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Seasonal affective disorder

Seasonal affective disorder is characterised by depression during the shorter day length of the winter months and remitting symptoms in the spring or summer. Phototherapy has been shown to be an effective treatment for this condition.

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'Now is the winter of our discontent…'
Shakespeare: Richard III, act 1, scene 1.

Wintertime has long been known to be a time of low mood or 'discontent', as observed by Shakespeare. A specific type of depression that regularly occurs in wintertime – seasonal affective disorder (SAD) – has now been recognised. Astute clinicians have been aware of the influence of seasons on mood since the times of antiquity. Peaks of depression and suicide were noted in the springtime. Depressive episodes were linked with the winter season and episodes of mania occurred in the summer months. This 'seasonality' of symptoms was considered to be related to a patient's poor adaptation to the changing seasons, particularly the changes in day length;[1] poor adaptation leads to disruption of circadian rhythms that trigger mood changes.

History of SAD

In the 1980s, the concept of seasonality was extended when a specific type of depression was identified. It was found to be linked to a particular season, and a new form of treatment (bright light therapy) based on its putative aetiology (short day length) was described.

Rosenthal and colleagues described a group of 29 patients with a syndrome that they labelled 'seasonal affective disorder'.[2] These patients had recurrent episodes of depression in the wintertime that spontaneously remitted in the spring or summer seasons. The depression was characterised by the 'atypical' features of hypersomnia and overeating, with carbohydrate craving.

The SAD syndrome has now been confirmed in many studies across many countries, including Australia.[3] There is a summer variant of the condition, which is characterised by depression in the summer months (with a more melancholic type of presentation) and remission in winter.

In the cohort of patients in Rosenthal's study, a patient from a northern state of the USA described how her depression remitted when she travelled to a southern state of the USA with a longer length of day.[2] This led to the development of using bright

IN SUMMARY

- **Seasonal affective disorder (SAD) has been identified in people in many countries, including Australia.**
- **The onset of SAD typically occurs in young adulthood and it affects women more commonly than men.**
- **The patient needs to be able to accurately recall his or her symptom history, particularly the timing of episodes, in order for a diagnosis of SAD to be made.**
- **There are several competing hypotheses for the cause of SAD including circadian rhythm disruption and altered neurotransmitter function.**
- **Bright light therapy, or phototherapy, is the current first-line treatment for patients with SAD.**

continued

Table 1. Diagnostic criteria for *DSM-IV* TR seasonal pattern specifier[4]

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 relation 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 autumn or winter).

   Note: Do not include cases in which there is an obvious effect of seasonal-related psychosocial stressors (e.g. regularly being unemployed every winter).

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 two years, two major depressive episodes have occurred that demonstrate the temporal seasonal relations 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.

lights to artificially extend the length of day in these patients with good therapeutic effect. The early success and novelty of bright light therapy generated a new industry in manufacturing light boxes specifically for the treatment of SAD.

Diagnosis and clinical features of SAD

SAD quickly gained official recognition. In 1987 a 'seasonal pattern' of depression or bipolar disorder was included in the US classification system, the *Diagnostic and Statistical Manual* (*DSM*).[4] The specifier requires that the person meets the diagnostic criteria for major depression and has symptoms that are present for at least two consecutive years with spontaneous remission occurring at a characteristic time of year (Table 1). The most common form of SAD is winter depression with spring or summer remission.

The onset of SAD typically occurs in young adulthood, and it affects women more commonly than men. The prevalence of SAD varies according to latitude, with the rates being highest in subpolar regions (characterised by long winter nights and a short photoperiod) and lowest in equatorial regions such as Africa, with an estimated overall annual prevalence of 0.8 to 1.2%.[5] The prevalence of SAD in Australia is unknown but it is estimated that it would be one of the lowest worldwide rates. SAD is not common in Australia, where the winter nights are not as long as those found in the northern hemisphere, but it does still occur and can lead to significant impairment.

The challenge when making a diagnosis of SAD is that the patient needs to be able to accurately recall his or her symptom history, particularly the timing of episodes.[6] This can be helped by anchoring the time when symptoms arose or remitted to 'seasonal' milestones such as the end of daylight saving, ANZAC Day or the June long weekend for those with winter depression.

Although it is largely the timing of symptom onset that determines a diagnosis of SAD, those who experience this type of depression tend to present with some 'atypical' depressive symptoms. Patients complain of hypersomnia and feeling fatigued. They tend to go to bed earlier than usual, but have poor-quality sleep and difficulty waking up in the morning. Rather than losing their appetite, patients have an increase in appetite with carbohydrate craving (eating 'junk' food) with associated weight gain (which they often lose in the summer).

They will also have some of the more 'typical' features of depression, such as decreased activity and complaints of sadness, irritability and anxiety (Table 2).[7] Patients will report poor functioning in winter, but when the spring comes their functioning improves. For example, a patient's work performance and productivity were very poor during the winter when he was depressed and his job was under threat. However, his performance changed dramatically when daylight saving started in October and his depression remitted; over one weekend he was able to catch up with everything he had not been able to do over the winter and he continued to function at a high level until the next winter.

Interpersonal relationships can also be

dramatically affected with deterioration of relationships during episodes of depression, especially when irritability is one of the symptoms. Patients with a summer variant of SAD will report the more classic melancholic symptoms of depression characterised by early morning awakening, loss of appetite and loss of weight.

SAD and bipolar disorder

There are links between SAD and bipolar disorder; Rosenthal and colleagues originally considered SAD to be a form of bipolar II disorder.[2] There are two reasons for this:

- patients with SAD, rather than simply returning to normal functioning in the spring or summer, may present with hypomanic or even manic symptoms at this time of year
- atypical depressive features (such as hypersomnia and increased appetite) present in patients with SAD are considered to be signals of bipolar depression.

SAD is a subtype of bipolar disorder, with the distinguishing feature being the seasonality of symptoms in SAD. Assessment of patients with SAD requires a thorough review of their functioning to determine whether they have episodes of mania that require treatment with mood-stabilising medication such as lithium or carbamazepine. They should also be counselled about the possibility that they may have hypomanic mood swings, although the hypomanic symptoms would be expected to switch to depression in the winter. It is unknown what percentage of patients with SAD have mania episodes.

Aetiology of SAD

The aetiology of SAD was initially considered to be a consequence of the short photoperiod of winter. This was supported by the efficacy of bright light therapy that was deemed to be effective because it artificially extended the photoperiod.[8,9] However, as research has progressed, the reason for the effectiveness of bright light therapy has become less clear. There are now several competing hypotheses for the cause of SAD, including circadian rhythm disruption and altered neurotransmitter function.

A number of climatic variables, including temperature and day length, were considered to be causal factors for SAD. SAD was found in countries that experienced the winter cold of the northern hemisphere, so it was thought that the cold was a factor. More robust was the evidence for an association with the changing photoperiod (day length) and the short photoperiod of winter being the climatic factor implicated in the onset of the depressive episode.[3]

Melatonin hypothesis

The pineal hormone melatonin is secreted at night, with bright artificial light suppressing its secretion. The initial hypothesis was that the secretion of melatonin was prolonged in those with SAD, leading to the onset of depressive symptoms. However, despite the differences in melatonin secretion between patients with SAD and controls, light therapy causes a similar effect in both. Bright light therapy will suppress melatonin in healthy controls, as well as in patients with SAD.

There is no antidepressant effect of suppressing melatonin in healthy subjects, but there is an antidepressant effect among patients with SAD. The reason for this is that the light affects the timing of melatonin secretion; when light therapy is used in the morning it pushes forward melatonin secretion, which is delayed in SAD. In addition, it has been shown that there is no difference in melatonin secretion after light therapy between responders and nonresponders; this suggests that perhaps melatonin secretion is not a direct cause of the symptoms in seasonal depression.[10]

Circadian rhythm hypothesis

It is well established that light is one of the most important environmental cues for entrainment of mammalian circadian rhythms.[9] As a result, circadian rhythm disruption was hypothesised to be a cause of SAD, with SAD being linked to a phase delay of internal circadian rhythms.[11] Bright light therapy, when applied in the morning, has been shown to have an effect on circadian rhythms, shifting them forward and therefore overcoming the phase delay of SAD.

Table 2. Symptoms of seasonal affective disorder

Atypical depressive symptoms
Hypersomnia
Fatigue
Difficulty sleeping
Difficulty waking after sleep
Increased appetite for carbohydrates
Typical depressive symptoms
Decreased activity
Sadness
Irritability
Anxiety
Summer variant: melancholic symptoms of early wakening, loss of appetite and loss of weight.

Serotonin hypothesis

An alternative hypothesis is that serotonin production is altered in patients with SAD. Whole brain serotonin turnover is decreased during the winter months even in healthy individuals,[12] and turnover is altered by acute changes in luminosity; levels are higher on brighter days, regardless of season. In addition, patients have been successfully treated for SAD relapse following tryptophan depletion.[13]

The possible involvement of serotonin in SAD also explains the atypical symptoms of carbohydrate craving and weight gain because of the role serotonin plays in satiety and feeding regulation.[14]

Treatment of SAD

Despite the divergent and often conflicting research into the aetiology of SAD, its treatment has demonstrated efficacy, is widely publicised and frequently used by clinicians.

Phototherapy

Bright light therapy, or phototherapy, is the current first-line treatment for patients with SAD. It is thought that the light produces a therapeutic effect by causing a phase advance of the circadian rhythm.[9] Research has examined the effect of varying the time of day during which phototherapy is administered. Although some researchers have found no difference, most findings show that treatment early in the morning demonstrates the best efficacy.[15]

Phototherapy involves exposing patients with SAD to light of between 2500 and 10,000 lux from a distance of about 0.5 to 1 metre.[6] This intensity of light is considerably higher than that given by normal artificial light – the luminosity of standard artificial light such as a standard light bulb used in the home is about 250 lux. The light intensity is directly related to the length of treatment, with patients using lights of 2500 lux requiring two hours of daily exposure, compared with just 30 minutes for those using lights of 10,000 lux.[16]

Phototherapy should be administered on awakening if possible. The patients need to have their eyes open; however, they do not need to look directly at the light, so they may perform other activities while sitting in front of the light box. This form of treatment can be administered at home or at a specialised clinic.[5] For patients who prefer the convenience of home treatment, portable light boxes are available (for more information see www.litebook. com or www.northernlighttechnologies.com).

Light therapy is the most natural and noninvasive treatment of SAD. The side effects of phototherapy, such as eye strain, headaches and irritability, are mild and relatively short lived and the treatment is, therefore, generally well tolerated.[6]

Pharmacotherapy

The second-line treatment for patients with SAD is based on the theory that altered serotonin production is part of the cause of SAD symptoms.[5] Therefore, treatment with SSRI antidepressants such as fluoxetine and sertraline has been tested, and shown to be effective.[6]

A new antidepressant, agomelatine, has been shown to be effective in the treatment of SAD. An agonist of melatonergic receptors (MT_1 and MT_2) and a $5HT_{2C}$ antagonist, agomelatine has been shown in patients with SAD to have a response rate of 75.7% and a remission rate of 70.3% in a recent open pilot study;[17] however, this may only reflect a placebo response. It is not yet available in Australia, but it has been licensed for use in Europe. It is hoped that it will be available in 2010.

Behavioural interventions

Behavioural interventions are also helpful in the treatment of patients with SAD. Based largely on the circadian rhythm hypothesis and successful treatment protocol of phototherapy, strategies implemented in the morning can help maintain the rhythms that are disrupted in these patients. As outdoor light is significantly brighter than indoor light, even on a cloudy and rainy day, spending time outside is a natural form of treatment for SAD.

Wirz-Justice and colleagues found that a one-hour morning walk significantly reduced depressive symptoms and improved sleep.[18] In addition, it was found that exposure to outdoor light, but not artificial light, suppressed sweet carbohydrate cravings in the second half of the day.

It may also be beneficial to make adjustments to the sleep environment, such as leaving curtains open to allow dawn light into the room, especially from an east-facing bedroom. Patients with SAD should also make an attempt to maintain regular rhythms, and force themselves to rise on first waking in the morning.

Stabilising rhythms by eating a regular breakfast and, where possible, exercising outdoors may be beneficial in decreasing depressive symptoms. Patients should maintain a regular sleep wake cycle, which can be enhanced by having regular daily routines, particularly fixed mealtimes.

Conclusion

SAD is characterised by depressive symptoms in autumn or winter, and spontaneous remission or hypomania in the spring or summer. Although the specific cause of the disorder is as yet unknown, it is widely believed that it is the decreased photoperiod that accompanies the winter months that triggers the depressive symptoms. Patients with SAD are likely to present with more atypical symptoms, such as hypersomnia and carbohydrate craving, than those with nonseasonal depression.

SAD can be effectively treated with light therapy or with antidepressants such as SSRIs. Medications should be limited, however, to the depressive period and behavioural interventions such as early rising and exposure to outdoor light should be encouraged. MT

References

A list of references is available on request to the editorial office.

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References

1. Boyce PM. 6-Sulphatoxy melatonin in melancholia. Am J Psychiatry 1985; 142: 125-127.
2. Rosenthal NE, Sack DA, Gillin JC, et al. Seasonal affective disorder – a description of the syndrome and preliminary findings with light therapy. Arch Gen Psychiatry 1984; 41: 72-80.
3. Boyce P, Parker G. Seasonal affective disorder in the southern hemisphere. Am J Psychiatry 1988; 145: 96-99.
4. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders. 4th ed, text revision. Washington DC: American Psychiatric Association; 2000.
5. Partonen T, Lonnqvist J. Seasonal affective disorder: a guide to diagnosis and management. CNS Drugs 1998; 9: 203-212.
6. Westrin A, Lam RW. Seasonal affective disorder: a clinical update. Ann Clin Psychiatry 2007; 19: 239-246.
7. Hellekson C. The phenomenology of seasonal affective disorder: an Alaskan perspective. In: Rosenthal NE, Blehar MC, eds. Seasonal affective disorder and phototherapy. New York: The Guilford Press; 1989. p. 33-45.
8. Golden RN, Gaynes BN, Ekstrom RD, et al. The efficacy of light therapy in the treatment of mood disorders: a review and meta-analysis of the evidence. Am J Psychiatry 2005; 162: 656-662.
9. Hill MA. Light, circadian rhythms, and mood disorders. Ann Clin Psychiatry 1992; 4: 131-146.
10. Miller AL. Epidemiology, etiology, and natural treatment of seasonal affective disorder. Altern Med Rev 2005; 10: 5-13.
11. Lewy AJ, Sack RL, Miller S, Hoban TM. Antidepressant and circadian phase-shifting effects of light. Science 1987; 235: 352-354.
12. Lambert GW, Reid C, Kaye DM, Jennings GL, Esler MD. Effect of sunlight and season on serotonin turnover in the brain. Lancet 2002; 360: 1840-1842.
13. Neumeister A, Turner EH, Matthews JR, et al. Effects of tryptophan depletion vs catecholamine depletion in patients with seasonal affective disorder in remission with light therapy. Arch Gen Psychiatry 1998; 55: 524-530.
14. Lam RW, Levitan RD. Pathophysiology of seasonal affective disorder: a review. J Psychiatry Neurosci 2000; 25: 469-480.
15. Lewy AJ, Bauer VK, Cutler NL, et al. Morning vs evening light treatment of patients with winter depression. Arch Gen Psychiatry 1998; 55: 890-896.
16. Lam RW, Tam EM, Yatham LN, Shiah I-S, Zis AP. Seasonal depression: the dual vulnerability hypothesis revisited. J Affective Disorders 2001; 63: 123-132.
17. Prjek E, Winkler D, Konstantinidis A, Williet M, Praschak-Rieder N, Kasper S. Agomelatine in the treatment of seasonal affective disorder. Psychopharmacology 2007; 190: 575-579.
18.Wirz-Justice A, Graw P, Krauchi K, et al. 'Natural' light treatment of seasonal affective disorder. J Affective Disord 1996; 37: 109-120.