Seasonal Affective Disorder

STEPHEN J. LURIE, M.D., PH.D., BARBARA GAWINSKI, PH.D., DEBORAH PIERCE, M.D., M.P.H., and SALLY J. ROUSSEAU, M.S.W., *University of Rochester School of Medicine and Dentistry, Rochester, New York*

Patients with seasonal affective disorder have episodes of major depression that tend to recur during specific times of the year, usually in winter. Like major depression, seasonal affective disorder probably is underdiagnosed in primary care settings. Although several screening instruments are available, such screening is unlikely to lead to improved outcomes without personalized and detailed attention to individual symptoms. Physicians should be aware of comorbid factors that could signal a need for further assessment. Specifically, some emerging evidence suggests that seasonal affective disorder may be associated with alcoholism and attention-deficit/hyperactivity disorder. Seasonal affective disorder often can be treated with light therapy, which appears to have a low risk of adverse effects. Light therapy is more effective if administered in the morning. It remains unclear whether light is equivalent to drug therapy, whether drug therapy can augment the effects of light therapy, or whether cognitive behavior therapy is a better treatment choice. (Am Fam Physician 2006;74:1521-24. Copyright © 2006 American Academy of Family Physicians.)

Patient information: A handout on seasonal affective disorder is available at http://familydoctor.org/267.xml.

The *Diagnostic and Statistical Manual of Mental Disorders,* 4th ed., (DSM-IV) categorizes seasonal affective disorder (SAD) not as a unique mood disorder, but as a specifier of major depression.[1] Thus, patients with SAD experience episodes of major depression that tend to recur at specific times of the year. These seasonal episodes may take the form of major depressive or bipolar disorders. *Table 1*[1] lists the DSM-IV criteria for the seasonal pattern specifier.

Epidemiology

The overall lifetime prevalence of SAD ranges from 0 to 9.7 percent.[2] This estimate depends on the specific population studied, as well as whether SAD is diagnosed by a screening questionnaire or a more rigorous clinical interview. In one U.S. study that used DSM-IV–based criteria, the lifetime prevalence of major depression with a seasonal pattern was 0.4 percent.[3] Prevalence may be higher at northern latitudes, and it may vary within ethnic groups at the same latitude.[4]

Patients with SAD are more likely to have family members with SAD, although this may be subject to reporting bias.[5] Twin studies have found that there may be a genetic component to susceptibility. Several genes code for serotonin transport, but the overall pattern of heritability likely is complex and polygenomic.[6]

Patients with SAD have more outpatient visits, more diagnostic testing, more prescriptions, and more referrals throughout the year compared with age- and sex-matched controls.[7] Patients with SAD visit their primary care physician more often in the winter than other patients, but rates between the groups are similar the rest of the year.[8]

Screening for SAD

Primary care physicians routinely fail to diagnose nearly one half of all patients who present with depression and other mental health problems.[9] Because SAD is a subtype of major depression, screening for depression should theoretically help identify patients with this disorder. The U.S. Preventive Services Task Force (USPSTF) concluded that there is good evidence that screening improves the accurate identification of patients with depression in primary care settings, and that treatment decreases clinical morbidity. The USPSTF concluded that the benefits of screening likely outweigh any potential harms.[10]

There are several instruments for detecting depression in primary care, ranging in length from one to 30 items with an average administration time of two to six minutes. Typically, the reading level of these instruments is between the third- and fifth-grade levels.[11] Some standardized instruments focus more narrowly on SAD. Reports on the sensitivity and specificity of these instruments can be difficult to interpret because of the small sizes and heterogeneity of patient samples

SORT: KEY RECOMMENDATIONS FOR PRACTICE

Clinical recommendation	*Evidence rating*	*References*
Standardized screening instruments for SAD probably are not sensitive enough to be used for routine screening.	C	12
Light therapy may be used for treating SAD, with effect sizes similar to those for antidepressant medications in treating depression. The total daily dosage should be approximately 5,000 lux, administered in the morning over 30 to 120 minutes.	A	23
Cognitive behavior therapy may be considered as an alternative to light therapy in the treatment of SAD.	B	28

SAD = seasonal affective disorder.

A = consistent, good-quality patient-oriented evidence; B = inconsistent or limited-quality patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, see page 1463 or http://www.aafp.org/afpsort.xml.

tested, the possibility of differential recall bias (depending on the time of year the test is administered), and ongoing controversy over the criteria standard for SAD.

The Seasonal Pattern Assessment Questionnaire (SPAQ) is perhaps the most widely studied tool. It has been reported to have a high specificity (94 percent) for SAD but a low sensitivity (41 percent).[12] Other authors, however, have reported a much lower specificity.[13] The Seasonal Health Questionnaire has been reported to have higher specificity and sensitivity than the SPAQ,[14] but these results must be confirmed in larger and more diverse patient groups.

Although benefits from screening are less likely to be achieved without an accurate diagnostic work-up, effective treatment interventions, and close follow-up, it is unclear whether screening ultimately improves the care and outcomes of patients with major depression. When deciding to implement a screening instrument in a practice, office personnel should consider the administration time, scoring ease, reading level, and usefulness in identifying major depression and assessing change in the depression scores over time.[15]

Once patients have been identified as having major depression, questions must be asked to determine if the depression is linked to SAD. These questions concern the relationship between depression and time of year (if remission occurs during certain times of the year) and whether the depression has occurred at the same time during the past two years.

TABLE 1
Criteria for Seasonal Pattern Specifier

There has been a regular temporal relationship between the onset of major depressive episodes in bipolar I or bipolar II disorder or major depressive disorder, recurrent, and a particular time of the year (e.g., regular appearance of the major depressive episode in the fall or winter).
NOTE: Do not include cases in which there is an obvious effect of seasonal-related psychosocial stressors (e.g., regularly being unemployed every winter).
Full remissions (or change from depression to mania or hypomania) also occur at a characteristic time of the year (e.g., depression disappears in the spring).
In the past two years, two major depressive episodes have occurred that demonstrate the temporal seasonal relationships, and no nonseasonal major depressive episodes have occurred during that same period.
Seasonal major depressive episodes (as described above) substantially outnumber the nonseasonal major depressive episodes that may have occurred over the individual's lifetime.

Reprinted with permission from American Psychiatric Association. Task Force on DSM-IV. Diagnostic and Statistical Manual of Mental Disorders. 4th ed. Washington, D.C.: American Psychiatric Association, 1994:390.

Associated Diagnoses

Because SAD is associated with serotonergic dysregulation and possibly with noradrenergic mechanisms, it may overlap with other diagnoses that share similar mechanisms, including generalized anxiety disorder, panic disorder, bulimia nervosa, late luteal phase dysphoric disorder, and chronic fatigue syndrome.[16] SAD also may be associated with attention-deficit/hyperactivity disorder (ADHD). Both conditions have been described as "disorders of central underarousal coupled with a heightened sensitivity to stimuli from the physical environment," and both are more common in women with a particular genotype for *HTR2A*, a gene that codes for a serotonin receptor.[17,18]

A pattern of seasonal alcohol use also may be associated with SAD. A summary of current research findings concluded that some patients with alcoholism may be self-medicating an underlying depression with alcohol or manifesting a seasonal pattern to alcohol-induced depression.[19] Such patterns appear to have a familial component and, like the link between ADHD and SAD, may be related to serotonergic functioning.

Treatment

Treatment options for SAD include light therapy, cognitive behavior therapy, and pharmacotherapy. Each option has been proven beneficial in treating SAD, but no large studies have found any treatment to be superior.

LIGHT THERAPY

Among susceptible persons, decreased seasonal exposure to light may mediate SAD through phase shifts in circadian rhythms, with resulting alterations in several aspects of serotonin metabolism. Thus, light replacement has been the most widely studied treatment for SAD.[20] In a review of studies of light therapy, an average dosage of 2,500 lux daily for one week was superior to placebo, as indicated by improvements on a depression rating scale.[21] The dosage most often found to be effective is 5,000 lux per day, given as 2,500 lux for two hours or 10,000 lux for 30 minutes.[22] A recent meta-analysis of 23 studies of light therapy found that the odds ratio for remission was 2.9 (95% confidence interval, 1.6 to 5.4); this ratio is similar to those of many pharmaceutical treatments for depression.[23] Like drug therapy for depression, light therapy carries some risk of precipitating mania.[24]

Light therapy generally is most effective when administered earlier in the day.[21,25,26] Early morning light therapy regulates the circadian pattern of melatonin secretion, whereas the use of light in the evening delays the normal melatonin phase shift.[27]

To ensure adequate response, patients should be treated with light therapy units that are specifically designed to treat SAD. Units that are not specifically designed for SAD treatment may not provide adequate brightness and may not have appropriate ultraviolet light filtration.[22]

COGNITIVE BEHAVIOR THERAPY

Although cognitive behavior therapy (CBT) has some effectiveness in improving dysfunctional automatic thoughts and attitudes, behavior withdrawal, low rates of positive reinforcement, and ruminations in patients with major depression, few studies have assessed its effectiveness in the treatment of SAD. In one small clinical trial, patients with SAD were randomized to six weeks of treatment with CBT or light therapy, or CBT plus light therapy.[28] At the end of treatment, all three groups had significantly decreased levels of depression, but there was no difference between groups. However, this study only enrolled 26 subjects. To date, there have been no studies large enough to establish the effectiveness of CBT in the treatment of SAD.

PHARMACOTHERAPY

Because patients with SAD also must fulfill criteria for depression, several randomized trials have assessed the use of antidepressants for this condition.[29-33] Most of these studies have compared pharmacotherapy with placebo rather than light therapy, making it difficult to determine if one treatment is superior. In the largest of these trials, patients with SAD had significantly better response on several measures of depression after eight weeks of sertraline (Zoloft) therapy compared with control patients.[29] Patients were excluded if they were receiving light therapy or other psychoactive medications, or if they had a history of alcoholism, drug abuse, or "emotional or intellectual problems."

A smaller study found that, in some statistical analyses, fluoxetine (Prozac) was better than placebo in the treatment of SAD.[30] Another small study found that the monoamine oxidase inhibitor moclobemide (not available in the United States) was similar to placebo in terms of changes on several general depression scales.[31]

Small trials of other agents (i.e., carbidopa/levodopa [Sinemet] and vitamin B_{12}) found no benefit over placebo.[34,35] Although there may be some theoretical justification for these treatments, there have not been trials of sufficient size to assess their effects.

Few randomized trials have assessed the effect of light therapy compared with pharmacotherapy.[32,36] These trials failed to find a difference between the effect of 6,000 lux and that of 20 mg of fluoxetine daily,[32] or between 10,000 lux and 20 mg of fluoxetine daily.[36] Larger trials will be required to establish whether there is a difference in effect size between light therapy and pharmacotherapy.

It is also possible that pharmacotherapy may preserve an initial therapeutic response to light therapy. Among 168 patients who had a positive response to light therapy, citalopram (Celexa) was found to be no more effective than placebo at preventing relapse; however, it was superior in terms of some secondary measures of depression.[33] In general, current evidence does not provide clear guidance as to whether antidepressant treatment is superior to light therapy, or whether antidepressants are useful as an adjunct to light therapy.

The Authors

STEPHEN J. LURIE, M.D., PH.D., is assistant professor of family medicine at the University of Rochester (N.Y.) School of Medicine and Dentistry.

BARBARA GAWINSKI, PH.D., is director of psychosocial curriculum in the Department of Family Medicine at the University of Rochester School of Medicine and Dentistry.

DEBORAH PIERCE, M.D., M.P.H., is clinical associate professor of family medicine at the University of Rochester School of Medicine and Dentistry.

SALLY J. ROUSSEAU, M.S.W, is administrator of the Family Medicine Research Center at the University of Rochester School of Medicine and Dentistry.

Address correspondence to Stephen J. Lurie, M.D., Ph.D., Dept. of Family Medicine, University of Rochester School of Medicine and Dentistry, 1381 South Ave., Rochester, NY 14620 (e-mail: Stephen_Lurie@urmc.rochester.edu). Reprints are not available from the authors.

Author disclosure: Nothing to disclose.

REFERENCES

1. American Psychiatric Association. Task Force on DSM-IV. Diagnostic and Statistical Manual of Mental Disorders. 4th ed. Washington, D.C.: American Psychiatric Association, 1994.
2. Magnusson A. An overview of epidemiological studies on seasonal affective disorder. Acta Psychiatr Scand 2000;101:176-84.
3. Blazer DG, Kessler RC, Swartz MS. Epidemiology of recurrent major and minor depression with a seasonal pattern. The National Comorbidity Survey. Br J Psychiatry 1998;172:164-7.
4. Mersch PP, Middendorp HM, Bouhuys AL, Beersma DG, van den Hoofdakker RH. Seasonal affective disorder and latitude: a review of the literature. J Affect Disord 1999;53:35-48.
5. Sher L, Goldman D, Ozaki N, Rosenthal NE. The role of genetic factors in the etiology of seasonal affective disorder and seasonality. J Affect Disord 1999;53:203-10.
6. Sher L. Genetic studies of seasonal affective disorder and seasonality. Compr Psychiatry 2001;42:105-10.
7. Eagles JM, Howie FL, Cameron IM, Wileman SM, Andrew JE, Robertson C, et al. Use of health care services in seasonal affective disorder. Br J Psychiatry 2002;180:449-54.
8. Andrew JE, Wileman SM, Howie FL, Cameron IM, Naji SA, Eagles JM. Comparison of consultation rates in primary care attenders with and without seasonal affective disorder. J Affect Disord 2001;62:199-205.
9. Higgins ES. A review of unrecognized mental illness in primary care. Prevalence, natural history, and efforts to change the course. Arch Fam Med 1994;3:908-17.
10. U.S. Preventive Services Task Force. Screening for depression: recommendations and rationale. Ann Intern Med 2002;136:760-4.
11. Williams JW Jr, Pignone M, Ramirez G, Perez Stellato C. Identifying depression in primary care: a literature synthesis of case-finding instruments. Gen Hosp Psychiatry 2002;24:225-37.
12. Mersch PP, Vastenburg NC, Meesters Y, Bouhuys AL, Beersma DG, van den Hoofdakker RH, et al. The reliability and validity of the Seasonal Pattern Assessment Questionnaire: a comparison between patient groups. J Affect Disorder 2004;80:209-19.
13. Raheja SK, King EA, Thompson C. The Seasonal Pattern Assessment Questionnaire for identifying seasonal affective disorders. J Affect Disord 1996;41:193-9.
14. Thompson C, Thompson S, Smith R. Prevalence of seasonal affective disorder in primary care; a comparison of the seasonal pattern assessment questionnaire. J Affect Disord 2004;78:219-26.
15. Nease DE Jr, Malouin JM. Depression screening: a practical strategy. J Fam Pract 2003;52:118-24.
16. Partonen T, Magnusson A. Seasonal Affective Disorder: Practice and Research. New York, N.Y.: Oxford University Press, 2001.
17. Levitan RD, Masellis M, Basile VS, Lam RW, Jain U, Kaplan AS, et al. Polymorphism of the serotonin-2A receptor gene (HTR2A) associated with childhood attention deficit hyperactivity disorder (ADHD) in adult women with seasonal affective disorder. J Affect Disord 2002;71:229-33.
18. Levitan RD, Jain UR, Katzman MA. Seasonal affective symptoms in adults with residual attention-deficit hyperactivity disorder. Compr Psychiatry 1999;40:261-7.
19. Sher L. Alcoholism and seasonal affective disorder. Compr Psychiatry 2004;45:51-6.
20. Partonen T, Lonnqvist J. Seasonal affective disorder. Lancet 1998;352:1369-74.
21. Terman M, Terman JS, Quitkin FM, McGrath PJ, Stewart JW, Rafferty B. Light therapy for seasonal affective disorder. A review of efficacy. Neuropsychopharmacology 1989;2:1-22.
22. Levitan RD. What is the optimal implementation of bright light therapy for seasonal affective disorder (SAD)? J Psychiatry Neurosci 2005;30:72.
23. Golden RN, Gaynes BN, Ekstrom RD, Hamer RM, Jacobsen FM, Suppes T, et al. The efficacy of light therapy in the treatment of mood disorders: a review and meta-analysis of the evidence. Am J Psychiatry 2005;162:656-62.
24. Sohn CH, Lam RW. Treatment of seasonal affective disorder: unipolar versus bipolar differences. Curr Psychiatry Rep 2004;6:478-85.
25. Eastman CI, Young MA, Fogg LF, Liu L, Meaden PM. Bright light treatment of winter depression: a placebo-controlled trial. Arch Gen Psychiatry 1998;55:883-9.
26. Terman M, Terman JS, Ross DC. A controlled trial of timed bright light and negative air ionization for treatment of winter depression. Arch Gen Psychiatry 1998;55:875-82.
27. Terman JS, Terman M, Lo ES, Cooper TB. Circadian time of morning light administration and therapeutic response in winter depression. Arch Gen Psychiatry 2001;58:69-75.
28. Rohan KJ, Lindsey KT, Roecklein KA, Lacy TJ. Cognitive-behavioral therapy, light therapy, and their combination in treating seasonal affective disorder. J Affect Disord 2004;80:273-83.
29. Moscovitch A, Blashko CA, Eagles JM, Darcourt G, Thompson C, Kasper S, et al., for the International Collaborative Group on Sertraline in the Treatment of Outpatients with Seasonal Affective Disorders. A placebo-controlled study of sertraline in the treatment of outpatients with seasonal affective disorder. Psychopharmacology (Berl) 2004;171:390-7.
30. Lam RW, Gorman CP, Michalon M, Steiner M, Levitt AJ, Corral MR, et al. Multicenter, placebo-controlled study of fluoxetine in seasonal affective disorder. Am J Psychiatry 1995;152:1765-70.
31. Lingjaerde O, Reichborn-Kjennerud T, Haggag A, Gartner I, Narud K, Berg EM. Treatment of winter depression in Norway. II. A comparison of the selective monoamine oxidase A inhibitor moclobemide and placebo. Acta Psychiatr Scand 1993;88:372-80.
32. Ruhrmann S, Kasper S, Hawellek B, Martinez B, Hoflich G, Nickelsen T, et al. Effects of fluoxetine versus bright light in the treatment of seasonal affective disorder. Psychol Med 1998;28:923-33.
33. Martiny K, Lunde M, Simonsen C, Clemmensen L, Poulsen DL, Solstad K, et al. Relapse prevention by citalopram in SAD patients responding to 1 week of light therapy. A placebo-controlled study. Acta Psychiatr Scand 2004;109:230-4.
34. Oren DA, Moul DE, Schwartz PJ, Wehr TA, Rosenthal NE. A controlled trial of levodopa plus carbidopa in the treatment of winter seasonal affective disorder: a test of the dopamine hypothesis. J Clin Psychopharmacol 1994;14:196-200.
35. Oren DA, Teicher MH, Schwartz PJ, Glod C, Turner EH, Ito YN, et al. A controlled trial of cyanocobalamin (vitamin B12) in the treatment of winter seasonal affective disorder. J Affect Disord 1994;32:197-200.
36. Lam RW, Levitt AJ, Levitan RD, Enns MW, Morehouse R, Michalek EE, et al. The Can-SAD study: a randomized controlled trial of the effectiveness of light therapy and fluoxetine in patients with winter seasonal affective disorder. Am J Psychiatry 2006;163:805-12.