

Winter Seasonal Affective Disorder: A Global, Biocultural Perspective



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Introduction

Winter Seasonal Affective Disorder (SAD) is a well-documented psychiatric disorder typically associated with the onset of winter and its periods of low light. The rates of this disorder are highly variable among different populations around the world. Some studies have demonstrated an increase in SAD rates with increasing geographic latitude while other studies have demonstrated that increased in latitude does not equate with increased SAD rates within certain populations (Haggarty et al., 2002). Genetic research has also addressed SAD through studies in familiarity, heritability and molecular genetics (Sher et al., 1999).

This paper will address SAD, its etiology and its prevalence on a global scale. It will demonstrate that SAD is a complex disorder with genes, environment and culture contributing to its etiology and that it is a disorder that is difficult to accurately identify in large scientific samples. Using a biocultural approach, factors that significantly contribute to documented SAD rates will be identified by a review of the literature and analysis of data extracted from this literature. Specifically this paper will address the geographic/environmental factors, genetic adaptations and cultural influences that may affect the variability of SAD rates among specific populations worldwide (McElroy, 1990). The problems of diagnosing SAD for the purposes of research will also be addressed as a potential influence on the variability of documented SAD rates (Haggarty et al., 2002).

Literature Review

The Pathophysiology of SAD

Winter Seasonal Affective Disorder (SAD) is a subtype of Seasonal Affective Disorder. Winter SAD occurs during the winter months in periods of low light. SAD is characterized by symptoms such as lowered mood, fatigue and decreased energy. Weight gain, hypersomnia and carbohydrate craving are less typical symptoms. SAD is considered a subtype of bipolar or major depressive disorder (Michalak, et al., 2001). SAD is defined by the Diagnostic and Statistical Manual for Mental Disorders IV as having the following diagnostic criteria: a temporal relationship between onset of depression and time of year, a seasonal pattern that has occurred over two years with no non-seasonal depression and a greater number of seasonal than non-seasonal episodes of depression (DSM-IV, 1994; Mickalak, et al., 2001). Age, gender, family history and melatonin sensitivity to light have all been identified as factors that contribute to SAD (Low and Feissner, 1998). SAD rates are significantly higher in women (Partonen and Lonquist, 1998), and SAD rates decrease with age (Magnusson et al., 2000).

Though SAD may appear to be caused by lack of sunlight, a direct causal relationship has not yet been identified. In fact, there is also a subtype of SAD that occurs in the summer months, though this is less common than winter SAD (Han et al., 2000; Partonen and Lonquist, 1998). This cause-effect relationship between reduced sunlight exposure and SAD is the basis for the photoperiod hypothesis (Lam, 2000; Partonen, Lonquist, 1998). Incorporated into this is the hypothesis that the duration of melatonin secretion at night reflects changes in the photoperiod in humans. This is based on the

findings that those with SAD have demonstrated abnormalities in their dim-light nocturnal melatonin profile (Lam, 2000).

According to Lam (2000) the major hypotheses regarding the etiology of SAD are phase-shifted circadian rhythms, serotonergic dysfunction and genetic vulnerability. Lam also cautions that all of these may not be mutually exclusive. According to the phase-shifted circadian rhythms hypothesis, sunlight acts as a type of synchronizer of the circadian pacemaker in humans, and exposure to light can shift the phase of these rhythms. The timing of light exposure, not necessarily the amount of light exposure, relative to the cycle of circadian rhythms influences the magnitude and direction of the circadian rhythm phase shift. SAD occurs as a result of internal circadian rhythms that are phase-delayed relative to an external clock. The external clock is periods of sunlight and darkness (Lam, 2000).

The hypothesis regarding serotonergic dysfunction is based on the findings that serotonin levels vary significantly in normal humans across seasons with lowest levels in the winter months. It states, based on research findings, that there is a dysfunction of the receptors in serotonergic neurons, resulting in abnormal neuroendocrine responses. These neurons are involved in the release of corticotrophin, cortisol, prolactin and growth hormone (Lam, 2000; Partonen and Lonquist, 1998).

Genetics, Geographic Latitude and SAD

The genetic vulnerability hypothesis states that certain individuals and homogeneous populations may be more or less vulnerable to SAD (Lam, 2000). The study of genetics has focused primarily on familiarity, heritability and molecular genetics (Sher et al., 1999). There have also been several studies comparing SAD rates of various

populations in different countries in an attempt to identify populations who are more resistant to SAD (Axelsson et al., 2002; Magnusson, 2000).

The studies of genetic factors in the etiology of SAD, looking at the prevalence of psychiatric disorders in relatives of patients with SAD, have suggested a familial contribution (Sher et al., 1999). In summarizing these studies Sher (2001) states that between 13% and 17% of first degree relatives were affected by SAD. This is significantly higher than the average rates of SAD in the general population according to Sher.

In a study of 4,369 twins, the effects of genes were identified to be responsible for at least 29% of variance in seasonality among individuals. Seasonality is defined as seasonal changes in mood, weight, energy level, appetite and social activity (Sher, 2001). Sher recommends caution in the interpretation of these studies as these studies did not evaluate the heritability of a diagnosis of SAD directly, only seasonality.

The field of molecular genetics has revealed some information useful in the identification of genes that may be associated with SAD. Spectrum disorders or complex phenotypes that contribute to a disorder best describe SAD from a genetic perspective (Lam, 2000). Sher (2001) suggests that many genes, possibly on many chromosomes, may influence vulnerability to SAD. He points to the findings of two genetic variants that have been associated with SAD. They are the 5-HTTLPR and the 5-HT_{2A}-1438G/A gene promoter polymorphisms, which are related to serotonergic transmission.

Epidemiological studies involving the comparison of SAD rates between populations in different countries have pointed to the possibility of a genetic resistance to the disorder among certain populations (Magnusson, 2000; Magnusson and Stefansson,

1993). In 1993 a study by Magnusson and Stefansson was published, and it demonstrated a significant difference in SAD rates among Icelanders living at 64-67 degrees north latitude compared to Americans living at much lower latitudes. The rates were 3.6% to 7.6% respectively. Magnusson also noted that the prevalence of other non-seasonal affective disorders as well as major depressive episodes are similar in Iceland and the United States. Magnusson and Axelsson (1993) studied prevalence SAD in a sample of Canadians of wholly Icelandic descent living in Manitoba, Canada. The SAD rate among this group was 1.2%. Since this rate was even lower than that of Icelanders living in Iceland (3.6%), they concluded that latitude could affect SAD rates within genetically similar populations (1993). Axelsson et al. (2002) followed up with a study comparing a population of Canadians of non-Icelandic descent with those of full Icelandic descent living, both living in Winnipeg, Manitoba. The SAD rates were found to be 4.8% in the Icelandic group and 9.10 in the non-Icelandic group. These studies not only demonstrate significant variation in SAD rates between genetically different populations, but they also bring into question the role of increasing geographic latitude in the prevalence of SAD (Magnusson, 1993).

A study of SAD rates in four locations in the United States demonstrated an increase in the prevalence of SAD that was correlated with an increase in latitude (Rosen, 1990). In this study the lowest SAD rate was 1.4% in Florida at 27 degrees north; the highest was 12.5 % at 40 degrees north in New York. Before Magnusson's study (1993) it was generally accepted that increasing latitude was the strongest predictor of SAD rates. This was supported by a number of studies showing low SAD rates at the low latitudes: For example, a study conducted in the Philippines at 15 degrees north found a

SAD rate of 0.0 (Ito et al. 1992). Axelsson et al. (2002) and Magnusson (1993) however conclude that it is likely a genetic adaptation of Icelanders that is revealed in the decreased SAD rates in this population residing in the far north. This conclusion is supported by the fact that the Icelandic population has been geographically isolated for about one thousand years (Magnusson, 1993). Iceland is considered one of four major European outliers based on its genetic distances from other groups, as well as historical information (Cavalli-Sforza et al., 1994). Icelanders' closest genetic relatives are the Norwegians with a genetic distance of 74 (all genetic distances mentioned are x10,000). The Swedes and the Norwegians have a genetic distance of 12 while the Danes and the Dutch have a genetic distance of 9 making them the two most closely related populations in Europe (Cavalli-Sforza et al., 1994). There has been no research comparing SAD rates between populations based on their genetic distance, however.

Since Magnusson's first study (1993) a number of studies, using the same assessment instrument, have demonstrated variability in SAD rates worldwide that do not appear to be strictly related to latitude. For instance Imai et al. (2003) found low rates of SAD in Japan. They were .89% at 39.75 degrees north and .48% at 33.35 degrees north latitude. Elbi, et al. (2002) found an average rate of 4.8% in Turkey at an average latitude of 39 degrees north. Eagles et al. (2002) recorded a SAD rate of 9.9 in Scotland at 57 degrees north latitude. Booker et al. (1991) identified a SAD rate of 16.2% in Siberia at 64 degrees north latitude. Though the changes in light and darkness cycles with increasing latitude are consistent, winter weather is not.

Weather, Acclimatization, Age and SAD

The effects of winter weather have been briefly addressed in regard to SAD. Boyce et al. (1988) found SAD rates in Australia at 35 degrees south latitude to be similar to the rates at 39 degrees north latitude in Maryland. According to Boyce, Maryland's average winter temperature is much lower than that of the study location in Australia. The relatively mild winter temperatures in Iceland have been considered as a possible factor in the low SAD rates there (Magnusson and Stefansson, 1993). In the discussion regarding the Icelandic descendants living in Manitoba, Canada, Magnusson and Axelsson (1993) state that if temperature extremes were a factor, Manitoba's extreme continental climate should exaggerate seasonal changes in mood, instead they found the opposite. While SAD rates and their corresponding latitudes have been widely investigated, less is known about acclimatization in regard to SAD on populations and individuals.

The effects of acclimatization on individuals living in the high latitudes have not been investigated as thoroughly in studies of SAD. Low and Feissner (1998) found in a study of college students living in Maine that students relocating to Maine from the south had higher rates than those relocating from other areas. A prospective study following Norwegians over a 9-year period in the northernmost county in Norway was completed by Nilsen et al. (2004). The authors looked specifically at improvement in mental health related to winter darkness over this 9-year period. They concluded that there was a significant decrease in reported depression related to the dark period over the 9-year time span. A study of two groups of Japanese living in Stockholm, Sweden revealed that the

group that had lived in Sweden longer demonstrated more seasonal mood variations than those living in the region for 2 years or less (Murase et al., 1995). In contrast, increased SAD rates have been found in those who had been living in Alaska for shorter periods of time (Booker and Hellekson, 1992). Though not directly linked to acclimatization in any of the research reviewed here, age and SAD rates have been studied thoroughly.

One commonality throughout the literature is that there are differences in SAD rates in adults, based on age. SAD rates are typically higher in younger people and decrease with age (Axelsson et al., 2002; Booker and Hellekson, 1992; Magnusson and Stefansson, 1993; Parslow et al., 2004). In the study of SAD rates between Canadians of Icelandic and non-Icelandic descent, both groups demonstrated age related differences. The authors state, "...if 2 individuals of the same descent and gender are separated in age by 10 years, the older individual is 29% less likely to suffer from SAD than the younger." (Axelsson et al., 2002:156).

The study conducted in Fairbanks, Alaska by Booker and Hellekson (1992) demonstrated those subjects under 40 years old were 2.84 times more likely to be affected by SAD. In Australia at seasonality was also found to decrease with age as higher seasonality rates were found in young adults (Parslow et al., 2004). Unlike the relationship between SAD rates and age, investigation of cultural adaptations and cultural concepts of disease and their influences on SAD rates has been minimal (Stuhlmiller, 1998).

Culture and the SAD Diagnosis

Kleinman and Good (1985) emphasize that the effect of culture on mental illness is important to recognize in studies of etiology. In Scotland, Eagles et al. (2002)

investigated the relationship between socioeconomic deprivation and SAD. No significant relationship was identified. In Iceland Magnusson and Stefansson (1993) investigated marital status, employment and residency. They found no difference in SAD rates in singles versus married individuals, those living in rural settings versus urban, those working outdoors versus indoors or those with active jobs versus sedentary jobs. They also discounted the possibility of Icelanders having a higher threshold for complaints based on findings of other studies that demonstrated no differences between Icelanders and Americans in measures of hypochondriac responses on standardized tests. In their study finding low rates of SAD in Canadians of Icelandic descent, Magnusson and Axelsson (1993) make an effort to point out that their sample of individuals of Icelandic descent was culturally Canadian. Magnusson and Axelsson (1993: 948) described their sample of 600 adults as being "...wholly descended from Icelandic emigrants. Their present culture and lifestyle are completely Canadian."

An ethnographic study was conducted by Stuhlmler (1998) at 69 degrees north latitude in Norway over a two-year period to study how Norwegians experience extreme seasonal change. This part of Norway is 386 miles above the Arctic Circle with 2 months of total darkness in the winter. Stuhlmler points out that Norwegians in this part of Norway are culturally different than those to the south. The southern Norwegians are more like the rest of Europe in their trends and thinking.

In regard to SAD, Stuhlmler reports among this population a general, widespread acceptance of emotions and changes in emotions as part of natural cycles. Being sad at times is acceptable to northern Norwegians, whereas the same type of sadness could be considered depression in an American's perspective. She contrasts the

northern Norwegians' acceptance of sadness to the Americans' concept of sadness being a disorder that should be treated.

A deep connection to nature and its changing seasons is rooted in all aspects of the northern Norwegians' lives. Social activities are common in the winter darkness and are typically linked to seasons (Stuhlmiller, 1998). The author describes extensive use of candles as natural light sources throughout the dark season, including their use in offices and during meetings. Children undergo, and have undergone for generations, a socialization process to prepare for the dark periods. Children are given cod liver oil daily as a sort of preventative treatment for general ailments. Cod liver oil is a source of Omega 3 fatty acids; some studies have demonstrated decreased Omega 3 levels in depressed subjects (Maidment, 2000). Other studies have demonstrated the effectiveness of Omega 3 for the treatment of bipolar disorder (Maidment, 2000). Active exercise, a healthy diet and participation of residents in the social and cultural events of the season are rituals that have also been passed from generation to generation in northern Norway (Stuhlmiller, 1998). This population resists the medicalization of what they consider everyday life, resulting in a lack of knowledge about disorders such as SAD. In this part of Norway there is general resistance to reliance on technology in daily activities; this, as Stuhlmiller (1998: 153) says, "...would simplify, standardize and impoverish nature-and with it, the potential for spiritual understanding."

Kleinman and Good (1985) state that cultural differences in the concepts of depression in general can be seen in language. For instance, there are many words in European languages that refer to depressive experience. In contrast no conceptually equivalent terms exist in Chinese, Japanese, Malaysian and Nigerian languages.

Kleinman and Good (1985) also state that so-called core symptoms of depressive syndromes are common across cultures though the manifestations are not universal. In non-European populations the so-called diagnostic symptoms are not present, while the somatic symptoms are present. Differing cultural perspectives on sadness bring into question the validity of the method used by scientists in the identification of SAD in different cultures around the world (Michalak et al., 2001).

All the SAD rates in the studies cited at this point in this paper were obtained by using the Seasonal Pattern Assessment Questionnaire (SPAQ). This assessment instrument is not without controversy (Michalak et al., 2001). The SPAQ was developed as a screening instrument by American N.E. Rosenthal in 1987, with the American population in mind (Magnusson, 2000; Parslow et al., 2004). Michalak et al. (2001) consider the SPAQ to be overly inclusive. They point to studies utilizing a full diagnostic interview such as that done by Blazer et al. (1998). In the United States a SAD rate of .4% was identified in the range of 27-43 degrees north latitude by this study. Magnusson (2000) addresses the issue of reliability and validity in using the SPAQ. He points to 6 studies that have demonstrated good reliability of the instrument. In regard to validity, he cites early studies that compared SPAQ scores to the clinical diagnosis of SAD. One found excellent agreement while the other 5 found moderate agreement. Magnusson states that in 3 more recent studies, the SPAQ has been shown to overrate the prevalence of SAD. There is no mention of cross-cultural studies of SPAQ SAD rates versus SAD rates obtained with a diagnostic interview in the literature.

Methods

In order to analyze SAD rates worldwide in regard to the effects of latitude, genetic distance and cultural influences, data was extracted from a number of studies in which SAD rates in percentage of population were obtained. A total of 30 cases comprise the sample used in the analysis. Only those studies using the SPAQ were used. This was done out of necessity since studies using other diagnostic methods were minimal in number and did not use a common method (Magnusson, 2000). This data and sources are summarized in Table 3 “Summary of Cases.” It lists the country in which the study was conducted, the SAD rate in percent and the author/authors of the study. A description of the sample showing the number of cases, mean latitude and mean SAD rate and standard deviations can be seen in Table 1 “Descriptive Statistics-Complete Sample.” Statistical analysis was conducted using SPSS 11.5 (2002) to determine if there is a correlation between SAD rates and increased latitude (Bindon, 2004; Oths 2004). Also the cases were divided into 2 categories based on latitude. One group contained cases with latitudes of 44.53 and below. The other category contained cases with latitudes higher than 44.53. A description of this can be seen in Table 2 “Group Statistics-High and Low Latitude.” The variance of SAD rate between groups was tested, as was the equality of the means between groups using parametric testing by means of a *t*-test (Oths, 2004; SPSS, 2002). Non-parametric testing of variance between the groups was done by means of a Moses 2 independent samples test (Oths, 2004; SPSS, 2002). This data was also examined for variation among similar populations and among different populations living in the same latitude range.

Genetic distance data for several northern European countries was extracted from The History and Geography of Human Genes by Cavalli-Sforza, Menozzi and Piazza (1994). It is summarized in Table 4 “Genetic Distances Between Countries.” This table shows a list of countries on the right and the left and the genetic distance is listed between them with a number that is x10,000. The genetic distance data was used to make comparisons with pairs of countries in regard to SAD rates and genetic distance. This was done in order to identify any trends in relatedness and SAD rates between genetically similar and genetically different groups in the same geographic region.

Analysis of the available qualitative data was incorporated into the results of the quantitative analysis in an attempt to identify possible influences other than genetics and latitude on SAD rates worldwide.

Table 1.

Descriptive Statistics-Complete Sample

	Mean	Std. Deviation	N
SAD rate in %	5.7793	4.13750	30
Latitude	46.0587	14.03263	30

Table 2.

Group Statistics-High and Low Latitude Categories

	Latitude Groups	N	Mean	Std. Deviation	Std. Error Mean
SAD rate in %	Low	16	4.2300	3.66741	.91685
	High	14	7.5500	4.04209	1.08029

Table 3.**Summary of Cases**

Author/s of Study	Location	Latitude in Degrees	SAD Rate in %
Morrissey et al., 1992	Australia	19 S	1.7
Parslow et al., 2004	Australia	35.5 S	5.35
Axelsson et al., 2002	Canada	50 N	9.1
Han et al., 2000	China	35.4 N	2.4
Dam et al., 1998	Denmark	55 N	12.4
Hagfors et al., 1995	Finland	60 N	7.1
Magnusson and Axelsson, 1993	Iceland	64.5 N	3.6
Muscoletta et al., 1995	Italy	41.5 N	4.4
Imai et al., 2003	Japan	39.75 N	.89
Imai et al., 2003	Japan	33.35 N	.48
Ozaki et al., 1995	Japan	35 N	.86
Mersch et al., 1995	Netherlands	53 N	3.0
Konradson, 1996	Norway	64 N	9.65
Ito et al., 1992	Philippines	15 N	0.0
Booker et al., 1991	Russia	64 N	16.2
Eagles et al., 2002	Scotland	57 N	9.9
Hagfors et al., 1995	Sweden	60 N	3.9
Broman et al., 1998	Sweden	61.1 N	3.5
Wirz-Justice et al., 1992	Switzerland	47.5 N	2.2
Elbi et al., 2002	Turkey	39 N	4.8
Booker et al., 1991	USA	55 N	6.6
Booker et al., 1992	USA	65 N	9.2
Hedge et al., 1996	USA	30 N	3.7
Levine, 1995	USA	65 N	9.35
Low et al., 1998	USA	44.53 N	13.2
Rohan and Sigmon, 2000	USA	44.53	7.8
Rosen et al., 1990	USA	27 N	1.4
Rosen et al., 1990	USA	39 N	6.3
Rosen et al., 1990	USA	40 N	4.7
Rosen et al., 1990	USA	42.5	9.7

Table 4.

Genetic Distances Between Countries
Northern Europe

Country	Genetic Distance (x10,000)	Country
Netherlands	9	Denmark
Norway	12	Sweden
Iceland	74	Norway
Sweden	106	Iceland

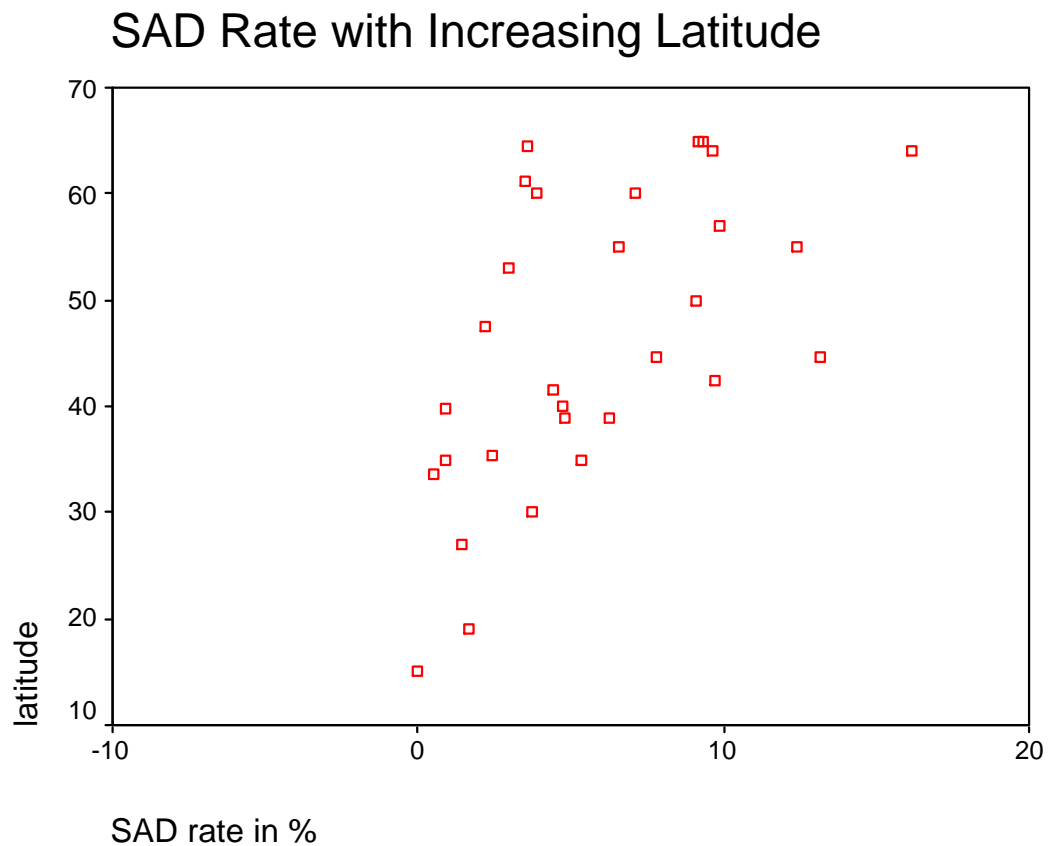
Results

In regard to the effects of latitude on SAD rate, a significant positive correlation was identified ($r=.590$). This can be observed in Graph 1 “SAD Rates With Increasing Latitude” and Graph 2 “SAD Rate and Latitude by Country.” No significant variance was identified in SAD rates between the low latitude and the high latitude groups ($p=.562$ and $p=.605$). However there was some variability noted within both groups and among populations living at the same latitude range. This can also be seen in Graph 1 and Graph 2. The difference in the mean SAD rates between the low latitude and the high latitude groups was found to be significant ($p=.026$).

Examination of the genetic distance data in comparison with the SAD rate data revealed no consistent observable connection between a close genetic relationship between populations and similar SAD rates. For example, the Dutch and the Danes are the most closely related populations in Europe with a genetic distance of 9 (x10,000) while there is a 9.4% difference in their SAD rates (Cavalli-Sforza, Menozzi and Piazza 1994; Dam et al., 1998; Mersch et al., 1995). The Icelanders and the Swedes are separated by a genetic distance of 106 (x10,000), yet the difference in SAD rates is .4%

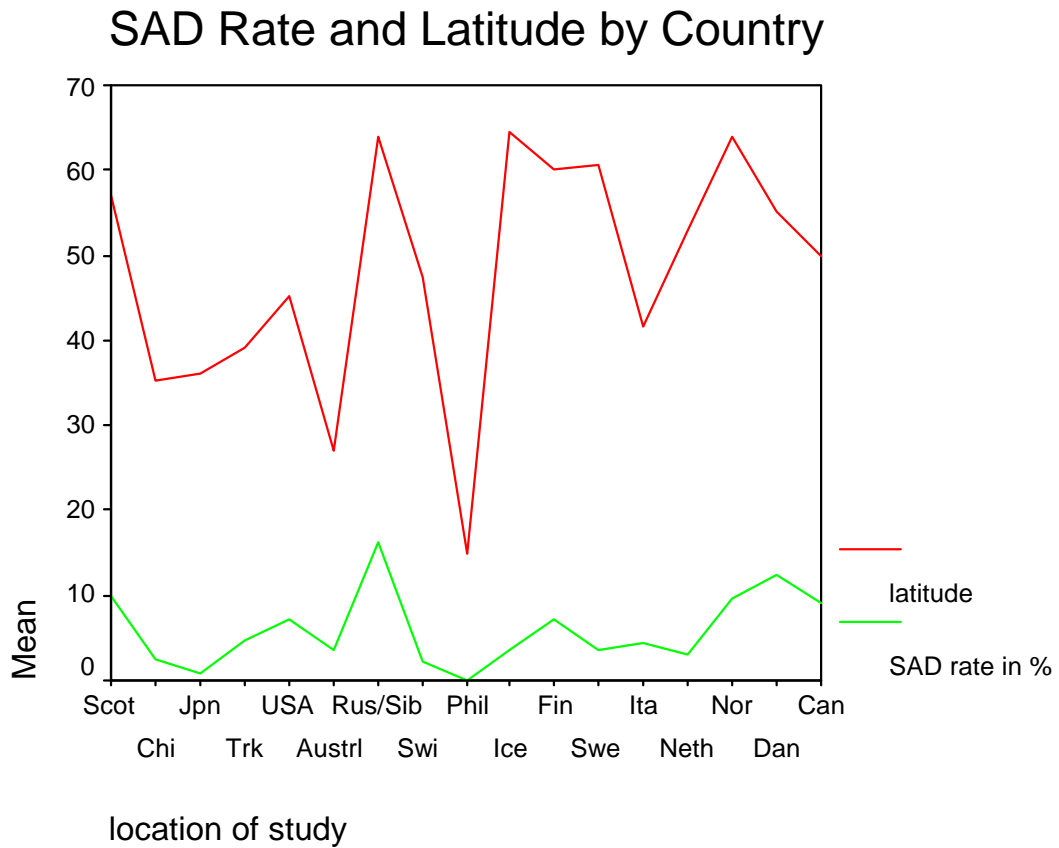
or .1% depending on the source (Broman et al., 1998; Hagfors et al., 1995; Magnusson and Axelsson, 1993). The closest genetic relatives of the Icelanders are the Norwegians. They are separated by a genetic distance of 74 (x10,000). Their average SAD rates differ by 6.05%. This can be seen in Graph 3 “SAD Rates and Genetic Distances.” Examination of the qualitative data revealed evidence that some trends in the quantitative data could be the result of influences other than genetics and/or geographic latitude.

Graph 1.



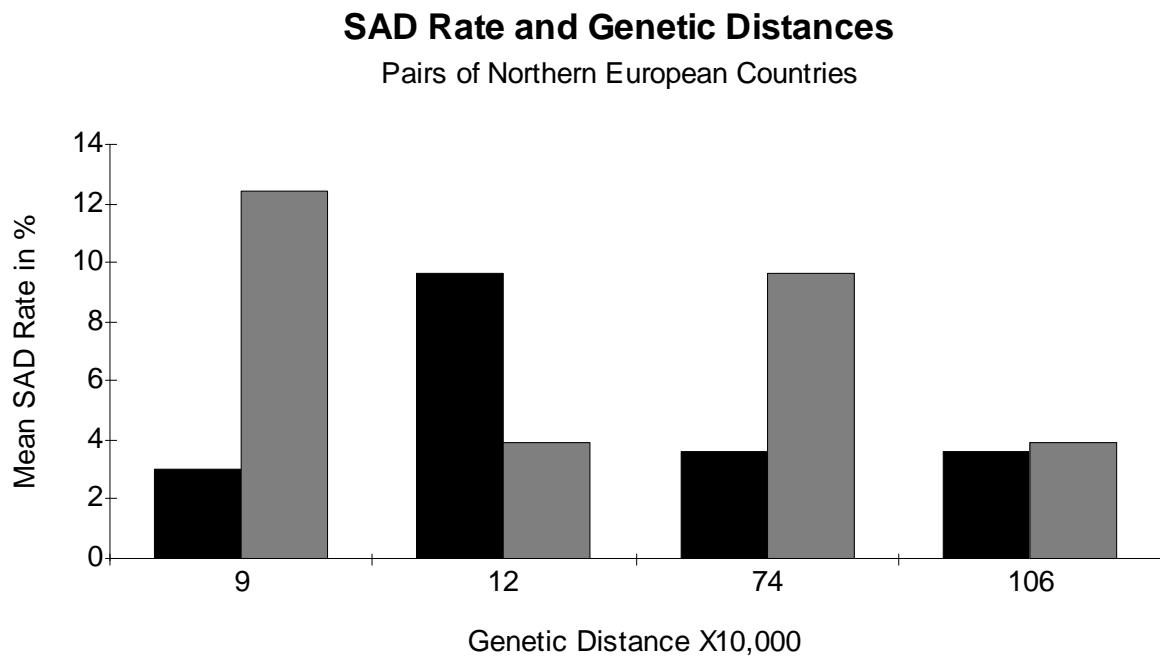
(reset X axis—0 is the minimum possible value)

Graph 2.



(sort countries by latitude for this graph)

Graph 3.



Discussion

Latitude

The findings of this study suggest a number of influences on the SAD rates identified the world. Though the effect of latitude has been minimized in its role in SAD rates by Magnusson (1993) based on his study of Icelanders living in Canada, the results of this study demonstrate that latitude does indeed have a general effect on SAD rates. As latitude increases SAD rates follow suit. The average SAD rate in the low latitudes is

significantly lower than the average SAD rate in the higher latitudes. An increase in SAD rates with an increase in latitude fits nicely with the phase-shifted circadian rhythms hypothesis (Lam, 2000). One would expect not only longer periods of darkness in higher latitudes, but also more month-to-month or even week-to-week variability in light and darkness with the onset of the dark season in the high latitudes. This would be more taxing on a phase-delayed system as it is constantly and rapidly having to adjust.

Increases in latitude and corresponding increases in SAD rates are not consistent among all populations. This can be seen in the case of the Icelanders (Magnusson and Stefansson, 1993). Also there is a noticeable variability of SAD rates in the same general latitude range. The rates in the Japanese have been found to be consistently low. Imai et al. (2003) found a rate of .89% at 39 degrees north latitude and Ozaki et al. (1995) found a rate of .86% at 35 degrees north latitude. Rosen et al. (1990) identified a SAD rate of 6.3% at 39 degrees north a rate of 4.7% at 40 degrees north in the United States. Since these findings were all at the same latitude, latitude alone cannot account for the differences in SAD rates between the Americans and the Japanese.

Genetics

A genetic adaptation among certain populations has been proposed as a possible reason for differing SAD rates across populations (Axelsson et al., 2002; Magnusson, 1993). Sher (2001) points out that there is some evidence of genetic predisposition to SAD in individuals based on studies on molecular genetics, familiarity and heritability. If a population has adapted genetically in such a way that they are more resistant to SAD, then one would expect to find similar SAD rates in populations that are genetically similar and greater differences in SAD rates with increased genetic distance. In the case

of northern Europe the opposite is true. Denmark's closest genetic relative is the Netherlands, yet there is a 9.4% difference in their SAD rates (Cavalli-Sforza, Menozzi and Piazza, 1994; Dam et al., 1998; Mersch et al., 1995). The genetic distance between Iceland and Sweden is almost tenfold that of Denmark and the Netherlands, yet their SAD rates are only .4% or .1% different depending on which rates one uses (Broman et al., 1998; Cavalli-Sforza, Menozzi and Piazza, 1994; Hagfors et al., 1995; Magnusson and Axelsson, 1993).

Culture

There is a variability of SAD rates among populations that is unable to be completely explained by geographic latitude and/or genetics. Assume, for the moment, that the assessment instrument is valid and reliable across cultures. The variability in SAD rates could be the result of differing cultural adaptations or lack thereof in different populations. This is demonstrated in Stuhlmiller's (1998) ethnographic study in northern Norway where she identified possible adaptive behaviors such as an increase in social events in the dark period, dietary supplementation with fish oil and the use of natural lighting (candlelight) in unusual settings. Stuhlmiller also addresses the northern Norwegians concepts of illness and sadness as possible influences on their perception of SAD symptoms during low-light periods. Differing perceptions of symptoms and concepts of disease raises the question of whether or not SAD even exists in all cultures (Stuhlmiller, 1998).

Kleinman and Good (1985) address differences in the perception of depressive symptoms across cultures. They point to differences in symptoms in depression among cultures and the existence or lack of words in the language appropriate for the description

of depressive experience. They point out the Chinese and Japanese have no words in which to adequately verbalize the depressive experience. It should be noted that low SAD rates have consistently been found in Japan. In 2 studies at 3 different latitudes in Japan the average of the combined SAD rates was .7433% (Imai et al., 2003; Ozaki et al., 1995). Among the Chinese at roughly the same latitude the rate was 2.4% (Han et al., 2000). Though this rate is higher than the Japanese it is considerably lower than rates in the US and in Turkey within 4 degrees of latitude (Elbi et al., 2000; Rosen et al., 1990).

These possible cultural differences bring into question the validity of the SPAQ for identifying SAD across cultures. The SPAQ is an American creation, originally created in English. It is in the form of a questionnaire (Magnusson, 2000). Looking at this sample of studies, it is evident that the SPAQ is testing something and appears to be reliable within populations. The question is whether it or not is actually identifying SAD. Also, the more thorough diagnostic interview gives lower SAD rates in the USA (Blazer et al., 1998). Given the differences in various cultures' perceptions of illnesses and their lexicons to express these perceptions, one should question if it is appropriate to administer the SPAQ across cultures and expect valid results.

Conclusions

The findings of this study conclude there is a correlation between an increase in latitude and an increase in SAD rates that is significant. However, given the variability of SAD rates at the same latitude across the globe other factors must be considered. There is some evidence for a genetic adaptation in some populations that may influence SAD rates. However there is variability in SAD rates between genetically similar populations which brings into question genetic adaptation as an explanation for variation at a given

latitude range. What have not been thoroughly considered, are all the cultural influences on the SAD diagnosis across the globe and the means by which SAD is identified in different cultures. The current method of identifying SAD for scientific research, the SPAQ, may not be the valid because of cultural bias within the instrument as well as bias on the part of those conducting research. Identification of SAD through any method may also have pitfalls depending on the culture being studied, their concept of depressive symptoms during winter and their adaptive or maladaptive behaviors.

SAD is likely the product of a combination of genetics, geography and culture. The geographic influences are well established. Further study in regard to the influences of culture and genetics on SAD rates is recommended. This should be done with careful consideration of the diagnostic criteria for SAD and the assessment instrument to be used as well as the culture of the population being studied.

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