Seasonal affective disorder (SAD)

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Primary Disciplinary Field(s): Psychiatry; Clinical Psychology; Chronobiology

1. Core Definition and Diagnostic Status

Seasonal affective disorder (SAD) is a pattern of recurrent, significant depressive episodes that exhibit a regular temporal relationship to a particular time of the year, followed by full remission during the remaining seasons. First formally conceptualized by Rosenthal and colleagues in 1984, SAD is characterized by its predictable onset and remission, most commonly involving depression during the fall and winter months (StatPearls, 2024). Although often colloquially referred to as "winter blues," SAD, when meeting full diagnostic criteria, represents a debilitating condition capable of significantly impairing daily functioning and quality of life.

In the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition, Text Revision (DSM-5-TR), SAD is not classified as a separate disorder. Instead, it is used as a specifier--"With Seasonal Pattern"--applied to recurrent **Major Depressive Disorder (MDD)** or **Bipolar I or Bipolar II Disorder**. The specifier requires a regular temporal relationship between the onset of mood episodes and a specific time of year for at least two consecutive years, with seasonal episodes substantially outnumbering non-seasonal episodes over the individual's lifetime. Furthermore, the pattern cannot be better explained by predictable, seasonally linked psychosocial stressors (American Psychiatric Association, 2022; StatPearls, 2024).

2. Clinical Presentation and Subtypes

SAD manifests primarily in two subtypes: the much more common **Winter-Pattern SAD** and the less frequent **Summer-Pattern SAD**. These two patterns are distinguished by their specific timing and contrasting vegetative symptom profiles.

The core symptoms of winter depression often align with **atypical depressive features**, reflecting a slowing down of physiological processes. Key characteristics include **hypersomnia** (oversleeping, difficulty waking, and daytime sleepiness), **increased appetite** (often accompanied by intense carbohydrate cravings), weight gain, and profound **fatigue** or lethargy, sometimes described as "leaden paralysis" in the limbs (StatPearls, 2024; PsychDB, 2021). These symptoms typically emerge in autumn and remit in spring.

In contrast, summer-pattern SAD often presents with features more typical of agitated or anxious depression. Symptoms include **insomnia** (difficulty sleeping), **decreased appetite** leading to weight loss, **restlessness**, agitation, and heightened anxiety. The mechanisms driving summer SAD are less clear but are hypothesized to involve sensitivity to excessive heat, humidity, and the extended duration of daylight hours disrupting sleep and circadian balance (NIMH; StatPearls,

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2024).

3. Etiology and Underlying Hypotheses

The etiology of Seasonal Affective Disorder is considered multifactorial, stemming from an interaction between reduced environmental light exposure and individual biological and genetic vulnerabilities. Research is centered on three main biological theories:

The Circadian Rhythm Hypothesis (Phase-Shift Hypothesis): This leading theory posits that winter depression results from a misalignment between the body's master pacemaker (the suprachiasmatic nucleus) and the shortened external light-dark cycle (Medscape, 2024). In susceptible individuals, the decrease in morning light in fall/winter leads to a **phase delay**--the internal clock runs later than the external clock--manifesting as morning grogginess and evening energy surges. The effectiveness of timed light therapy supports this model.

The Melatonin Hypothesis: Melatonin, the hormone signaling nighttime, is secreted primarily in darkness. In the context of SAD, the **timing and duration** of melatonin secretion are critical. While early theories suggested simple overproduction of melatonin, current research focuses on how the longer duration of darkness in winter extends the melatonin signal, reflecting the underlying circadian phase delay (Lewy et al., 1987; Wehr et al., 2001). Correcting this timing is central to light therapy's efficacy.

The Serotonin Hypothesis: Serotonin (5-HT) activity is implicated in mood, appetite, and sleep regulation. Studies using positron emission tomography (PET) scans suggest that individuals with SAD exhibit seasonal fluctuations in the levels of the serotonin transporter (SERT), the protein responsible for removing serotonin from the synapse. Higher SERT levels observed during winter compared to summer, potentially due to reduced sunlight exposure, lead to decreased serotonin availability, contributing to depressive symptoms (Willeit et al., 2000; Praschak-Rieder et al., 2008).

4. Epidemiology and Risk Factors

SAD exhibits distinct epidemiological patterns related to geography and demographics. Prevalence rates range from 0.5% to 3% in the general population but are significantly higher (10% to 20%) among those with recurrent major depression (Magnusson & Partonen, 2005; AAFP, 2012).

The most consistent finding is the pronounced **latitude gradient** for winter-pattern SAD. Prevalence rates increase dramatically at higher northern or southern latitudes--for example, ranging from 1.4% in low-latitude Florida to nearly 10% in high-latitude New Hampshire or Alaska (Rosen et al., 1990). This strong correlation underscores the environmental light component. The disorder typically begins in **young adulthood** (average onset 18 to 30 years), and it occurs substantially more often in **women** than in men, with reported ratios generally between 2:1 and 4:1

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(NIMH; Galima et al., 2020).

A significant **genetic component** is also indicated by family and twin studies. Individuals with SAD are more likely to have relatives with SAD or other mood disorders (Mayo Clinic, 2022). Research focuses on candidate genes involved in the serotonin system (e.g., 5-HTTLPR) and core **circadian clock genes** (e.g., PER2, PER3, ARNTL), suggesting that SAD is a polygenic disorder resulting from the complex interplay between multiple genetic vulnerabilities and environmental light exposure.

5. Assessment and Differential Diagnosis

Accurate diagnosis of SAD requires a meticulous clinical assessment focused on symptom chronology and severity. The cornerstone of evaluation is a detailed clinical history to confirm that depressive episodes have occurred reliably during the same season for at least two consecutive years, with full remission outside that period (StatPearls, 2024). Clinicians must also rule out non-seasonal mood episodes and ensure the pattern is not due to predictable psychosocial stressors (American Psychiatric Association, 2022).

Standardized tools, such as the **Seasonal Pattern Assessment Questionnaire (SPAQ)**, are widely used as screening instruments to quantify the degree of seasonal variation in mood and behavior, although they require clinical confirmation for formal diagnosis.

Differential diagnosis is crucial, particularly differentiating SAD from:

Bipolar Disorder: Up to 25% of individuals with Bipolar Disorder may exhibit a seasonal pattern (often winter depression/summer hypomania). Screening for lifetime manic or hypomanic episodes is essential, as antidepressant monotherapy can trigger mania in these individuals.

Non-Seasonal MDD or Persistent Depressive Disorder (Dysthymia): These lack the strict, predictable onset and remission pattern necessary for the seasonal specifier.

Medical Conditions: Hypothyroidism, chronic fatigue syndrome, and sleep disorders can mimic SAD symptoms like fatigue and hypersomnia and must be ruled out through appropriate medical workup (Kurlansik & Ibay, 2012).

6. Treatment Modalities

SAD is highly responsive to treatment, employing modalities that target the underlying neurobiological and circadian dysfunctions, alongside psychological interventions.

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Light Therapy (Phototherapy)

Light therapy is considered a **first-line treatment** for winter-pattern SAD, with a strong evidence base demonstrating its efficacy compared to placebo (Golden et al., 2005). The mechanism of action is primarily thought to involve **circadian phase advancement**--using morning light exposure to reset the body clock--and acute suppression of melatonin (StatPearls - Light Therapy). Standard protocols involve exposure to a light box emitting **10,000 lux** of UV-filtered, broad-spectrum white light for 30 minutes daily, typically within the first hour of waking (Terman & Terman, 2005). Response usually occurs within one to two weeks, and treatment should continue throughout the symptomatic season. While generally safe, caution is necessary in patients with retinal disease or underlying Bipolar Disorder due to the risk of inducing hypomania.

Pharmacotherapy

Antidepressant medications are effective options, particularly for severe cases, lack of response to light therapy, or for prophylactic use.

Bupropion XL (Extended-Release) is the only medication approved by the U.S. Food and Drug Administration (FDA) specifically for the **prevention** of seasonal major depressive episodes in patients with SAD. Trials have shown that starting Bupropion XL in the autumn, before symptom onset, significantly reduces recurrence (Karger, 2019).

Selective Serotonin Reuptake Inhibitors (SSRIs) such as fluoxetine are widely used due to the hypothesized role of serotonin dysfunction and have demonstrated efficacy comparable to light therapy in some acute trials (Lam et al., 2006).

Psychotherapy

Cognitive Behavioral Therapy for SAD (CBT-SAD) is a structured, time-limited therapy that targets maladaptive cognitive patterns associated with winter (e.g., negative beliefs about darkness) and employs behavioral activation to counteract seasonal social withdrawal. RCTs have shown that CBT-SAD is as effective as light therapy for acute treatment (Rohan et al., 2015). Importantly, long-term follow-up studies suggest that CBT-SAD may offer **superior long-term durability** compared to light therapy, with significantly lower recurrence rates one and two winters following treatment, implying that learned coping skills provide lasting protection (Rohan et al., 2016; Healio, 2015).

7. Further Reading

StatPearls: Seasonal Affective Disorder (2024)
Mayo Clinic: Seasonal affective disorder (SAD)

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National Institute of Mental Health (NIMH): Seasonal Affective Disorder
Medscape: Seasonal Affective Disorder (SAD): Background, Pathophysiology, Epidemiology
Rohan, K. J., et al. (2016). Outcomes one and two winters following cognitive-behavioral therapy or light therapy for seasonal affective disorder.

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