Seasonal Affective Disorder: Diagnosis And Management

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Over a decade of research has refined and clarified the diagnosis of seasonal affective disorder (SAD), a condition characterized by recurrent major depressive episodes in the fall and winter. Primary care physicians are likely to encounter SAD in their practice because it is a common condition, SAD patients generally have mild to moderate depressions, and they may present with somatic complaints. Numerous studies have shown that exposure to bright, artificial light, termed light therapy (or phototherapy) is a safe and effective treatment for SAD. Although lack of environmental light is widely thought to be a factor in the etiology of SAD, the pathophysiology of SAD and the mechanism of action of light therapy remain elusive. Bright light clearly has significant, predictable effects on human circadian rhythms, but a circadian hypothesis for SAD remains unconfirmed. Other studies have implicated serotonergic dysfunction in the pathophysiology of SAD, and serotonergic medications (e.g., SSRI antidepressants) appear to be effective in the treatment of SAD. The role of light therapy versus medications requires more systematic evaluation, but the choice of treatment depends on various factors such as the severity of the episode, side effects of treatment, cost, and patient compliance. Recent research has begun to explore seasonality and the use of light in other psychiatric conditions, including nonseasonal depression, bulimia nervosa, and premenstrual depression.

Primary Care Psychiatry 1998; 4:63-74

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A number of subtypes of major depressive disorder have been identified, based on the cross-sectional clinical features or the course of depressive episodes (Table 1). These subtypes have important differences in clinical course, treatment response, and possibly etiology and pathophysiology. Seasonal affective disorder (SAD) is one such subtype, termed seasonal pattern in DSM-IV. SAD consists of recurrent fall and winter depressive episodes with full remissions (or switch to hypomania or mania) in the spring and summer. Although seasonality of depression has been recognized for centuries, the concept of SAD was first systematically developed and described in 1984 by Rosenthal and his group at the U.S. National Institute of Mental Health [1]. Their studies showed that these patients experienced dramatic and rapid relief of symptoms when exposed to bright, artificial fluorescent light, which they initially called phototherapy (and later was changed to light therapy, to distinguish it from other forms of phototherapy, i.e., for hyperbilirubinemia).

Diagnostic criteria for SAD are similar across the different classification systems (Table 2). The major tasks for diagnosis consists of identifying the specific onset and offset (remission) of depressive episodes, and excluding any depressive symptoms in the summer. For most patients, the usual onset of an episode is in October, and the typical offset is in April (Figure 1).

TABLE 1. Clinical subtypes (specifiers) of mood disorders identified in the DSM-IV.

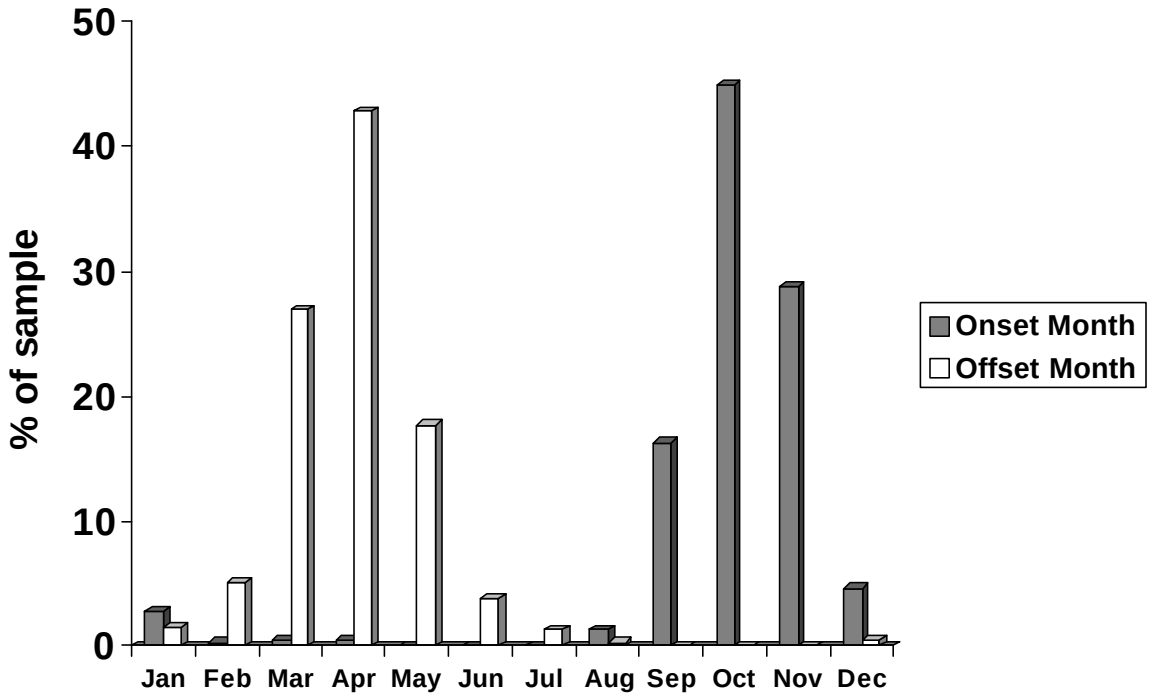
Episode Specifiers	Course Specifiers
With psychotic features	With seasonal pattern
With melancholic features	With postpartum onset
With atypical features	With rapid-cycling
With catatonic features	

Patients may not be reliable about the specific times for their episode onsets and offsets, so collateral information from family and friends is important to ensure that the depressive episodes are strictly seasonal. The seasonal specifier can be applied to either recurrent major depressive disorder, or to bipolar disorder. In our Vancouver clinic sample of 454 SAD patients diagnosed with DSM-III-R criteria (similar to ICD-10 criteria), the majority had unipolar depressions (89%), while 8.5% had spring/summer hypomanic episodes (bipolar disorder, type II), and 2.5% had full-blown mania (bipolar disorder, type I).

TABLE 2. Diagnostic Criteria for Seasonal Affective Disorder

DSM-IV Criteria	ICD-10	Rosenthal Criteria
2 or more episodes meet DSM-IV criteria for Major Depressive Disorder Last 2 episodes must be consecutive	3 or more mood episodes meeting ICD-10 criteria for Major Depression 3 or more episodes must be consecutive	1 or more episodes lifetime meet Research Diagnostic Criteria for Major Depression 2 or more episodes must be consecutive
Onset and remission of episodes must occur regularly in the same seasons	Onset and remission of episodes must occur regularly within particular 90-day periods of the year	Onset and remission of episodes must occur regularly in the same seasons
Seasonal episodes must greatly outnumber any nonseasonal episodes No nonseasonal episodes in the last 2 episodes	Seasonal episodes must substantially outnumber any nonseasonal episodes	
Exclude seasonal psychosocial stressors		Exclude seasonal psychosocial stressors

FIGURE 1. Month of onset and month of offset (remission) of symptoms reported by a clinic sample of 454 patients with SAD, diagnosed using DSM-III-R criteria.



CLINICAL FEATURES OF SAD

While the diagnostic criteria for the diagnosis of winter depression only include identifying a specific pattern of recurrent depressive episodes, clinic samples have shown that SAD is associated with a specific symptom cluster [1-4]. This cluster consists of the so-called "atypical" vegetative symptoms of depression, including hypersomnia, increased appetite, carbohydrate craving, and weight gain. Table 3 shows the prevalence of these clinical features in the 454 SAD patients assessed at our SAD Clinic. The hypersomnia seen in SAD may present as increased hours of sleep during the winter, often 2 to 4 hours more per night than in summer, or as increased need for sleep and difficulty arising in the morning.

Despite sleeping more hours, patients remain fatigued and tired during the day, with marked afternoon slumps in mood and/or energy to the point where they may feel compelled to nap.

The increased appetite is typified by carbohydrate craving for sugars and starches that is often described as uncontrollable. Binge-type eating can occur, although purging behaviours (e.g., vomiting) are uncommon [5,6]. The increased eating and reduced activity usually leads to significant weight gain. 10% of SAD patients seen in our clinic experience winter weight gains of greater than 20 pounds. Some patients report that they require two wardrobes, with their winter clothes being two or three sizes larger than their summer clothes. With initial winter episodes, patients lose the weight during the summer months when their appetite returns to normal and they are more active. However, with increasing age it becomes more difficult to shed the winter weight gain, and there is a gradual year-round increase in weight.

These atypical symptoms have led some investigators to suggest that SAD may be a form of atypical depression [7]. Atypical depression is characterized by mood reactivity, where patients experience marked but temporary improvement in mood in response to favourable external circumstances. The mood reactivity is also associated with at least two symptoms of hypersomnia, hyperphagia with weight gain, leaden paralysis (a severe form of fatigue that is experienced as a physical sensation of heaviness), and interpersonal rejection sensitivity (a long-standing pattern of exquisite sensitivity to rejection, especially romantic rejection). However, studies have shown that SAD patients do not have more mood reactivity, leaden paralysis, or rejection sensitivity than nonseasonal depressed patients [8]. Therefore, the overlap between the two subtypes appears to be limited to the atypical vegetative symptoms.

TABLE 3. Clinical features reported by a Vancouver (latitude 49°N) clinic sample of 454 patients with SAD, diagnosed using DSM-III-R criteria. In this group, the female to male ratio was 74% to 26%, and the mean age was 37.7 ± 10.8 years.

Vegetative Symptoms	% of Sample	Other Symptoms	% of Sample	Psychosocial Function	% of Sample
Sleep– Increased	71	Diurnal Variation		Occupational	73
Decreased	26	Morning worse	47	Impairment	
No change	3	Evening worse	26		
Appetite– Increased	57	Anxiety	79	Impaired	93
Decreased	28	Panic Attacks	12	Social	
No change	15			Function	
Weight– Increased	53	Suicidal Thoughts	47	Past	70
Decreased	14	Past Attempts	10	Psychiatric	
No change	33			Contact	
Carbohydrate Craving	77	Feelings of Guilt	82	Hospitalization	12
Loss of Interest	93	Irritability	82		
Loss of Energy	97	Poor Concentration	95		

Cognitive symptoms of depression are also present in SAD, including feelings of guilt and self-blame. SAD patients have similar neuropsychological deficits in memory and concentration as do nonseasonal depressed patients [9]. Interestingly, suicidal ideation and attempts are not as prominent or frequent in SAD compared to nonseasonal depression [10]. In part, this may be because SAD patients recognize the seasonal nature of their mood change and that they will likely improve in a few months with the onset of spring. The fact that an end to their depression is "in sight" may reduce the hopelessness found in nonseasonal depression, when patients never know how long they will be depressed.

Patients with SAD also notice that their symptoms remit when they are at lower latitudes (i.e., closer to the equator) [1]. Thus, it is informative to ask whether they have taken holidays or spent time in a more southerly location during the symptomatic winter months. Patients will often report that their mood improves markedly within a few days at the new latitude. Unfortunately, symptoms usually return within a week or two upon return to their usual locale. Additionally, patients will often notice winter symptoms only when they move to higher latitudes or to an area where there is greater winter cloud cover.

Primary care physicians are likely to encounter SAD patients because the depressions are usually mild to moderate in severity. A study of 303 patients attending a primary care clinic in the winter identified a clinical diagnosis of SAD in 9%, with another 29% having significant winter depressive symptoms without meeting criteria for major depression (subsyndromal SAD) [11]. The functional impairment of these patients, whether SAD or subsyndromal SAD, exceeded that of all the common chronic medical conditions measured. Detection of SAD is important since many patients do not recognize their disorder. In our clinic, 30% of patients diagnosed with SAD had never before sought professional help for their condition, even though they had suffered through, on average, 10.3 ± 8.0 previous winter depressive episodes. The reasons cited for not seeking help include that they believed they had "winter blues", that no treatment was available, that the winter problems were related to physical illness, and that their physicians did not take the symptoms seriously. A degree of vigilance is required since patients often do not associate their winter symptoms with a depression. Therefore, patients seen in the winter should be screened for SAD if they complain of recurrent bouts of the "flu", excessive fatigue, chronic sleepiness, excessive weight gain, or unexplained pain.

PREVALENCE AND COURSE

Studies of the prevalence of SAD have predominantly relied on questionnaires, such as the Seasonal Pattern Assessment Questionnaire (SPAQ) [12], which assess seasonality rather than clinical diagnoses. The questionnaire studies from the United States indicated that the prevalence of SAD increased with higher (more northern) latitudes, ranging from 1.4% in Florida to 9.7% in New Hampshire [13,14], and 9.2% in Alaska [15]. European and Asian studies, using translated versions of the same questionnaire, found lower rates of SAD at high latitudes, including 3.8% in Iceland [16], less than 1% in Finland [17], and 1% to 2% in Japan [18,19], although a significant correlation of SAD with higher latitude was still observed. This suggests that these questionnaires (or their translations) may not be consistent in identifying diagnoses of SAD [20-22] or that there are other factors that influence seasonality, such as culture or genetics. Other research has found that 15% to 20% of patients with mood disorders have distinct seasonal patterns [23-25]. Since the lifetime prevalence of mood disorders in the general population is about 10%, these data suggest that the prevalence of seasonal depression should be about 1% to 2%. A recent epidemiologic study from Canada supports these figures. In a telephone interview study conducted in the province of Ontario, Canada, 1.7% of the general population were found to have a clinical diagnosis of SAD [26].

Longitudinal follow-up studies of 2 to 11 years suggest that a percentage of patients diagnosed as SAD do not continue to have seasonal major depressive episodes [27-30]. About a third of SAD patients (22% to 42%) continued to have definite, recurrent seasonal depressive episodes. A similar proportion (28% to 44%) had complicated patterns suggesting a more nonseasonal course, although some patients in this group were taking antidepressants constantly throughout the year, so that their seasonal patterns may have been obscured. Another third (14% to 38%) either had subsyndromal episodes, or went into clinical remission. This shifting of episode pattern is also seen in other clinical subtypes of depression, including atypical and melancholic specifiers [31].

DIFFERENTIAL DIAGNOSIS

The differential diagnosis of SAD is similar to that of major depressive disorder. Organic conditions such as hypothyroidism need to be ruled out, as do other conditions such as phase-delayed sleep disorder, anniversary grief reactions, and seasonal occupational or psychosocial stressors.

There is also some debate as to whether SAD is a categorical diagnosis or an extreme form of a dimensional seasonality trait. Some people have marked symptoms during the winter, but not to the point where they meet criteria for major depressive disorder. The term "subsyndromal" SAD has been used to describe these patients [13]. These patients usually have the vegetative features of hypersomnia and hyperphagia, and prominent winter fatigue and lethargy. However, they may not have the cognitive symptoms of depression, such as depressed mood, feelings of guilt, and suicidal ideation. While they do not meet criteria for major depressive disorder, patients with subsyndromal SAD have significant distress and impairment of function [11,13]. Preliminary studies suggest that these patients also show good response to light therapy [32].

Many other patients with nonseasonal depressions, such as dysthymia and chronic major depression, may have winter worsening of their symptoms [33]. These patients can be differentiated from SAD proper because they are still symptomatic in the summer. Patients with bipolar disorder [20] also report marked worsening of mood, sometimes to the point of syndromal depression, but sometimes not, in the winter. Recent findings suggest that these patients also benefit from addition of light therapy to their treatment regimen during the winter.

Finally, seasonality is becoming increasingly recognized in other psychiatric conditions, including anorexia and bulimia nervosa [34-42], premenstrual depression [43], panic disorder [44], obsessive-compulsive disorder [45], and post-traumatic stress disorder [46].

ETIOLOGY AND PATHOPHYSIOLOGY OF SAD

Research into the etiology of SAD is intimately tied to that of the mechanisms of action of light therapy. Initial theories focused on the light-dark cycle or photoperiodic (relating to the length of the day) mechanisms that mediate seasonal rhythms in animals [1]. These theories hypothesized that patients with SAD were unable to adapt to the shorter winter photoperiod. Thus, the first successful study of light therapy exposed patients to bright (2500 lux) light from 6:00 to 9:00 a.m. and 6:00 to 9:00 p.m., daily, to extend the winter photoperiod and simulate a summer day [1]. However, subsequent studies showed that a pulse of bright light (e.g., 2 hours of 2,500 lux daily) was sufficient for the antidepressant effect. Attention shifted to abnormalities of circadian rhythms, such as phase-delayed [47] or reduced amplitude [48] circadian rhythms that were corrected by appropriately timed bright light pulses. However, studies have not consistently demonstrated that SAD patients have disturbed circadian rhythms compared to normal controls, or to themselves in summer, or that the clinical effect of light therapy is dependent on normalizing circadian rhythm abnormalities.

Other studies have focused on neurotransmitter or neurohormonal systems, including melatonin, serotonin [49] and dopamine [50,51]. Melatonin is a hormone synthesized from tryptophan and secreted only at night by the pineal gland. Melatonin secretion is controlled by two major influences. It is under circadian control by the biological pacemaker located in the suprachiasmatic nucleus (SCN) of the hypothalamus. Melatonin secretion can also be directly suppressed by bright light acting through the retina, to the SCN via the retinohypothalamic tract, and from there to the pineal gland via a complicated neural pathway. Seasonal changes in many animal behaviours are mediated by the duration of melatonin secretion, which reflects the photoperiod. Melatonin secretion does not appear to be primarily dysregulated in SAD, and experimental tests of a melatonin hypothesis have been primarily negative [52,53]. However, recent studies of propranolol, a beta-blocker that suppresses melatonin production, suggest that melatonin may still be involved in SAD. Morning doses of propranolol, which would suppress the early morning secretion of melatonin in the same way as bright light, are effective in SAD [54].

Serotonin is of particular interest in SAD because serotonin, of all the neurotransmitters of interest in depression, is the only one to clearly show a seasonal variation in normal metabolism (for review, see [55]). Neuroendocrine challenge studies using nonspecific and specific serotonin agonists have found evidence for serotonergic dysregulation [56-61]. Serotonergic medications are effective in SAD, including serotonin precursors (e.g., tryptophan [62,63]), serotonin reuptake inhibitors (e.g., fluoxetine [64,65], sertraline [66]), and serotonin releasing agents (e.g., d-fenfluramine [67,68]). Finally, tryptophan depletion studies, in which blood tryptophan levels (and presumably brain serotonin levels), are experimentally manipulated, show that the antidepressant effect of light therapy can be reversed if blood tryptophan levels are rapidly reduced [69,70].

Recent behavioural genetics studies have also shown that there may be a genetic basis for SAD. Studies of monozygotic and dizygotic twins, utilizing the SPAQ and multivariate statistical techniques, have shown that seasonality is a heritable trait. A genetic factor accounts for 29% to 83% of the variance in seasonality scores between twins [71,72].

TREATMENT OF SAD

Light Therapy

Light therapy (previously known as phototherapy) is recognized as a safe and effective treatment for SAD [73,74]. More than 3 dozen controlled studies have shown efficacy of light therapy with response rates of 60% to 90% [75-77]. The most widely studied protocol is 2500 lux fluorescent light for 2 hours per day, although studies of higher intensity light have shown that 10,000 lux light for 30 minutes per day gives similar response rates [78,79]. Lux is a unit of illumination intensity that corrects for the photopic spectral sensitivity of the human eye. For comparison, indoor evening room light is usually less than 100 lux, a brightly-lit office is less than 500 lux, a cloudy, gray winter day is around 4,000 lux, and bright sunshine can be 50,000 to 100,000 lux or more.

Although light therapy is regarded to be clinically effective, there are still some critiques about the evidence for its efficacy. Like other non-pharmacologic treatments, the studies are not funded by multinational companies, and so sample sizes tend to be small (usually less than 20 patients per condition) and the duration of treatment short (usually 1 to 2 weeks). There is also difficulty in designing a suitable placebo condition. Since the light cannot be "blinded", some deception is usually required to control for non-specific effects of treatment and biases inherent in expectations of response. Not surprisingly, given the small sample sizes, some studies have not found superiority of bright light over putative placebo conditions [80,81]. Other studies have not found that bright light is more effective than dim light of intensity found in ordinary indoor room light [82]. In these studies, the possibility of statistical Type II errors (i.e., missing a true effect) was high.

Two multi-year, large-sample, placebo-controlled studies were recently reported [83,84] that may finally answer the efficacy question. Both showed significant effects of the active bright light condition against plausible placebo controls. Additionally, a recent meta-analysis (where many similar studies are analyzed together using standardized effect sizes) also showed significant effects of bright light over dim or no light controls [85]. Together, these studies should provide sufficient confirmatory evidence that light therapy does have significant clinical benefit over placebo in SAD.

Various studies have investigated clinical parameters of light therapy including intensity of light, wavelength of light, duration of daily exposure, and timing of light exposure within the day. Results of this research are summarized by current clinical guidelines for the use of light therapy [74,86]. The protocol used in our clinic is exposure to 10,000 lux cool-white light produced by a fluorescent light box, fitted with a ultraviolet filter, for 30 to 45 minutes daily. Light therapy is usually administered in the early morning upon awakening (e.g., 7:00 a.m.) because many studies found that morning light exposure is superior to exposure at other times of the day [83,84,87-89] (but not all, see [56,90,91]). Patients use the light therapy for at least 2-3 weeks to determine response.

Patients usually obtain a light device (see below) and use light therapy at home, although some hospital and outpatient clinics have designed light therapy rooms for patient use. The onset of action of light therapy is rapid, with significant clinical improvement found in studies of 1 or 2 weeks duration. However, relapse usually occurs after a similar period once light therapy is discontinued [92]. Therefore, most patients must use light therapy regularly during their symptomatic winter season, until the time of their usual spring/summer remission. Once patients have remitted, they can often experiment with individual dosing required to stay well. Thus, they may be able to maintain their response while reducing the daily time of exposure to 15 or 20 minutes, or by using the light box on weekdays only [93]. In subsequent years, patients may be advised to begin light treatments in the early fall, before the onset of symptoms, thus avoiding any gradual or insidious impairment of function [94].

Several studies have shown that various atypical depressive symptoms predict positive response to light therapy [95-99]. Similarly, the balance of melancholic symptoms (e.g., insomnia, appetite and weight loss) over atypical symptoms was correlated to poor response to light therapy [100].

Side effects to light therapy are generally mild and transient, and consist of headache, nausea, eyestrain, blurred vision, and feelings of edginess [101,102]. Bright light exposure in the later evening may disrupt onset and maintenance of sleep. Like any effective antidepressant treatment, there is a risk of precipitating a hypomanic or manic episode with light therapy [103,104], and Bipolar I patients (those with a history of manic episodes) should be on mood-stabilizing medications if light therapy is used. Current dosing guidelines for intensity of light should not prove to be harmful to the eyes, and two long-term follow-up studies did not find any ophthalmologic changes with chronic use of light therapy [105,106]. However, caution should be exercised when treating patients at higher risk of bright light induced eye damage, including patients with pre-existing retinal disease (e.g., retinitis pigmentosa), patients who are taking photosensitizing medications (e.g., lithium, antipsychotics, chloroquine), and elderly patients (due to the higher risk for senile macular degeneration, which may be asymptomatic). For those patients, an ophthalmologic examination is

recommended before initiating light therapy, as well as regular follow-up monitoring.

Other light devices also have been studied for winter SAD. Three light therapy studies used a similar portable light visor. These studies, with large sample sizes and rigorous designs, found no differences between bright, medium, and dim intensity light, although the response rates of all conditions were similar to those of light box studies [107-109]. Other head-mounted devices also have not demonstrated a dose-response relationship or a superior response compared to a putative placebo [81,110]. It is possible that less light is required for therapeutic effect using light visors because of the close proximity of the light source to the eye. Physiologic studies using the light visor have shown that biological effects of light can be demonstrated with lower intensity light [111].

"Dawn simulator" devices are also marketed. These devices gradually increase the indirect light in a bedroom, while the patient is sleeping [112], to a final illumination of less than 500 lux, to simulate a summer dawn during the symptomatic winter. Preliminary studies of efficacy are promising [113], but not yet replicated, so dawn simulation remains an experimental treatment.

Light therapy has also been studied for nonseasonal depression, although not as extensively as for SAD. Several studies have shown positive effects with light therapy [82,114-116], although other studies have been negative [117,118]. These studies generally had smaller effect sizes than light therapy studies of SAD, and were all of relatively short duration (1-4 weeks) compared to most antidepressant studies of nonseasonal depression. Thus, further replication or more definitive studies are required before light therapy can be endorsed as effective for nonseasonal depression.

Other Treatments For SAD

Medications have not been studied in SAD as extensively as light therapy. Only 3 placebo-controlled studies have been reported for antidepressants in SAD. Selective serotonin reuptake inhibitors (SSRIs) are the best-studied medications, with multi-centre, placebo-controlled studies showing that fluoxetine and sertraline are effective in SAD. The fluoxetine study (N=68 SAD patients) used a fixed 20 mg/day dose for 5 weeks. Although there was no significant difference in the raw depression scores, the clinical response rate of fluoxetine was superior to that of placebo (59% vs. 34%, respectively) [64]. The sertraline study (N=170 SAD patients) used doses of 50 to 200 mg/day for 8 weeks. Sertraline was superior to placebo in both the depression scores and the clinical response rates (62% vs. 46%, respectively) [66]. A study of moclobemide (N=31 SAD and subsyndromal SAD patients) used low doses of 300 mg/day for only 3 weeks, and found no differences between drug and placebo [119].

Although not placebo-controlled, a 6-week comparison study of moclobemide versus fluoxetine (N=29 SAD patients), found no significant difference in response rate (64% vs. 44%, respectively) [65]. Other smaller controlled studies of tryptophan (N=11 SAD) [62], d-fenfluramine (N=29 SAD patients in 2 studies) [67,68], and hypericum (an extract of St. John's Wort, N=20 SAD patients) [120] suggest that these treatments, if the positive results can be replicated, may be effective for SAD.

A number of case series studies suggest that other antidepressants may also be beneficial for SAD, including bupropion (N=15) [121], tranylcypromine (N=14) [122] and alprazolam (N=6) [123].

Although psychological treatments like cognitive-behavioural therapy and interpersonal psychotherapy have been demonstrated to be effective in nonseasonal depression, there are as yet no studies of such treatments in SAD.

In summary, the first-line medication treatment for SAD is with SSRI medications such as fluoxetine and sertraline, followed possibly by moclobemide, then with other medications such as d-fenfluramine, tryptophan, bupropion and tranylcypromine.

HOW TO CHOOSE A TREATMENT FOR SAD

There are no published studies comparing the efficacy of light therapy versus medications for SAD. Thus, the choice of treatment for SAD requires individual risk/benefit assessment. There are more studies demonstrating efficacy of light therapy than there are of medications, but the studies of SSRI antidepressants are much larger than any individual light therapy study. Clinically, light therapy seems to work faster than antidepressants, and generally has fewer side effects. Many patients also prefer a non-pharmacologic treatment for their symptoms. For these patients, light therapy should be the first-line treatment of choice. However, compliance is an issue, since even the newer light therapy protocols mandate spending a half-hour per day or more using the light device. Many patients do not have the interest or motivation required to use light therapy effectively. For those patients, daily medication use is more convenient. For more severely depressed inpatients, antidepressant medications are indicated as first-line

treatment, although light therapy is often useful as an adjunctive treatment.

Light boxes are now widely available commercially, at a cost of US$150 to US$350. Thus, the cost of a light box is approximately the same as one season of the newer antidepressant medications. For recurrent use, light therapy appears to be more cost-effective. However, insurance plans may not reimburse light boxes, while medications may be covered, and some patients may not be able to afford a light device. Many light device companies have rental programs or money-back guarantees so patients can have a trial of light therapy before purchasing a light device.

Some patients find that a combination of light therapy and medications works best for them, and that the dose of antidepressant can be reduced when light therapy is combined. Unfortunately, there are as yet no studies of combined use of light therapy and antidepressant medications.

CONCLUSION

SAD is a common depressive condition that results in significant psychosocial dysfunction and disability. Primary care practitioners should be vigilant for the presenting features of SAD and subsyndromal SAD when seeing patients during the winter. SAD is a very treatable condition with a good prognosis. Sample sizes in light therapy studies have been limited, but the efficacy of light therapy in the treatment SAD has been established by multiple replications in independent laboratories around the world. Medications, notably SSRI antidepressants such as fluoxetine and sertraline, have also been demonstrated to be effective in SAD. Further research is required to elucidate the pathophysiology of SAD and light therapy, and the optimal treatment (light therapy, medications, psychotherapies, or a combination) for individual patients with SAD.

INFORMATION RESOURCES FOR SAD

Seasonal Affective Disorder Association

As a registered UK charity, SADA is a self-help organization that promotes information about the disorder and its treatment.

Contact: The Secretary, SADA, PO Box 989, London SW7 2PZ

Society for Light Treatment and Biological Rhythms

As a non-profit international scientific organization founded in 1988, SLTBR is dedicated to fostering research, professional development and clinical applications in the fields of light therapy and biological rhythms.

Web site: www.sltbr.org
(includes a list of Corporate Members that manufacture and distribute light devices)

Other Web Sites

Dr. Lam's SAD Page at the University of B.C.

www.psychiatry.ubc.ca/mood/sad/

Centre for Environmental Therapeutics

Includes a FAQ (Frequently Asked Questions) and resources about SAD.

www.cet.org

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