

Seasonal Influences on Human Physical and Mental Performance

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Introduction

Many biological processes are controlled by rhythmic cycles. While a large number of cycles are about a day in period (circadian), other significant activities occur on much longer term cycles, such as seasonal or annual (circannual) rhythms. This paper proposes checklists that could be used to evaluate a person's risk for, and assess potential severity of, changes in physical and/or mental performance due to Seasonal Affective Disorder (SAD) or other seasonal disorders. Gender-specific normal infradian rhythms, such as menstrual cycles, are omitted from this paper's scope.

Outline

The rest of this paper will:

- Define SAD and describe its epidemiology
- Describe how SAD is diagnosed and its possible mechanisms
- Describe current SAD treatment approaches
- Describe other conditions commonly comorbid with SAD
- Address novel ways to resist SAD symptoms, or predict how external factors may affect mood and performance
- Collect all the above to create checklists of long- and short-term risks for SAD, which could be implemented as a standalone software application or web-based application.

Naturally, this approach isn't a substitute for formal diagnostic measures.

SAD Definition

Seasonal Affective Disorder (SAD) is a recurrent major depression which generally results in lethargy and depression symptoms during the Fall and Winter seasons (known as 'winter SAD'). The seminal paper formally describing SAD was (Rosenthal, 1984; cited in Winkler, 2005). The most common symptoms include loss of energy, depressed mood, daytime fatigue, irritability, loss of libido, and hypersomnia (sleeping excessively). (Winkler, 2005; hypersomnia definition from NINDS, 2007)

There is also a summer SAD, mainly seen in tropical climates or in people with severe mood disorders, in which the spring or summer months are most depressing. Summer SAD “appears to be less common than winter SAD” (Magnusson, 2000), however no specific data on its frequency were obtained. The rest of this paper assumes SAD refers to winter SAD.

Epidemiology

SAD shows significant variability in frequency of occurrence based on age, gender, and latitude, but not race or ethnicity.

Age and Gender

SAD “can occur in children as young as age 6, although the onset is usually at puberty,” and it is in puberty that the rates of males and females diverge. (Boehnert, 2003; Eagles, 2003) A review of 20 epidemiological studies of SAD showed 14 of those studies reported higher incidence of SAD among women, and 13 reported higher incidence among the young. (Magnusson, 2000)

Latitude

SAD and subsyndromal SAD (SSAD or s-SAD; see SAD Diagnosis section) occur in much higher rates further from the equator, as shown in Table 1. Exceptions to this rule have been noted in Toronto (2.7% SAD rate), Iceland (3.8%), and Sweden (3.9%). (Sher, 2001) This may be due to a genetic component of SAD (see Genetic Abnormalities section later), or simply natural selection (people very susceptible to SAD should choose not to live far from the equator!).

Table 1. Prevalence of SAD and SSAD at various latitudes

SAD, seasonal affective disorder; SSAD, subsyndromal seasonal affective disorder.

Most of table is from (Boehnert, 2003); SAD2 and SSAD2 are similar data from (Sher, 2001).

N Latitude	Location	SAD	SAD2	SSAD	SSAD2
45 – 50	Canada, Seattle, Minneapolis	10.2%	4%, 2.7%	20.2%	10%
40 – 45	Boston, Cleveland, New York City	8.0%	9.7%, 6%	17.1%	20%
35 – 40	Baltimore, Raleigh (North Carolina), St. Louis	5.8%		13.9%	
30 – 35	Phoenix, Atlanta	3.6%		10.6%	
25 – 30	Houston, Tampa (Florida), Baja California (Mexico)	1.4%	1.4%	7.5%	

Race or Ethnicity

While some geographically isolated groups of people have unusually high or low incidence of SAD (e.g. Iceland, Japan, and northern Scandinavia), there is no evidence of racial or ethnic bias in the incidence of SAD. (Magnusson, 2000)

SAD Diagnosis

SAD is a clinically recognized psychiatric disorder. There are three major tools used to diagnose and quantify the extent to which a person has SAD.

- One tool is the Global Seasonality Score (GSS) from the Seasonal Pattern Assessment Questionnaire (SPAQ). (Rosenthal, 1987) ‘The overall degree of impairment during the worst time of the year has to be at least “moderate” to be considered SAD; less severe symptoms may indicate subclinical or subsyndromal SAD (s-SAD). (Winkler, 2005) Some regard the SPAQ as being over-inclusive, which may inflate epidemiological data. (Eagles, 2003; Magnusson, 2000; McCarthy, 2002)
- The Diagnostic and Statistical Manual of Mental Disorders, 4th edition (DSM-IV) is used to diagnose and quantify the extent of SAD depression. (APA, 2000)
- Finally, the extent of SAD depression may be measured with the Structured Interview Guide for the Hamilton Depression Rating Scale: Seasonal Affective Disorder Version (SIGH-SAD). (Williams, 1988; cited in Lewy, 2006)

The World Health Organization (WHO) is still indecisive about the status of SAD. Their “ICD-10 Classification of Mental and Behavioral Disorders” gives only provisional diagnostic criteria for SAD. (WHO, 1992; cited in Tsuno, 2005)

SAD Mechanisms

Major approaches for describing the mechanisms which cause SAD are based on phase shift, melatonin production, serotonin deficiency, or genetic abnormalities.

Phase Shift Hypothesis

The Phase Shift Hypothesis (Lewy, 1989) proposes that SAD is based on abnormal delay of circadian rhythms in winter, which can therefore be fixed by bright light early in the day. Lewy

(2006) refines this to describe affective disorders ‘might be at least partly due to a mismatch in circadian rhythms, ... related to the sleep/wake cycle and those that are more tightly coupled to the endogenous circadian pacemaker’ in the suprachiasmatic nuclei. Later dawn causes a phase delay in their circadian rhythms with respect to clock time, and hence with respect to the sleep/wake cycles. While the phase shift patterns explained by Lewy are statistically significant ($p < 0.01$ in all cases), they are often very weak (R^2 often between 0.1 and 0.5), suggesting that the Phase Shift Hypothesis may be a significant element in SAD, but isn’t the whole story. Murray (2006) tried to verify some of Lewy’s work and could get agreement on the predicted directions of changes, but not quantitative agreement on their magnitudes.

The phase shift hypothesis would suggest that the core body temperature of SAD patients would also be phase delayed, but this was not observed by Lam (2000).

Melatonin

Melatonin is a neurotransmitter-like hormone produced mainly by the pineal gland. It helps regulate many body functions, however its role in circadian rhythms is critical because the ‘enzymes of melatonin synthesis are activated ... by darkness.’ (Malhotra, 2004) Therefore melatonin synthesis is triggered by nightfall under normal conditions, and is suppressed by sunrise. According to Sher (2001), “SAD may be the result of abnormal secretion or or sensitivity to melatonin,” however attempts to prove this hypothesis have been inconclusive. Melatonin can be measured by saliva sample, in blood plasma, or indirectly by looking for its metabolite 6-sulphatoxymelatonin in urine. (Arnedt, 2003)

Only SAD patients demonstrated a seasonal variation in melatonin profile under dim light at night, suggesting that they respond to seasonal photoperiodic signals that the control subjects did not. (Lam, 2000)

A possible insight into SAD was found by (Wehr, 2001) – in people with SAD, the duration of melatonin secretion is longer in winter than normal, which could produce some of the characteristic symptoms of SAD. A similar result was also cited by (Lam, 2000). Wehr’s work contradicts the phase shift hypothesis, in that the onset of melatonin secretion was not different for SAD subjects compared to controls.

Serotonin Deficiency

The serotonin levels in many animals (including humans) have been shown to have strong seasonal variations. L-tryptophan levels peak in April and May, and are lowest in late summer and early fall. (Lam, 2000) Some symptoms of SAD, such as craving carbohydrates, point to a serotonin deficiency or dysfunction. Based on this observation, SAD has been treated successfully using selective serotonin reuptake inhibitors (SSRIs). (Sher, 2001)

L-tryptophan is a precursor of serotonin, which is why the former has also been investigated as a SAD treatment option, sometimes in conjunction with light therapy. The serum level of tryptophan can be reduced to 20% of normal through oral ingestion of a mixture of other large neutral amino acids, causing a reduction in serotonin synthesis. (Lam, 2000)

These results suggest that serotonin and tryptophan play a significant role in SAD.

Genetic Abnormalities

Mood disorders such as SAD are complex phenotypes, or ‘spectrum’ disorders, hence it is not likely that a single genetic answer will be found. Genetic approaches for predicting SAD or understanding its mechanisms have taken three tracks – looking for epidemiological similarities which point to genetic factors, looking for serotonin system defects, and looking for clock defects.

Epidemiological studies have typically looked at twins or close family members. Genetic effects have predicted from 29 to 69% of variance in seasonality scores, with the strongest variance prediction for men. Family studies have shown slightly increased likelihood of first degree relatives having SAD, and a much stronger increase in likelihood of having other mood disorders. (Lam, 2000; Sher, 2001)

Promising serotonin system defect gene candidates are serotonin transporter promoter 5-HT gene polymorphisms, however studies have yielded mixed results. (Lam, 2000) More specifically, the serotonin transporter promoter repeat length polymorphism 5-HTTLPR has been extensively studied in connection with both SAD and alcoholism. While results are not one-sided, most evidence favors ‘the hypothesis that the 5-HTTLPR is involved in the etiology of both seasonality and alcoholism.’ (Sher, 2004)

Clock-related gene studies have also had mixed results, but promising candidates for circadian sleep disorders include mutations and missense variants in the Clock, Per1, Per2, and casein kinase 1 delta (CK1 δ) genes. (Ebisawa, 2007)

Traditional SAD Treatment Options

SAD is typically treated with light therapy and/or pharmaceutical intervention.

Light Therapy

The retina has circadian sensors, ganglion cells containing melanopsin (Barinaga, 2002), which are separate from the rods and cones used for receiving visual information. As summarized well by Bierman (2005), the circadian system in the retina differs from the visual or optical system in five ways:

- “The circadian system has a much higher threshold for activation
- It has a peak spectral sensitivity at a much shorter wavelength
- It has greater sensitivity to light in the inferior retina (viewing the sky) than in the superior retina
- It requires much longer exposures for activation
- It is differentially sensitive to light depending upon the time of day”

Based on these characteristics of the circadian sensors in the retina, treatment of SAD using light therapy (a.k.a. phototherapy) tends to use very bright light (2500 to 10,000 lux) for a fairly extended time period (15 to 30 minutes), early in the day, as summarized in a statistically rigorous review by Golden (2005).

Per the phase shift hypothesis, the reason light therapy works is that the phase response curve for people near dawn produces a phase advance, which counters the phase delay inherent in their bodies. This also works from the melatonin viewpoint, since bright light stops melatonin production, and therefore encourages one to be more alert. (Arendt, 2003)

The International Society for Affective Disorders (ISAD) also recommended wake therapy (i.e. controlled sleep deprivation) after a November 2004 review of the current literature.

Specifically, their recommendations were:

“(1) Wake therapy is the most rapid antidepressant available today: approximately 60% of patients, independent of diagnostic subtype, respond with marked improvement within hours. Treatment can be a single or repeated sleep deprivation, total (all night) or partial (second half of the night). Relapse can be prevented by daily light therapy, concomitant administration of SSRIs, lithium (for bipolar patients), or a short phase advance of sleep over

3 days following a single night of wake therapy. Combinations of these interventions show great promise.

(2) Light therapy is effective for major depression – not only for the seasonal subtype. As an adjuvant to conventional antidepressants in unipolar patients, or lithium in bipolar patients, morning light hastens and potentiates the antidepressant response. Light therapy shows benefit even for patients with chronic depression of 2 years or more, outperforming their weak response to drugs. This method provides a viable alternative for patients who refuse, resist or cannot tolerate medication, or for whom drugs may be contraindicated, as in antepartum depression.

(3) Given the urgent need for new strategies to treat patients with residual depressive symptoms, clinical trials of wake therapy and/or adjuvant light therapy, coupled with follow-up studies of long-term recurrence, are a high priority.”

(Wirz-Justice, 2005)

In spite of this clear endorsement, light and wake therapies are noted by Wirz-Justice not to be pursued as much in research because of “the commercial drawback that they cannot be patented” unlike neuropharmacological treatments of depression.

Pharmaceutical Intervention

As noted earlier, SSRIs have been used to treat SAD, such as Prozac and Zoloft. The effectiveness of these treatments is about the same as light therapy, 60%. (Blumberger, 2005) Other drugs such as Wellbutrin and Provigil have shown promise in small studies. Melatonin, beta-blockers, L-tryptophan, and St John’s Wort have been tried, with mixed results. (Blumberger, 2005)

Comorbidity

SAD and related seasonal disorders are often found in conjunction with other diseases or disorders. Since SAD affects mood, sleep, and melatonin, it is a prime candidate for affecting a wide range of seemingly unrelated conditions.

Beyond the conditions discussed below, SAD or seasonal variations in symptoms have also been comorbid with panic disorders, social phobia, chronic fatigue, anxiety disorders, and obsessive-compulsive disorders. (Eagles, 2003; Magnusson, 2000)

Bulimia

Bulimia nervosa is also a seasonal disorder, and it has been successfully treated with light therapy. (Eagles, 2003)

It has been suggested that SAD and binge eating during winter might be a leftover evolutionary adaptive mechanism – the tendency to eat as much as possible during sparse food supply months might have been good initially, until high calorie food became readily available. The 7R allele of the dopamine-4 receptor (DRD4) gene has been shown to be significantly more common in binge eaters. (Levitan, 2004)

Premenstrual Dysphoric Disorder

Premenstrual dysphoric disorder (PMDD), a severe form of premenstrual syndrome (PMS), often accompanies SAD. (Portella, 2006) Since fertility is somewhat seasonal in humans, it isn't surprising that premenstrual symptoms are also seasonal. Even when no SAD or s-SAD symptoms were present, PMDD exhibits seasonal variations. Some symptoms of PMDD may overlap with SAD, such as craving carbohydrates, depressed mood, and lethargy. (Bhatia, 2007) Because of this, Portella recommends that "Future research testing possible explanations of the co-occurrence of seasonal and premenstrual symptoms should incorporate the full range of severity on symptom variables, treating them as continua rather than solely as binary categories."

Alcoholism

People with a history of a mood disorder are twice as likely (odds ratio 1.9) to be alcoholics. As noted previously under the Genetic Abnormalities section, there is good evidence that the same serotonin gene polymorphism 5-HTTLPR contributes to both SAD and alcoholism. (Sher, 2004)

Reduced Odor Detection Threshold

Experiments with hamsters, squirrels, and lemurs showed that a lack of sense of smell, due to olfactory bulbectomy, disrupted their seasonal rhythms. Based on this, experiments were done by Postolache (2002) which showed that the odor detection threshold of people with SAD were statistically lower ($p=.006$) than control patients; hence the SAD patients were more sensitive to odor.

It is suggested that odor sensitivity is associated with depression rather than seasonality. Neuroimaging suggests that olfactory processing is overstimulated, producing increased

sensitivity. Olfactory stimulation also augments phase shifting, so it's possible that it could be used to increase the effectiveness of light therapy. (Postolache, 2002)

Novel Mood Enhancement Methods

Many other approaches have been tried to counteract the effects of SAD, or more generally respond to seasonal mood changes.

Diurnal Preference

Diurnal preference is the time of day when a person prefers to be active. Those who tend to wake early (a.k.a. morningness, early risers, larks) should be less likely to be affected by SAD, given the effect of early light on causing a phase advance. Those who prefer to wake late in the day (eveningness, late risers, night owls) should be more affected by SAD since they lose that benefit. One's tendency to morningness or eveningness is typically measured using the Morningness Eveningness Questionnaire (MEQ). (Horne, 1976 cited in Bernert, 2005) Surprisingly, some studies have shown that diurnal preference is not a factor in SAD. (Natale, 2005; Bernert, 2005) Eveningness has been linked to more variability in when sleep occurs, and other studies have shown some association between eveningness and symptoms of depression, but often the statistical significance of these associations is weak (p around 0.03 to 0.05). (Bernert, 2005)

Dawn Simulators and Negative Ions

While bright morning light therapy is a common treatment for SAD, a gradual increase of light, simulating dawn, has also proven effective. Terman (2006) compared a gradual dawn light (0 to 250 lux imitating 45 deg latitude); a fixed low intensity dawn pulse (250 lux for 13 min); bright light (10,000 lux for 30 min); and low and high flow rate negative ions (1.3E11 or 4.5E14 ions/sec for 93 minutes). All of these except the low rate negative ions were equally effective (42.7 to 57.1% improvement based on the SIGH-SAD questionnaire), and the dawn pulse led to residual depressive symptoms. They concluded that the dawn simulator or high rate negative ions are just as effective as "traditional" bright light for treating SAD.

Vitamin D

In order to counter a normal drop in serum vitamin D levels during winter, a study by (Vieth, 2004) examined the effect of high doses of vitamin D on a group of otherwise normal thyroid clinic outpatients. A normal dose of 15 mcg/day (mcg = microgram) was compared to an unusually high dose of 100 mcg/day, over a 15 month time period. Those on the high dose showed a significantly improved sense of well-being, even though there was not a significant difference biochemically. While the study design wasn't perfect (e.g. there was no control group, since all of the subjects needed some level of vitamin D supplement), it points to the possibility that other vitamin-based treatment approaches could help cope with seasonal conditions.

Social Factors

A naturalistic study by McCarthy (2002) followed a group of people with SAD to see what influenced the severity of symptoms. Specifically, they were interested in the effect of self-esteem and the perceived level of social support (both measured in the summer), and how that related to the subsequent severity of depression the following winter. SAD was measured with the SPAQ cited earlier. Anxiety, depression, and self-esteem were measured with other surveys at regular two-week intervals. Key observations from this study included:

- The worst levels of depression were in the January-February time period.
- Physiological symptoms (appetite, sleep, and fatigue) emerged before cognitive symptoms (dislike, criticism, worthlessness).
- Anxiety was highly correlated with depressive symptoms.
- Low self-esteem and perceived lack of social support resulted in earlier depressive symptoms, especially when both were present
- Poor social support was associated with earlier anxiety

About two thirds of the test subjects used a light box during the study, but it had no measurable effect on their levels of depression or anxiety. This is a critical observation, since we may be fixing the physical symptoms with light therapy without addressing the psychological ones.

Weather Influences

It is widely known that good weather tends to improve one's mood. Keller (2004) put this to the test, to investigate whether time spent outside, ambient temperature, and/or barometric pressure

affected mood, memory capacity (as measured by digit span), and openness to new information. Recall that high barometric pressure generally results in sunnier weather.

Multiple linear regression was done to assess the influence of each independent parameter (time, temp, pressure) on the dependent variables (mood, memory, openness). In addition, regression parameters were obtained for (time x temp) and (time x pressure) as predictors. The results are summarized in Table 2. The time spent outdoors was judged by whether the person spent more than the median 30 minutes outside that day, or less. If they spent >30 minutes outside, then high temperature and pressure associated with good mood, but if they spent less than 30 minutes outside, the same conditions produced a bad mood.

Table 2. Effects of weather on mood, memory, and openness to new information
Northern USA location in springtime (Keller, 2004)
Dependent variable

Independent var.	Mood	Memory	Openness
Temperature	NS	NS	NS
Pressure	NS	NS	P<.10
Time outside	NS	P<.10	NS
Time x Temp	P<.05	NS	NS
Time x Pressure	P<.10	P<.05	P<.01

NS = not significant (p>0.10)

To be more predictive, this type of data would be needed for a variety of geographic locations and times of year. It confirms the general belief that time spent outdoors improves one’s mood and cognitive ability.

Mood Assessment Checklists

Based on all the previous analysis of seasonal disorders and external influences on one’s mood, two checklists are proposed – one to assess one’s risk for SAD-related symptoms based on relatively fixed parameters (gender, family history, etc.), and the second checklist to provide day-to-day assessment of risk factors (weather, location, behavior, etc.). The first checklist is called the Personal History Form (PHF), and the second checklist is the Recent Habits Survey

(RHS). Table 3 summarizes the data used to create both checklists. It is assumed that hormone levels (e.g. melatonin) and genetic details (other than personal and family history) are not available to the “clients” who use these checklists. Weather information could be looked up online (wunderground.com, weather.com, etc.) for a given location and date.

Table 3. Data Sources for PHF and RHS

Data Description	PHF	RHS	Data Source
Age	X		Calculate from Date of birth in Personal history
Gender	X		Personal history
Latitude and elevation		X	Look up from Location
Race/Ethnicity			Not used
Personal history of SAD	X		Personal history
Family history of SAD	X		Personal history
Twin history of SAD	X		Personal history
History of bulimia	X		Personal history
History of PMDD	X		Personal history, if female
History of alcoholism	X		Personal history
Sensitivity to odors	X		Personal history
Diurnal preference			Not used
Season		X	Determined from the current Date
Vitamin usage		X	Recent habits survey
Level of self-esteem		X	Recent habits survey
Level of social support network		X	Recent habits survey
Calendar date		X	Look up from computer, or have user enter
Time spent outdoors		X	Recent habits survey
Outside temperature		X	Weather for current location and date
Outside barometric pressure		X	Weather for current location and date

Table 3 is used in the following two sections to construct checklists for the PHF and RHS.

Personal History Form

The Personal History Form (PHF) examines invariant or slowly changing parameters to assess one’s risk of SAD or other seasonal disorders. The PHF is shown in Table 4 – it collects the personal history information described in Table 3, plus general client identification information.

The scale used to quantify the relative risk of each response generally follows these rules:

- A risk of 0 is given to responses which contribute no significant risk of SAD

- A risk of ½ is given to responses which contribute a slight risk of SAD. This is generally used for responses which show a possibility of contributing to SAD risk, or show mixed results in studies as to their strength of association.
- A risk of 1 is given to responses which contribute a clear risk of SAD. This is used when studies show an association at the $p < .05$ level or stronger.

For special cases, other risk values may be assigned. A negative risk value indicates factors which reduce the risk of SAD or lessen symptoms. Risk values larger than 1 are possible for very strong indicators, such as a personal previous diagnosis of SAD.

Table 4. The Personal History Form

Field	Risk Value for Yes	Risk Value for No	Notes
Name	N/A	N/A	Basic contact info
Email address	N/A	N/A	Basic contact info
What is your gender?	F = 1	M = 0	Answer other than F or M has risk value = 0
What is your birthday?	N/A	N/A	See below – used to calculate age.
Have you reached puberty yet? If you don't know, say no.	0	-1	Question only used if client is under 15 years old.
Have you completed menopause?	-1	0	Question only used if client is female and over age 40
Have you ever been diagnosed with Seasonal Affective Disorder (SAD)?	+2	0	Is +2 too conservative?
Have you ever been diagnosed with bulimia?	1	0	
Have you ever been diagnosed with premenstrual dysphoric disorder (severe PMS)?	1	0	Question only used if client is female
Have you ever been diagnosed as an alcoholic?	1	0	
Are you more sensitive to odors than others?	1	0	
Do you have a twin?	0	0	
If yes, do they have SAD?	1	0	
Does anyone in your immediate family (other than your twin, if you have one) have seasonal affective disorder?	½	0	

The birthday is used to calculate the client's age. If they are less than 15 years old, it's possible they haven't gone through puberty yet, which makes it a little less likely they could have SAD. If they are over 40 and female, they could have gone through menopause, which would reduce the risk of SAD. While not specifically addressed in the literature studied, having a personal history with SAD would make one much more likely to have it now – perhaps almost certain. The risk scores from the Personal History Form are added, which gives a possible range of -1 to +8½. Exactly how that total risk score should be interpreted would take some additional study, but clearly a higher total risk score indicates a stronger probability that one has SAD or will develop it.

Recent Habits Survey

The Recent Habits Survey (RHS) assesses the impact of changing conditions on one's risk of having SAD, or on the severity of likely SAD symptoms. The RHS includes both conditions one can change (going outside) and conditions one can't change (the weather). The RHS is given in Table 5.

The Season risk value reflects the plateau effect shown in (Keller, 2005). During Spring and Summer, the risk value is zero. During the transition months (September and April) the risk value is ½. During the winter months between transition months, the risk value is 1.

From the location, look up the Latitude. Assign risk value to Latitude based on:

- For Latitude less than 30 degrees (North or South), risk value is zero.
- For Latitude from 30-40 degrees (North or South), risk value is ½.
- For Latitude greater than 40 degrees (North or South), risk value is 1.

Table 5. Recent Habits Survey

Field	Risk Value for Yes	Risk Value for No	Notes
What is your location?	N/A	N/A	Used to determine latitude, average atmospheric pressure, and look up weather.
What is today's date?	-	-	Might be looked up automatically, hence not needed on the survey.
-	N/A	N/A	Determine Season from the date. See rules above for assessing risk value based on Season (range 0-1).
What vitamin supplements are you taking?	-	-	Provide choices of typical vitamins. Assign risk value = -1/2 for taking Vitamin D or a multivitamin. Risk value is zero for all others.
Do you feel good about yourself today?	0	1	
Do you have friends you can talk to easily?	0	1	
How much time per day have you spent outdoors during the daytime?	-	-	For response > 30 minutes assign "Dvalue" of 1, otherwise Dvalue is zero. Dvalue is used below with weather calculations.
-	-	-	Latitude – see above for calculation of risk values, which range from 0 to 1.
Outside temperature and barometric pressure	-	-	See discussion below for weather risk values, which range from -1 1/2 to 0.

Risk value due to weather is complex. If data like Table 2 are available, they could be interpreted as follows:

- For the current location and date, look up the average temperature and atmospheric pressure.
- If the today's high temperature is above average, give it a Tvalue of 1. Otherwise Tvalue is zero.
- Likewise if today's pressure is above normal, give it a Pvalue of 1, otherwise Pvalue is zero.
- Get the Dvalue (D as in Daylight) from Table 5, which is based on the time spent outdoors.

- If $Dvalue * Tvalue = 1$, the weather gets a risk value of $-\frac{1}{2}$. This corresponds to the “Time x Temp” row in Table 2. The risk value is negative because good weather reduces the risk of SAD.
- If $Dvalue * Pvalue = 1$, the weather gets a risk value of -1 . This corresponds to the “Time x Pressure” row in Table 2.
- For all other $Dvalue$, $Pvalue$, and $Tvalue$ combinations, the risk value is 0. This ignores the $P < .10$ entries in Table 2 for Memory and Openness, since they are isolated, low significance results.
- The net result for weather risk value is a range from $-1\frac{1}{2}$ to 0.

The risk values for the RHS are added up, which produces a total range from -2 to $+4$. Like the PHF, exact interpretation of the RHS total would require additional study, but the general trend of ‘positive risk value total means higher risk of SAD’ applies.

Conclusions

This paper examined seasonal affective disorder (SAD) and other seasonal disorders to help understand what puts a person at risk for developing them, and what conditions and activities either exacerbate or diminish their effects.

Two checklists were developed, the Personal History Form (PHF), and the Recent Habits Survey (RHS), to measure the quasistatic and dynamic aspects of one’s life that could influence the risk of SAD. The result of those checklists is a total risk value, ranging from -1 to $+8\frac{1}{2}$ for the PHF, and -2 to $+4$ for the RHS, which indicate the relative magnitude of risk for developing SAD or the severity its symptoms may have.

The numbers used for risk value are attempts to quantify very broad concepts, so further discussion of the ‘correct’ values is certainly possible – the intent here is to provide a starting point for further study and reflection.

The output from the PHF and RHS could also be used to provide recommendations on how the client could reduce their risk of severe SAD symptoms, such as through getting outside more, taking vitamins, developing a support network, etc. in addition to considering treatment options.

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