Seasonal Affective Disorder

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Raymond W. Lam, M.D.

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Summary

Recent research on seasonal affective disorder (SAD) has refined the diagnostic criteria and highlighted the heterogeneous nature of the disorder. Recent light therapy studies show improved methodology, but some negative studies add to the controversy over non-specific (placebo) effects of light exposure. Several psychobiologic studies generated interesting data, but the pathophysiology of SAD remains elusive and unconfirmed.

Introduction

The decade since the publication of Rosenthal et al's 1984 paper [1] defining seasonal affective disorder (SAD) has seen remarkable research interest into the seasonality of mood disorders and treatment with bright light. For example, Medline has included a separate Medical Subject Heading (MeSH) for seasonal affective disorder, and over 100 articles have been indexed since-1991 alone. In the past year, publications have focused on the diagnosis, symptomatology, epidemiology, and light therapy of SAD.

Symptoms And Diagnosis

The validity of SAD as a diagnosis has received attention because of the impending publication of the Diagnostic and Statistical Manual of Mental Disorders, 4th edition (DSM-IV) [2,3*]. Draft diagnostic criteria for seasonal pattern (Table 1) were revised according to empiric data and consensus in the field [4*]. SAD continues to be classified as a 'seasonal pattern' for unipolar and bipolar mood disorders, and is included as one of several 'course specifiers' of the major mood disorders (along with rapid cycling and post partum).

Although SAD is defined by the pattern of depressive episodes in DSM-IV, Allen et al [5**] confirmed previous reports that so called atypical depressive symptoms of hypersomnia, hyperphagia, and weight gain were more frequently found in SAD patients compared to matched nonseasonal patients. They also determined family psychiatric history in first-degree relatives using the Family History Method. The genetic loading for mood disorders (of unspecified seasonality) was similar for both seasonal and nonseasonal patients, but the SAD patients were more likely to have alcoholism in their families.

The similarity of symptoms between SAD and atypical depression prompted studies of the clinical overlap between the two diagnoses. Pande et al [6] found high seasonality scores in 30 patients with defined atypical depression: 63% had seasonality scores in the range of SAD or 'subsyndromal' SAD. Conversely, Terman and Stewart [7] also found high rates of cardinal symptoms of atypical depression (mood reactivity and rejection

sensitivity) in a cohort of SAD patients. Previous attempts at treating nonseasonal patients with atypical depression with light therapy were negative [8], but these reports suggest further study is indicated.

Epidemiology

Several studies using different methodologies shed light on the prevalence of SAD. In Alaska, a study using a screening questionnaire estimated a prevalence of 9.2% for SAD [9]. The prevalence in Alaska is similar to those reported, using the same instrument and criteria, at much lower latitudes [10]. One plausible explanation is a 'ceiling effect' with prevalence rates reaching a plateau above a certain latitude. Other studies examined general clinic populations using prospectively recorded data on depression onset and course, thereby avoiding potential selection bias in assessing seasonality. Williams and Schmidt [11] reported that 20% of patients treated for recurrent depression at a northern Canadian Mental Health Center (latitude 54 to 60 degrees) met operational criteria for winter depression. They also noted that this was no higher than data reported at lower latitudes. Using retrospective data from a research-oriented psychiatric clinic in Italy, Faedda and associates [12**] reported that 9.7% of patients with mood disorders had a seasonal pattern of recurrence. Interestingly, the seasonal patients were evenly divided between spring/summer and autumn/winter depression patterns. Finally, a large scale longitudinal epidemiologic study of young adults in Zurich identified 10.4% of the sample as having SAD, defined as at least two consecutive years of seasonal depression in the three year study period [13*]. Because self-reports of seasonality were unreliable between the two assessments, the investigators suggested that the diagnosis of SAD should not rely solely on patient self-report, but should also include prospective follow-up or collateral information.

Treatment of SAD

Treatment studies of light therapy have shown increasingly rigorous methodology with larger sample sizes, less diagnostic heterogeneity, longer treatment periods, and parallel

instead of crossover designs. Wavelength of light used in light therapy was examined in two studies. In one study, the ultraviolet (UV) spectrum did not add to the therapeutic efficacy of light therapy [14*]. Because of the potential harmful effects of long-term W exposure, light therapy devices should have W filters that block wavelengths below 400 nm. In a comparison light box study, cool-white fluorescent lights were as effective as full-spectrum fluorescent lights [15], adding evidence to other studies showing that various light sources (including incandescent lights) are effective for treating SAD.

Devices other than light boxes were also studied for light therapy. Two recent studies, with the largest sample sizes in light therapy studies to date, used a light visor [16*,17*]. In both studies, there was no relationship between the intensity of light and various measures of response to treatment, despite the fact that very low intensity light (60 lux) was used. This contrasts to most light box studies where a dear intensity-response relationship is found. Several explanations may explain this discrepancy. The proximity of the visor light source to the eye may increase the amount of light that reaches the retina, as compared to a light box. Lux, a unit of illumination, may also not be the best measure of the biologic or therapeutic effect of light. There is increasing evidence that even low illumination can affect biologic parameters [18], so that for some patients, light as low as 100 lux may be therapeutically effective. Finally, although the response rate was high in both studies (over 60% by strictly defined criteria), a non-specific (placebo) effect of light therapy must also be considered. In this regard, a light box study by Eastman and associates [19**] using a non-light control condition (a negative ion generator that, unknown to subjects, was turned off), found no differences between the control condition and bright light treatment (7000 lux for 1 hour in the morning). However, the response rate for the bright light condition (29%) was unusually low compared to other treatment studies. The selection criteria and unusually sunny weather during
the course of their study may have excluded more light-responsive patients. Thus, the issue of placebo effects in light therapy remains unresolved.

The Seattle group conducted a series of studies investigating dawn simulation in SAD [20,21,22*]. Dawn simulation uses a device that gradually increases illumination

exposure, while the patient is sleeping, to simulate a summer dawn during the winter. Significant improvement occurred using dawn simulation compared to various control conditions, despite a final illumination as low as 250 lux.

Two groups studying predictors for light therapy found that hypersomnia and hyperphagia predicted clinical response [23*,24,25*]. Another study, however, reported that only high consumption of sweets in the latter half of the day predicted response to treatment [26*]. Of interest is that prospective measures of sleep and eating were used in the latter study, whereas the other studies used global patient self-report.

Light therapy has been considered a rather benign treatment with few side effects. A systematic report of side effects to light therapy using a light visor showed that approximately 20% of patients reported mild side effects, including headache, eyestrain, and "feeling wired" [27]. A more controversial topic is the potential for prolonged bright light exposure to produce harmful effects on the retina. The intensities of light used in light therapy regimens are not considered harmful to the human retina based on short term studies, but the retinal effects of long term bright light exposure are not known. Some investigators have called for routine ophthalmologic evaluation prior to starting light therapy because of the small, potential risk of aggravating previously unrecognized retinal conditions (e.g. macular degeneration) [28*]. Others suggest ophthalmologic screening only in patients with a history of pre-existing retinal disease, patients taking highly photosensitizing drugs, and the elderly [29*]. Empiric data are still sparse, but a recently reported five-year prospective study of patients on chronic light therapy has not shown any significant clinical or electrophysiologic changes in the eyes [30].

Finally, antidepressant drugs are also being studied in SAD. An open study showed efficacy of bupropion in treating SAD [31*]. One case-study suggested that citaloprim, a selective serotonin reuptake inhibitor, was as effective as light therapy [321. Fluoxetine was reported to be as effective as light therapy for SAD [33*], and results from at least two double-blind studies of serotonin reuptake inhibitors in SAD will soon be available. What remains a question is whether a combination of medications and light therapy is more effective than either alone.

Psychobiology of SAD

Despite the heuristic appeal of a circadian hypothesis for SAD, there are as yet no consistently replicated data to support abnormal circadian rhythms as an etiology for SAD or for the therapeutic effects of light [34]. One study did not find abnormalities of CSF norepinephrine, serotonin, or dopamine activity [35*] in SAD patients, although other studies have shown changes in peripheral noradrenergic measures [36]. Using a challenge paradigm, the NIMH group have shown consistently different behavioural responses to the serotonergic agent, m-CPP, in SAD patients compared to matched controls [37*,38*], suggesting that serotonin dysregulation is a fruitful area for further study.

Retinal mechanisms have been proposed as an etiology of SAD. Using flash electroretinography, Lam et al [39*] found reduced b-wave amplitudes in female SAD patients, but not in males. Ozaki et al [40**] replicated the finding of reduced electrooculographic (EOG) ratios in SAD. These results support a hypothesis of reduced retinal light sensitivity in SAD [41]. In contrast, Oren et al [42*] did not find differences in a number of different electrophysiologic measures of ophthalmologic function.

In preliminary brain imaging studies, Cohen et al [43*] studied seven winter SAD patients with positron emission tomography (PET) and found abnormalities in the prefrontal and parietal cortex areas. In another PET study of nine patients with a summer pattern of SAD, abnormalities were noted in the orbital frontal cortex and in the left inferior parietal lobule [44]. These interesting findings will need replication in a larger sample of subjects.

Conclusions

When SAD was first investigated there was hope that the apparent link between an animal model (photoperiodism) and treatment (with bright light exposure) would soon

lead to confirmation of an underlying etiology for winter depression. The past decade of research has shown SAD to be more complicated and heterogeneous than first thought, and the mechanism of light therapy is proving as elusive as that of antidepressant medications. Further research is therefore needed to determine the pathophysiology of SAD and the specific effects of light therapy.

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1. Rosenthal NE, Sack DA, Gillin JC, Lewy AJ, Goodwin FK, Davenport Y, Mueller PS, Newsome DA, Wehr TA: Seasonal Affective Disorder: A Description of the Syndrome and Preliminary Findings with Light Therapy. Arch Gen Psychiatry 1984; 41:72--80.

2. Bauer MS: Defining Seasonal Affective Disorder(s). Biol Psychiatry 1992, 31:1185--1189 [published erratum appears in Biol Psychiatry 1992, 32: 1062].

**3. Bauer MS, Dunner D: Validity of Seasonal Pattern as a Modifier for Recurrent Mood Disorders in DSM-IV. Compr Psychiatry, 1993, in press.

An excellent review of the issues relating to validating the diagnosis of SAD. Specifically, the authors discuss the limitations of past SAD studies, e.g., the recruitment of subjects, the use of self-report questionnaire data, the brief duration (1-2 weeks) of light therapy studies, the lack of comparison studies with traditional antidepressants, and the spectrum of disorder issue (is SAD a distinct clinical entity, or is it better conceptualized as a dimension similar to neuroticism?).

*4. Task Force on DSM-IV: DSM-IV Draft Criteria. Washington DC, American Psychiatric Association, 1993.

Revisions of DSM-III-R criteria for SAD as a 'seasonal pattern' of recurrent mood disorders. Likely to be published without change in the DSM-IV in September, 1993.

**5. Allen JM, Lam RW, Remick RA, Sadovnick AD: Depressive Symptoms and Family History in Seasonal and Nonseasonal Mood Disorders. Am J Psychiatry 1993, 150:443--448.

A case-control study showing that SAD patients are more likely to have atypical symptoms of depression compared to matched nonseasonal patients, although the genetic loading for mood disorders in both groups was similar.

6. Pande AC, Haskett RF, Greden JF: Seasonality in Atypical Depression. Biol Psychiatry 1992, 31:965 967.

7. Terman M, Stewart JW: Is Seasonal Affective Disorder a Variant of Atypical Depression. IL Diagnostic Similarities [Abstract]. Abstracts of the 5th Annual Meeting of the Society of Light Treatment and Biological Rhythms, San Diego, June, 1993, p.21.

8. Stewart JW, Quitkin FM, Terman M, Terman JS: Is Seasonal Affective Disorder a Variant of Atypical Depression? Differential Response to Light Therapy. Psychiatry Res 1990; 33:121-128.

9. Booker JM, Hellekson CJ: Prevalence of Seasonal Affective Disorder in Alaska. Am J Psychiatry 1992, 149: 1176--1182.

10. Rosen LN, Targum SD, Terman M, Bryant MT, Hoffman H, Kasper SF, Hamovit 1R, Docherty JP, Welch B, Rosenthal NE: Prevalence of Seasonal Affective Disorder at Four Latitudes. Psychiatry Res 1990; 31:131--144.

11. Williams RJ, Schmidt GG: Frequency of Seasonal Affective Disorder Among Individuals Seeking Treatment at a Northern Canadian Mental Health Center. Psychiatry Res 1993; 46:41--45.

**12. Faedda GL, Tondo L, Teicher MH, Baldessarini RJ, Gelbard HA, Floris GF: Seasonal Mood Disorders. Patterns of Seasonal Recurrence in Mania and Depression. Arch Gen Psychiatry 1993, 50:17--23.

This study applied DSM-III-R criteria retrospectively to a large sample (N=2381) of outpatients with mood disorders followed in a research clinic in Italy during a period prior to the popularization of SAD. 9.7% of patients met criteria for seasonal pattern; the characteristics of this sample are described and compared to other studies.

*13. Wicki W, Angst J, Merikangas KR: The Zurich Study. XIV. Epidemiology of Seasonal Depression.

An epidemiologic study of 417 subjects, age 27-28 years, interviewed twice over a three year period. Significant seasonal patterns were found in depression and in other disorders, with 2.4% of the group having consistent winter depressions and 10.4% reporting at least two consecutive seasonal episodes. The self-reports of seasonality, however, had a Kappa of only 0.25 between interviews, indicating a low level of agreement.

*14. Lam RW, Buchanan A, Mador JA, Corral MR, Remick RA: The Effects of Ultraviolet A Wavelengths in Light Therapy for Seasonal Depression. J Affect Dis 1992, 24:237--243.

Thirty three SAD patients were randomized to light therapy with or without UV-A exposure. No difference in response was found between conditions, indicating that UV was not critical for effective light therapy. The investigators recommended that light therapy devices should screen out UV wavelengths.

15. Bielski RJ, Mayor J, Rice J: Phototherapy with Broad Spectrum White Fluorescent Light: A Comparative Study. Psychiatry Res 1992, 43:167--175.

This cross-over study found no differences in the response of 11 SAD patients treated with full-spectrum or cool-white fluorescent lights; both types of lights significantly improved symptoms.

*16. Joffe RT, Moul DE, Lam RW, Levitt AJ, Teicher MH, Lebegue B, Oren DA, Buchanan A, Glod CA, Murray MG, Brown J, Schwartz P: Light Visor Treatment for Seasonal Affective Disorder. Psychiatry Res 1993; 46:29--39.

The largest treatment study to date, with 105 subjects randomized to three intensities of light using a light visor in a parallel design. Results did not show any differences between 3200, 600, and 60 lux visors, although the response rate was high in all three conditions.

*17. Rosenthal NE, Moul DE, Hellekson CJ, Oren DA, Frank A, Brainard GC, Murray MG, Wehr TA: A Multicenter Study of the Light Visor for Seasonal Affective Disorder No Differences in Efficacy Found Between Two Different Intensities. Neuropsychopharmacology 1993, 8:151-160.

This light visor study randomized 55 patients to intensities of 6000 lux and 400 lux in a parallel design. No significant differences were noted in clinical response, with patients responding well to both conditions. However, relapse occurred more often in patients withdrawn from the 400 lux visor.

18. Lewy AJ, Saeeduddin A, Bauer V, Cutler N, Waldman M, Sack R: Circadian Phase Shifting Effects of "Dim" Green Light [Abstract]. Abstracts of the 5th Annual Meeting of the Society of Light treatment and Biological Rhythms, San Diego, June, 1993, p.11.

**19. Eastman CI, Lahmeyer HW, Watell LG, Good GD, Young MA: A Placebo-Controlled Trial of Light Treatment for Winter Depression. J Affect Dis 1992, 26:211--221.

A well-designed negative study of light therapy in 32 patients using a non-light placebo (an inactivated negative ion generator). Both conditions significantly reduced depression scores with no differences between conditions.

20. Avery D, Bolte MA, Millet M: Bright Dawn Simulation Compared with Bright Morning Light in the Treatment of Winter Depression. Acta Psychiatr Scand 1992, 85:430--434.

21. Avery DH, Bolte MA, Cohen S, Millet MS: Gradual Versus Rapid Dawn Simulation Treatment of Winter Depression. J Clin Psychiatry 1992, 53:359--363.

*22. Avery DH, Bolte MA, Dager SR, Wilson LG, Weyer M, Cox GB, Dunner DL: Dawn Simulation Treatment of Winter Depression: A Controlled Study. Am J Psychiatry 1993, 150:113--117.

One of several reports by the Seattle group on the efficacy of dawn simulation. In this parallel design study, 22 patients were treated with either a 2-hour dawn signal peaking at 250 lux (active condition) or a 30-minute dawn signal peaking at 0.2 lux (control condition). Depression scores were significantly lower after one week of the 'summer' dawn compared to the control dawn.

*23. Oren DA, Jacobsen FM, Wehr TA, Cameron CL, Rosenthal NE: Predictors of Response to Phototherapy in Seasonal Affective Disorder. Compr Psychiatry 1992, 33:111--114 [published erratum appears in Psychiatry 1992, 33:419].

The NIMH group combined data from several studies of different light therapy regimens, and found that hypersomnia, hyperphagia, and suicidality predicted clinical response to light in 44 female patients.

24. Lam RW, Buchanan A, Mador JA, Corral MR: Bypersomnia and Morning Light Therapy for Winter Depression. Biol Psychiatry 1992, 31: 1062--1064.

*25. Lam RW: Morning Light Therapy for Winter Depression: Predictors of Response. Acta Psychiatr Scand 1994, in press.

Regression analysis of 43 SAD patients treated with 2500 lux x 2 hours in the early morning found that hypersomnia, hyperphagia, and younger age predicted response.

*26. Krauchi K, Wirz-Justice A, Graw P: High Intake of Sweets Late in the Day Predicts a Rapid and Persistent Response to Light Therapy in Winter Depression. Psychiatry Res 1993, 46:107--117.

This study found that only hyperphagia, in the form of increased eating of sweets in the afternoon and evening, was a consistent and significant predictor of response to light in 51 SAD patients. This study used prospective ratings of hyperphagia and hypersomnia

(daily sleep logs and eating questionnaires) instead of the global ratings used in the previous studies.

27. Levitt AJ, Joffe RT, Moul DE, Lam RW, Teicher MH, Lebegue B, Murray MG, Oren DA, Schwartz P, Buchanan A, Glod CA, Brown J: Side Effects of Phototherapy in Seasonal Affective Disorder. Am J Psychiatry 1993, 150:650--652.

*28. Reme CE, Terman M: Does Light Therapy Present an Ocular Hazard? [Letter]. Am J Psychiatry 1992; 149:1762--1763.

This letter presents an argument for mandatory ophthalmologic assessment prior to light therapy.

*29. Waxler M, James RH, Brainard GC, Moul DE, Oren DA, Rosenthal NE: Retinopathy and Bright Light Therapy [Letter]. Am J Psychiatry 1992, 149: 1610--1611.

This letter argues for pre-light therapy ophthalmologic assessment only upon a positive history of risk factors for phototoxicity.

30. Gorman CP, Wyse PH, Demjen S, Caldwell LH, Chorney MY, Samek NP: Ophthalmological Profile of 71 SAD Patients: A Significant Correlation Between Myopia and SAD Abstract . Abstracts of the 5th Annual Meeting of the Society of Light Treatment and Biological Rhythms, San Diego, June, 1993, p.8.

*31. Dilsaver SC, Qamar AB, Del Medico VJ: The Efficacy of Bupropion in Winter Depression: Results of an Open Trial. J Clin Psychiatry 1992, 53:252--255.

A case series of 15 patients treated in an open trial with this novel antidepressant. Two-thirds of the patients had an excellent response to bupropion, while the rest had partial improvement.

32. Wirz-Justice A, van der Velde P, Bucher A, Nil R: Comparison of Light Treatment with Citalopram in Winter Depression: A Longitudinal Single Case Study. Int Clin Psychopharmacol 1992, 7:109--116.

*33. Ruhrmann S, Kasper S, Hawellek B, Maranez B, Hoflich G, Nlskelsen T, Moller H-J: Fluowetine Versus Light Therapy in the Treatment of SAD [Abstract]. Biol Psychiatry 1993, 33:83A.

This German group reported a study comparing fluoxetine plus dim light versus placebo plus bright light, and found both conditions significantly improved depression scores.

34. Eastman CL Gallo LC, Lahmeyer HW, Fogg LF: Searching for Clues to SAD in the Circadian Rhythm of Temperature [Abstract] . Abstracts of the 5th Annual Meeting of the Society of Light Treatment and Biological Rhythms, San Diego, June, 1993, p.6.

35. Rudorfer MV, Skewerer R, Rosenthal NE: Biogenic Amines in Seasonal Affective Disorder - Effects of Light Therapy. Psychiatry Res 1993; 46: 19--28.

Seventeen SAD patients were compared to eight control subjects. No abnormalities in CSF metabolites of norepinephrine, serotonin, or dopamine were found, or in plasma measures of norepinephrine levels. Light therapy also did not alter these measures.

36. Anderson JL, Vasile RG, Mooney n, Bloomingdale KL, Samson JA, Schildkraut JJ: Changes in Norepinephrine Output Following Light Therapy for Fall/Winter Seasonal Depression. Biol Psychiatry 1992, 32:700--704.

37. Joseph-Vanderpool JR, Jacobsen FM, Murphy DL, Hill JL, Rosenthal NE: Seasonal Variation in Behavioral Responses to m-CPP in Patients with Seasonal Affective Disorder and Controls. Biol Psychiatry 1993, 33:496-504.

This study found that the behavioral responses of SAD patients to the serotonergic agonist, m-CPP, were state dependent measures, since they were reversed by light therapy and in the summer remitted state.

*38. Rosenthal NE, Garcia-Borreguero D, Schwartz PJ, Oren DA, Snelbaker A, Ozaki N, Falouji W, Moul DE, Murphy DL: Abnormal Behavioural and Hormonal Responses to m-CPP in SAD [Abstract]. Abstracts of the 5th Annual Meeting of the Society of Light Treatment and Biological Rhythms, San Diego, June, 1993, p.15.

This report by the NIMH group replicated their earlier finding of differences between SAD patients and controls in behavioral and hormonal responses to m-CPP. Their data were also opposite in direction to those seen in nonseasonal depression.

*39. Lam RW, Beattie CW, Buchanan A, Mador JA: Electroretinography in Seasonal Affective Disorder. Psychiatry Res 1992, 43:55--63.

An electrophysiologic study showing that 17 female SAD patients had lower flash ERG b-wave amplitudes than matched controls, suggesting retinal subsensitivity to light, while 6 male SAD patients had higher b-wave amplitudes than their matched controls.

**40. Ozaki N, Rosenthal NE, Moul DE, Schwartz PJ, Oren DA: Effects of Phototherapy on Electrooculographic Ratio in Winter Seasonal Affective Disorder. Psychiatry Res, 1993, in press.

This study also showed low EOG ratios in SAD patients in winter, and found that the EOG ratios remained low after light therapy. Low mean EOG ratio is one of the few psychobiologic findings in SAD to be replicated by a different research group.

41. Reme C, Terman M, Wirz-Justice A: Are Deficient Retinal Photoreceptor Renewal Mechanisms Involved in Pathogenesis of Winter Depression? [Letter]. Arch Gen Psychiatry 1990; 47:878--879.

*42. Oren DA, Moul DE, Schwartz PJ, Alexander JR, Yamada EM, Rosenthal NE: An Investigation of Ophthalmic Function in Winter Seasonal Affective Disorder. Depression 1993; 1:29--37.

This study did not find any abnormalities in SAD patients in a variety of electrophysiologic measures of ophthalmologic function, including dark adaptation, intraocular pressure, pupillary size, color vision, pattern electroretinograms, and pattern evoked potentials.

*43. Cohen RM, Gross M, Nordahl TE, Semple WE, Oren DA, Rosenthal N: Preliminary Data on the Metabolic Brain Pattern of Patients with Winter Seasonal Affective Disorder. Arch Gen Psychiatry 1992, 49:545--552.

The first PET study of SAD, by the NIMH group. Seven SAD patients (5 Bipolar, type II; 2 unipolar) were compared to 38 normal controls; SAD patients showed lower global metabolic rates before and after light therapy. Other positive findings were similar to those reported in bipolar patients not selected for seasonality. The effect of light treatment was seen primarily as increased regional metabolism in the occipital cortex.

44. Goyer PF, Schulz PM, Semple WE, Gross M, Nordahl TE, King AC, Wehr TA, Cohen RM: Cerebral Glucose Metabolism in Patients with Summer Seasonal Affective Disorder. Neuropsychopharmacology 1992, 7:233--240.

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Light Therapy Devices for SAD

Seasonal affective disorder (SAD) is a type of clinical depression that regularly occurs in the winter, with normal mood in the summer. Light therapy is an effective and safe treatment for SAD. Other treatments for depression (for example, medications) are also effective. Self-diagnosis or treatment of SAD is not recommended because there are other medical causes for depressive symptoms, and because light therapy may be harmful to people with certain medical conditions (for example, eye disease). See your doctor first!

Although light therapy is effective for SAD, we still do not fully understand how the light works and what is the best method for light therapy. There are now many light therapy devices available on the market that make claims about light treatment, but light therapy devices are not well regulated in Canada. Therefore, we believe it is wise to be cautious about recommending light therapy devices. We have no financial interest in any light device companies. Our recommendations are based on the following principles: 1) the light device should be tested in scientifically valid studies, 2) the light device should have

a filter that blocks the ultraviolet rays, 3) the light device should be CSA approved in Canada, and 4) the light device company should have a track record of reliability.

We recommend either a 10,000 lux light box or a light visor because they have been extensively tested in scientific studies, and we have experience with these devices. These two devices fullfill all the criteria above. The other light devices marketed, to our knowledge, do not meet all of these criteria.

We currently recommend 3 Devices:

Medic-Light 10,000 Light Box
Medic-Light, Inc.
Yacht Club Drive
Lake Hopatcong, NJ 07849

SunRay I Light Box
SunBox Company
19217 Orbit Drive
Gaithersburg, MD 20879-4149
1-800-548-3968
sunbox@aol.com

Light Visor
BioBrite, Inc.
7315 Wisconsin Avenue, Suite 900E
Bethesda, MD 20814-3202

Canadian Suppliers:

Uplift Technologies, Inc.
Box 102, CRO
Halifax, Nova Scotia
B3J 2L4

CANADA

Toll Free: 1-800-387-0896

Tel: (902) 422-0804

Fax: (902) 422-0798

E-Mail: info@day-lights.com

VitalAire (formerly WinterLites and ARS VitalAire)

#1003, 7495 - 132 Street

Surrey, BC V3W 1J8

604-572-4000

800-663-0794

Health Light Inc.

P.O. Box 3899, Station C

Hamilton, Ont. L8H 7P2

800-265-6020 (Toll free, Canada and USA)

Phone: 905-545-4997

Fax: 905-545-8963

e-mail: healthlight@excite.com

Other Light Device Companies

Apollo BriteLite

352 West 1060 South

Orem, UT 84058

800-545-9667

801-226-2370

Enviro-Med Bio-Light

1600 SE 141st Ave

Vancouver, WA 98683 800-222-DAWN

360-256-6989

Medic-Light, Inc.
Yacht Club Dr.
Lake Hopatcong, NJ 07849
201-663-1214
800-668-2110

Northern Light Technologies
3070 Brabant-Marineau,
St. Laurent, PQ H4S 1K7
514-335-1763

Sunnex Biotechnologies Inc.
Suite 465, 435 Ellice Avenue
Winnipeg, Man. R3B 1Y6
204-956-2476

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Thompson C, Silverstone T (eds): *Seasonal Affective Disorder*. London, CNS (Clinical Neuroscience) Publishers, 1989.

Patient Education

Seasons of the Mind, by Dr. Norman Rosenthal. New York, Bantam Books, 1989, about $13.00.

Winter Depression, by Angela Smyth in consultation with Professor Chris Thompson. London, Unwin Paperbacks, 1990, about 13.00.

The Light Book, by Jane Wegscheider Hyman. Toronto, Random House, 1990, about $7.00.

For Further Information

Society for Light Treatment and Biological Rhythms.
A not-for-profit international organization dedicated to fostering research, professional development, and clinical applications.

Francine Butler, Ph.D., Executive Director
Society for Light Treatment and Biological Rhythms

10200 West 44th Avenue, Suite 304
Wheat Ridge, Colorado, 80033-2840, USA

Telephone: (303) 424-3697
Fax: (303) 422-8894
email: sltbr@resourcenter.com

Address Correspondence to:

Dr. Raymond W. Lam
Department of Psychiatry
Vancouver Hospital and Health Sciences Center, UBC Pavilions
2255 Wesbrook Mall
Vancouver, B.C. V6T 2A1
Tel: (604) 822-7325
Fax: (604) 822-7756
