SAD Behind the Knees

Lawrence P. Morin, PhD

The report in Science by our colleagues, Scott Campbell and Patricia Murphy, that light presented through the skin at the back of the knees can phase shift human circadian rhythms is nothing short of astonishing. The electronic and print media carried the story repeatedly for several days. I am sure that many of you experienced the same types of questions posed to me by friends from a variety of science and non-science backgrounds. That’s crazy, isn’t it? What did they really do? What does it mean?

Well, no. Apparently it is not crazy. Some years ago, Tom Wehr and co-workers applied light to skin or eyes and determined that application of light to the eyes of SAD patients attenuated the symptoms to a much greater degree than in the same patients who received light applied to the skin, but not eyes. However, embedded in a group of two individuals who had very large improvement to light on the skin. One of these improved only with light on the skin. Dr. Wehr, while speaking publicly, often repeated his interest in those individuals, suggesting that someone with the right amount of experimental courage should pursue the issue of light acting through the skin to alter rhythm-related variables.

Dr. Dan Oren also put his stamp on the topic by publishing two speculative articles describing reasonable biochemical mechanisms by which light hitting the skin could affect rhythm physiology and behavior. He has remarked upon the ability of light to dissociate putative neural signaling gases, CO and NO, from hemie molecules. One of the molecules discussed by Dr. Oren as a putative blood-borne phototransducing substance is bilirubin which is eliminated by skin exposure to bright light.

Drs. Campbell and Murphy employed the bright light-low heat technology of a BiliBlanket Plus (Ohmeda). Light from a quartz lamp was transmitted to the back of each knee for 3 hours via 2400 optic fibers with participants seated in a reclining chair. General illumination at eye level during stimulus presentation was about 20 lux; at the target site intensity was about 13,000 lux. At all other times, illumination was a continuous 50 lux. The body temperature rhythm was recorded continuously and saliva was collected hourly for melatonin rhythm assays.

An unambiguous phase response curve resulted from timed presentation of the extracocular light stimulus. There were maximal phase delays of about 2 hours when the stimulus occurred about 3-4 hours before the body temperature minimum; maximal phase advances were at about 1 hour after the minimum. An interval of no phase response may exist from 4 to 16 hours after the temperature minimum, although this remains to be more thoroughly tested.

In this one experiment, Campbell and Murphy have a) provided solid evidence that extracocular light can alter circadian rhythm function in mammals, b) legitimized the case for extracocular photoreception in mammals, c) provided a method that may finally allow adequate control procedures for experiments involving human photostimulation particularly those involved with seasonal affective disorder, and d) stimulated a host of ideas concerning new experiments that now can be conducted and new ways of providing timed light to ambulatory patients or experimental subjects.

References


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January 25, 1998
Letter to Scott Campbell
Dear Scott,

Now that all the hullaballoo over this peculiar, light-on-the-back-of-the-knees stuff has been superseded by Bill Clinton's purported White House bedroom (or perhaps, closet or other kinky location of your/choice) antics, could you spare a moment for a lowly, SLTBR reporter skulking about on the outskirts of a hot story, but about 10 days late?

The story I had in mind concerns the rumor that you have been nominated to receive the Nobel Prize for Scientific Irony. The nominators are (I am told by sources who cannot be revealed for fear of phase shifting themselves to another time/place — which of these depends entirely upon the current version of Einsteinian theory to which you currently adhere) senior patent attorneys from Harvard University.

Incidently, the Nobel Prize for Scientific Irony is sponsored, in part, by Bud Lite who made his fortune in a remarkable joint marketing venture that combined solar radiation, geographic locale, and fruit. His product was oranges which he sold as "a bit of Florida Sunshine to brighten an otherwise dreary day." These have been used as folk remedies ever since he declared that his Florida Sunshine was good for the soul. There are presently over 5,280 internet sites advocating the use of Florida Sunshine for the treatment of winter blues, indigestion and gout. (The only down side to the Bud Lite story is the alleged ongoing lawsuit by the Celestial Seasonings tea company that claims a patent infringement by Lite and his company. Celestial apparently charges that not only is Florida sunshine in the public domain and, hence, lower case letters, but that even if it weren't, they would have been there first because, if you look carefully, orange is a common flavoring ingredient in their teas).

Now, Harvard is steeped in history and the folks there like to show their knowledge of, and connections to, the past literature on all topics. So, not surprisingly, the patent attorneys considered the soul to be, as declared by Descartes, the pineal body. (There is emerging doubt about the Descartes declaration, however. It is possible that his spelling was wrong and he really meant "popliteal," see below). It follows that Florida Sunshine, being good for the soul, could also be patented for novel use as a diagnostic tool that would allow examination of the potency of the visual-circadian-pineal pathway. Logic dictates that a bit of Florida Sunshine would suppress pineal melatonin, thereby altering all human physiology and creating beauty and goodness in the world for all humankind. (Or is that what happens when melatonin is elevated? I forget).

Now, the irony. The world has been thoroughly misled by the Harvard patent attorneys to believe that route to the soul is through the eyes! And, yes, on behalf of Harvard, the attorneys have claimed an exclusive route to the soul! Indeed, there are patents on paper that prove to their satisfaction that, in fact, Harvard scientists demonstrated that Florida Sunshine acts through the eyes. Not only did the Harvard scientists perform such a demonstration, they did it first and anybody else who now tries to show something similar (e.g., the Celestial Seasonings tea company) must, if so requested, turn over the results of their demonstrations to the Harvard patent attorneys.

Oh, the irony. The irony, indeed! Just as night usually follows day, it is clear to this naive reporter that just as the way to a man's heart is through his stomach, the way to a man's soul is through the popliteus! It is not through the eyes at all! It's through the back of the knees! Ah, a novel use patent of some real significance is there for the asking. In fact, this humble reporter himself has already received an informal inquiry from a large anonymous airline company about potential commercial use of the "Pop-Lite," as they call it. The company is prepared to immediately modify all seats on all airplanes flying across more than two time zones to include "Pop-Lites" on the front of each seat where the knees drape over the edge. Lites will be computer controlled to shoot the backs of the knees with photic energy at appropriate time for enabling travelers to arrive feeling fully refreshed and in sync with local time. Scotsmen and women wearing skirts or dresses will find the "Pop-Lite" method of curbing the agony of jet lag particularly satisfying.

Anyway, I find all of this behind-the-knee stuff breathtakingly thought provoking and am seriously considering writing something about it for the SLTBR bulletin. Would you please give me a call so we could talk?

Best,
Larry
Bright Light Therapy in Focus: Lamp Emission Spectra and Ocular Safety


In recent years, bright light treatment of seasonal affective disorder (SAD), recurrent depressions in fall and winter, has been discovered. Newer applications include circadian sleep phase disorder, shift work and jet lag. Apart from creating the visual signal, light can modify retinal structure and physiology. UV and visible light lead to distinct lesions of ocular tissues under certain experimental and naturalistic conditions. In light therapy, a large variety of fixtures is used but the spectral emission of lamps is mostly unknown to the user and clinician leading to the potential hazard of ocular lesions. Therefore, we have analyzed a wide selection of light sources commonly used for treatment. We measured the spectral emission and calculated irradiant doses for several light therapy regimens. Based on these measurements, potential hazards are analyzed, physiological mechanisms of light action are discussed and safety measures for bright light therapy are proposed. They include recommendations for lamps devoid of damaging spectral emissions and standardized therapy fixtures, ophthalmological monitoring of patients with eye diseases and control by optometrists for patients with healthy eyes who are likely to undergo light treatment for extended periods.

In the present study we analyzed a wide selection of light sources and diffusing screens commonly used for light treatment. We measured spectral emission and screen transmission and calculated irradiant doses for several light therapy regimens. Based on these measurements, we outline the potential hazards of extensive exposure to bright light. In addition, we discuss the physiological mechanisms of light action in the context of chronobiology.

There is widespread evidence for acute and chronic damage to ocular tissues by both UV and visible light (Organisciak and Winkler, 1994; Remé et al., 1996; Terman et al., 1990). Although there are few unequivocal action spectra available for the human eye, photochemical, photophysical and animal studies have revealed action spectra that suggest basic damage mechanisms. Exposure regimens, intensity and spectral range determine the extent of lesions.

Given the likelihood the patients will use bright light therapy for decades, two types of potential lesions must be considered: acute lesions and chronic cumulative damage. The cornea and conjunctiva are the primary loci of UV-induced acute lesions such as the keratoconjunctivitis in snow blindness or after exposure to welding arcs. A number of studies report acute and subacute light damage to the human retina (Remé et al., 1996).

Chronic degenerative diseases linked to UV-exposure include climatic droplet keratopathy in the cornea, the pterygium and pingeuscula of the conjunctiva, and cataracts of the lens (Young, 1994). Light exposure might accelerate ageing processes in the retina or potentiate age-related macular degeneration (Young, 1994). A crucial factor that influences ocular irradiant dose is the individual exposure geometry. Disparate results of epidemiological studies may be attributed to inadequate specification of individual's exposure conditions (Sliney, 1994).

Several factors increase light induced lesions. In particular, certain drugs and other compounds cause photosensitization of ocular tissues. They show absorption in the near UV and visible ranges, pass the blood-ocular interfaces and may bind to ocular tissues. The chemical structures include tricyclic-heterocyclic, amphiphilic-lipophilic and porphyrin ring systems (Roberts and Dillon, 1990; Roberts et al., 1992). Exogenous melatonin has been administered at specific times of day to assist circadian phase adjustment (Terman et al., 1995, Deacon et al., 1994) and it will likely be used in conjunction with light treatment. This hormone is widely investigated in chronobiology (Arendt, 1995) and is thought to provide a physiological "dark signal." It may increase retinal sensitivity by suppressing light adaptive retinal dopamine (Dubocovic, 1988). Indeed, melatonin administration in animals increases susceptibility to light induced retinal lesions (Leino et al., 1984; Souëtre et al., 1989). Under supervised clinical use the timing of melatonin and bright light would be strictly separated, because their phase-response curves bear an approximate inverse relation. However, if the drug were taken at high dose or concurrently with bright light exposure there would be a potential retinal hazard.

Results and Discussion

[For details about methods, see original publication.]

In general, a quarter to a third of fluorescent lamp spectral power is provided by UV and blue light, which has greater photon energy than the longer wavelengths. In sunlight, the corresponding proportion is 1.5% UVB and 6.3% UVA, whereas 91.7% of spectral power derives from visible light and IR. The proportion of UV and blue in sunlight impinging on the human eye, however, varies greatly with time of day and environmental conditions including grass or trees and reflecting surfaces such as snow or cement. Furthermore, protective behavioral factors such as aversion and squinting strongly modify the exposure of the eyes to natural sunlight in an outdoor environment (Sliney, 1991,1994). Thus, there is no simple correspondence to the conditions of artificial bright light therapy despite the fact that
light levels used for therapy are below or within the illuminance range encountered on a bright sunny day outdoors.

Out of nine lamps analyzed, seven showed distinct patterns of UVA and UVB emission (Fig. 1). Most of the bulbs also showed a strong component of blue and green light (Table 1), a wavelength range that has caused damage to photoreceptors and the pigment epithelium in laboratory studies (Organisciak and Winkler, 1994). Tube no. 9 showed minimal levels of UV emission and moderate levels of blue and green light. Furthermore, the spectral pattern was relatively evenly balanced across the broad range of visible wavelengths, a fact that would increase subjective comfort at high intensity. Therefore, this lamp type may be preferable for therapeutic applications.

![Graph showing irradiance at 1 m for different tubes](image)

**Fig. 1. Emission of UVB and UVA by fluorescent lamps used in light therapy boxes.** Measurements were made with a spectrometer in 1 nm steps at 1 m distance from the lamps. The emission of UVB from 300-320 nm and the emission of UVA are shown. Tube nos. 5 and 7 show the highest values for UVA and UVB, while tube nos. 8 and 9 emit practically no UV: Tube 1, Osram Lumilux deluxe L 36/12; tube 2, Osram Biolux L 36/72; tube 3, Philips TLD 36/93; tube 4, Philips TLD 36/96; tube 5, Duro-Test Vita-Lite (Truelite) 40 TH 12; Tube 6, Duro-Test Vita-Lite (Truelite) II 40 TH 10; tube 7, Duro-Test Vita-Lite (Truelite) Plus 40 TH 10; tube 8, Duro-Test ColorGard 50 (40 w); tube 9, Duro-Test ColorGard 50 (60 w).

The number of lamps in a light box and the characteristics of rear-reflectors and plastic diffusing screens all contribute to the irradiance level and the spectral composition of the emitted light. Therefore, we also measured the transmission characteristics of several common plastic diffusing screens, which varied greatly (Fig. 2).

The radiometric measurements have to be translated into the therapeutic situation, where a patient is supposed to receive about 2500 lux or 10,000 lux, respectively, at eye level. We have calculated the approximate irradiant doses for UVB, UVA and blue light for different therapeutic regimens over a wide range of treatment durations for two contrasting bulbs, one of the recommended type and another that showed unfavorable emission characteristics (Table 1). Tube no. 5 shows a high UVA emission with 4.6 μW/cm². With treatment at 10,000 lux, the corresponding irradiance would be approximately 220 μW/cm². A treatment session of 30 minutes using bulb no. 5 would result in a UVA exposure of about 0.8 J/cm²; with 30 sessions per month the cumulative dose would be 16.0 J/cm². A treatment period of five months per year over 10 years would yield a cumulative dose of 800.9 J/cm². By comparison, tube no. 9, with its strict cutoff near 400 nm, would yield a cumulative dose of 6.35 J/cm². These values assume that the patient's head position is 1 m from the lamps. Closer distance would increase the irradiant dose; at 30 cm, the distance commonly employed for 10,000 lux treatment, the dose would be approximately ten times higher. These calculations demonstrate that over extended treatment periods the potential for UV-induced ocular lesions cannot be disregarded. For example, calculations were made for daily SAD treatment at 2500 lux during the winter months, resulting in the equivalent of a daily erythemally effective radiant exposure of 40 J/m² (Diffey, 1993).

A satisfactory therapy regimen would thus include lamps with negligible UV emission, diffusing screens which block UV and reduce blue light, and careful control of patients potentially susceptible to light induced lesions. Worst case situations would include UV-emitting lamps with insufficient absorption of UV and blue light by the diffusing screen or even the lack of a diffusing screen with therapy applied over

<table>
<thead>
<tr>
<th>Winter depression (SAD)</th>
<th>Cumulative doses in J/cm²</th>
<th>Shift work phase-adjustment</th>
<th>Cumulative doses in J/cm²</th>
</tr>
</thead>
<tbody>
<tr>
<td>10,000 lx, 0.5 h/session</td>
<td><strong>per day</strong></td>
<td><strong>per month</strong></td>
<td><strong>per year</strong></td>
</tr>
<tr>
<td><strong>per 5 months</strong></td>
<td><strong>per 30 months</strong></td>
<td><strong>per 5 years</strong></td>
<td><strong>per 10 years</strong></td>
</tr>
<tr>
<td>Tube 3</td>
<td>UVB (300-320 nm)</td>
<td>0.06</td>
<td>1.18</td>
</tr>
<tr>
<td></td>
<td>UVA (320-400 nm)</td>
<td>0.80</td>
<td>16.02</td>
</tr>
<tr>
<td></td>
<td>Blue (400-470 nm)</td>
<td>2.30</td>
<td>46.07</td>
</tr>
<tr>
<td>Tube 9</td>
<td>UVB (300-320 nm)</td>
<td>0.005</td>
<td>0.10</td>
</tr>
<tr>
<td></td>
<td>UVA (320-400 nm)</td>
<td>0.006</td>
<td>0.13</td>
</tr>
<tr>
<td></td>
<td>Blue (400-470 nm)</td>
<td>1.372</td>
<td>27.45</td>
</tr>
</tbody>
</table>

| 5000 lx, 3 h/session | **per day** | **per month** | **per year** | **per 5 years** | **per 10 years** |
|-------------------------|---------------------------|-----------------------------|---------------------------|
| **per 5 months** | **per 30 months** | **per 5 years** | **per 10 years** | **per 5 years** | **per 10 years** |
| Tube 5 | UVB (300-320 nm) | 0.12 | 0.59 | 7.05 | 70.51 |
| | UVA (320-400 nm) | 1.60 | 8.01 | 96.10 | 961.03 |
| | Blue (400-470 nm) | 4.61 | 23.03 | 276.41 | 2764.09 |
| Tube 9 | UVB (300-320 nm) | 0.010 | 0.05 | 0.58 | 5.78 |
| | UVA (320-400 nm) | 0.013 | 0.06 | 0.76 | 7.62 |
| | Blue (400-470 nm) | 2.745 | 13.72 | 164.69 | 1646.94 |

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a For protocol, see Terman et al. (1995).
b For protocol, see Eastman et al. (1995).
light attenuation and side shields. Furthermore, we propose the identification of patients at risk as determined by ophthalmological consultation, because eyes affected by a preexisting disease may be more susceptible than healthy eyes to light induced changes. Finally, consultation and follow-up should be sought for patients likely to use light treatment for extended periods in their lives.

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References


Fig. 2. Spectral transmission curves of selected plastic (a) and Plexiglas (b) diffusing screen materials. Transmission was measured in individual samples with a spectrophotometer in 1 nm steps, and expressed as percent transmission. Distinct differences in the transmission of UVA and blue light are apparent, which underscores the importance of standardization not only of fluorescent lamps but also the interposed screens. The "See-More" filter represents an ideal transmission property for ocular safety, but as yet has been applied only to eyeglass lenses.

e xtended time periods. Patients taking photosensitizing medication and/or suffering from preexisting ocular disease would be in considerable danger if exposed to UV-emitting lamps with insufficient or absent diffusing screens. In general, because scattering is higher in the UV range than in the visible range of the spectrum, the use of a diffusing screen reduces the risk of radiation hazard. On the other hand, there is a drastic increase of the UV dose with decreasing distance from the screen and light source.

Conclusion

We propose that the field develop lamp and treatment standards not only to facilitate controlled studies but also to prevent overexposure to damaging radiation by uncontrolled spectral characteristics of lamps. This can be accomplished by modification of the spectral output of the lamps and use of a plastic diffusing screen, or untinted eyeglasses with UV blockers, blue
A Placebo-Controlled Study of Sertraline in the treatment of Outpatients with Seasonal Affective Disorder

The following is a summary of findings from a study of Sertraline in winter depression. The findings were presented by Adam Moscovitch, principle investigator of the study, in his review of pharmacotherapy of SAD at last June’s meeting in Vancouver. The findings have been published previously in abstract form.

Introduction

The pathophysiology of SAD is thought to involve altered 5-hydroxytryptamine (5-HT) neurotransmission (Wirz-Justice et al., 1996; Neumeister et al., In Press; Parton et al., 1994), a proposition supported by the efficacy of a number of serotonin enhancing agents as treatment of SAD (O’Rourke et al., 1989; McGrath et al., 1990; Dilsaver et al., 1992; Linjaerde et al., 1993; Ruhrmann et al., 1993; Lam et al., 1996). Sertraline is a potent and selective serotonin reuptake inhibitor with little or no affinity for other neurotransmitter receptors (Koe et al., 1983). In view of the atypical symptom profile in SAD, sertraline’s lack of sedation and tendency not to cause weight gain may be particularly advantageous features of the medication. The objective of this study was to evaluate the efficacy and tolerability of Sertraline therapy in the treatment of SAD.

Hamilton and 10 or more on the eight SIGH-SAD supplementary items – a total 29-item SIGH-SAD score of 22 – at baseline and after a one to two week single-blind placebo washout. After washout, subjects whose scores were still at least 75% of baseline in both subscores were randomized. Dosing began with one 50mg Sertraline or placebo pill and were titrated upwards at two-week intervals according to clinical need to a maximum of 4 pills (200 mg Sertraline or placebo), taken once daily, over an 8-week study period.

Primary efficacy analysis was performed on an intention-to-treat (ITT) basis, including all patients who had at least one post-randomization assessment. An analysis of patients completing the 8 weeks was also performed (Completers). Between-group differences in depression severity change during treatment were assessed with ANCOVA, using baseline score as covariate. Within-group changes during treatment were also evaluated using least square methods from ANCOVA models.

Mean Depression Severity Scores for End Point (ITT) Population and at Week Eight (Completers)*

<table>
<thead>
<tr>
<th>END POINT GROUP (ITT)</th>
<th>COMPLETERS</th>
</tr>
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<tbody>
<tr>
<td></td>
<td>SIGH-SAD</td>
</tr>
<tr>
<td>Baseline</td>
<td>36.3</td>
</tr>
<tr>
<td>SERTRALINE Post-Rx</td>
<td>17.5</td>
</tr>
<tr>
<td>Between-Group Difference</td>
<td>p=.02</td>
</tr>
<tr>
<td>Baseline</td>
<td>35.4</td>
</tr>
<tr>
<td>PLACEBO Post-Rx</td>
<td>20.2</td>
</tr>
<tr>
<td></td>
<td>36.3</td>
</tr>
<tr>
<td></td>
<td>15.7</td>
</tr>
<tr>
<td></td>
<td>p=.06</td>
</tr>
<tr>
<td></td>
<td>35.4</td>
</tr>
<tr>
<td></td>
<td>16.8</td>
</tr>
</tbody>
</table>

*Within-group changes from baseline to post-treatment were significant to p<.001 level within active drug and placebo groups and among both ITT and completer populations.

Methods

This was an eight week randomized, double-blind, placebo-controlled, parallel-group study conducted at 28 sites in Austria, Canada, Finland, France, and the United Kingdom. Subjects were studied between November 1991 and May 1994. Outpatients 18 years or older with DSM-III-R seasonal pattern of recurrent depression (Unipolar, Bipolar, or NOS) were studied. To enter, subjects were required to score 12 or more on the 21-item

Results

204 subjects were enrolled in the study, of which 17 were withdrawn prior to randomization for various reasons, including 9 patients (4.4%) considered placebo responders during single-blind washout. Of the 186 subjects randomized, 148 (79.6%) completed the full eight week study. Subjects were mostly female (77%), mean age 40 ± 11 years, with mainly recurrent unipolar
major depression (84%). Drug and placebo groups did not differ significantly on any of these variables.

Mean duration of treatment in both groups was 52 ± 13.5 days. Final daily dose of study medication was 111 ± 45 mg (mean) and 100 mg (mode) for Sertraline and 2.6 ± 1 pill (mean) and 3 pills (mode) for placebo.

Depression severity ratings and their change during treatment are shown in the Table. Significant differences between drug and placebo were seen in HAM-D scores among both End Point (ITT) and Completer populations. The between group difference at eight weeks for 29-item SIGH-SAD showed a trend toward significance. Though not denoted in the table, for both Completer and End-Point populations, Sertraline and placebo groups each showed significant within-group improvement during treatment, to p<0.001 levels.

Sertraline subjects demonstrated a mean weight loss at end point of 1.08 ± 2.53 kg compared to a slight weight gain of 0.06 ± 1.43 kg among placebo subjects (p=0.002).

Adverse events reported with statistically significant greater frequency among active drug compared to placebo subjects include nausea (35.5% vs. 8.5%, respectively), insomnia (25% vs. 11%), diarrhea (19% vs. 5%), dry mouth (13% vs. 2%), anorexia (6.5% vs. 1%), and vomiting (6.5% vs. 0%). Subjects withdrawn for reason of side effects numbered 10 (10.9%) and 4 (4.3%) in the Sertraline and placebo groups, respectively. Subjects withdrawn for inadequate treatment response numbered 3 (3.2%) and 14 (14.9%) among Sertraline and placebo, respectively (p<.01).

Discussion

The finding demonstrate that Sertraline possesses efficacy that is significantly superior to placebo in the treatment of SAD. Clear-cut and significant improvement over time was also observed within among placebo subjects, an effect that may have been partially attributable to lengthening daylight over a relatively long, 8-week study period among patients in whom spontaneous remission is expected by definition.

References


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10th Annual Meeting of the
Society for Light Treatment and Biological Rhythms
Combined with the Society for Research on Biological Rhythms
May 6-10, 1998—Amelia Island Plantation, Florida

Preliminary Program

Three Parallel Sessions for Symposia, Workshops, Slide Sessions.

WEDNESDAY, MAY 6 SRBR
12:00 – 18:00 Registration SRBR
19:30 – 22:00 Opening Reception

THURSDAY, MAY 7 SRBR
8:30 – 10:30 SYMPOSIA
• Organization of the SCN
  Chair: R. Silver
• Genetic Dissection of Clocks: A Quarter Century of Progress
  Chair: J. Dunlap
• Circadian Pacemakers: Essential or Merely Convenient?
  Chair: I. Zucker

10:30 – 11:00 Coffee Break
11:00 – 13:00 SLIDE SESSIONS SRBR

14:30 – 16:30 TUTORIALS
• The Three P’s Primer on Oscillators: Period, Phase and Perturbation
  W. O. Friesen
• Circadian Rhythm Data Analysis
  E. N. Brown

REVIEWS LECTURES
• B. K. Follert
  The Photoregulatory Clock: From Theory Through Formal Experiments to Physiology
• K. K. Siwicki
  How Flies Time

16:30 – 18:30 WORKSHOPS
• Do Non-Photic Stimuli Actually Participate in Normal Entrainment, and, If So, How?
  Discussion Leader: R. Y. Moore
• Identifying and Assembling Clock Parts: Criteria, Candidates and Claptrap
  Discussion Leader: M. Zatz
• Interactions Between Sleep and Circadian Rhythms
  Discussion Leader: F. W. Turek

FRIDAY, MAY 8 SLTBR Officially Begins
8:30 – 10:30 SYMPOSIA
• By the Dawn’s Early Light: Effects of Twilight on Entrainment
  Chair: A. Wirz-Justice
• Mechanisms of Rhythmic Transcription
  Chair: P. Hardin
• Evolutionary and Comparative Studies of Circadian Systems
  Chair: C. H. Johnson

10:30 – 11:00 Coffee Break
11:00 – 13:00 SLIDE SESSIONS SRBR/SLTBR
15:30 – 16:30 SLTBR BOARD MEETING
16:30 – 18:30 WORKSHOPS
• Chronobiologic Studies of Melatonin in Humans
  Discussion Leader: A. J. Lewy
• What Value Should We Place on Human Tau?
  Discussion Leaders: T. H. Monk and S. S. Campell
• Pacemaker Cells in Insects
  Discussion Leader: D. Saunders
• Why the Cultured Retina Should Replace the SCN as the In Vitro Model of Choice
  Discussion Leader: M. Menaker

18:30 – 19:30 SRBR BUSINESS MEETING
20:00 – POSTER SESSION SRBR/SLTBR

SATURDAY, MAY 9
8:30 – 10:30 SYMPOSIA
• The Impact of Blindness on Human Circadian Rhythms
  Co-chairs: J. Arendt and A. J. Lewy
• Molecular and Cellular Interactions in the SCN
  Chair: S. M. Reppert
• The Role of Clocks in Photoperiodic Flower Initiation
  Chair: C. R. McClung

10:30 – 11:00 Coffee Break
11:00 – 13:00 SLIDE SESSIONS SRBR/SLTBR
16:30 – 18:00 PITTENDRIGH MEMORIAL LECTURE
  S. Daan
18:00 – 19:00 SLTBR BUSINESS MEETING
20:00 – COMBINED BANQUET SRBR/SLTBR
10th Annual Meeting

Preliminary Program

(continued)

SUNDAY, MAY 10

8:30 – 10:30 SLTBR ROUND TABLE

Invited discussants from the Boards of SLTBR, SRBR and SRS. The theme of this Round Table will include an overview of a decade of SLTBR achievements, a discussion on the future roles of the three Societies (where do they overlap, where are they complementary); problems of standards, and the future of clinical chronobiology, particularly light and melatonin.

Chairs: Anna Wirz-Justice (SLTBR), Mary Carskadon (SRS), Larry Morin (SRBR)

Invited participants:

SLTBR: Ray Lam, Al Lewy, Dan Oren, Norm Rosenthal, Michael Terman
SRS: Mary Carskadon, Chuck Czeisler, Dave Dinges, Tim Roehrs
SRBR: Jay Dunlap, Fred Turek
ISC: Rae Silver, Jim Waterhouse and: Christine Acebo, Jo Arendt, Stuart Armstrong, Scott Campbell, Charmaine Eastman, Dan Kripke, Masako Okawa, Eve van Cauter, Tom Wehr

TOPICS

Introduction: a history of light therapy (Norm Rosenthal)
A decade of SLTBR: Why it began and what it has achieved (Michael Terman)
Light therapy around the world: how it is accepted and used
Stuart Armstrong (Australia), Bengt Kjellman (Scandinavia), Wilfried Köhler (Germany), Ray Lam (Canada), Alexander Neumeister (Austria), Masako Okawa (Japan), Dan Oren (USA), Anna Wirz-Justice (Switzerland).
Summary of new applications.
Light in circadian sleep disorders (Scott Campbell)
The reality of applications in shift work (Charmaine Eastman)
Melatonin overview (Jo Arendt)
The future of applied chronobiology
Scenario 1 Al Lewy SLTBR
Scenario 2 Dave Dinges SRS
Scenario 3 Fred Turek SRBR
with Mary Carskadon, Chuck Czeisler, Dan Kripke, Tim Roehrs, Eve van Cauter, Tom Wehr
Functional issues (suggested themes)
- Coordination of teaching issues (diagnosis/therapy)
- Consensus statements
- LTBR Bulletin – a newsletter for the SRBR as well! To provide in-between-meetings information.
- Public/patient brochures on light therapy, melatonin
- Standards, e.g. with respect to medical devices (lamps) or melatonin (what else is in those pills?)
- Patents
- Publicity

10:30 – 11:00 Coffee Break

END OF MEETING
HIGH Instruments Revised

Namni Goel, PhD

The Hypomania Interview Guide (Including Hyperthymia) — Retrospective Assessment Version (HIGH-R), Current Assessment Version (HIGH-C), and Current Assessment Self-Rating Version (HIGH-C-SR) — which are available as part of SLTBR's SAD Clinical Assessment Tools Packet — have been revised. The HIGH-R is the successor to the original instrument, the Hypomania Interview Guide (Including Hyperthymia), for Seasonal Affective Disorder (HIGH-SAD). As a result of the absence of a statistically valid conversion scale to compare HIGH-SAD and HIGH-R total scores, the revised editions eliminate the first edition’s “HIGH-SAD approximate score,” otherwise preserving their original format.

The HIGH-R retrospectively rates the frequency and severity of DSM diagnostic criterion features (e.g., elevated mood, flight of ideas, rapid speech) as well as several non-DSM features (e.g., sharpened thinking, increased energy) for spring/summer mood states that recur seasonally in alternation with winter depression. The HIGH-R has demonstrated validity for assessing non-depressed mood states in SAD patients with DSM-IV diagnoses of Recurrent Bipolar Disorder (I, II) or Recurrent Major Depressive Disorder (MDD; unipolar), both with Seasonal Pattern (Goel et al., 1997). The instrument has high internal consistency. Unipolar patients have lower total scores than bipolar patients, with total score classifying 85% of SAD patients into the correct unipolar or bipolar group. Based on total score, MDD patients can be divided into 3 subgroups: euthymes (normal mood), hyperthymes (slightly elevated mood) and high-hyperthymes (scores overlapping with hypomania). For boundary mood cases, small subsets of non-depressed mood features provide better classification accuracy. With the exception of sharpened thinking, DSM items dominate patient classifications. In addition, distinct clusters of “positive” (pleasant, agreeable) or “negative” (impairing) features describe the various mood states. Thus, the HIGH-R is useful for discriminating and classifying hypomania and mania in bipolar patients, and euthymia and hypothympnia in unipolar patients.

In addition to the HIGH family of scales, the new assessment packet includes the latest (1994) versions of Structured Interview Guide for the Hamilton Depression Rating Scale — Seasonal Affective Disorder Version (SIGH-SAD) and Self-Rating Version (SIGH-SAD-SR), with scoring and interpretation guides. Packets, with masters formatted for copying, are available $20; members; $40, non-members) from SLTBR, 10200 W 44th Ave Ste 304, Wheat Ridge CO 80033-2840; Phone 303-422-3697; FAX 303-422-8894; e-mail csdb@resourcener.com.

References


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Journal of Alternative and Complementary Medicine

This peer-reviewed journal, now in its third year, offers an opportunity for SLTBR members to communicate their results and perspectives to a very wide audience interested in nonpharmacological treatments. The journal was founded to promote rigorous scientific evaluation in an oft-questioned but active field. Editors are Kim Jobst (Editor-in-Chief, University of Oxford), Fredi Kronenberg (Associate Editor, Columbia University) and Jackie Wootten (Managing Editor, jackiew@clarknet.net), who also serves as Informatics Project Director at the Rosenthal Center for Alternative and Complementary Medicine at Columbia. JACM is listed by Index Medicus, MEDLINE, and the British Library. Review and publication are prompt. The journal has published David Schlag's provocative review of SLTBR's Seventh Annual Meeting (1995; 1:107-109), Michael and Juan Su Terman's report on the antidepressant effects of high-dose negative air ionization (1995:1:87-92), and Katherine Rex, Daniel Kripke, Roger Cole and Melville Klauber's case report on nocturnal light and menstrual cycle length (1997:3:387-390). As a member of the JACM editorial board, Michael Terman urges SLTBR members to contribute reports on novel applications of light therapy (beyond SAD and sleep-phase disorders), and combination of light therapy with other interventions. Members are invited to discuss potential submissions (Michael Terman, PhD, Dept. of Psychiatry, Columbia University, 722 West 168th Street, Unit 50, New York, NY 10032; tel 212-543-5712; fax -5184; mt12@columbia.edu).

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**Society for Light Treatment and Biological Rhythms**

**Publications available to our members**

These publications are available through our executive office on a prepaid basis (U.S. funds only). Please include your check payable to the SLTBR or include your Visa/MasterCard information with your order. Mail this form with your payment to SLTBR at the address on the back of this form.

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