PUBLIC HEALTH

Winter needn't be the SAD season

Epidemiology: Seasonal affective disorder (SAD) is a form of bipolar or major depressive disorder characterized by recurrent depressive episodes associated with the changing seasons. It begins insidiously, often appearing in people aged 20 to 30 years in the form of mild to moderate depressive episodes that are typically endured for several years before medical assistance is sought. Sadness, anxiety, irritability and social withdrawal are common features, as are "atypical" depressive symptoms such as increased sleep duration, increased appetite and weight, and cravings for carbohydrates.[1]

SAD is a relatively common subtype of major depression, occurring in 11% of patients with major depression.[2] Its prevalence in Canada is 1%–3%,[2,3] as compared with 1.3%–3% in Europe and less than 0.9% in Asia.[2] Some studies have shown an association between increasing prevalence of SAD and increasing latitude, but a review of the evidence suggests that the influence of latitude is small[4] and that other factors — climate, social–cultural context and, more recently, genetic vulnerability — contribute to this disorder's complex etiology. Between 13% and 17% of first-degree relatives of people with SAD appear also to be affected.[5]

Clinical management: SAD is one of those perplexing diseases for which medicine can offer effective therapy without being able to offer a complete explanation of how and why it works. A meta-analysis of controlled trials involving 332 patients with SAD in winter revealed that exposure to 2500-lux light from a light box for 2 hours every morning for 1 week led to improvements in 67% of patients with mild depressive episodes and in 40% of those with moderate to severe episodes.[6] (About 8 fluorescent lamps are needed to produce 2500 lux.)

Clinical consensus guidelines recommend light therapy as a first-line treatment for SAD on the basis of evidence from numerous studies showing efficacy, including large randomized controlled trials and meta-analyses.[7] Selective serotonin re-uptake inhibitors (sertraline or fluoxetine) or moclobemide, a reversible monoamine oxidase A inhibitor, have also demonstrated efficacy as a supplement or alternative to phototherapy.[1]

Several hypotheses have been offered to explain the intriguing response of SAD patients to bright light. One of the first was simply that the shorter winter photo period (dark-light cycle) led to depressive symptoms. Were this so, exposure to bright light at the beginning and end of the winter day, to simulate a summer photo period, should restore summer behaviour. Subsequent evidence that single daily pulses of light are as effective as morning and evening pulses is inconsistent with this hypothesis.[3]

Attention has also focused on melatonin, the endogenous hormone that is secreted nocturnally by the pineal gland. Melatonin can shift the phase of the circadian rhythm, induce drowsiness and be suppressed by bright light — all of which implicate it in the pathophysiology of SAD. However, observations that the 24-hour melatonin rhythm in winter does not differ between people with SAD and control subjects, and that melatonin suppression alone does not produce a therapeutic effect, suggest it is too simplistic to attribute SAD to the direct influence of melatonin.[3]

Substantial evidence supports a third theory, the phase-delay hypothesis.[3] According to this theory, SAD results from internal circadian rhythms that are phase delayed relative to the external clock and other endogenous rhythms (e.g., the sleep–wake cycle). Morning light is predicted to be superior to evening light because exposure to morning light results in a corrective "phase advancement" of cortisol, temperature and melatonin rhythms in patients with SAD. Although some of the evidence is conflicting, studies involving the most reliable measures of the endogenous circadian phase (dim-light melatonin onset) do demonstrate a relation between the level of clinical response to light therapy and melatonin and the degree of corrective phase advances.[3]

There is also a unique rationale for hypothesizing that serotonergic dysfunction plays a major role in SAD. Serotonin activity in both animals and humans is known to fluctuate markedly across the seasons.[3] For example, serotonin levels in the hypothalamus have marked seasonal variations, with the lowest levels found in winter.[8] Given the role of hypothalamic serotonin in satiety and feeding regulation, this could explain the tendency of patients with SAD to crave carbohydrates and gain weight during depressive episodes.[3,5] The recent discovery that a serotonin transporter promoter polymorphism (the 5-HTTLPR "s" allele) is more prevalent among SAD patients than among control subjects suggests that genetic vulnerability is an underlying factor in this disorder.[5]

Prevention: The conflicting theories and results indicate that there is likely substantial heterogeneity in the etiologic and pathophysiologic features of SAD. The task of identifying primary preventive factors is therefore difficult. Given that there is effective therapy, it may be more helpful if physicians practised secondary prevention in patients presenting with a major depression in the winter. — *Erica Weir*, CMAJ

References

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