

# Seasonal affective disorder

## How to help patients beat the winter blues

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If your patient's depression recurs in autumn and winter,  
a trial of bright light therapy may provide good results at minimal risk.

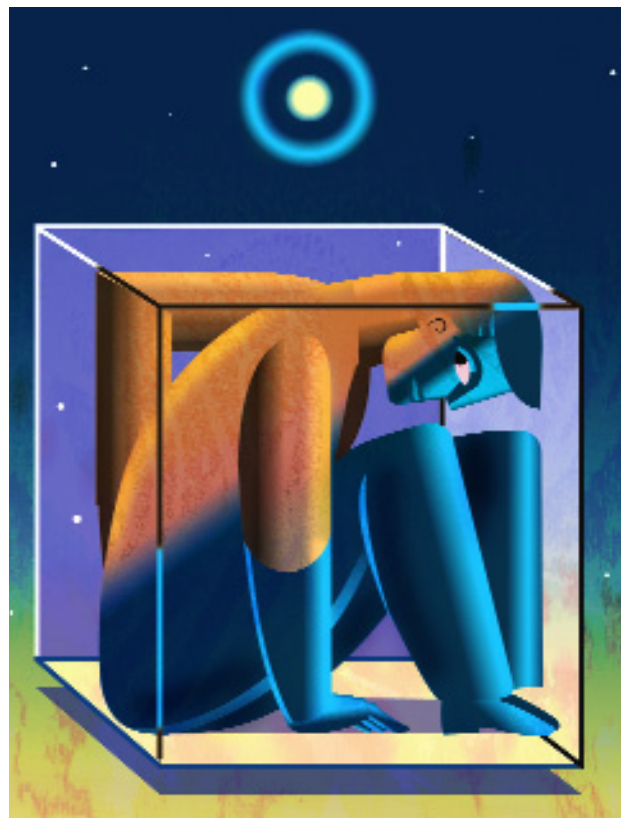
**A**ll of us see patients whose recurrent depressions seem to have a seasonal component. Should we treat them differently than patients whose recurrent depressions are not related to seasons? Is there adequate evidence for the existence of seasonal affective disorder (SAD), or—as it is called in DSM-IV-TR—mood disorder with a seasonal pattern? Is bright light therapy supported by the literature, or is it just some sort of fad?

As December brings the shortest days of the year, we shine the spotlight on SAD and examine the latest evidence on its causes, diagnosis, and treatment.

### Moods with a seasonal rhythm

Moods have been observed to change with the seasons since ancient times (*Box 1*).<sup>1</sup> As recently as 25 years ago, however, seasonal affective disorder was not recognized as a psychiatric diagnosis.

In the early 1980s, when researchers at the National Institute of Mental Health (NIMH) were studying the effect of bright light on melatonin secretion, they were contacted by Herbert E. Kern, a research engineer who suffered from recurrent depression. A methodical person, Kern had kept a journal of his mood variations and noticed a pattern that appeared to



Box 1

**'LAPP SICKNESS' AND THE EFFECT OF LIGHT ON MOOD**

Hippocrates, with his knack for keen observation, observed the variation of moods with the seasons. Aretaeus went a step further in the 2nd century by proposing that "lethargics are to be laid in the light and exposed to the rays of the sun."

More recently, the physicist Angstrom—for whom the unit of light wavelength is named—was one of the first to mention the Swedish word "Lappsjuka," which means "Lapp sickness" (Lapp refers to Scandinavian aborigines). He wrote, "Many people are sensitive to the lack of light, while others are less so. The former will in arctic winters suffer from Lappsjuka."<sup>1</sup>

follow the seasons. His depression worsened in the fall and winter and improved in the spring and summer. Kern subsequently participated in an NIMH trial with phototherapy, his mood improved, and the results were published in 1982.<sup>2</sup>

Two years later, the researchers published the first paper that described SAD as a psychiatric diagnosis.<sup>3</sup> Criteria for the diagnosis included:

- presence of a major affective disorder
- affective episodes occurring during fall or winter and remitting in spring or summer for at least 2 consecutive years.

The paper also discussed treatment of winter depression with phototherapy.

DSM-IV-TR describes SAD as a course specifier for mood disorders, including major depressive episodes in bipolar I and II disorders and major depressive disorder (Box 2). In other words, as used in DSM-IV-TR and this article, SAD is not an independent disorder but a type of major affective disorder.

**Characteristics of SAD**

**Symptoms.** Patients with SAD suffer the typical symptoms of depression—decreased energy, guilt, and decreased libido—as well as atypical symptoms—carbohydrate craving, hypersomnia, and weight gain. They also appear less likely to exhibit psychotic symptoms and may

be at lower risk for suicide than persons with major mood disorders but without SAD.<sup>1</sup>

Changes in sleep patterns also have been observed. Rosenthal et al<sup>4</sup> found increased sleep latency and increased total sleep time in patients with SAD. Delta or slow-wave sleep—the restorative part of the sleep cycle—decreased by nearly one-half (mean 46%). REM latency did not change, contrary to typical findings in depressed patients. Anderson et al<sup>5</sup> also reported no change in REM latency in patients with SAD.

**Comorbid conditions.** Eating disorders—particularly bulimia nervosa—are more prevalent in patients with SAD.<sup>1</sup> Binge eating tends to worsen in the fall and winter.

Personality disorders are also common in these patients, with cluster C over-represented. Avoidant personality disorder is most common. In a sample of 45 patients with SAD, Reichborn-Kjennerud et al<sup>6</sup> found any personality disorder in 58% and avoidant personality disorder in 31%. Patients with comorbid personality disorders were less likely to respond to bright light therapy.

**Prevalence.** The prevalence of SAD in North America is approximately 1 to 6%, with four times as many women affected as men.<sup>1</sup> Data on the effect of latitude on prevalence of SAD are inconclusive.<sup>7</sup>

**Making the diagnosis**

For patients with depression, clinicians should ask about seasonality of symptoms. Onset of major depressive symptoms in the fall or winter for at least two consecutive years or remission of depressive symptoms in the spring for two consecutive years (without onset of depressive syndromes during the spring or summer) probably merits a diagnosis of SAD. The diagnosis is confirmed if seasonal patterns of depressive symptoms substantially outnumber nonseasonal occurrences over the patient's lifetime. The diagnosis may not be appropriate if there are obvious seasonal psychosocial stressors, such as anniversary reactions in posttraumatic stress disorder.

Some patients have sub-syndromal depressive symptoms that occur seasonally. DSM-IV would probably classify them as "mood disorder, not otherwise specified," and that group has not been studied extensively.

Atypical depressive symptoms of SAD include carbohydrate craving, hypersomnia, and weight gain

## Box 2

## DSM-IV-TR: SEASONAL PATTERN SPECIFIER FOR MOOD DISORDER

Specify if:

**With seasonal pattern** (can be applied to the pattern of major depressive episodes in bipolar I disorder, bipolar II disorder, or major depressive disorder, recurrent)

- A. 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 seasonal-related psychosocial stressors (e.g., regularly being unemployed every winter) create an obvious effect.
- B. Full remissions (or a change from depression to mania or hypomania) also occur at a characteristic

time of the year (e.g., depression disappears in the spring).

- C. In the past 2 years, two major depressive episodes have occurred that demonstrate the temporal seasonal relationships defined in criteria A and B, and no nonseasonal major depressive episodes have occurred during that same period.
- D. Seasonal major depressive episodes (as described above) substantially outnumber the nonseasonal major depressive episodes that may have occurred over the individual's lifetime.

Source: DSM-IV-TR

### What causes SAD?

Research is ongoing, but the cause of SAD is not yet fully understood, although hypotheses have been developed. The four main hypotheses relate to duration of sunlight, changes in the circadian cycle, and secretion of the “hormone of darkness,” melatonin.

**Photoperiod hypothesis.** The shortening of the photoperiod—duration of sunlight—during autumn and winter may explain winter depression. Some research suggests that patients with SAD have an exaggerated melatonin response to shorter days and longer nights. For example, Wehr et al<sup>8</sup> found that SAD patients secrete melatonin approximately 30 minutes longer per day in the winter, compared with controls.

**Phase-delay hypothesis.** Core body temperature is considered one of the most reliable markers of circadian rhythm. The nadir core body temperature occurs earlier than normal in a person whose circadian rhythm is “phase-advanced” and later than usual in those with “phase-delayed” circadian rhythms. Patients with seasonal affective symptoms generally reach their lowest body temperature of the day earlier than do controls.

Lewy et al,<sup>9</sup> who proposed the phase-delay hypothesis, observed that melatonin secretion appeared to be delayed in patients with SAD. Some studies have supported this hypoth-

esis, demonstrating greater benefit of bright light treatment when administered early in the morning than later in the day. Other studies, however, have shown benefit from light exposure late in the day.<sup>10</sup>

**Reduced-amplitude hypothesis.** SAD sufferers have dampened circadian rhythms, and bright light may increase the amplitude of the rhythms. There is little evidence for this hypothesis.

**Melatonin hypothesis.** Melatonin does not appear to cause depression. Looking at melatonin secretion patterns in conjunction with circadian phases, however, may offer new insights.<sup>8</sup> Several studies have shown that manipulating the timing of melatonin secretion affects mood.

Cryptochrome, a photoreceptor in the retina, may be responsible for transmitting the photosignal to the elements of the circadian clock that regulate melatonin secretion. The pineal gland modifies its secretion of melatonin in response to the amount of light exposure (*Box 3*).<sup>8,11</sup>

### Light therapy

Bright light therapy has been supported by placebo-controlled trials and is first-line treatment for patients with SAD.<sup>1</sup> Bright light therapy is usually dosed at 2,500 lux for 2 hr/day or 10,000 lux for 30 min/day at eye level.<sup>12</sup> The best evidence supports

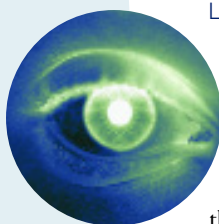
Box 3

## HOW SUNLIGHT MAY SET THE CIRCADIAN CLOCK

When light strikes the eye, it is well known that vitamin A-based photo pigments, cones, and rods receive the signal and transmit it via the optic tracts to the occipital cortex. But if light plays a role in the pathophysiology of seasonal affective disorder, how does light signal the other elements of the circadian system?

**Cryptochrome**, a photoreceptor in the retina, may be responsible for transmitting the phot signal to the circadian clock.<sup>11</sup> Cryptochrome is a vitamin B2-based pigment found in the ganglion cells and on cells in the inner nuclear layer of the retina. It is thought to transmit light through the optic nerve to the suprachiasmatic nucleus, the circadian pacemaker in the hypothalamus. The signal is then transmitted through the paraventricular nucleus, down the sympathetic chain, through the superior cervical ganglion, up the nervi canarii, to its final destination—the pineal gland.<sup>8</sup> The pineal gland then modifies its secretion of melatonin in response to the amount of light exposure.

**Although mammals** have an intrinsic rhythmicity, the circadian clock must be entrained by exogenous light. The retinohypothalamic tract is believed to be the vehicle that sets the circadian clock. If the optic nerve is cut, a person becomes blind and unable to entrain the circadian rhythm.



administering light therapy in the morning, generally between 6 and 10 AM. As described by Terman,<sup>13</sup> “larks”—people who go to bed early and get up early—need earlier light than “owls”—people who stay up later and sleep later.

Light therapy can be used alone or in addition to pharmacotherapy in patients whose previously well-controlled depressive symptoms worsen in the fall or winter. Light therapy also can be used as prophylaxis—starting in early fall—in patients with a history of a seasonal pattern of depression. Either way, light treatments generally should continue until early spring.

Given the relatively few side effects, light thera-

py may be used as monotherapy in patients with mild, sub-syndromal mood symptoms occurring on a seasonal basis. Light therapy should not be used without pharmacotherapy to treat a full-blown major depressive episode.

Light boxes can be found via the Internet at an average cost of \$180 to \$300 for a 10,000-lux unit. The boxes are small enough to be placed on a table while the patient reads or eats breakfast. Artificial lights for this therapy do not emit ultraviolet rays, which have been associated with skin cancers.

**Light visors** also have shown some promise in SAD treatment, as demonstrated by a 2-week, randomized, controlled trial by Joffe et al.<sup>14</sup> Compared with light boxes, light visors are more portable, so the patient can move around during treatment. Generally, the patient wears the visor 30 minutes in the morning.

Light visors appear to be as effective as table models, although no studies have compared the two devices. A visor costs \$250 to \$300.

**Dawn simulation** in SAD treatment has been examined in a few small studies and one placebo-controlled trial with 95 patients.<sup>10</sup> In dawn simulation, a white light gradually increases between 4:30 and 6 AM to a peak intensity of 250 lux. Dawn simulation can be done while the patient is sleeping, whereas other light treatments require the patient to wake up early enough each morning to sit before a light box for 30 minutes. More study is needed to assess this modality’s efficacy.

**Light therapy precautions.** Review the patient’s medications before starting light therapy. Drugs that can magnify the effects of short wave-length light—leading to severe sunburns or rashes—include tetracycline, sulfon-

amides, and some older antipsychotics such as chlorpromazine. Some authors recommend an ophthalmologic examination before starting light therapy and every 2 to 3 years afterwards if no complications are apparent.<sup>1</sup> Others believe that no ophthalmologic examination is necessary unless the patient is older than 70 or has a history of retinal disease.

Side effects of bright light therapy are usually few and mild and include headaches, eye irritation, and nausea. In some anecdotal cases, patients with bipolar disorder appear to have switched

Light therapy should not be used without pharmacotherapy to treat a major depressive episode

from depression to mania upon starting light therapy,<sup>1</sup> but such switches appear to be rare. Still, patients with bipolar disorder and their family members should be advised to watch out for switches when using light therapy.

### Pharmacologic therapy

Drug therapy in SAD has not been well studied, and many of the placebo-controlled trials that have examined this mode of treatment have been small. Serotonergic agents have been most studied because serotonin, with its effects on sleep and appetite, is thought to be related to SAD pathogenesis. The largest study of a selective serotonin reuptake inhibitor for SAD<sup>15</sup> compared sertraline with placebo. Patients who received sertraline at a mean dosage of 111 mg/d had significantly fewer depressive symptoms than did the placebo group.

A placebo-controlled, double-blind study by Thorell<sup>16</sup> found that adding citalopram to light therapy improved measures of depressed mood, compared with light therapy alone. This study is limited by small sample size but provides direction for further research.

An open trial of reboxetine—a noradrenaline reuptake inhibitor not available in the United States—suggests that further research of agents affecting catecholamines may be worthwhile in SAD treatment.<sup>17</sup>

### Psychotherapy

Psychotherapy has not been researched sufficiently to be considered a proven treatment for SAD. However, some have observed that SAD patients have a negative cognitive style that

may benefit from cognitive therapy. Thus, behavioral therapy may alter a patient's response to light.<sup>1</sup>

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### Related resources

- ▶ National Organization for Seasonal Affective Disorder [www.nosad.org](http://www.nosad.org)
- ▶ Dr. Ivan's (Ivan Goldberg, MD) Depression Central [www.psycom.net/depression.central.seasonal.html](http://www.psycom.net/depression.central.seasonal.html)
- ▶ Columbia/Presbyterian Hospital Program in Clinical Chronobiology [www.light-and-iontherapy.org](http://www.light-and-iontherapy.org)

#### DRUG BRAND NAMES

Citalopram • Celexa  
Sertraline • Zoloft

#### DISCLOSURE

Dr. Paradies reports no financial relationship with any company whose products are mentioned in this article, or with manufacturers of competing products.

Dr. Hillard reports that he is a consultant to Pfizer Inc. and Janssen Pharmaceutica, and serves on the speakers bureau of Pfizer Inc., Janssen Pharmaceutica, and Eli Lilly and Co.

Seasonal affective disorder (SAD) may be characterized by lethargy, guilt, carbohydrate craving, weight gain, and disordered sleep. SAD is related to circadian rhythms, which are entrained by light exposure. A trial of bright light therapy may provide good results at minimal risk if a patient's depression recurs in autumn and winter.

BottomLine