts of 6.5-10 h) were instructed to expose themselves to either bright white light (2000 lux) or dim red light (<100 lux) for 2-3 h upon awakening in the morning. This schedule was followed for three days, during which the subjects kept daily sleep logs. No differences were observed between the group means for any sleep measure, but on at least one of the treatment days, the time of light exposure in the white light group correlated negatively with total daily sleep and percent nighttime sleep, and positively with percent daytime sleep. Thus, exposure to bright light early in the morning appeared to facilitate the consolidation of sleep into a single nighttime episode, while later exposure times may have actually hindered it. In the second study (Sasaki et al., 1989), polysomnographic recordings were obtained from four subjects before and after a flight from Tokyo to San Francisco (8-h advance). In San Francisco, the subjects were required to go to bed at 2300h and be up by 1000h, and were exposed on three consecutive days to either bright (>3000 lux) or dim light (<500 lux) for 3 h starting at 1100h. On the first four nights, the bright light subjects showed higher sleep efficiency and less wake after sleep onset than dim light subjects, the latter exhibiting prolonged wakefulness during the first half of the night.

For air crew scheduled to return to home base after brief layovers, staying on home time may be preferable to trying to adjust to local time, as this eliminates the need for readjustment after the return flight. Indeed, a survey of flight personnel (Lavernhe et al., 1965) showed that, after flights from Paris to North America, 23% of all respondents (and 41% of those over 50 years) reported going to bed before 1900h local time, or 0100h Paris time. In such cases, timed exposure to bright light may be used

to maintain entrainment to home time rather than to accelerate reentrainment to new local times.

The development of small, head-mounted, battery-operated light sources may prove useful in field applications of bright light treatment, as these are easy to carry along on international trips, and can even be used on the day of travel, aboard the aircraft. Finally, recent computer software (Houpt et al., 1992) will help those who, in trying to determine light exposure times for jet lag therapy, have had to struggle with home, layover, destination, standard, daylight savings, and circadian times. Given a travel itinerary and a user-defined PRC, the program will indicate when exposure to bright light should be sought, and when it should be avoided.

In summary, although decades of animal and human research attest to the efficacy of timed exposure to light as a means of manipulating circadian timing systems, much remains to be learned before procedures can be developed that are at once effective, reliable, and practical. For this to happen, optimal combinations of several light exposure parameters — timing, intensity, spectral composition, duration, and number of repetitions — must first be defined, then tailored to specific flight situations.

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SAD IN ICELAND

Phototherapy is not new. One of the first recipients of the Nobel Prize was Dr. Finsen who received it in 1903 for light therapy of skin disorders, especially for dermal tuberculosis which was common at that time. Dr. Finsen had an Icelandic mother and a Danish father. The story goes that his interest in the therapeutic effect of light began when he was a college student in Reykjavik and found that his energy and concentration greatly improved when he could catch the sun's rays by moving his desk towards the window.

Skammdegistunglyndi is a well known word in Icelandic. Skamm means short, degi=day, tung=heavy,

lyndi=mood. Thus the word *Skammdegistunglyndi* means *the heavy mood of the short days* which shows that the concept of a winter depression has long existed in Iceland. The first reports of SAD and light therapy were therefore immediately received with great interest in Iceland.

In spring 1988 we mailed out the *Seasonal Pattern Assessment Questionnaire* (Rosenthal et al., 1984) to a random sample of 1000 Icelanders. Prevalence rates of SAD and S-SAD were significantly lower in Iceland (11%) than along the eastern seaboard of the USA (Magnússon and Stefansson, 1993). This was unexpected, since in the USA the prevalence of SAD increases with more northern latitudes. We speculated that, since the Icelandic population has lived in virtual isolation and has endured harsh living conditions for the past 1000 years, Icelanders might have become genetically adapted to the long arctic winter. To test this hypotheses we measured the prevalence of SAD and S-SAD in a group of Canadian immigrants of wholly Icelandic descent who had lived in Canada for the past 3-4 generations. The prevalence of SAD and S-SAD was approx. 6- and 3 times lower, respectively, in this group than in the USA (Magnússon and Axelsson, 1993). These results are consistent with the idea that the Icelandic population has acclimatized to long and dark winters.

We are currently surveying a new group of urbanized Canadian immigrants of wholly Icelandic descent together with a sex and age matched control group from the same city in Canada. We are also currently organizing, together with Carl Hagfors from Finland, a Scandinavian multicenter study which examines the epidemiological features of SAD in the Nordic countries.

From the epidemiological study in Iceland, 20 subjects from each of the three groups; SAD, S-SAD and those with Seasonality Score = zero, were interviewed. The purpose was to compare the *SPAQ* with a clinical interview, the main emphases being on how well the two methods agree in the diagnoses of SAD and S-SAD. A manuscript is in preparation. After a short visit to Michael Terman's laboratory in New York we embarked on a light therapy study during the winter 1988-89. The details of this study have already been published (Magnússon and Kristbjarnarson, 1991) but some of the specific features are as follows: 1. We obtained excellent results with only 40 minutes light exposure by using 10,000 lux light intensity. 2. We interviewed the patients the following summer at which time point all were symptom free. 3. A confounding factor in many outpatient light therapy studies is that the SAD patients may have a considerable exposure

to daylight during the study period. This is a minor problem in Reykjavik since the winters are very dark and ambient light exposure is minimal relative to the light received from the light boxes.

We also carried out hormonal analyses (morning, noon and evening) in SAD patients before and after light therapy, and in summer, as well as in controls in winter and summer. No significant differences in the levels of the hormones T3, T4, TSH, cortisol, testosterone and prolactin have been detected between any categories.

In a pilot study for light therapy in narcolepsy we interviewed a group of 24 carefully diagnosed narcoleptics. Five out of the 24 patients felt that their symptoms improved in bright weather conditions, but in most cases this was modest. Only three out of 24 felt that their symptoms improved during the summer. We felt that these reports were not encouraging enough to embark on a light therapy study. Since then there have appeared at least two negative reports on light therapy in narcolepsy.

During the winter 1989-90 we went to three colleges in Reykjavik and selected a group of pupils who had problems with school attendance in the winter and who also showed circadian rhythm instabilities and certain symptoms of SAD. We treated this group with light therapy but did not find a statistically significant improvement in attendance.

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BOOK REVIEW

The 24-Hour Society

By M.C. Moore-Ede (London: Judy Paitkus Publishers Ltd., 1993, British edition, 230 pp., £18.00. Available in USA from Addison-Wesley, Reading, MA, \$22.95).

In this book, Moore-Ede describes and explains the cost, in terms of increased accidents, human discomfort and even illness, of living in a society that no longer enables us all to sleep each night. This requirement stems from the need to provide emergency cover, utilities and transport throughout the 24h. In addition, many industrial processes must be run continuously and the cost of some equipment is so high that uninterrupted operation is required to recoup the initial financial outlay before it becomes obsolete. Finally, with our planet ever-shrinking and increasing intercontinental travel, business executives and politicians must contend with time zone transitions and jet lag.

In the first part of his book, Moore-Ede emphasizes these needs together with the inability of us humans to cope easily. Our problems arise because of the slow adjustment to a changed schedule of our body clock, with poor performance at night and poor sleep in the daytime. Sleep loss compounds the problems of nocturnal fatigue, as a result of which the night worker works less efficiently, makes more mistakes — sometimes with catastrophic consequences — and often suffers a poor social life and a lower standard of health into the bargain!

In the second part, specific areas of the work force are dealt with in more detail. These are the medical profession (particularly junior doctors, for the author once was amongst their ranks), aviation, power stations, rail and road transport, and globe-trotting executives and statesmen. Often based on his consulting experiences, Moore-Ede describes the problems; but a distinction between those due to excessive hours of work, to poor or lost sleep, or to working during the night (on body time) is not always made, even when this is possible. This reduces the ability to focus on the major culprit in some of these cases. The British edition includes examples from the UK in this section, though by far the majority of the text has a North American bias. The last chapter in this section centers on two examples in which Moore-Ede acted as an expert witness and describes how his Institute (Circadian Technologies) estimated the amount of impairment of mental alertness that would have been experienced in two specific legal cases. (The computer

algorithm is not discussed in any detail.). These examples do separate the components of fatigue (see above); fascinating.

In the third section, ways to overcome some of these problems are considered. These include the use of appropriately timed bright light and melatonin ingestion to shift the body clock, and ways to overcome fatigue during a spell of duty. There is also mention of some methods that have been developed to monitor whether a person falls asleep when on duty. In this section, the author tends to be dismissive of those areas in which he or his colleagues have not been involved, and the section as a whole cannot be considered as a balanced account of this area. Nevertheless, he makes the very interesting and important point that some of the very things that managers incorporate into night shifts in order to improve performance when the body is working below its peak — increased comfort and reduced distractions from background music and casual conversation, for example — can all promote boredom and sleep rather than improve efficiency.

The last section deals with the broader picture of attempting to produce humans to inhabit this non-stop society. The idea of flexibility of an individual's lifestyle and working hours is discussed, together with the differences between individuals (though this last issue is considered far too briefly). The synthesis that emerges is that of individuals producing their own "time cocoon" in which, according to their particular characteristics — a "lark" or an "owl", say — they can attempt to organize a pattern of work, leisure and sleep that overcomes many of the difficulties. While the aim cannot be faulted, of course, it is unclear if this scheme would produce sufficient numbers of alert and socially fulfilled long-distance pilots or lorry drivers, for example; it is so much easier for researchers and those whose time is their own anyway!

This book is not intended as a systematic account, but rather as a distillation of Moore-Ede's and his colleagues' important research as well as his experiences as a consultant over the past 10 years. At times, therefore, there is a hint of advertisement for Circadian Technologies and for those who have been associated with the author.

There is one area which is not covered and which must limit its usefulness for a European market; no mention of weekly or rapidly rotating shift systems. Since these are common in that part of the world (I don't have enough information to comment on shift systems in Australia and

China for which markets I believe other editions of the book are aimed), the advice that is contained in this book will not always be appropriate to these work forces.

In summary, the book is entertaining, stimulating, well written and abundantly illustrated, provided that one remembers that other and valid points of view exist also.

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RESEARCH OPPORTUNITIES

Post-Doctoral Research Position: Participate in all aspects of our research on the phase shifting of human circadian rhythms by bright light during simulated night shifts using body temperature as a circadian system marker. Collaborate in the development of new experiments exploring other methods of phase shifting, or modifying factors for light-induced phase shifts, such as melatonin, exercise and naps. Contact: Dr. Charmane Eastman, Biological Rhythms Research Laboratory, Rush-Presbyterian-St. Luke's Medical Center, 1653 W. Congress Parkway, Chicago, IL 60612-3864. Tel 312-942-4472; fax 312-955-3958.

Post-Doctoral Research Fellowship in Clinical Psychobiology: Post-doctoral Intramural Research Training Award (IRTA) available for individual interested in coordinating large ongoing clinical research program, investigating psychobiological aspects of seasonal affective disorder and its response to light therapy and other forms of treatment. Position requires active involvement in research planning, patient contact, data gathering and processing, as well as supervision of research assistants and program management. Salary range from \$25,000 to \$31,000 depending on qualifications. Please forward curriculum vitae and the names of three references to: Dr. Norman E. Rosenthal, Section on Environmental Psychiatry, Clinical Psychobiology Branch, National Institute of Mental Health, Bldg. 10/4S-239, 9000 Rockville Pike, Bethesda, MD 20892. Fax 301-496-5439. NIH is an equal opportunity employer.

Pre- or Post-doctoral Positions: Light Therapy Unit, New York State Psychiatric Institute. NIMH-sponsored projects include clinical evaluation of SAD patients, treatment monitoring and assessment. Data analysis, contribution to publications. Experience, skill and interest in computer applications (statistical, database, word processing, programming). Excellent writing, oral communication and quantitative skills. Specific job description may be tailored to qualifications. Opportunity for training in areas without prior experience. At senior levels, opportunity for self-initiated research. At M.D. or Ph.D. post-doctoral levels, opportunity to apply for supplementary fellowship funding through Columbia's NIH-sponsored cross-disciplinary program in psychobiology (U.S. citizens only), and opportunity for limited time effort in personal clinical practice. Two years minimum commitment. Salary commensurate with experience. Begin late summer or early fall 1993. Write or fax, with resume and personal statement: Dr. Michael Terman, NYS Psychiatric Institute, 722 West 168th St., Unit 50, New York, NY 10032. Fax 212-960-2584.