Seasonal Affective Disorder (SAD)

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Introduction

For thousands of years, mankind's mood and behaviour had been subject to the changing of the seasons. Though the advent of electricity had ostensibly left human beings independent of the changing seasons, some still tend to feel listless during the cold and dark winter or the hot and radiant summer. Despite the significant number of seasonally depressed individuals, it was not until the early 1980s that the scientific community, led by Norman E. Rosenthal, M.D., and his colleagues at the National Institute of Mental Health (NIMH) in the United States, brought forth the concept of Seasonal Affective Disorder (SAD).

The vast majority of SAD patients have their worst symptoms sometime during the fall or the winter (winter SAD), while a smaller yet statistically significant group characterize the summer as their most dreaded time of the year (summer SAD). Typical symptoms of the more common winter SAD include increased appetite (particularly carbohydrate-rich and starchy foods), increased sleeping, irritability, forgetfulness, reduced libido, and feelings of inadequacy. People with the most severe cases of the winter form of SAD can suffer from September until April, while others find symptoms are only present during the harshest winter months, setting in around Christmas and subsiding in March. Rosenthal and his colleagues have postulated that SAD is approximately four times as common among women than men. They have also reported that those aged between 20 and 40 appear to be the most susceptible to SAD (1993). Yet despite more than twenty years of literature on SAD, the disorder is still under diagnosed. In three studies testing the efficacy of a potential SAD drug, only 41% of those with

symptoms characteristic of SAD reported having received prior treatment for a major mood disorder (Modell et al., 2005).

Screening for SAD

The principal tool used in determining if a patient has the symptoms of SAD is the Seasonal Pattern Assessment Questionnaire (SPAQ), developed by Rosenthal and colleagues at the NIMH. Essentially, the SPAQ asks the individual to recall their experience living in a single climatic region over the course of the past two or three years. First, the subjects are asked during what months of the year they feel the best/worst, eat the most/least, sleep the most/least, gain/lose the most weight, and socialize the most/least. Second, the individuals grade the effect the changing of the seasons has on them in six areas (sleep length, social activity, mood, weight, appetite, and energy level) on a scale of 0 to 4, with 0 being "no change" and 4 being "extremely marked change." To determine their degree of seasonality, subjects add up their scores for the six questions. A score equal to or greater than 11 can qualify for a formal diagnosis of SAD while a score of 8 to 11 is characterized as subsyndromal SAD (S-SAD or "winter blues"), a less severe and less prolonged form of SAD. Third, they are asked if the changes they experience over the course of the seasons are a problem, and if so, they are asked to grade the effect of the problem on a scale from "mild" to "disabling." Fourth, participants are asked how much their weight fluctuates through the seasons. Fifth, they are asked how many hours per day they sleep in each of the four seasons. Finally, they are asked if they have any food preference during any period of the year (Rosenthal, 1993). Despite its efficacy in screening possible SAD patients, the SPAQ has been critiqued by some in the scientific community for its reliance upon the potentially biased

opinions and memories of its subjects (Magnusson et al., 2000). The results of the SPAQ, therefore, cannot be the sole basis for a diagnosis of SAD.

Etiology

Researchers have noticed clear disparities in the prevalence of SAD from region to region. The SAD rate for North America has been estimated at anywhere from 1.4% to 9.7%. Europe, on the other hand, has a much smaller estimated prevalence of between 1.3% and 3%. Asia, meanwhile, has been estimated to have SAD rates as low as 0.9%. Though these disparities might be attributable to differing study methods, socioeconomic and cultural issues could also play a part in the discrepancy. A study done in Toronto of 781 respondents using the SPAQ and the Depression and Seasonality Interview (DSI), the first structured interview to diagnose and assess SAD, resulted in 39 (5%) individuals meeting the SPAQ-SAD criteria and 23 (2.9%) meeting the DSI-SAD criteria. Among people meeting the SAD criteria of DSI, a ratio of approximately two to one were female; the SPAQ study had a SAD female to male ratio of approximately 1.4 to one. Though the overall numbers differed significantly, the two standards agreed that SAD was most prevalent among women aged 40 to 49 (slightly higher than the NIMH standard), and also that SAD was nearly nonexistent in people over the age of 59 (Levitt et al., 2000).

Though the precise etiology of SAD has not yet been pinpointed, a number of hypotheses abound, with the reduction of daylight hours in winter being the primary culprit. Scientists point to the fact that areas of extreme latitude, which receive far less daylight during winter than southern latitude areas, generally have higher recorded levels of winter SAD. Studies done in the United States reveal that people in Sarasota, Florida (42 N in latitude) dislike the winter far less than do people in New Hampshire (27 N). Conversely, people in New Hampshire tend to enjoy their relatively fleeting summer

months significantly more than do people in Florida. One notable exception to this "latitude hypothesis" is the tiny northern nation of Iceland (Axelsson et al., 2002). The capital and largest city in the country, Reykjavik (64 N) , typically receives as little as four hours of daylight during the peak of bleak midwinter (Iceland Travel Information, 2002). Despite this, Iceland has a markedly lower prevalence of SAD and S-SAD than several US cities of moderate latitude. Moreover, a study of people of Icelandic descent in Interlake, Manitoba (50.5 N) concluded that the rate of SAD and S-SAD was only slightly higher than in Sarasota, despite a marked difference in latitude. A recent study of Winnipeg (roughly 50 N) residents of both Icelandic and non-Icelandic descent found that 9.1% of participating non-Icelandic residents had SAD while only 4.8% of Icelandic descendents exhibited signs of SAD. The cumulative average of the Interlake and Winnipeg rate of SAD among people of Icelandic descent, 3%, was similar to that of Iceland itself, 3.6% (Axelsson et al., 2002). Though it has not yet been fully grasped, these studies strongly suggest a genetic component to the disorder. There is also evidence that people with a family history of mental illness might be at greater risk for SAD. A study cited in the New York Times found that more than two-thirds of patients with SAD have a relative with a major mood disorder (2002).

Other studies have signalled a similarly ambiguous relationship between latitude and SAD prevalence. While Japan's rate of SAD is consistent with the latitude hypothesis, the populations of Italy, Turkey, and Australia have shown no such correlation. There is also the possibility of acclimatization to one's environment. Scientific research has shown that Asian women immigrants living in the UK were far less energetic than were women of UK descent. Some suggest Iceland's uncharacteristically low rate of SAD can be explained by natural selection. Simply put,

over many generations, the population of Iceland has become acclimated to their mercurial surroundings (Magnusson et al., 2005).

Generally speaking, most SAD research has shown a positive correlation between an area's latitude and its SAD rate. However, other climatic factors may be at play, including weather and geography. In the Manitoba study, for instance, a higher rate of people of Icelandic descent had symptoms of SAD in the urban sprawl of Winnipeg, 4.8%, than in rural Interlake, 1.3%. This calls into consideration the effect environmental factors such as population density and population might have on SAD rate.

Other external factors that might contribute to SAD are the prevalence of negative life experiences (NLEs) and the lack of social support (SS). A study of 26 patients with seasonal depression and 66 patients with non-seasonal depression examining the effects of these factors concluded that these elements, along with patient being female and being non-native to his/her residential area, contributed to higher levels of seasonal depression. NLEs were quantified using the List of Threatening Experiences questionnaire and SS was assessed using the Medical Outcomes Study-Social Support Survey. Seasonally depressed patients reported approximately two NLEs over the course of the previous six months. It was found that the social support networks of seasonally and non-seasonally depressed patients networks of SAD patients were similarly underdeveloped or impaired (Michalak et al., 2004).

The fact that the overwhelming majority of SAD patients are women of childbearing age has inspired a number of yet unproven hypotheses. Some have suggested that the changes in mood, appetite, and sleep patterns are attributable to the female body's preparation for pregnancy. Historically, the myriad physical limitations caused by the dark and cold days of winter made it the optimal season for conception,

gestation, and lactation. There is evidence that births would traditionally peak in the late winter and early spring. Proponents of this theory point not only to the fact that men are considerably less likely to be diagnosed with SAD but also that seasonality appears to remit as patients age (Davis et al., 2005).

Forms of Treatment

Some suggest the general malaise caused by winter is attributable to changes in biorhythms. Individuals afflicted with SAD find their circadian rhythm out of step with clock times, and feel they are waking too early. Essentially, because people are waking before the sun rises, their bodies believe it is night. Alfred Lewy of Oregon Health Sciences University likens the effect to jet lag (New York Times, 1998). One physiological explanation concerns the decrease in hypothalamic levels of the neurotransmitter serotonin – the neurotransmitter tied to mood, appetite, and sleep patterns – during the winter. As a result, serotonin reuptake inhibitors are often prescribed to patients with SAD (Johansson et al., 2001). Corresponding with the drop in serotonin is the increase of melatonin, a hormone produced by the pineal gland at night that helps regulate biorhythms and immune function. Its secretion is suppressed by light exposure to the retina. Though its role in SAD has not yet been precisely determined, studies have shown that melatonin levels among SAD patients are as much as 2.4 times higher than in controls. In view of melatonin's seemingly negative effects for SAD sufferers, some have suggested that neuroimmune dysfunction might actually mediate winter SAD (Lam et al., 2004). However, Lewy and his colleagues considered the effect of small doses of melatonin to regulate the body rhythms of SAD patients. They concluded that daily 0.125 mg doses of melatonin generally synchronized internal timing mechanisms and lessened the effect of SAD symptoms (New York Times, 1998).

The US Food and Drug Administration (FDA) has just recently approved the first drug for seasonal depression. In three separate studies, 150 mg daily doses – gradually upped to 300 mg – of Wellbutrin XL (Bupropion), a norepinephrine-dopamine reuptake inhibitor developed by GlaxoSmithKline, left patients depression-free 84% of the time versus 72% with the placebo (2006). Also, only 16% of those taking bupropion had a recurrence of depression, versus 28% for the placebo group (Modell et al., 2005). In order to be prescribed Wellbutrin, patients must exhibit the diagnostic criteria of seasonal depression. Potential side effects of Wellbutrin include agitation, anxiety, and insomnia (FDA, 2006).

Aside from prescription medication, the most common form of treatment for SAD is phototherapy, also known as light therapy. Rosenthal and Thomas Wehr, also of the NIMH, were among the first to consider the effect of sustained light therapy on patients with SAD. Over the course of a number of studies to determine the therapeutic effects of light boxes, they observed antidepressant effects among those who sat facing a fluorescent light for a sustained period of time. They also found that those who merely had light directed at their skin exhibited little to no symptomatic changes. They viewed the findings as evidence that light must be absorbed through the eye in order to treat SAD (Rosenthal, 1993). A study conducted at Yale University concluded that therapeutic light stimulates production of reactive oxygen species, which play a role in muscle contraction, cell growth, and possibly brain functions (New York Times, 2001). Light therapy has been determined to be most effective when conducted in the morning for a period of between 15 and 90 minutes a day. A large proportion of light therapy patients also require adjunctive psychopharmacologic treatment (Modell et al., 2005). In the years since light therapy was first studied, researchers have considered the effects of coloured bulbs.

George C. Brainard of Thomas Jefferson University conducted a five-year study examining whether SAD patients responded better to certain elements of the light spectrum and found they were most responsive to narrow bands, particularly sky blue. One study Brainard conducted of 24 SAD patients involved half using red LED lights and the other half using blue LED lights for 45 minutes a day each. While 60% of those subject to blue light had reduced symptoms, only 40% of those using the red lights showed signs of improvement. Furthermore, the effect of a mere 400 lux blue light was found to be the same as that of a 10,000 lux white light (Raloff, 2006).

Specialists also suggest means of stress reduction to mitigate the effects of SAD. Two ways this can be done are diet improvement and increased exercise. Choosing fun, non-restrictive exercise routines has been shown to increase energy levels among patients with depressive disorders. As well, it can help to temper the weight fluctuations symptomatic of SAD. Researchers disagree as to the merits of a carbohydrate-free diet when grappling with seasonal depression. Some suggest the body craves carbs because it needs them in order to cope. Indeed, some afflicted with SAD report boosts of energy after indulging themselves with carb-rich foods. Others believe this rise in energy is temporary and only leads to further feelings of fatigue and renewed cravings. Rosenthal posits that diet approaches depend on the individual (1993). Other means of reducing stress include closely monitoring sleep and seasonal stress cycles (Rosenthal, 1993).

Conclusion

Unfortunately, much remains clouded about the nature of SAD. However, what is known is seasonal depression is very real and affects a significant portion of the North American population. It is reasonable to suggest that the general under diagnosis of SAD is a result of the medical community's casual dismissal of the disorder as mere "winter

blues." While it is true that many people tend to feel weighed down by the shroud of winter, a number these individuals have symptoms characteristic of a major mood disorder. Although the precise cause of SAD has not yet been ascertained, there is strong evidence to suggest the disorder is the result of neurotransmitter irregularities. Twenty years on, SAD is now on the map as a serious yet treatable mood disorder. Ongoing research on the positive effects of supplementation, prescription medication, light therapy, and stress management has shown encouraging signs and promises to continue doing so well into the future.

Works Cited

Axelsson, J., Stefansson, J. G., Magnusson, A., Sigvaldason, H., Karlsson, M. M. (2002). Seasonal Affective Disorders: Relavence of Icelandic and Icelandic-Canadian evidence to etiologic hypotheses. *Canadian Journal of Psychiatry, 47*, 153-158.

Brody, J. E. (1998). New tack promising on winter depression. *New York Times*, March 31.

Davis, C., & Levitan, R. D. (2005). Seasonality and Seasonal Affective Disorder (SAD): An evolutionary viewpoint tied to energy conservation and reproductive cycles. *Journal of Affective Disorders, 87*, 3-10.

FDA News Release. (2006). FDA Approves the First Drug for Seasonal Depression. *United States Food and Drug Administration*. Retrieved June 13, 2006, from http://www.fda.gov/bbs/topics/NEWS/2006/NEW01388.html.

Ivry, S. (2002). Seasonal depression can accompany summer sun. *New York Times*, August 13.

Johansson, C., Smedh, C., Partonen, T., Pekkarinen, P., Paunio, T., Ekholm, J., Peltonen, L., Lichtermann, D., Palmgren, J., Adolfsson, R., & Schalling, M. (2001). Seasonal Affective Disorder and serotonin-related polymorphisms. *Neurobiology of Disease, 8*, 351-357.

Levitt, A. J., Boyle, M., Joffe, R.T., & Boumal, Z. (2000). Estimated prevalence of the seasonal subtype of major depression in a Canadian community sample. *Canadian Journal of Psychiatry*, *45*, 650-654.

Magnusson, A., & Karlsson, M. M. (2000). Lack of seasonal mood change in the Icelandic population: Results of a cross-sectional study. *American Journal of Psychiatry, 157*, 234-238.

Magnusson, A., & Partonen, T. (2005). The diagnosis, symptomatology, and epidemiology of Seasonal Affective Disorder. *CNS Spectrums, 10*, 625-634.

Michalak, E. E., Tom, E. M., Manjunath, C. V., Yatham, L. N., Levitt, A. J., Levitan, R. D., & Lam, R. W. (2004). Hard times and good friends: Negative life events and social support in patients with seasonal and nonseasonal depression. *Canadian Journal of Psychiatry, 49*, 408-412.

Modell, J. G., Rosenthal, N. E., Harriett, A. E., Krishen, A., Asgharian, A., Foster, V. J., Metz, A., Rockett, C. B., & Wightman, D. S. (2005). Seasonal Affective Disorder and the prevention by anticipatory treatment with Bupropion XL. *Biological Psychiatry, 58*, 658-667.

Nagourney, E. (2001). Of light, tissue and better moods. *New York Times*, April 24.

Raloff, J. (2006). Light impacts. *Science News, 169*, 314-316.

Rosenthal, N. E. (1993). *Winter Blues: Seasonal Affective Disorder: What is it and how to overcome it.* New York: Guilford Press.