Seasonal affective disorder: an overview

Cheryl Zauderer and C Anne Ganzer outline the diagnosis, causes and treatment options for people who experience the winter blues

Abstract

The definition of seasonal affective disorder (SAD) is a history of major depressive episodes and remissions that are seasonal, affecting mostly women during the winter months in colder countries. Symptoms commonly begin in the autumn, peak in midwinter and subside in the spring. The exact cause is unknown; however, there are several contributing factors including seasonal change of light affecting internal circadian rhythms and secretion of the hormone melatonin, genetic inheritance and diet. Mental health care clinicians are in an ideal position to promptly recognise the condition and implement treatment that may help limit the severity of the depressive symptoms. The degree of illness can vary, as well as the timing of onset and resolution and may depend on geographical location. Treatments include light therapy, diet, exercise, psychotherapy and antidepressant medication.

Keywords
Circadian rhythm, depression, light therapy, seasonal affective disorder

COMMONLY KNOWN as ‘winter blues’, seasonal affective disorder (SAD) is defined as a history of depressive episodes that recur regularly, typically in autumn or winter, and completely remit in the spring (Howland 2009). According to Frontier Behavioral Health (2014) and Psychcentral (2003), women make up 60-90% of people with SAD. The condition is predominantly seen in women between the ages of 20 and 50, occurring more often in climates where there is less sunlight and winter months are colder (Zender and Olshansky 2009). In the general population, the annual worldwide incidence of SAD is believed to be around 5%. The average duration of a SAD episode is approximately 3.9 months, or about 33% of the year (Kurlansik and Ibay 2012). SAD, also known as winter depression, can be seen in any country or state where there is not tropical weather all the year round and therefore there is some form of winter. Some countries where the population consumes a lot of fish, such as Iceland, have been shown to have lower rates of SAD; this is perhaps associated with higher levels of omega-3 fats in the diet (Magnússon and Axelsson 1993, Magnússon et al 2000).

There is also a strong genetic component to SAD. The melanopsin gene is a light-sensitive protein that is believed to originate in the retina of individuals. This gene is not concerned with vision, but can be linked to non-visual responses, for example, circadian rhythms, hormones, alertness and sleep. These mutations may cause some individuals to be predisposed to SAD. Individuals who carry two copies of this gene mutation will have a higher probability of experiencing the disorder (University of Virginia 2008).

The disorder can be distinguished from depressive disorder in that onset of symptoms is triggered by a particular season, typically the darker winter months. A small percentage of people may experience ‘summer blues’ in the warmer months, triggered by discomfort from heat and humidity particularly if they live in more tropical climates (Gill and Saligan 2008).

Diagnosis
According to the American Psychiatric Association’s DSM-5 (2013) criteria, SAD is not to be regarded as a separate psychiatric mental health disorder. The condition is considered a non-specific major depressive disorder with recurrent episodes in a seasonal pattern. The diagnosis depends on meeting four criteria (Rosenthal 2009, American Psychiatric Association 2013):

- A regular temporal relationship between onset of major depressive episodes in major depressive disorder and a particular time of the year (in the autumn or winter).
- Full remissions (or change from major depression to mania or hypomania) also occur at a characteristic time of the year (depression disappears in the spring).
In the past two years, two major depressive episodes have occurred that demonstrate the temporal seasonal relationships defined above, and no non-seasonal major depressive episodes have occurred during that same period.

Seasonal major depressive episodes (as described above) substantially outnumber the non-seasonal major depressive episodes that may have occurred over the individual’s lifetime. The onset of SAD is generally in October or November for those who live north of the equator, and June or July for those who live south of it, and symptoms do not resolve until the spring (Moreland 2010). The degree of illness can vary, as well as the timing of onset and resolution and may depend on geographical location (Timby and Smith 2005). SAD is characterised by minor changes in behaviour, such as low energy levels, disturbed sleep patterns, poor appetite or eating too much and general mood swings and sadness (Timby and Smith 2005). Symptoms of SAD can also affect sex drive (Gagne et al 2010).

Healthcare practitioners evaluating patients should be alerted by functional impairment severe enough to interfere with work schedules, not being able to complete tasks that were once easily managed, excessive guilt and crying spells, negative thoughts about self, and diminished physical functions, such as generalised fatigue throughout the day, and a lack of control over eating (Howland 2009, Rosenthal 2009). People who experience SAD may report that they prefer to be socially isolated, much like being in ‘hibernation mode’. Clinicians should determine the relationship between a client’s depression and the time of year and establish whether seasonal patterns occur (Rankin 2011). Screening tools such as the Seasonal Pattern Assessment Questionnaire (SPAQ) (Box 1) can be used to determine whether an individual’s experience meets the DSM-5 criteria of SAD, but should not replace sound clinical judgement (Gupta and Sharma 2008).

Physically, SAD is also associated with somatic complaints such as backaches, muscle aches, headaches, influenza-like symptoms and fibromyalgia, and a worsening of premenstrual syndrome. People commonly report that they self-medicate with large amounts of caffeine, alcohol, nicotine and illegal substances (Virk et al 2009). Differential diagnosis should include the evaluation of possible mental health disorders such as dysthymia, major depression, premenstrual syndrome and eating disorders.

There are several medical conditions that can mimic SAD, and clinicians should assess for underlying clinical syndromes such as hypothyroidism and hypoglycaemia and viral illnesses such as Epstein Barr infection and chronic fatigue syndrome. A complete physical examination, blood tests including full blood count, thyroid levels, a metabolic panel and liver function, and a pregnancy test should be performed to rule out general medical conditions. Hypothyroidism and dementia can mimic depression, and pregnancy, menopause and other hormonal conditions can affect mood also (Birtwistle and Martin 1999).

Circadian rhythm

Clinically, SAD is considered a chronic condition that does not necessarily predispose individuals to major depression (Laskoski 2010). Specific causes of SAD remain unknown; however, there is an established link to a seasonal deprivation from light, which changes the internal clock that determines our circadian rhythm.
A circadian rhythm is a repeated physical, mental and behavioural change that goes through generally a 24-hour cycle and responds to light and darkness in the environment. Humans, animals, plants and many other organisms can exhibit the effects of a circadian rhythm. The study of this phenomenon is known as chronobiology (National Institute of General Medical Sciences 2012)

A reduced amount of natural sunlight causes changes in the body’s circadian rhythm, affecting mood, energy levels, sleep and eating patterns, focus and attention (Targum and Rosenthal 2008). A decrease in exposure to sunlight has been shown to extend the production of the sleep-inducing ‘hormone of darkness’ or melatonin that functions to regulate sleep cycles (Pail et al 2011). People experiencing SAD have elevated levels of melatonin that remain high until the spring, when there is more sunlight and the days become longer (Timby and Smith 2005). Another possible aetiological factor has to do with the level of serotonin in the brain, which is typically stimulated by sunlight (Birtwistle and Martin 1999). Low levels of serotonin have been linked to mood disorders, especially depression (Gagne et al 2010). Other causes may include genetic or familial susceptibility or other seasonal environment factors (Virk et al 2009).

**Light therapy**

Non-pharmacological treatments for SAD are primarily aimed at increasing exposure to direct, natural sunlight. People with the condition should be encouraged to obtain as much sunlight as possible during the day, taking walks outside, limiting the use of sunglasses and keeping the home and workplace well lit with lamps and bright lights (Timby and Smith 2005). For some individuals, travelling to a sunny climate in the middle of the winter can help disrupt the cycle of SAD (Rosenthal 2009); in some cases, individuals with severe SAD may consider relocating permanently to a sunnier climate.

Light therapy is an effective treatment as, properly used, it rectifies the individual’s circadian rhythm and targets the depressive symptoms promptly (Virk et al 2009). The person needs to sit by a light replacement for between 30 minutes and two hours per day, if there is no available natural sunlight. A typical effective dose would be 2,500lux for a duration of two to three hours, or a dose of 10,000lux for a shorter duration of minutes (Howland 2009, Privitera et al 2010, Pail et al 2011). There are commercial light boxes available specifically for the treatment of SAD. Many of these light boxes can be purchased online. Light therapy for treating SAD helps make up for lost exposure to the sun and reboots the body’s internal clock.

Light therapy can be used alone or in conjunction with antidepressants in the treatment of SAD, and is safe and easy (Bhattacharjee 2007). It can be used during pregnancy and in lactating women in lieu of antidepressant medication, if the new mother does not wish to take any medication while she is breastfeeding. Bright light therapy can be received from 30 minutes to three hours, once or twice a day, depending on the symptoms and the type of light used. Studies have shown that those who used a 10,000-lux system for 30 minutes each day had marked improvement in their depressive symptoms (Columbia University 2012). Side effects of light therapy include eyestrain, headache, nausea, anxiety, nervousness and irritability (Eastman 2011).

Exercise is an excellent way to enhance the mood (Dinas et al 2011). Alternative mind-body therapies that may help relieve depression symptoms include acupuncture, yoga, meditation, guided imagery and massage therapy (Javnbakht et al 2009).

Cognitive behaviour therapy and interpersonal psychotherapy have been found helpful in the treatment of SAD (Stangier et al 2011). Both of these methods tend to accentuate the effectiveness of treatment and therefore should be included in the total approach to addressing this disorder or as adjuvant to medical treatment.

Non-pharmacological treatments for SAD include dietary supplementation with vitamin D at 1,000-2,000IU (international unit) per day (Howland 2011). The addition of supplements in the category of omega-3 fatty acids has also been shown to improve mood disorders and is a safe alternative or adjunct to other treatment for depression (Freeman et al 2006).

Another dietary consideration is the maintenance of high levels of protein, including nuts and low-fat cheeses, and green vegetables and fruits (Palinkas 2010). Although individuals who experience SAD often crave carbohydrates, it is important to encourage them to limit refined carbohydrates such as sugar and bleached white flour. Carbohydrate consumption can increase serotonin release, so people may tend to overeat carbohydrates (especially snack foods, like potato chips or pastries, which are high in carbohydrates and fats) to feel better. This tendency to use comfort foods as though they were medicine can be a cause of undesirable weight gain, and may be observed in some people experiencing SAD (Wurtman and Wurtman 1995).

**Pharmacological therapies**

SAD is a treatable illness, with many therapeutic options available including antidepressant therapy. In general, the treatment choice depends on the degree of depression and other accompanying factors. Options include serotonin reuptake inhibitors (SSRIs), serotonin-norepinephrine reuptake inhibitors (SNRIs), atypical antidepressants,monoamine oxidase inhibitors (MAOIs), tricyclic antidepressants (TCAs) and monoaminergic agents. Among the available antidepressants are selective serotonin reuptake inhibitors (SSRIs), which are effective, well tolerated and well studied as first-line treatment for SAD.

Many of these SSRIs are effective for SAD (American Psychiatric Association 2000). Venlafaxine (Effexor) and duloxetine (Cymbalta) may also be considered. It is prudent to treat SAD with an antidepressant by itself, without the need for mood stabilizers. Antidepressants are generally started at low doses and titrated upward to achieve an effective dose.

Other treatment options include psychotherapy, such as cognitive-behavioural therapy (CBT) and interpersonal psychotherapy (IPT). CBT focuses on the individual’s core beliefs about themselves and the world, while IPT focuses on the individual’s relationships with others and the interpersonal relationships that may be contributing to their depression or anxiety. These therapies can be provided as individual or group therapy and can be delivered by mental health professionals such as psychologists, psychiatrists, social workers, nurses and nurse practitioners. Cognitive-behavioural therapy is a form of psychotherapy that helps people change the negative thought patterns and patterns of living that contribute to their depression, anxiety, panic attacks, phobias, eating disorders or other mental health problems. It is based on the assumption that people are largely responsible for their thoughts, feelings and actions and that many of these are learned and can be unlearned. Cognitive-behavioural therapy helps people identify and change these negative patterns of thinking.

Antidepressant therapy and psychotherapy are often used together to help people with SAD, and both are often recommended as the first-line treatment for depression. Antidepressants can help improve mood, energy, sleep, appetite, concentration and the ability to participate in everyday activities. Psychotherapy can help people understand their thoughts and feelings and develop strategies to manage their symptoms. Cognitive-behavioural therapy can help people develop new thinking patterns and learn new ways of coping with stress. However, antidepressant therapy may be more effective than psychotherapy for some people with SAD. Antidepressant therapy can help reduce the severity of symptoms and prevent future episodes of depression. Psychotherapy can help people develop new coping strategies and improve their overall well-being. 

In addition to antidepressant therapy and psychotherapy, other treatment options include non-pharmacological treatments such as light therapy, exercise, dietary changes and alternative therapies such as acupuncture, yoga, meditation, guided imagery and massage therapy. These treatments can help improve mood, energy, sleep, appetite, concentration and the ability to participate in everyday activities.

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conditions. It also may depend on age, pregnancy status or other individual factors. Antidepressants have been shown to be an effective therapy in the psychopharmacological treatment of SAD. Clinicians should target medications such as those that inhibit the reuptake of serotonin (Erren et al 2011). Examples include fluoxetine, sertraline, paroxetine and citalopram. Bupropion XL is also an approved treatment for SAD (Thaler et al 2011). Research into the use of bupropion antidepressant therapy before symptoms typically begin has shown to decrease symptoms and improve outcomes (Modell et al 2005).

Alternative pharmacological therapies include the use of stimulants such as modafinil (Provigil) as an effective treatment for hyposomnia (Lundt 2004). Due to the lack of research in this subject in general and the effects of the treatments available for SAD it is difficult to offer patients an accurate prognosis (Fallowfield and Jenkins 2004).

Conclusion

Seasonal affective disorder is a common, often undiagnosed, mood disorder that affects women more than men. Nurses working in the area of women’s health should have an understanding of identification, diagnosis and available treatment of SAD. Clinicians should evaluate clients who report repeated seasonal depression and advise about prevention methods. Starting treatment during the autumn or early winter, before the symptoms of SAD begin, and continuing therapy through the early spring season may diminish symptoms and possibly eliminate episodes.

Implications for practice

- Seasonal affective disorder (SAD) is a form of major depressive disorder that exhibits predictable, seasonal episodes and remissions.
- SAD manifests most commonly among women aged 20 to 50 years experiencing winter in colder, dark countries.
- The degree of illness can vary, as well as the timing of onset and resolution, and may depend on geographical location and climate.
- Treatments include light therapy, diet, various forms of exercise, psychotherapy and antidepressant medication.

References


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