# Seasonal affective disorder: Epidemiology, clinical features, assessment, and diagnosis

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## INTRODUCTION

The term seasonal affective disorder (SAD) describes episodes of major depression, mania, or hypomania that regularly occur during particular seasons. The most prevalent form of SAD is winter depression, marked by recurrent episodes of unipolar depression that begin in the fall or winter and, if left untreated, generally remit in the following spring or summer. Recognizing the disorder is important because SAD is common and associated with psychosocial impairment [1,2]. In addition, acute treatment is often effective and maintenance treatment can prevent future episodes [3]. Among patients who were recruited for randomized trials studying treatment of winter depression, nearly 60 percent had never been treated for depression [4].

This topic discusses the epidemiology, pathogenesis, clinical features, assessment, diagnosis, and validity of SAD; most of the topic is devoted to recurrent unipolar major depression with winter seasonal pattern (winter depression). Choosing treatment is reviewed separately. (See "Seasonal affective disorder: Treatment".)

## DEFINITIONS

**Seasonal affective disorder** — Seasonal affective disorder (SAD) is defined as recurrent episodes of major depression, mania, or hypomania with seasonal onset and remission [5]. It is

not considered a separate mood disorder; rather, SAD is a subtype of the following mood disorders:

- Unipolar major depression (major depressive disorder)
- Bipolar I disorder
- Bipolar II disorder

Thus, patients with SAD have recurrent episodes of unipolar major depression ( table 1), bipolar major depression ( table 2), mania ( table 3), or hypomania ( table 4); the essential feature is that onset and remission of the mood episodes occurs at characteristic times of the year [5].

Subsyndromal SAD consists of recurrent periods of clinically significant mood symptoms that occur with seasonal onset and remission; however, the symptoms do not rise to the level to meet criteria for mood syndromes, such as major depression, and do not substantially impair functioning [1,6].

This topic focuses primarily upon recurrent unipolar major depression with seasonal pattern because it is more common than bipolar disorder with seasonal pattern [7-9]. Among patients with unipolar major depression with seasonal pattern, the winter seasonal pattern is far more common than the summer pattern. (See 'Clinical features' below.)

The clinical features and diagnosis of unipolar major depression and bipolar disorder are discussed separately. (See "Unipolar depression in adults: Clinical features" and "Unipolar depression in adults: Assessment and diagnosis" and "Bipolar disorder in adults: Clinical features" and "Bipolar disorder in adults: Assessment and diagnosis".)

**Seasonality** — SAD is a clinical disorder that is distinguished from seasonality, which represents seasonal changes in mood and behavior [7,10]. Some authorities think that seasonality is distributed along a continuum of severity and that SAD represents one end of the continuum, whereas others conceptualize SAD as a categorical diagnosis [11].

Although seasonality may occur widely in the general population [10,12], it appears that for most individuals in the community, and many patients with mood disorders, the effect of seasonality is modest at most [13-16]. However, seasonal variation in mood and behavior may be greater in patients with depressive syndromes than healthy controls [17].

In addition, placebo response rates in patients with depression may vary in different seasons [18]. A study examined patients with depressive syndromes (n = 432) who were not selected for seasonality and who received a 10-day course of placebo as a “washout” or lead-in to a

randomized trial [19]. Response to placebo occurred in fewer patients during the late fall and winter months than the rest of year (15 versus 25 percent).

## EPIDEMIOLOGY

**General population** — In studies from Canada, the United States, and Wales that used interviews rather than self-report questionnaires and used standardized diagnostic criteria for major depression with seasonal pattern, the lifetime prevalence of seasonal affective disorder (SAD) in the general population was approximately 0.5 to 3 percent [20-23]. Studies from the Netherlands and Switzerland using less rigorous methods have found comparable rates [24,25].

**Clinical settings** — The estimated point prevalence of SAD in outpatients is as follows:

- Primary care patients – 5 to 10 percent [26,27]
- Depressed patients – 15 percent [28,29]

**Risk factors** — Although risk factors for SAD have been identified, they are likely to have little predictive power for any specific patient.

Most community surveys [29,30] and clinical studies [1] indicate that SAD is more common among women. However, this finding may be due to methodologic problems. As an example, a nationally representative survey using more rigorous methods (interviews and standardized diagnostic criteria) found that the lifetime prevalence of SAD was greater in men than women [20]. The finding that SAD is more common among women in clinical studies may be due to selection bias, and simply reflect the observation that the prevalence of unipolar major depression in general is greater in women. (See "Unipolar depression in adults: Epidemiology", section on 'Sex'.)

The prevalence of SAD in different age groups is not clear, but seems to be greater in adults than either older adults or children and adolescents [1,21]. Onset of SAD is usually between age 20 and 30 years [1].

Individuals residing at higher northern latitudes, which receive less light in winter, may be at increased risk for SAD; however, this is controversial. Some studies have found that the prevalence of SAD was greater in higher latitudes, but case finding relied upon screening instruments [31]. Methodologically more rigorous studies using standardized criteria to diagnose SAD found that latitude was not associated with the prevalence of SAD [1].

## PATHOGENESIS

The pathogenesis of seasonal affective disorder (SAD) is not known; the primary hypotheses involve disturbances of circadian rhythms, decreased sensitivity of the retina, genetic factors, and dysregulation of neurotransmitters such as serotonin [32]. These hypotheses are not mutually exclusive.

In one model, the pathogenesis of SAD includes two components: a seasonal factor and a depression factor [7]. Different pathophysiologic mechanisms may underlie each factor. As an example, the seasonal factor may be due to phase delayed circadian rhythms and the depression factor may be due to monoamine (eg, serotonin) dysregulation.

- **Circadian rhythms** – Circadian (daily) rhythms are physiologic and behavioral changes that oscillate over a 24-hour cycle, primarily in response to light and darkness in the environment. It is hypothesized that the genesis of SAD involves changes in circadian rhythms due to seasonal changes in the length of daylight each day (also referred to as the light-dark cycle) [33-35]. The circadian rhythms affect the timing and duration of sleep [36].

  Two hypotheses based upon circadian rhythms have been proposed for the pathogenesis of SAD: the photoperiod hypothesis and the phase shift hypothesis.

  - **Photoperiod hypothesis** – The length of natural daylight each day is called photoperiod, and is shorter in winter and longer in summer. The photoperiod hypothesis of SAD proposes that in vulnerable individuals, the shorter photoperiod in winter induces depression [34,35]. Active secretion of melatonin occurs at night, and the longer duration of melatonin secretion in winter may trigger depression. A study found that in patients with SAD (winter depression; n = 55), the duration of nocturnal melatonin secretion was longer in winter than summer; among the healthy controls (n = 55), the duration of melatonin secretion was similar in winter and summer [37]. These results suggest that patients with SAD respond to photoperiod in a manner analogous to other mammals, who respond to the shorter photoperiod of winter (and longer duration of melatonin secretion) with a hibernation response. The hypersomnia, increased appetite, and weight gain that characterize winter depression in patients with SAD may be viewed as a type of hibernation response [35]. Other evidence supporting the photoperiod model of SAD comes from studies of light therapy, which suggest that artificial light may extend the photoperiod. Additional information about

melatonin is discussed separately. (See "Pharmacotherapy for insomnia in adults", section on 'Melatonin'.)

- **Phase shift hypothesis** – The phase shift hypothesis proposes that there is an optimal relationship between the timing of circadian rhythms (eg, the body's minimum temperature and secretion of melatonin) and the timing of sleep, and that misalignment between circadian rhythms and sleep leads to SAD [33,38]. In most patients with SAD, the later dawn in winter and diminished light delays circadian rhythms relative to external clock time and sleep; this shift of circadian rhythms to a later time is called phase delay. These patients respond to artificial morning light that realigns the circadian rhythms with the sleep-wake cycle [39,40]. Among a smaller group of patients with SAD, circadian rhythms shift to an earlier time with respect to sleep (phase advance); these patients may respond to evening light [33,38]. Properly timed doses of melatonin may also correct circadian misalignment and relieve SAD [41].

  The suprachiasmatic nucleus in the hypothalamus is often referred to as the master biological clock or pacemaker [35,42]. This nucleus generates and synchronizes (entrains) internal circadian rhythms with external time cues such as light, and helps control multiple circadian rhythms, such as daily fluctuations in core body temperature, as well as melatonin secretion by the pineal gland.

  Bright light can shift the timing of circadian rhythms. Light just before the temperature minimum will typically shift the temperature minimum clockwise to a later time (phase delay). Light soon after the temperature minimum will shift the temperature minimum counterclockwise to an earlier time (phase advance). The timing of the light relative to the temperature minimum will determine how much the circadian rhythms shift.

  Darkness generally has the opposite effect of light upon circadian rhythms. Darkness in the morning will cause a phase delay in the rhythms; darkness in the evening will cause a phase advance in the rhythms.

- **Noncircadian effects of light** – Light may have a direct positive effect on mood. The intrinsically photoactive retinal ganglion cells are especially sensitive to blue light and connect to not only the suprachiasmatic nucleus of the hypothalamus, but also areas of the brain involved in mood regulation, such as the amygdala and the lateral habenular areas [43].

- **Retinal subsensitivity** – The sensitivity of the retina normally increases during winter in response to decreased light levels; the retinal subsensitivity hypothesis posits that this

response is impaired in SAD [34]. Retinal ganglion cells, which contain a photopigment (melanopsin) that is involved in circadian entrainment, are connected to the suprachiasmatic nucleus. The diminished retinal sensitivity to light may thus affect circadian function. The prevalence of SAD appears to be greater among individuals with severe visual impairment, compared with individual with full sight [32]. In addition, observational studies indicate that bright light therapy can normalize retinal sensitivity anomalies in patients with SAD [44].

- **Genetics** – There have not been any twin studies of SAD and it is not known if SAD has a heritable component. One study found that a missense variant of the melanopsin gene was associated with an increased risk of SAD, but the variant was seen in only 5 percent of patients with SAD [45]. Other studies have found that SAD is associated with variants of specific genes involved in synthesis of dopamine, guanyl-nucleotide-binding protein (G protein), and serotonin, but none of these results have been consistently replicated across different studies [35]. In family history studies, a family history of mood disorders is present in 25 to 67 percent of patients with SAD, and approximately 15 percent of patients with SAD have a first degree relative with SAD [35]. Although these rates exceed what is found in the general population, a positive family history may be due to environmental effects, genetic factors, or both.

  Twin studies have examined seasonality, which represents seasonal changes in mood and behavior that are less severe than what occur in SAD. These studies suggest that genetic factors account for approximately 30 percent or more of the phenotypic variance [35].

  The activity of many genes appears to vary in different seasons. A study of more than 22,000 genes from volunteers (n >1200) found that seasonal variation in gene expression occurred in nearly 25 percent of the genes [46]. Approximately 2300 genes were more active in summer and 2800 were more active in winter. Seasonal variation in gene expression was observed in 9 of the 16 clock genes tested. As an example, the circadian clock gene ARNTL was 50 percent more active in August than February.

- **Serotonin** – Multiple studies suggest that abnormal functioning of serotonergic neurons in the central nervous system may be involved in SAD:

  - Decreased serotonergic activity may occur in SAD because of hyperfunctional activity of the serotonin transporter; this enhanced activity may increase clearance of serotonin from the synaptic cleft and reduce synaptic serotonin concentrations. A study observed that activity of the serotonin transporter was elevated in patients with winter depression compared with healthy controls; in addition, the enhanced activity of the

transporter in patients normalized after bright light therapy and during natural summer remission [47].

- Increased levels of the serotonin transporter protein (which are associated with lower synaptic serotonin levels) may also decrease central serotonergic activity in SAD. A study that examined healthy volunteers found that levels of the protein in the brain were greater during the fall and winter, compared with the spring and summer [48].

- A crossover study compared an amino acid beverage containing tryptophan with an amino acid beverage containing no tryptophan in patients with SAD (n = 11) who were in remission during the summer [49]. Tryptophan is the precursor amino acid required for central synthesis of serotonin, and administration of a large amount of amino acids without tryptophan induces hepatic metabolism that transiently depletes tryptophan and reduces brain serotonin. The patients served as their own controls and were randomly assigned to the order in which they received the two beverages; the oral solutions were administered approximately one week apart. Relapse of depression occurred more often during active tryptophan depletion than sham depletion (8 versus 0 patients [73 versus 0 percent]). Other crossover trials have similarly found that tryptophan depletion increased depressive symptoms in patients with SAD who had remitted after bright light therapy [50,51].

In addition, dysregulation of neurotransmitters other than serotonin may be involved in SAD [7].

Information about neurotransmitters in the general population of patients with unipolar depression is discussed separately. (See "Unipolar depression: Neurobiology", section on 'Neurotransmitters'.)

## CLINICAL FEATURES

The clinical features of seasonal affective disorder (SAD) depend upon the specific mood disorder that is present (unipolar major depression, bipolar I disorder, or bipolar II disorder). This section focuses primarily upon patients who have recurrent unipolar major depression with seasonal pattern because it is more common than bipolar disorder with seasonal pattern. Among patients with SAD, it is estimated that bipolar I or II disorder is the underlying condition in no more than 30 percent [7,8]. As an example, a study of consecutive patients treated for SAD (n = 454) at a specialty clinic found that bipolar disorder was present in approximately 10 percent [9].

**Seasonal pattern** — Among patients with recurrent unipolar major depression with seasonal pattern, two specific patterns have been described [1,5]:

- **Fall-winter onset** – Fall-winter onset SAD is also known as winter depression. Major depressive episodes begin in the fall to early winter and, if left untreated, generally remit during the following spring and summer. These episodes are usually characterized by increased sleep, increased appetite, carbohydrate craving, and weight gain (symptoms that are also found in major depression with atypical features). Depressive symptoms may recur during the summer if exposure to ambient light is reduced (eg, during persistent cloudy weather).

- **Spring-summer onset** – Spring-summer onset SAD is also known summer depression; major depressive episodes begin in the spring or summer and remit during the following fall and winter. These episodes are usually marked by typical symptoms of depression, such as insomnia and decreased sleep, and decreased appetite and weight loss.

This topic focuses primarily upon winter depression because it is far more common and widely studied than summer depression [1,5,8]. As an example, a registry study found that the prevalence of winter SAD and summer SAD was 3.0 and 0.1 percent [24].

**Symptoms** — The symptoms of major depression in patients with SAD and in patients with nonseasonal major depression are the same ( table 1):

- Depressed mood
- Loss of pleasure or interest
- Change in appetite or weight (decreased or increased)
- Sleep disturbance (insomnia or hypersomnia)
- Fatigue or loss of energy
- Neurocognitive dysfunction
- Psychomotor agitation or retardation
- Feelings of worthlessness or guilt
- Suicidal ideation or behavior

Additional information about these symptoms is discussed separately. (See "Unipolar depression in adults: Clinical features", section on 'Symptoms'.)

Major depressive episodes in patients with SAD usually include atypical features, such as [4,5,7,42]:

- Increased appetite (especially for carbohydrates)

- Weight gain (eg, 3 to 4 kg [6.6 to 8.8 pounds])
- Hypersomnia (eg, sleeping at least one hour more during winter depression than during summer euthymia)

As an example, a retrospective study compared patients with SAD (recurrent unipolar major depression with seasonal pattern; n = 53) and patients with nonseasonal unipolar major depression (n = 54) who were randomly selected from outpatients attending a specialty mood disorders clinic at a university teaching hospital [52]. Hyperphagia and hypersomnia were more prominent in SAD than nonseasonal depression. However, patients with SAD were less prone to rejection sensitivity than patients with nonseasonal depression, indicating that SAD overlaps with but is not the same as major depression with atypical features. (See "Unipolar depression in adults: Clinical features", section on 'Atypical'.)

It is worth noting that hypersomnia and insomnia are not mutually exclusive, and that both can occur in SAD. A study of patients with SAD (n = 51) found that both hypersomnia and insomnia were present in 47 percent of patients [53].

Among patients with SAD, the symptom profile of winter depression appears to differ from the symptoms of summer depression. A prospective study of patients with winter SAD (n = 30) and summer SAD (n = 30) found that both groups of patients were characterized by dysphoria, fatigue, and decreased activity and functioning [54]. However, hypersomnia, hyperphagia, carbohydrate craving, and weight gain occurred more frequently in winter depressives than summer depressives. By contrast, decreased appetite and sleep occurred more often in summer depressives.

The frequency of psychotic symptoms in winter depression appears to be rare [1].

**Functioning and quality of life** — By definition, psychosocial functioning is impaired during episodes of unipolar major depression ( table 1), whether or not a seasonal pattern is present [5]. One study examined patients with SAD (n = 20) and patients with nonseasonal depression who were hospitalized for suicide attempts (n = 20), and found that global functioning and social functioning were each comparable in the two groups [2].

Quality of life, which refers to subjective satisfaction with one's physical, psychological, and social functioning, is generally comparable in patients with SAD and patients with nonseasonal unipolar depression [55].

**Comorbidity** — Patients with SAD often have comorbid psychopathology, including [1,6,7,56-63]:

- Alcohol use disorders
- Attention deficit hyperactivity disorder
- Binge eating disorder
- Bulimia nervosa
- Delayed sleep phase disorder
- Generalized anxiety disorder
- Panic disorder
- Personality disorders
- Premenstrual dysphoric disorder
- Social anxiety disorder

The presence of comorbidity may lead to a chief complaint that is not a key symptom of SAD and thus interfere with recognition of SAD [1]. In addition, the severity of depression appears to be greater in patients with comorbid disorders than patients without comorbidity [63].

High rates of comorbid psychopathology are also found in the general population of patients with major depression. (See "Unipolar depression in adults: Clinical features", section on 'Comorbidity'.)

**Course of illness** — The short-term course of illness in SAD may vary during the fall and winter months. As an example, bright sunny days are associated with improvement of symptoms [64]. In addition, residual symptoms may persist during summer; these symptoms are often amenable to bright-light therapy [65]. It is estimated that over the course of one year, symptoms are present 40 percent of the time [66].

Many patients with winter depression do not suffer a depressive episode the following winter. Prospective studies have found that from one winter to the next, recurrence of depression occurs in only 50 to 70 percent of patients [67].

The seasonal pattern of recurrent unipolar major depression is generally not a long-term phenomenon. In several longitudinal studies of patients who were initially diagnosed with SAD and subsequently evaluated (eg, 5 to 11 years later), recurrent depression with seasonal pattern persisted in less than 50 percent [1]. Approximately 40 percent of patients continued to suffer recurrent major depressive episodes that no longer retained a pattern of winter depression followed by summer remission, and approximately 15 percent recovered with no further episodes of major depression.

## ASSESSMENT

Patients with a possible diagnosis of seasonal affective disorder (SAD) are assessed for a recurrent mood disorder in which the onset and remission of the mood episodes occur at characteristic times of the year.

**When to suspect the disorder** — The presence of recurrent unipolar depression with seasonal pattern (winter depression) is suggested by the following clues [1]:

- Seasonal pattern to the depressive episodes
- Depressive syndrome is usually worse in winter
- Depression is ameliorated by sunshine
- Atypical depressive symptoms
  - Hypersomnia
  - Hyperphagia (especially carbohydrate craving)
  - Weight gain
- Somatic symptoms

In one study of primary care patients, health care utilization (eg, number of outpatient visits, diagnostic tests, and prescriptions) throughout the year was greater in patients with SAD than patients without SAD [68]. During winter, the number of outpatient visits was greater in patients with SAD.

**Mood disorder** — The evaluation of patients for a diagnosis of SAD begins with an assessment for unipolar major depression or bipolar disorder. (See "Unipolar depression in adults: Assessment and diagnosis", section on 'Assessment' and "Bipolar disorder in adults: Assessment and diagnosis", section on 'Assessment'.)

**Suicide** — All depressed patients should be assessed for suicidal ideation (including plans) and behavior. (See "Unipolar depression in adults: Assessment and diagnosis", section on 'Suicide risk' and "Suicidal ideation and behavior in adults", section on 'Patient evaluation'.)

**Seasonal pattern** — Patients with unipolar major depression ( table 1) and a possible diagnosis of fall-winter onset SAD are assessed for recurrent depressive episodes with a seasonal pattern. The interview includes the following questions:

- Do you feel worst (more sad) in the winter?
- Do you eat more in the winter?
- Do you gain weight in the winter?
- Do you sleep more in the winter?

- Do you have less energy in the winter?
- Do you socialize with other people less often in the winter?
- Is the depression ameliorated by sunnier weather?
- Does the depression resolve during travel to a sunnier location or a location with a longer photoperiod (length of natural daylight each day)?
- Does the depression remit during the spring and/or summer?

Hypersomnia may present as difficulty awakening for patients who are disciplined and have to wake up to go to work [69]. Asking about sleep on days when patients are free to sleep in may uncover hypersomnia.

It is useful to ask about exposure to outdoor light. Depressed patients who isolate themselves indoors may create an "eternal winter." Darkness during the summer may perpetuate the depressive syndrome because the patient is not receiving summer light.

The initial assessment asks about mood episodes that may have occurred several years in the past, and it can thus be helpful to gather information from collateral sources, such as family and friends.

**Screening** — We generally do not use an instrument to screen for SAD. Rather, we verbally ask patients who present with a history of recurrent depressive episodes if the episodes occur with a seasonal pattern. (See 'Seasonal pattern' above.)

For clinicians who choose to screen for SAD, we suggest the self-administered Seasonal Pattern Assessment Questionnaire ( table 5) [70]. The questionnaire includes a six-item scale that assesses seasonal changes in sleep, social activity, mood, weight, appetite, and energy. Each item is rated on a Likert scale ranging from 0 (no change) to 4 (extremely marked change). The scores for each item are summed and the total score ranges from 0 to 24; a score ≥11 is usually used to identify SAD [6,53]. However, this cut-off score is considered overinclusive [1,71]. The psychometric properties of the Seasonal Pattern Assessment Questionnaire range from poor to good, with a sensitivity of 38 to 94 percent, specificity 46 to 79 percent, and positive predictive value 45 to 71 percent [27,72-74]. The questionnaire has good internal consistency, test-retest reliability, and construct validity [74-76], but prospective validity is poor [77].

An alternative to the Seasonal Pattern Assessment Questionnaire is the Seasonal Health Questionnaire, which is longer and has six sections and multiple items within each section [78]. The sensitivity is 59 percent, specificity 97 percent, and positive predictive value 93 percent [27].

Screening questionnaires do not generate a diagnosis of SAD; thus, patients who screen positive require a clinical interview to make the diagnosis. In addition, studies indicate that screening for depression is beneficial only in settings that can provide follow-up to ensure accurate diagnosis and effective treatment. General information about screening for major depression, including the effectiveness of screening, is discussed separately. (See "Screening for depression in adults".)

## DIAGNOSIS

**Diagnostic criteria** — We recommend diagnosing SAD according to the criteria in the American Psychiatric Association's Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5) [5]. In DSM-5, SAD is defined as recurrent episodes of major depression, mania, or hypomania with seasonal onset and remission. SAD is not considered a separate mood disorder; rather, SAD is a subtype of the following mood disorders:

- Unipolar major depression (major depressive disorder)
- Bipolar I disorder
- Bipolar II disorder

Patients with SAD thus have recurrent episodes of unipolar major depression ( table 1), bipolar major depression ( table 2), mania ( table 3), or hypomania ( table 4); the essential feature is that onset and remission of the mood episodes occurs at characteristic times of the year [5]. This topic focuses primarily upon patients with recurrent unipolar major depression with seasonal pattern because most of what is known about SAD comes from studies of these patients.

The World Health Organization's International Classification of Diseases, 10$^{th}$ Revision (ICD-10) mentions “seasonal depressive disorder” as a subtype of recurrent unipolar depressive disorder, but does not provide any specific diagnostic criteria for seasonal depressive disorder. The formal ICD-10 diagnostic term for SAD is “recurrent depressive disorder, unspecified” [79,80].

**Unipolar major depression** — According to DSM-5, an episode of unipolar major depression manifests with five or more of the following symptoms, present most of the day nearly every day for a minimum of two consecutive weeks ( table 1) [5]. At least one symptom is either depressed mood or loss of interest or pleasure.

- Depressed mood
- Loss of interest or pleasure in most or all activities
- Insomnia or hypersomnia

- Change in appetite or weight
- Psychomotor retardation or agitation
- Low energy
- Poor concentration
- Thoughts of worthlessness or guilt
- Recurrent thoughts about death or suicide

Additional information about the diagnosis of unipolar major depression is discussed separately. (See "Unipolar depression in adults: Assessment and diagnosis".)

**With seasonal pattern** — The specifier "with seasonal pattern" applies to recurrent major depressive episodes that meet each of the following criteria [5]:

- There is a regular temporal relationship between the onset of major depressive episodes and a particular time of year (eg, fall or winter). However, the specifier is not used for patients with episodes that occur in response to seasonally related psychosocial stressors (eg, unemployment every winter).

- Remission of the episodes (in the absence of treatment) also occurs at a characteristic time of year (eg, spring or summer).

- In each of the last two years, onset and offset of an episode of major depression has occurred at a characteristic time of year, and there have not been any nonseasonal episodes.

- The lifetime course of illness is such that seasonal episodes of major depression substantially outnumber the nonseasonal episodes (eg, by a ratio of two to one).

**Bipolar disorder** — The diagnosis of bipolar disorder, including bipolar disorder with a seasonal pattern, is discussed separately. (See "Bipolar disorder in adults: Assessment and diagnosis", section on 'Diagnosis'.)

**Differential diagnosis** — The disorder at the top of the differential diagnosis for SAD is nonseasonal major depression, particularly nonseasonal major depression with atypical features. Both SAD and nonseasonal major depression consist of major depressive episodes (see 'Unipolar major depression' above). However, SAD is distinguished by onset and remission of recurrent episodes at characteristic times of the year (eg, winter) [5]. In nonseasonal major depression, there is no temporal relationship between recurrence/remission of major depressive episodes and a particular time of year. Although SAD and nonseasonal major depression with atypical features can share certain symptoms (such as hyperphagia, weight

gain, and hypersomnia), the two disorders are differentiated by whether the depressive episodes occur with a seasonal pattern. (See 'With seasonal pattern' above.)

Other disorders that constitute the differential diagnosis for SAD are similar to disorders found in the differential diagnosis for unipolar major depression and bipolar disorder [7]. (See "Unipolar depression in adults: Assessment and diagnosis", section on 'Differential diagnosis' and "Bipolar disorder in adults: Assessment and diagnosis", section on 'Differential diagnosis'.)

## SYNDROMAL VALIDITY

DSM-5 recognizes SAD as a subtype of recurrent mood disorders [5], and ICD-10 recognizes seasonal depressive disorder as a subtype of recurrent depressive disorders [79,80]. SAD is thought to be a valid clinical syndrome, based upon its face validity (extent to which experts think the concept is distinct), descriptive validity (extent to which the defining characteristics of the illness are unique to the disease), predictive validity (extent to which one can predict the course of illness and response to treatment), and construct validity (extent to which the defining characteristics, boundaries, and pathophysiology of a disorder are known) [10].

Evidence supporting the syndromal validity of SAD includes the following:

- The clinical manifestation of winter depression is distinct and predictable, such that patients regularly present with depression in the fall or winter and remit in the spring or summer [11]. As an example, prospective studies that recruited euthymic patients with SAD during summer months have found that patients became depressed in the fall and winter.
- Winter depressive episodes characteristically and consistently manifest some atypical features [11] (see 'Symptoms' above).
- SAD has been observed in both the southern and northern hemispheres [81].
- Neurobiologic studies suggest that patients with SAD differ from patients with nonseasonal major depression and healthy controls. As an example, studies of patients with SAD have observed seasonal changes in melatonin secretion and found that processing of visual light is impaired [11].

Nevertheless, some clinicians doubt that SAD is a valid syndrome [8,82]. Arguments put forth that SAD is not a distinct entity include the following:

- SAD symptoms (eg, hyperphagia and hypersomnia) overlap with symptoms of major depression with atypical features [5,10]. However, this argument is undermined by lack of overlap with regard to other atypical symptoms (see 'Symptoms' above). In addition, an observational study found that bright light therapy was more effective for SAD than atypical depression [83].

- The boundary between SAD and the seasonal variations in mood and behavior found in the general population (called seasonality) is not clearly delineated [10]. When the Seasonal Pattern Assessment Questionnaire is administered to the general population, the histogram of the frequencies of each score does not have a bimodal distribution; instead, there is a continuum of the frequencies ranging from no seasonality to mild seasonality to SAD [12]. However, this lack of distinct boundaries exists for other psychiatric syndromes.

- SAD responds to a variety of antidepressants that are also efficacious for nonseasonal major depression [84-87]. However, this argument is undermined by the fact that antidepressants are efficacious for several disorders other than nonseasonal major depression, including anxiety disorders, bulimia nervosa, and fibromyalgia.

- Response to light therapy is not specific to SAD. Multiple randomized trials, especially high quality trials, have demonstrated that bright light therapy is efficacious for nonseasonal depression [88-93]. However, three observational studies each found that the benefit of bright light therapy was greater in patients with SAD than patients with nonseasonal depression [89].

- SAD is uncommon in at least one northern country, Iceland [94]. However, this finding may be explained by genetic factors; the prevalence of SAD is lower in Canadians of Icelandic descent compared with non-Icelandic Canadians [95].

- One study examined depression in a random sample of adults (n >34,000) who lived in different geographical areas in the United States and were assessed with a single, cross-sectional telephone survey at various times of the year [96,97]. The study found no relationship between the level of depression and the season of the year or latitude, and questioned the validity of seasonal affective disorder. However, the study did not ask subjects about a seasonal pattern, had no longitudinal assessment in an illness characterized by its longitudinal course, and had other methodologic problems [98].

## INFORMATION FOR PATIENTS

UpToDate offers two types of patient education materials, “The Basics” and “Beyond the Basics.” The Basics patient education pieces are written in plain language, at the 5th to 6th grade reading level, and they answer the four or five key questions a patient might have about a given condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials. Beyond the Basics patient education pieces are longer, more sophisticated, and more detailed. These articles are written at the 10th to 12th grade reading level and are best for patients who want in-depth information and are comfortable with some medical jargon.

Here are the patient education articles that are relevant to this topic. We encourage you to print or e-mail these topics to your patients. (You can also locate patient education articles on a variety of subjects by searching on “patient info” and the keyword(s) of interest.)

- Basics topics (see "Patient education: Seasonal affective disorder (The Basics)")

Additional information for patients about seasonal affective disorder is provided by the Society for Light Therapy and Biological Rhythms and Center for Environmental Therapeutics.

## SUMMARY

- Seasonal affective disorder (SAD) is defined as recurrent episodes of major depression, mania, or hypomania with seasonal onset and remission. It is not considered a separate mood disorder; rather, SAD is a subtype of the following mood disorders: unipolar major depressive disorder, bipolar I disorder, and bipolar II disorder. (See 'Seasonal affective disorder' above.)

- The estimated lifetime prevalence of SAD in the general population is approximately 0.5 to 3 percent. The point prevalence in primary care outpatients is approximately 5 to 10 percent, and in depressed outpatients is 15 percent. (See 'Epidemiology' above.)

- The pathogenesis of SAD is not known; the primary hypotheses involve disturbances of circadian rhythms, decreased sensitivity of the retina, genetic factors, and dysregulation of neurotransmitters. (See 'Pathogenesis' above.)

- Two specific patterns of recurrent unipolar major depression with seasonal pattern have been described. Fall-winter onset SAD (winter depression) begins in the fall to early winter and, if left untreated, generally remits during the following spring and summer. Spring-summer onset SAD (summer depression) begins in the spring or summer and remits

during the following fall and winter. Winter depression is far more common than summer depression. (See 'Seasonal pattern' above.)

- The symptoms of major depression in patients with fall-winter onset SAD and in patients with nonseasonal major depression are the same, and include depressed mood, loss of pleasure or interest, change in appetite or weight, sleep disturbance, loss of energy, neurocognitive dysfunction, psychomotor agitation or retardation, feelings of worthlessness or guilt, and suicidal ideation or behavior ( table 1). However, major depressive episodes in patients with SAD usually include atypical features, such as hypersomnia, hyperphagia (especially carbohydrates), and weight gain. (See 'Symptoms' above.)

- The seasonal pattern of recurrent unipolar major depression is generally not a long-term phenomenon. Among patients who are initially diagnosed with fall-winter onset SAD and subsequently evaluated (eg, 5 to 11 years later), recurrent depression with seasonal pattern persists in less than 50 percent. (See 'Course of illness' above.)

- Patients with unipolar major depression and a possible diagnosis of fall-winter onset SAD are assessed for recurrent depressive episodes with a seasonal pattern. The interview includes the following questions:

  - Do you feel worst (more sad) in the winter?
  - Do you eat more in the winter?
  - Do you gain weight in the winter?
  - Do you sleep more in the winter?
  - Do you have less energy in the winter?
  - Do you socialize with other people less often in the winter?
  - Is the depression ameliorated by sunnier weather?
  - Does the depression resolve during travel to a sunnier location or a location with a longer photoperiod?
  - Does the depression remit during the spring and/or summer?

(See 'Seasonal pattern' above.)

- The diagnosis of SAD requires that patients meet criteria for a recurrent mood disorder with seasonal pattern. In patients who have recurrent unipolar major depression ( table 1) with a fall-winter onset seasonal pattern, each of the following is present:

  - There is a regular temporal relationship between the onset of major depressive episodes and a particular time of year (fall or winter). However, episodes that occur in response to seasonally related psychosocial stressors (eg, unemployment every winter) do not count toward meeting this criterion.

  - Remission of the episodes also occurs at a characteristic time of year (spring or summer).

  - In each of the last two years, onset and offset of a major depressive episode has occurred at a characteristic time of year, and there have not been any nonseasonal episodes.

  - The lifetime course of illness is such that seasonal depressive episodes substantially outnumber the nonseasonal episodes (eg, by a ratio of two to one).

  (See 'Unipolar major depression' above.)

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