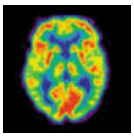


Seasonal affective disorder

Module 4 13.7.1 mood disorders

This activity will help you to…

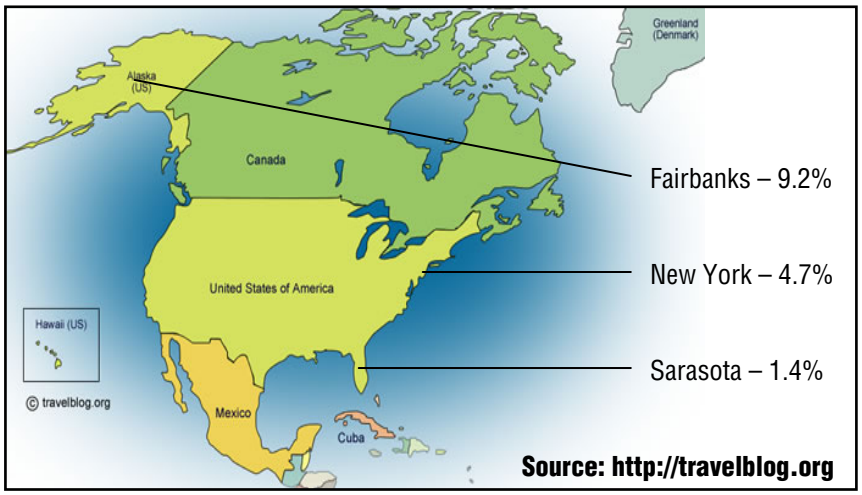
- Understand research into the nature, causes and treatment of SAD

What is seasonal affective disorder?

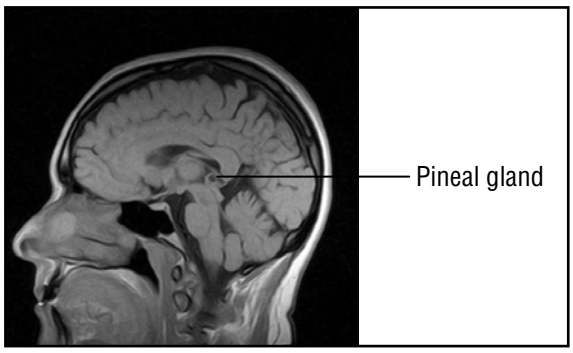
Because the earth tilts on its axis, the number of daylight hours increases in summer and decreases in winter. It is now widely acknowledged that, in northerly latitudes, a reduction in exposure to natural light is connected to an apparent rise in depressive symptoms during the winter. This 'winter depression' or **Seasonal Affective Disorder** (SAD) shares many features with other depressive disorders, being characterised by low mood, feelings of guilt and worthlessness, increased irritability and lack of energy. However, SAD sufferers tend to show increased sleep, appetite and weight gain rather than the disrupted sleep and weight loss associated with more typical depressive episodes (Barlow and Durand, 1995). Under the DSM IV system, 'seasonal pattern' is an additional specifier for mood disorder rather than a discrete diagnostic category. Seasonal pattern for mood disorder can be diagnosed if there is a clear association between the onset of mood disorder symptoms and the time of year, with depressed symptoms appearing during winter and manic symptoms (if present) appearing in summer. It is necessary for the association between time of year and symptom onset to have been present for at least two years.

Why would people get depressed in winter?

A range of evidence suggests a link between seasonal depression and exposure to daylight. For example, it appears to become more common the further away one moves from the equator. This should be expected, as at more extreme latitudes the days are shorter in winter. Brooker and Hellekson (1992) estimated the prevalence of SAD in different areas of the US using mail and telephone surveys. They found that in Satasota, Florida (27° North) the prevalence of SAD was 1.4%, rising to 4.7% in New York City (41° North) and 9.2% in Fairbanks, Alaska (65° North). The same trend was evident in the prevalence of subsyndromal (i.e. mild) SAD, which was 2.6% in Sarasota, compared to 19.1% in Fairbanks. Other studies have found that SAD sufferers experience no symptoms if they spend the winter in tropical latitudes (Pande, 1985) and that they show a preference for brightly lit environments (Heerwagen, 1990).



The apparent link between SAD and daylight has led research to focus the search for explanations on the hormone **melatonin**. This chemical, whose main function is the regulation of the sleep-wake cycle is produced by the pineal gland in a rising and falling 24 hour pattern (i.e. it follows a **circadian rhythm**). Melatonin levels rise in darkness and production is inhibited by light. The link between SAD and abnormalities in melatonin-dependent brain systems is supported by a number of observations. First, the relative lack of daylight during the non-tropical winter leads to generally



higher levels of melatonin. Since melatonin increases sleepiness, this is consistent with the increased lethargy and extended sleep periods found in SAD sufferers. Second, the circadian rhythms of SAD sufferers differ from those of clinically normal individuals. Generally, body temperature rises and falls over a circadian cycle, peaking at about 3pm and reaching a low point at about 3am. In SAD sufferers, this rhythm appears to be phase advanced, so that it peaks and troughs later than in non-sufferers (Kalat, 1998). As circadian cycles in body temperature are usually synchronised with the sleep-wake cycle, this further implicates melatonin as a factor in SAD.

Direct evidence is provided by Wehr et al (2001) who found that, in winter, SAD patients produced melatonin for significantly longer than a matched sample of healthy volunteers. They suggest that this change in melatonin secretions is analogous to the changes that signal seasonal behaviour changes (e.g. migration, food gathering) in non-human animals, and it has been suggested that SAD represents an evolutionary remnant of seasonal behaviour change from an earlier stage in human development. However, the causes of SAD are likely to involve many more brain systems than just those dependent on melatonin. A number of studies also implicate the neurotransmitter serotonin (Garcia-Borreguero et al, 1995; Schwartz et al, 1997) which is not surprising given that serotonin levels are closely related to melatonin secretion and that serotonin is itself implicated in a variety of mood disorders. Other researchers have linked SAD to retinal abnormalities. Lam et al (1992) and Ozaki (1993) found that, compared to controls, SAD patients showed abnormal **electrooculograph** (EOG) responses to flashes of light. However, their findings were not consistent (female and male patients differed in their responses) and a study by Oren et al (1993), using a variety of different measures, failed to detect any visual or retinal abnormalities in SAD patients.

Treating SAD using light (phototherapy)

Whatever its causes, one thing that has become clear about SAD is that its symptoms respond well to **phototherapy**. Exposure to bright light for about two hours a day seems to alleviate depressive symptoms within two to four days in the majority of patients (Rosenthal et al, 1985; Jacobsen et al, 1989). Treatment must continue on a daily basis throughout the winter for this improvement to be maintained (James et al, 1985) and the light used must be sufficiently bright to have a therapeutic effect (at least 2500 **lux**, compared to the 200-500 lux output of most domestic lightbulbs). SAD patients prefer phototherapy to other forms of treatment, as it does not have the side effects associated with many antidepressant drugs (although about 20 per cent of users report mild side effects such as headaches and eye strain; Levitt et al, 1993). However, those patients with the severest SAD symptoms may also require drug treatment to control the disorder effectively (Schwartz et al, 1996).

The main drawback of phototherapy is the size and weight of the light-box commonly used, which, not being portable, requires the patient to sit in front of it for two hours each day, interfering with their normal activities. Promisingly, studies using a ‘light visor’ worn by the patient have suggested that this can be as effective as treatment using a cumbersome light box (Joffe et al, 1993) with the added advantage that lower intensities of light can be used to bring about a therapeutic effect, although relapse rates appear to be higher when low intensity light is used (Rosenthal et al, 1993).