

## Seasonal Affective Disorder: A Narrative Review

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### ABSTRACT

This narrative review of research includes summaries of 46 papers on seasonal affective disorder that were published during the period 2019-2023. The publications are primarily cross-sectional studies of risk factors, randomized controlled trials for light therapy and cognitive behavioral therapy and systematic reviews. A few of the papers are focused on the prevalence of seasonal affective disorder which has varied by latitudes and climates from 3% in Saudi Arabia to 21% in Norway, suggesting length of day and sunlight effects. Only a few studies focused on the negative effects of seasonal affective disorder including sleep problems, depression and poor performance on memory tasks. Most of the research has addressed risk factors for developing seasonal affective disorder including younger age, female gender, dependent personality, seasonal beliefs, chronotype (eveningness), inactivity, insomnia and depression. Very few potential underlying mechanism studies appeared in this literature with the exception of MRI studies suggesting reduced brainstem volume. In contrast, many intervention studies were published recently including light therapy and cognitive behavioral therapy. Methodological problems relate to the variability in the assessments of seasonal affective disorder, the reliance on self-report measures and the cross-sectional data that are not definitive about directionality of predictors and effects of seasonal affective disorder.

Seasonal affective disorder (SAD) is a mood disorder that is a subtype of major depression disorder that occurs typically in the fall or winter, with a remission in spring or summer and has also been called winter depression [1]. The risk factors frequently given include being young (18 to 30 years of age), female, having a family history of seasonal affective disorder and living in a northern latitude. Diagnosis requires two consecutive years of winter episodes and summer remissions. The typical treatments have included light therapy, cognitive behavioral therapy and antidepressants either alone or in combination. The light therapy is usually somewhere between 2,000 and 10,000 lux (intensity) for 30 to 60 minutes at the same time every day, most preferably in the morning. For shorter sessions (20-30 minutes a day) 10,000 lux is recommended. Exercise and exposure to natural light are also encouraged.

This narrative review involved entering the terms seasonal affective disorder and the years 2019-2023 into PubMed and PsycINFO. Although the search yielded 168 papers for this period, exclusion criteria including case studies, non-English papers and study protocols reduced the number to 46 papers. This narrative review of research includes summaries of those 46 papers on seasonal affective disorder.

The publications of this period are primarily randomized controlled trials and systematic reviews of randomized controlled trials. A few of these are focused on the prevalence of seasonal affective disorder which varied from 2 to 21% in different countries. Surprisingly very few researchers focused on negative effects of seasonal affective disorder including memory loss and sleep problems. Most of the studies addressed predictors/risk factors of seasonal affective disorder including female gender, young age, eveningness, sensory sensitivity, sleep problems and inactivity. Potential underlying mechanisms included attenuated cortisol levels and reduced brainstem volume. Interventions that appeared in this recent literature on seasonal affective disorder included light therapy, cognitive behavioral therapy and antidepressants. Methodological problems relate to the variability in the assessments of seasonal affective disorder, the reliance on self-report measures and the cross-sectional data that are not definitive about directionality of predictors and effects of seasonal affective disorder. This review is accordingly divided into sections on the prevalence of seasonal affective disorder, the effects of this condition, predictors/ risk factors, interventions, potential underlying biological mechanisms and methodological limitations of the literature.

### Prevalence of Seasonal Affective Disorder

The prevalence of seasonal affective disorder (SAD) has varied widely from 2% to 21% across countries of varying latitudes (see table 1). In a study from Saudi Arabia, health students were compared to non-health students (N=391) [2]. The prevalence of SAD was 3% while the prevalence of major depressive disorder (MDD) was 5%. The non-health students experienced more severe depression and anxiety while the health students experienced more severe stress. These groups are rarely compared, but their differences suggest the need for further research. The health students may have had more stress related to the significant demands of their studies and their exposure to medical conditions and less time for feeling depressed or anxious.

**Table 1: Prevalence of seasonal affective disorder (and first authors)**

Prevalence	First author
3% Saudi Arabia	Alhenoki
19% Alaska	Drew
21% Norway	Bjorvarn
8% Zurich	Wirz-Justice
13% Portugal	Fonte
2-9% worldwide	Nussbaumer-Streit

At the other extreme, in a study from Alaska, the prevalence of SAD was 19% and the prevalence of subsyndromal SAD (early symptoms but failure to meet the diagnostic criteria) was 44% [3]. The predictors were female gender, younger age, and being social versus being independent during work-out sessions at a gym. Similarly, the prevalence of SAD was high at 21% in Norway (N=45,338) [4]. In this study, the predictor variables were again being female and younger, but also being unmarried, having low education and no children. High seasonality was associated with late chronotype (eveningness or preferring evening hours). These high prevalence data for two high latitude countries suggest northern latitude as a risk factor, although other confounding risk factors like sociodemographic variables and chronotypes were implicated in this study.

Different chronotypes have been given animal labels based on the sleep/wake habits of animals. The bear is the most common at 50% of samples. This chronotype is characterized by the sleep and wake cycle happening according to the sun, with greater productivity in the morning and no trouble sleeping, but a post-lunch energy dip between two and four in the afternoon. The wolf is like a night owl who goes to sleep late and awakens early. It's not clear why this chronotype wasn't named after the night owl. The lion prefers early mornings, one would presume because that's typically when the lion goes hunting. The dolphin is the least common (10% of samples) which is characterized by being "wired and tired". The dolphin is tired during the day and wired with nervous energy at night. It's not clear why this chronotype was named after the dolphin.

Prevalence data for countries in between the extremes just reviewed include 8% for Zurich for minor or major autumn/winter episodes [5]. In this sample (N=499), repeated winter episodes occurred in 3% and one winter episode occurred in

10%. Women were noted to have five times more episodes than men. These were comorbid with social anxiety disorder and agoraphobia as well as diurnal variation of mood which suggested improvement in the evenings. These gender differences in SAD episodes are not surprising given that social anxiety disorder occurs more often in women (6% in women versus 4% in men) as well as agoraphobia (5% in women versus 2% in men). Research exploring the mediating/moderating variables for the relationships between gender, SAD, social anxiety disorder and agoraphobia would help inform the intervention literature for each of these disorders.

Seasonal sensitivity as a precursor to SAD has also been assessed in higher education students in Portugal (N=324) [6]. In this sample, a relatively high prevalence of SAD (13%) was noted as well as a high prevalence of subsyndromal SAD (29%). But the SAD group also had a high prevalence of psychiatric comorbidities (29%), while only 3% of those without SAD had psychiatric comorbidities. These high prevalences in higher education students are consistent with the greater prevalence of SAD noted in young adults.

Latitude would seemingly be a factor, as already mentioned, since the prevalence for both Norway and Alaska at the high prevalence extremes are 40 degrees north of the latitude of Saudi Arabia which has reported a low prevalence. This is also exemplified by a review of studies worldwide that suggested a prevalence that ranged from 1.5% to 9% as a function of varying latitude [7].

Surprisingly, at least one research group noted no evidence of seasonal variation in mild forms of depression [8]. And, 30% of the 41 studies in a systematic review on winter depression either reported no seasonality or inconclusive data [9]. These authors suggested, however, that the studies did not meet criteria for a meta-analysis because of "heterogeneity of symptoms, disorder definitions, operationalism and measurement and publication bias".

### Effects of Seasonal Affective Disorder

Several effects have been noted for seasonal affective disorder (see table 2). They include memory impairment, lack of well-being, psychological morbidity, depression, and sleep problems, all of which are notably comorbid.

**Table 2: Effects of seasonal affective disorder (and first authors)**

Effects	First author
Memory impairment	Iorio
Less Well-being	Alvarado
Depression symptoms	Lukmanji
Sleep problems	Delaney

In one of the rare experimental studies comparing adults with SAD and adults without SAD (N=120), neutral or arousal stimuli were presented [10]. The researchers reported that both attention bias to negative stimuli and declarative **memory impairment** occurred in those with SAD on recall and recognition tasks. Those data were not surprising given that impairment in those two cognitive functions has been associated with depression.

In a study that explored the effects of seasonal sensitivity on those with SAD who were living in extreme environments, the Seasonal Pattern Assessment Questionnaire was completed (N=370) [11]. Those who had greater seasonal sensitivity had less **well-being**. Presumably seasonal sensitivity would be an early marker for SAD, especially in extreme climates and for those who had not experienced seasonal variety.

In a paper entitled "Seasonal variations in symptoms of depression", the Patient Health Questionnaire was given (N=8000 who were 12-24 years-old and 45,000 who were greater than 25 years-old) [12]. The younger group with mild symptoms had greater depression in the winter months. The symptoms included lack of interest/pleasure, feeling depressed/down, hypersomnia/insomnia, lacking energy, poor appetite/overeating, feeling bad about yourself/like a failure, being slow/fidgety, and trouble concentrating. In the adult group, only those with sleep and poor appetite/overeating experienced SAD. Adults expressing self-harm/suicidal thoughts did not report seasonality likely because their depressive symptoms were chronic rather than limited to the winter season. The negative effects could also relate to the participants having less exercise during the winter which would lead to decreased serotonin levels and greater depression. This study had the frequent limitation of being cross-sectional and having unequal sample sizes for group comparisons. In addition, the use of antidepressants was not given.

In research on **sleep and circadian rhythm** profiles in adults with seasonal depression, both SAD and non-SAD adults participated (N=103). Based on cluster analysis, two sleep/ circadian profiles were noted in those with SAD. The "disrupted sleep" cluster involved irregular and fragmented sleep and the "advanced sleep" cluster involved early sleep as well as greater total sleep time. Those with the advanced sleep cluster would be expected to have more severe or chronic depression symptoms.

### Risk Factors/Predictor Variables for Seasonal Affective Disorder

Many risk factors/predictor variables for seasonal affective disorder appear in this recent literature (see table 3). They include the demographic variables of being young and female, chronotype (morningness/ eveningness), body mass index, dependent personality, seasonal beliefs and sensitivity, depression, insomnia, inactivity, living in a seasonal home and being bipolar type II.

**Table 3: Predictors/Risk factors of seasonal affective disorder (and first authors)**

Predictors/Risk factors	First author
Young and female	Drew, Holler
Eveningness	Bjorvatn, Camuso
Cold temperature	Nevares-Flores
Short length of day	Landsall-Welfare
Seasonal beliefs	Rohan
Sensory processing sensitivity	Hjardt
Dependent personality	Terman
Sleep problems	Hobeika, Wescott
Seasonal home	Bertrand
Bipolar II disorder	Yeom

As already mentioned in the study from Alaska, predictor variables for seasonal affective disorder were **being young and being female** [3]. Being young and female has been a risk factor in several studies in this literature including a study entitled "The effect of age and chronotype on seasonality, sleep problems and mood" (N= 410) [13]. In this research, the oldest group (greater than 59 years) was more resilient to seasonal change, and they had lower scores on sleep problems, anxiety, depression, stress, seasonality, and chronotype. These authors suggested that their research was inconclusive because of its cross-sectionality and its being limited to one culture, as most studies are in this recent literature. Longitudinal and cross-cultural studies have rarely appeared, likely because of their cost and inconvenience.

**Chronotype (eveningness)** has been a risk factor for SAD in a number of studies, including the research in Norway already described [4]. And in another study, morningness was associated with less severe depression [14]. These results are not surprising given that morning typically involves more light for greater activity and the evening features less light which in turn is a risk factor for SAD. Surprisingly, researchers in countries like Norway that have an all-day dark season and an all-day light season haven't reported greater severity of SAD, although they have reported greater prevalence of SAD. Also surprisingly, comparisons have not appeared in this literature on all-day light and all-day dark seasons even though researchers from several Scandinavian countries could make this comparison.

The importance of **natural light** is demonstrated in a review on 13 studies entitled "The under-explored presence of seasonal affective disorder in the southern atmosphere" [15]. This research revealed greater prevalence in Tasmania, the southern-most state and the coldest state in Australia. However, it is extremely sunny in their winter with a UV index of 11 (an extreme index), and their temperatures are warmer than the UK. These results might seem paradoxical given that Tasmania is closer to the polar zone, but it is also warmer there than mainland Australia, likely because of their greater sunshine. In contrast to light, temperature has not been considered as a predictor or even a potentially confounding variable for the effects of light.

Low environmental light and **short length of day** have also been associated with the prevalence of anti-depressant prescriptions [16]. These data are not surprising, although the use of anti-depressants was less often reported than light therapy and cognitive behavioral therapy as an intervention for SAD.

**Seasonal beliefs** also have predictive validity for SAD. For example, in a study entitled "Predictive validity of the Seasonal Beliefs Questionnaire for discriminating between seasonal and non-seasonal major depressive disorder", this scale was developed from a qualitative study during a course of CBT-SAD therapy [17]. This 26-item scale significantly predicted SAD with its items on maladaptive thoughts about the seasons, light availability, and weather conditions. It would be interesting to compare the predictive validity of this SAD-specific scale versus the more traditional measures of depression, especially since this seasonal scale is not often given in a diagnostic workup when depression symptoms are present.

In another study, the Highly Sensitive Person Scale (HSPS) was used to assess seasonal affective disorder. In this paper entitled “**Sensory processing sensitivity** and its association with seasonal affective disorder”, those individuals with SAD during both symptomatic and remission phases had higher scores on the Highly Sensitive Person Scale [18]. 25% of the individuals with SAD had higher scores on the HSPS while only 5% of the controls had higher scores on the HSPS. Higher scores during the summer predicted SAD in the winter. That this scale was significantly predictive of SAD is surprising as its items relate to sensitivities to non-visual senses like smell and noise. If these scores were entered into a regression model, seasonal sensitivity scale scores would be expected to explain more of the variance than scores on the HSPS given that high sensitivity regarding other senses would be expected to persist across seasons, not just winter.

Personality pathology has also been implicated as a risk factor for seasonal affective disorder [19]. In a sample of 174 adults with SAD, 56% were noted to have pathological personality traits and disorders and 37% reportedly had any disorder. Dependent personality was the most common disorder. Personality pathology was related to higher depression scores but not to responses to cognitive behavioral therapy or light therapy treatment. More significant relationships between personality pathology and depression than between personality pathology and responses to various forms of treatment would be expected given that personality pathology is often non-responsive to treatment.

REM sleep has also been a risk factor for seasonal affective disorder. In a study on Lebanese adults with SAD, both **insomnia** and depression led to greater odds of SAD [20]. These are not surprising data given that depression and REM sleep are highly comorbid. These data are further supported by findings reported in a paper entitled “Elusive hypersomnolence in SAD: actigraphic and self-reported sleep in and out of depressive episodes”, which suggested that sleep time was 72 minutes greater on clinical interviews and 23 minutes on actigraphy during the winter (N=64) [21]. No differences were reported on sleep diaries or retrospective self-reports. Endorsement of winter insomnia by participants with SAD was predicted by greater fatigue, total sleep time, time in bed, naps, and later sleep. These data highlight the greater accuracy of actigraphy than sleep diaries and retroactive sleep report, although the endorsement of winter insomnia by participants seems inconsistent with their self-report and diaries and lack of awareness of them.

In a meta-analysis of seven polysomnography studies (183 subjects, 109 with SAD and 74 controls), **REM sleep** and REM latency were greater in SAD participants even during remissions [22]. Although no differences were noted in other sleep forms including slow wave sleep, sleep efficiency, or total time in sleep during remissions, the data suggested that REM sleep and REM latency were more sensitive biomarkers of individuals who experienced SAD as well as summer remissions of winter SAD.

In a study entitled "Effect of hometown seasonality on undergraduate students' risk of developing SAD", students from **non-seasonal** hometowns (towns that didn't change seasons) were compared to those from seasonal hometowns on the Seasonal Patterns Assessment Questionnaire (N=115) [23]. The scores on that scale for seasonal hometown students did not

change on campus in the winter, but the scores increased for students from non-seasonal hometowns, suggesting that they are at greater risk for the development of SAD. These findings are not surprising because non-seasonal hometown students would have less experience with the change of seasons and likely less resilience for adapting to seasonal changes as they attended their university campuses in winter.

In a paper entitled "Bipolar II disorder has the highest prevalence of seasonal affective disorder (N =378), data were taken from the Mood Disorder Cohort Research Consortium [24]. Greater seasonality scores were noted in mood disorders with the highest scores for those experiencing **bipolar II disorder** versus bipolar I and major depression. Individuals with bipolar II disorder would experience greater depression than those with bipolar I and less need for sleep than either those with depression or bipolar I which may explain their higher scores on seasonality and the propensity for SAD.

#### Interventions for Seasonal Affective Disorder

Most of the research on interventions for seasonal affective disorder have involved light, either natural or artificial light (see table 4). Other studies have compared light therapy with cognitive behavioral therapy and with antidepressants or the combinations of these therapies.

**Table 4: Interventions for seasonal affective disorder (and first authors)**

Interventions	First author
Sunshine/summer light	Kanazawa, Rohan
Light therapy	Tao
Bright light	Nussbaumer-Streit, Prjek
Blue light	Do
White light	Wirz-Justice
Light therapy plus antidepressants	Geoffrey
Light therapy plus CBT	Rohan
Tourism therapy	Sha
Dietary intervention	Yang

Natural light studies have assessed the effects of sunshine and summer light. Data from the National Longitudinal Study on Adolescent and Adult Health have suggested that exposure to sunlight contributes to greater happiness [25]. This effect was surprisingly weaker for more intelligent individuals. More intelligent individuals may be more inclined to spend time indoors engaging in intellectual activities which make them happier than being outdoors in sunlight.

Greater prevalence of summer remissions has been noted. Those remissions have typically been attributed to increased sunlight. For example, in a recent sample experiencing treatment for SAD, 79% experienced summer remissions and 13% showed partial remission [19]. These data are not surprising given that the summer sunshine is typically 32,000 to 100,000 lux, which is significantly greater lux than any of the light therapy interventions that have been effective which more closely approximate 1,000-10,000 lux.

Studies on artificial light have compared bright light to dim light and bright light to blue light and have assessed the different intensities of light. In a review of 23 randomized controlled trials on light therapy for individuals with seasonal depression (N=1120 adults), light therapy had mild to moderate treatment effects in reducing depression symptoms [26]. In a light therapy study for preventing SAD, bright-light reduced the risk of SAD by 36% while infrared light reduced the risk of SAD by 50% [7]. These are problematic findings insofar as infrared light has been noted to lead to gradual but irreversible opacity of the lens as well as damage to the retina.

In a meta-analysis on 19 randomized controlled trials (610 participants) on bright light (greater than 1000 lux in a light box or light visor) versus dim light (less than 400 lux), bright light was superior [27]. If 32,000 lux is the minimal lux for sunshine, it is not surprising that greater than 1000 lux was more effective than less than 400 lux. This review was methodologically limited to heterogeneous studies on small to medium sample sizes.

Blue light therapy has also been tried for seasonal and non-seasonal depression, as reported in a recent review of nine randomized controlled trials (N =347) [28]. In most of these studies on adults with SAD, the Structured Interview Guide for the Hamilton Depression Rating Scale (SIGH - SAD) was used. The data reported in this review are mixed, suggesting that they are inconclusive. As the authors suggested, future trials should be of greater duration on larger samples using standardized parameters of light therapy.

In a study that assessed specific parameters of light therapy, the optimal dose for white light was considered 10,000 lux [5]. Thirty to 60 minutes was the optimal exposure and morning was considered the optimal timing. Again, this is significantly lower lux than natural sunlight (34,000 lux), highlighting the importance of those with SAD finding natural sunlight.

In a study entitled "Efficacy of light therapy versus anti-depressant drugs, and of a combination versus monotherapy in major depression episodes", adults (N=397) with moderate to severe major depression from seven different populations were assessed [29]. The median treatment duration was five weeks. Light therapy effects were equivalent to those of anti-depressants and, not surprisingly, the combination of light therapy and antidepressants was the most effective therapy.

Light therapy has also been compared to cognitive behavioral therapy for winter depression (Rohan et al, 2020). In this study, adults (N=177, mean age= 46) were randomly assigned to six weeks of light therapy or to group CBT-SAD.

CBT-SAD was adapted from traditional CBT for depression by these authors. It addresses behaviors and cognitions that are specifically related to winter depression by targeting behavioral activation and cognitive restructuring. Behavioral activation is focused on wintertime anhedonia by identifying, scheduling, and completing pleasant events, and attempts to decrease disengagement and depressed mood by positive reinforcement. Cognitive restructuring involves reframing negative thoughts and core beliefs related to the winter season, lack of light, and bad weather. CBT-SAD was delivered in 12 1.5-hour face-to-

face group sessions held twice weekly. At pre-treatment, body weight and symptoms were assessed on the Seasonal Beliefs Questionnaire and the Chronotype (morningness-eveningness) Questionnaire and depression was assessed on the Beck Depression Inventory. CBT-SAD led to a decrease in negative symptoms while light therapy corrected misaligned circadian rhythms and decreased eveningness. In a subsequent publication, the same research group reported on the same sample that a change in seasonal beliefs mediated the advantages of CBT versus light therapy for winter depression [19]. The CBT group had more flexible seasonal beliefs. This study highlights the cognitive and physical aspects of SAD as well as the therapeutic efficacy of combining a cognitive and a physical therapy for SAD. The limitation of this study is the comparison between a one-on-one (light therapy) and a group therapy (CBT-SAD).

Other interventions have appeared in this recent literature including "tourism therapy" and "diet therapy". In a study entitled "Can rehabilitative travel mobility improve the quality of life of seasonal affective disorder tourists?", samples from Western China (N = 397 tourists of a total sample of 695 adults) were seen before and after a cold escape in winter to the warmer Hainan Island [30]. This intervention was said to be effective which is not surprising as Hainan is at the southernmost point of China and is noted for its tropical climates.

In contrast, a dietary intervention was ineffective. Adults with SAD have been noted to eat larger dinners and more evening snacks and have more cravings for starch - rich food and high fiber [31]. In this review of 11 studies, including five interventions, no effects were noted for nutrition interventions or for vitamin D or B12. The authors suggested that there is a need for larger samples, longer duration interventions, and more randomized controlled trials.

### Potential Underlying Biological Mechanisms for Seasonal Affective Disorder

Several different potential underlying biological mechanisms have been suggested for seasonal affective disorder (see table 5). These include attenuated cortisol levels, changes in EEG alpha waves and reduced brainstem volume.

**Table 5: Potential underlying biological mechanisms for seasonal affective disorder (and first authors)**

Mechanisms	First author
Attenuated cortisol levels	Augustini
EEG alpha power	Theodorsdottir
Change in alpha frequency	Holler
Lower brainstem volume	Majrashi

In a systematic review of 13 studies, an attenuated cortisol awakening response was noted in winter, but not in the summer months [32]. A dexamethasone suppression test suggested a normal suppression of the hypothalamic pituitary adrenal axis. These data highlight another way that SAD can be differentiated from major depression inasmuch as the latter is typically accompanied by elevated cortisol.

In a study that was conducted during summer remissions for adults with SAD (N = 34 adults with SAD from a larger

sample of 110), EEG was recorded during the presentation of 60 emotional pictures and a recognition condition [33]. During the recognition phase, larger alpha power occurred in response to negative versus neutral stimuli, suggesting an inhibition of attention to the negative stimuli. This normal response would be expected during summer remissions of SAD. It's surprising that EEG was not recorded during winter episodes of SAD to determine if alpha power could be used as a biomarker of SAD.

In a study entitled "EEG responses to mood induction interact with seasonality and age", EEG was recorded during rest and during induced mood in the summertime (N=114) [34]. Seasonality in this study was defined as a pre-clinical form of SAD including seasonal variation in mood, appetite, weight, sleep, energy, and socializing. A greater seasonality score was associated with greater changes in the EEG power from rest to sad mood, specifically in the alpha frequency range. This was noted in the greater than 50-year-old adults with an opposite pattern occurring in the less than 50-year-old adults. Although this differential response by age is difficult to interpret, it suggests that future studies need to control for age and for the varying EEG characteristics in the different age groups.

In a different paper published by the same research group entitled "Predictability of seasonal mood fluctuations based on self-report questionnaires and EEG biomarkers in a non-clinical sample", several questionnaires were given [34]. The questionnaire data had 82% accuracy of predicting seasonality as did the EEG alone data. When the questionnaire data were combined with the EEG data, the prediction accuracy rate was 86%. This study was limited by having too many measures for a small sample (N =64), and again being sampled during the summer when the participants were in remission.

In a paper entitled "Brainstem volume mediates seasonal variation in depressive symptoms: a cross-sectional study in the UK biobank cohort", a large sample of MRIs (magnetic resonance images) were reported (N=9,289, age range= 44-79) [35]. The results suggested that photoperiod (a recurring cycle of light and dark periods of constant length also defined as day length) was negatively correlated with low mood and anhedonia and positively correlated with brainstem volume. The negative relationship between photoperiod and low mood and anhedonia was mediated by volumes of the brainstem, pons and medulla. It is not clear how these data would compare to those of adults with major depression and directionality of this relationship, again, cannot be assumed from these cross-sectional data.

### **Methodological Limitations and Future Research Directions**

This recent literature on seasonal affective disorder has several methodological limitations that relate to definitions, diagnoses, sampling, measures, and methods. These limitations are highlighted by several systematic reviews that could not be submitted to meta-analyses because of significant heterogeneity of methods and measures across studies that resulted in their failure to meet criteria for meta-analyses [9].

The definition and diagnostic criteria have varied across studies with some researchers sampling folks who meet the SAD diagnostic criteria of two continuous winter episodes of depressed mood with remissions in the summer and others

sampling the less severe and less chronic seasonality condition. And no studies could be found that traced the longitudinal course of transitions from seasonality to SAD in the same individuals.

Another sampling problem is the reliance on clinical samples without their being compared to non-clinical non-SAD groups. Further, comparisons of data from summers and winters on the same individuals, again using a longitudinal design, are non-existent. The researchers have relied on self-report measures of changes in mood between winter episodes and summer remissions.

Some studies have occurred in northern and some in southern latitudes with the northern latitudes like Norway and Alaska having a greater prevalence of SAD than more southern latitudes or warmer climes like Saudi Arabia and Tasmania. A comparison of samples from different latitudes and different climates in the same study using the same measures would be important for future research. It hasn't been clearly demonstrated whether the critical variable is light, length of day and/or temperature and related activities like exercise that would also decrease during winter and result in depression related to low serotonin levels.

Heterogeneity of measurement by the use of different questionnaires in different studies has also made it more difficult to compare results of different research. And, the reliance on self-report on questionnaires has made the data more subjective and less definitive than the more objective physiological measures like cortisol levels, EEGs and MRIs. In addition, measures like vagal activity and neurotransmitters like serotonin and dopamine that have been widely studied in major depression have also been missing from this literature on SAD.

Most of the studies have focused on single predictor variables rather than assessing multiple variables and conducting regression analysis, discriminant function analysis or structural equations models to determine the relative variance in the outcome variables that is explained by the predictor variables. In these single predictor variable studies, limited control for potential confounding variables or entrance of covariates into the data analyses suggests the possibility of confounded results. The occasional significance of mediating variables highlights the importance of assessing multiple variables in the same samples. The limited literature on effects of SAD and the infrequent consideration of comorbidities is surprising given that depression research has suggested that depressive states are often accompanied by stress, anxiety and sleep disorders.

The recent intervention literature has primarily focused on light therapy and cognitive behavioral therapy which seem to be very different forms of physical versus psychological therapy making it surprising that they are compared, although it suggests that SAD is not simply a physical but also a psychological phenomenon. CBT is one of the most popular therapies for depression, so its appearance in this literature is not surprising. However, better comparisons for light therapy might be natural versus artificial light, light therapy versus different length of day and light therapy versus some form of heat therapy. The higher temperatures in southern climes where SAD is less prevalent has not been considered as an alternative mediating variable for less SAD. Other therapies like physical therapy, massage therapy,

yoga, tai chi and exercise might be effective for SAD as they have been for major depression [36-38].

The potential underlying mechanism literature is also very limited, possibly because research funding has been less available for the more expensive mechanism research. Although the few studies on cortisol, EEGs and MRIs are suggestive of potential mechanisms, they need replication. Having limited this review to studies on humans may have meant overlooking an animal literature that might inform potential mechanisms.

Despite these methodological limitations, this literature has highlighted the prevalence of seasonal affective disorder, with the possibility that the prevalence may have also increased as climate change has increased the number of overcast days in most places in the world. Surprisingly, climate change activists have not mentioned the potential increase in SAD in people along with their expressed concerns about melting icebergs, sea level rises and the loss of animal life. The increasing prevalence of SAD will highlight the need for more intervention, the data on predictor variables will help identify individuals in need of therapy and the intervention data will inform clinicians on potential treatments for those with seasonal affective disorder.

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