Which Environmental Variables Are Related to the Onset of Seasonal Affective Disorder?

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Seasonal affective disorder (SAD) consists of recurrent depressive episodes in which onset and offset exhibit a regular pattern related to the calendar year: Episodes begin in the fall to winter months and remit in the spring to summer months (Rosenthal et al., 1984). Presumably, this temporal pattern occurs as the result of environmental factors that vary systematically across the year: Rosenthal and colleagues (1984) suggested that photoperiod (hours from sunrise to sunset), daily hours of sunshine, and mean daily temperature are "the most promising candidates for future study" (p. 77). Since then, most theories have focused on the duration or intensity of light (Oren & Rosenthal, 1992) and its importance in terms of the amount of light received (the photon-counting hypothesis), the phase shifting of circadian rhythms, or the secretion of melatonin, a neurohormone that is secreted only during the dark (Lewy, Kern, Rosenthal, & Wehr, 1982). This approach also has generated substantial research evaluating artificial light as a treatment for SAD (Terman et al., 1989).

Despite the general acceptance of environmental theories of SAD, few studies have investigated the relationship between SAD and natural environmental change. Several studies have found a strong relationship between the lifetime prevalence of SAD and latitude, which is a major determinant of photoperiod (Potkin, Zein, Stamenkovic, Kripke, & Bunney, 1986; Rosen et al., 1990; Sakamoto, Kamo, Nakadaira, Tamura, & Takahashi, 1993). At very high latitudes, however, prevalence has been lower than expected (Magnusson & Stefansson, 1993; Murase, Murase, Kitabatake, Yamauchi, & Mathe, 1995; Partonen, Partinen, & Lonqvist, 1993). Sakamoto et al. (1993) found that prevalence at different latitudes in Japan correlated with total hours of sunshine from September through March but not with mean December temperature (both averaged over 30 years). In a multiple regression, hours of sunshine, rather than latitude, appeared to be related to prevalence. Our confidence in these results is limited, however, because of possible site differences in sampling and diagnostic methods.

Of particular relevance to the issue of causality are several studies that examined the relationship of environmental factors to when SAD patients are depressed. Rosenthal and colleagues (1984) and Wirz-Justice, Buchelli, and Graw (1986) observed high correlations between (a) the proportion of SAD patients that reported feeling depressed during each month and (b) the mean monthly photoperiod and the mean monthly temperature. Molin, Mellerup, Bolwig, Scheike, and Dam (1996) used a random regression analysis that assessed variation in scores on the Beck Depression Inventory (BDI; Bock & Steer, 1987) over time within subjects (SAD patients). BDI scores were associated with shorter photoperiods and cooler temperatures 2 weeks before; BDI scores were not related to daily minutes of sunshine, total daily solar radiation (energy at the earth's surface), cloud cover, precipitation, or barometric pressure.

From another perspective, Albert, Rosenthal, Alexander, and Rosenthal (1991) found that statistically removing the effects of daily weather on energy level did not eliminate annual rhythm

1 Each minute, an instrument assesses whether a shadow can be detected. If so, the minute is considered sunny; if not, the minute is considered cloudy.
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Figure 1. Annual rhythms of photoperiod, mean daily temperature, daily solar radiation, and daily hours of sunshine in Chicago, Illinois (latitude 42°N). All data points are mean weekly level averaged across 7 years, 1988–1994. To present all four variables clearly on one scale, the magnitude consists of the standard score of each variable with a different constant added. The original means, standard deviations, and the added constants for each variable, respectively, were as follows: photoperiod (13.26 hr, 2.16, 2); temperature (50.34°F, 17.56, 4); radiation (14.24 MJ/m², 6.19, 6); sunshine (6.87 hr, 2.08).

in energy for seven of eight SAD patients with annual energy rhythms. This suggests that these weather factors (temperature, sunshine, precipitation, barometric pressure, and relative humidity) were not responsible for the annual energy rhythms observed. Another study (Kotobuki, Kamei, Fukuzako, Iwagawa, & Morioka, 1994) obtained mixed results in assessing the relationship between the proportion of participants feeling at their worst in each month and the mean monthly temperature and mean monthly hours of sunshine. Finally, Summers and Shur (1992) reported a case study with 13 years of daily ratings in which many onsets of depression followed sudden decreases in solar radiation, although this occurred regardless of season.

A major problem in this research is that the environmental factors that may be related to SAD are highly correlated with each other, so that it is difficult to distinguish which ones may be playing a causal role. For example, Potkin et al. (1986) reported high correlations between prevalence in each of 32 states and (a) the photoperiod on December 15, (b) the mean temperature in December, and (c) the mean daily hours of sunshine in December. The similar annual rhythms of photoperiod, daily hours of sunshine (see footnote 1), mean daily temperature, and total daily solar radiation at the earth’s surface are shown in Figure 1. The correlations between pairs of these variables are all greater than .82.

Photoperiod, however, differs from the rest of these variables in an important way. Photoperiod follows a periodic function that is completely determined by the latitude and the day of the year (Figure 2; Kiesling, 1982); therefore, it varies identically in every year. The phase of the function is set by the calendar date, with the minimum photoperiod occurring on the winter solstice (December 22). The amplitude of the photoperiod function varies with latitude; the further from the equator, the shorter the minimum photoperiod. Because photoperiod varies across latitude and calendar time, the photoperiod hypothesis predicts that the occurrence of SAD will vary by latitude and by calendar date. At higher latitudes, the onset of symptoms should begin earlier because shorter photoperiods occur earlier (see Figure 2). Although not previously noted in the SAD literature, this is true only after the fall equinox (September 22). Prior to the equinox, the photoperiod is longer at higher latitudes (Figure 2). This reverse effect is not taken into account if one examines simply the relationship of onset to latitude. Although the majority of SAD onsets occur after the equinox, about 10% occur prior to it.

Other variables, such as temperature and hours of sunshine, vary from day to day and from year to year. The annual rhythms of these variables are their typical patterns averaged across years. Therefore, these environmental factors should produce both the typical seasonal onset pattern observed with SAD and year-to-year differences in onset time. A number of SAD sufferers do report that their symptoms start earlier (or later) than usual when the fall conditions are particularly cloudy/cold (or sunny/warm). Reacting to such accounts, Mayor, Rice, and Bielski (1991) suggested that a person’s symptoms may begin at different times in different years because of differences in available light.

If we examine a sample of SAD patients in a particular year, episode onsets will be distributed over the weeks of fall and winter.2 We can view onset as a probabilistic event that is the result of the onset risk associated with the level of an environ-

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2 One reason that all onsets are not at the same time is that there presumably are individual differences in vulnerability. There also may be other factors that advance or delay onset.
mental factor at a particular time. If a particular environmental factor is related to SAD, then the risk of onset should be high in weeks when its magnitude is high (assuming the direction of the relationship is positive), and the risk of onset should be low in weeks when its magnitude is low. The general seasonal pattern of SAD onset is the average pattern of risk across years. If we observe onsets over a number of years, then deviations in risk from the average seasonal pattern should correspond to the deviations in the environmental factor from its annual rhythm. In other words, weeks in which the environmental factor is particularly high (or low) should have onset risks that are particularly high (or low), assuming the direction of the relationship is positive. This approach corresponds to separating smooth (annual rhythms and seasonal patterns) and rough (deviations) in Tukey's (1977) *Exploratory Data Analysis*.

On the basis of these formulations, we conducted two studies. In the first study, we used data from published reports and new data of our own to examine the relationship between the onset risk of SAD and photoperiod as it varies across both time and latitude. Prior studies have examined only the effects of either time or latitude. In the second study, we collected data in one location across 7 years to examine whether the weekly risk of SAD onset could be accounted for by any of three environmental variables that vary from year to year: daily hours of sunshine, mean daily temperature, and total daily solar radiation at the earth's surface.

### Study 1

#### Method

**Participants and Procedures**

Data from the four locations other than Chicago were taken from previously published reports (see Table 1). The Chicago data came from years 1987–1995 of our own ongoing study of the onset of SAD (Young, Watzl, Lahmeyer, & Eastman, 1991). All five studies used similar methods. Altogether, 387 participants were recruited from referrals and media notices soliciting persons with symptoms of SAD. All participants were interviewed in person and met diagnostic criteria for a current episode of depression, and they met Rosenthal's criteria (Rosenthal et al., 1984) for a lifetime diagnosis of SAD. All participants were interviewed in person and met diagnostic criteria for a current episode of depression, and they met Rosenthal's criteria (Rosenthal et al., 1984) for a lifetime diagnosis of SAD. The five samples did not differ significantly in mean age (range: 36–44) or gender distribution (range: 73%–86% women). The typical month of episode onset was determined from interviews, questionnaires, or both. The daily photoperiod for each location was computed (Kiesling, 1982), and an average was calculated for each month and location.

#### Statistical Analyses

We conducted a survival analysis (Lee, 1992; Singer & Willett, 1991) for each location using the actuarial life table method (Norusis & SPSS, 1993a). From this analysis, we obtained the risk of onset (hazard rate) for each month at each location. Hazard is a measure of the probability of an onset in a month for the persons at risk in that month, although its upper limit is not one. It is this "force of morbidity" that we

3 In most of these articles, data on the onset of SAD were a minor part of the report.

4 Hazard is a measure of the conditional probability that an onset will occur at a particular time, given that it has not already occurred. If no participants drop out of the study during the period of observation, one could simply compute the conditional probability. In this case, the conditional probability (p) has an exact relationship to hazard (h), such that p = h/(1 + h). However, when there is participant dropout, only the hazard can be estimated. Although there were no dropouts in our data, we chose to follow the traditional survival analysis approach by reporting hazard. The life-table method estimates the hazard at the center of an interval. This is appropriate for our data because (a) onsets occur at various, unknown times within a month (Kalbfleisch & Prentice,
be relatively difficult to demonstrate empirically. For descriptive purposes, we fit the data to an exponential (exp) curve: risk = \( 729.8 \exp(-0.6712 \times \text{photoperiod}) \); \( R^2 = .96 \); data point at 7.32 hr omitted (Jandel Scientific Software, 1994).

The data point for November, 60°N (7.32 hr) did not follow the pattern of onset in the rest of the data. This is consistent with other reports that the prevalence of SAD is lower than would be expected at latitudes from 60°N to 70°N (Magnusson & Stefansson, 1993; Murase et al., 1995; Partonen et al., 1993). We believe that these findings are due to a ceiling effect in the ability of the organism to respond to short photoperiods. This appears to occur at a photoperiod of about 9.5 hr. This explanation is supported by our finding that onset risk was lower than expected at 60°, but only in November when the photoperiod is exceptionally short. Earlier in the fall at 60°N, onset risk was at expected levels. Other explanations of lowered prevalence have focused on cultural differences (Murase et al., 1995) and on population selection in stable populations (Magnusson & Axelson, 1993; Magnusson & Stefansson, 1993). However, these factors are less likely because they should affect onset risk throughout the year. We also note that prior to the fall equinox, fewer onsets should be expected at higher latitudes because the photoperiod is actually longer at 60° than it is at lower latitudes. However, because the onset risk at all latitudes is low this early in the year, this effect will account for a relatively small proportion of “missing” cases at high latitudes and will be relatively difficult to demonstrate empirically.

In our data, there is an absence of information for photoperiods between 7.32 and 9.23 hr (Figure 3). Extrapolation of the fitted curve much beyond 9.23 hr would produce risk values outside a meaningful range. What does the risk curve look like in this range? To fill in this part of the curve, data must come from a range of locations between 47°N and 60°N, where photoperiods this short occur. In addition, onset data with a shorter time resolution (e.g., 1 week) would help to fill in sparse portions of the curve both below and above a photoperiod of 9 hr. In Study 2, we collected weekly onset data in Chicago, but we used methods that are not comparable with those used at other locations.

**Results and Discussion**

Risk of onset exhibited a highly regular relationship with photoperiod (see Figure 3). This supports the hypothesis that the onset of SAD is related to a short photoperiod, regardless of whether it is due to latitude or calendar date. For descriptive purposes, we fit the data to an exponential (exp) curve: risk = \( 729.8 \exp(-0.6712 \times \text{photoperiod}) \); \( R^2 = .96 \); data point at 7.32 hr omitted (Jandel Scientific Software, 1994).

The study included a number of methodological refinements compared with previous work: (a) We used specialized procedures to collect episode onset data, (b) we obtained onset data for each participant’s current episode rather than his or her typical experience, (c) we determined onsets by week rather than by month as in most studies, and (d) data on episode onset were collected in 7 different years. To our knowledge, such data have not been collected previously.

**Method**

**Environmental Data**

From the National Oceanographic and Climatic Data Center, we obtained data collected at O’Hare International airport in Chicago on the
Photoperiod

Figure 3. Risk of onset (hazard) of seasonal affective disorder as a function of photoperiod. The outlying point was omitted in computing the fitted curve.

The following variables for 1988–1994: (a) daily mean temperature, (b) daily hours of sunshine (see Footnote 1), and (c) total daily solar radiation (energy reaching the earth's surface in megajoules per square meter). Photoperiod was calculated using the formula given by Kiesling (1982). For each year, daily data were aggregated into 7-day averages, corresponding to the calendar weeks used for collecting onset data. To compute the annual rhythm for each weather variable, we averaged these values across years, and the means were smoothed using the T4253H smoothing algorithm (Norusis & SPSS, 1993b). For each environmental variable, the deviation for each week of each year was the raw value of the variable for that year and week minus the value of the annual rhythm for that week.

To assess delayed effects of the environment, we also computed 7-day averages for periods beginning 1 to 7 days prior to the beginning of each onset week. This produced eight variables for each environmental factor, the first corresponding exactly to each week in the onset data and the last corresponding to the week prior to the week in the onset data. Although there is no direct evidence as to how long the delay in effect might be, the therapeutic effects of artificial light usually begin in 3 to 4 days (Terman et al., 1989). We calculated the deviations for these lagged variables as described above.

Participants

Participants (N = 190) were recruited in the fall from the Chicago metropolitan area (latitude 42°N) as part of a treatment study for winter depression. Trained clinical raters assessed criteria using the Schedule for Affective Disorders and Schizophrenia (Endicott & Spitzer, 1978; modified for the Diagnostic and Statistical Manual of Mental Disorders: DSM–III–R; American Psychiatric Association, 1987). At the time of evaluation, all participants met DSM–III–R criteria for a current major depressive episode and did not meet criteria for any other current Axis I disorder. Participants also met Rosenthal's criteria (Rosenthal et al., 1984) for SAD (winter pattern): (a) recurrent fall–winter depressions, at least two of which occurred in consecutive years; (b) no seasonally varying psychosocial factors that might account for the recurrent depressions; (c) regularly occurring nondepressed periods in the spring and summer; and (d) no other major psychiatric disorder. In addition, all participants experienced the symptoms most typical of SAD: fatigue, hypersomnia, and increased appetite or weight. No participant received any pharmacological treatment during his or her current episode; a few were in psychotherapy at some time during the current episode but not at the time of their evaluation.

The number of participants in each study year (i.e., the year in the fall from 1988–1994) were 21, 24, 37, 23, 32, 25, and 28, respectively. Each participant provided data in 1 year only. The mean age of participants was 35.9 (SD = 8.8); 153 (80%) participants were women. There were no significant differences among the years in gender, x²(6, N = 190) = 4.13, or mean age, F(6, 182) = 0.22. The 45 participants from 1988 and 1989 were also part of the sample whose individual symptom onsets were reported on by Young et al. (1991).

Procedures

Participants entered the study between mid-November and early March (median, mid-January). At his or her assessment, the participant recalled the week of the current episode during which each of 15 individual symptoms began (see Appendix). To facilitate this process, we gave a participant a set of cards, each of which described one symptom of depression. After selecting the symptoms he or she had experienced in the current episode, the participant was given calendars for the fall and winter months and wrote in personal events that might help trigger and organize recall. The participant then placed the symptom cards on the calendar week in which each symptom first occurred. (Any number of symptoms could begin in any given week.) The week containing August 1 was arbitrarily designated Week 1.

Episode Onset Data

The week of onset of the episode was defined as the week of onset of the first symptom(s). This corresponds to the usual definition of an episode. Symptom onset in this study, and for SAD in general, is simpli-
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Odd because inclusion criteria required the absence of other Axis I disorders prior to the onset of the episode of SAD. The mean time from episode onset to the evaluation was 13.7 weeks (SD = 5.0). Seventy-seven participants repeated the data collection task 1 to 7 weeks later (M = 2.7, SD = 1.7). The test-retest reliability for the week of episode onset was .88; the mean onset week (11.0 and 11.1) was virtually identical for the two administrations.

To obtain the overall seasonal pattern of onset risk, we performed a survival analysis using data pooled across all years. The resulting sequence of weekly risks (hazards) was smoothed using the T4253H smoothing algorithm (Norussis & SPSS, 1993a, p. 123.) Risks were smoothed because we believed that the pattern of risk over time is a smooth continuous function. We included risks through the 3rd week in November because most onsets (174 of 190) had occurred by this time.

To obtain the deviations in risk, we first performed a separate survival analysis for each year's data. The deviation for each week of each year was the risk value obtained for that year and week minus the risk value of the seasonal pattern for that week, as obtained from analysis of the pooled data. Risks were minimal in August (10 of 190 onsets occurred then), so we examined risks beginning with the 1st week in September. For 7 year weeks (the last week for 1988, the last 4 weeks for 1991, and the last 2 weeks for 1994), there was only 1 participant whose episode had not yet begun, and meaningful risk values could not be calculated. As a result, there were data for 77 weeks spread across the 7 years to examine associations between onset risk and environmental variables.

Results

The frequency distribution of SAD onset pooled across years is shown in Table 2. Seventy-four percent of the onsets occurred in the 8 weeks from mid-September through the 1st week in November. In general, the risk of episode onset increased from September to mid-October and then leveled off. Patients often mention the change from daylight saving time to standard time, when it begins getting darker earlier in the day, as contributing to their difficulties. This occurred each year during the week of October 31. However, onset risk had already reached its maximum prior to this time. Of the 5 week years in which risk appeared exceptionally high, 2 occurred in this week, but 3 occurred prior to it. These five points were distributed across the years 1988, 1989, 1991 (two occurred here), and 1994. Thus, the change from daylight saving to standard time was not associated with an elevated risk of onset. The overall seasonal pattern in risk accounted for 29% of the variance in risk (r = .54, N = 77). The remaining 71% of the variance in risk represents the deviations potentially accounted for by deviations in environmental factors.

Next, we examined the correlations between the weekly deviations in risk and those of the three environmental variables, with varying lag times between the environmental and risk variables. As expected, there was an orderly change in the correlations across lags because the data overlapped across the different lags. The peak correlations for sunshine deviations (—.32, p < .005) and solar radiation deviations (—.22, p < .06) occurred with no lag. The peak correlation for temperature deviations (.28, p < .02) occurred with a lag of 5 days. A multiple regression, with these three environmental variables as the independent variables and onset risk deviation as the dependent variable, was statistically significant, but hours of sunshine was the only individual predictor that approached significance (p < .06; see Table 3). From this, we concluded that only hours of sunshine potentially was related to onset risk. Examination of the scatterplot of sunshine and onset risk indicated that this result was due mostly to 1 data point (October 20–26, 1991). When this point was omitted, the correlation dropped from —.32 (p < .005) to —.21 (p < .07). Because omitting 1 of 77 data points should not substantially affect the finding of a true relationship, we considered these results to indicate that none of the three environmental variables were related to onset risk.

Next, we returned to the association of photoperiod and SAD onset risk. Photoperiod correlated —.97 with the overall seasonal pattern in risk. Although photoperiod, as an annually fixed variable, only can generate an annually fixed seasonal pattern in onset risk, we examined how much of the total weekly and yearly variation in actual risk (not deviations) was accounted for by photoperiod. Photoperiod accounted for 26% of the total variance in the weekly risks (r = .51, N = 76, p < .001). The correlations of the three environment variables with the seasonal pattern in risk were all very high (.98 > r > .82), but none of the annually varying environmental variables, or their interaction with photoperiod, accounted for significant additional variance when entered into a multiple regression after photoperiod. 5 The bivariate relationship between risk and temperature lagged back 5 days had also been significant, (r = .28, p < .02) but was due to two outlying points (October 20–26, 1991, and October 27–November 2, 1989). Note also that this relationship was in the opposite direction of that predicted, with exceptionally high risk associated with exceptionally high temperature. 6 The maximum correlation was obtained with no lag in photoperiod.

<table>
<thead>
<tr>
<th>Week beginning</th>
<th>N</th>
<th>%</th>
<th>Onset risk*</th>
</tr>
</thead>
<tbody>
<tr>
<td>August 1</td>
<td>1</td>
<td>0.5</td>
<td></td>
</tr>
<tr>
<td>August 8</td>
<td>1</td>
<td>0.5</td>
<td></td>
</tr>
<tr>
<td>August 15</td>
<td>2</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>August 22</td>
<td>2</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>August 29</td>
<td>7</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>September 5</td>
<td>7</td>
<td>4</td>
<td>.05</td>
</tr>
<tr>
<td>September 12</td>
<td>12</td>
<td>6</td>
<td>.07</td>
</tr>
<tr>
<td>September 19</td>
<td>20</td>
<td>11</td>
<td>.09</td>
</tr>
<tr>
<td>September 26</td>
<td>12</td>
<td>6</td>
<td>.11</td>
</tr>
<tr>
<td>October 3</td>
<td>10</td>
<td>5</td>
<td>.15</td>
</tr>
<tr>
<td>October 10</td>
<td>24</td>
<td>13</td>
<td>.22</td>
</tr>
<tr>
<td>October 17</td>
<td>21</td>
<td>11</td>
<td>.29</td>
</tr>
<tr>
<td>October 24</td>
<td>21</td>
<td>11</td>
<td>.32</td>
</tr>
<tr>
<td>October 31</td>
<td>20</td>
<td>11</td>
<td>.33</td>
</tr>
<tr>
<td>November 7</td>
<td>6</td>
<td>3</td>
<td>.34</td>
</tr>
<tr>
<td>November 14</td>
<td>3</td>
<td>2</td>
<td>.35</td>
</tr>
<tr>
<td>November 21</td>
<td>6</td>
<td>3</td>
<td>.36</td>
</tr>
<tr>
<td>November 28</td>
<td>7</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>December 5</td>
<td>3</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>December 12</td>
<td>1</td>
<td>0.5</td>
<td></td>
</tr>
</tbody>
</table>

Note. Dashes represent onset risks that were computed for those weeks in which there was substantial risk and in which the sample still at risk was large enough to generate stable estimates of risk (i.e., September through mid-November).

* Onset risk is the hazard rate computed from data pooled across all 7 years and then smoothed (see Episode Onset Data section).
Deviations in Environmental Variables

Table 3

<table>
<thead>
<tr>
<th>Variable</th>
<th>B</th>
<th>SE B</th>
<th>( \beta )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sunshine deviations</td>
<td>-0.050</td>
<td>0.026</td>
<td>-0.47*</td>
</tr>
<tr>
<td>Temperature deviations</td>
<td>0.008</td>
<td>0.025</td>
<td>0.18</td>
</tr>
<tr>
<td>Solar radiation deviations</td>
<td>0.026</td>
<td>0.026</td>
<td>0.24</td>
</tr>
</tbody>
</table>

Note. \( R^2 = .15 \), \( p < .01 \). Temperature deviations are for the week laged 5 days prior to the onset risk deviations. Descriptive statistics, \( N = 77 \): onset risk deviations, \( M = 0.3, SD = 0.23 \); sunshine deviations, \( M = -0.12 \) hr, \( SD = 2.13 \); temperature deviations, \( M = -0.32 \) \(^\circ\)F, \( SD = 5.41 \); and solar radiation deviations, \( M = -0.10 \) joules/m\(^2\), \( SD = 2.08 \).

\(* p < .05\)

Discussion

These results failed to find a relationship between risk of SAD onset and hours of sunshine, mean daily temperature, or total daily radiation. Confidence in these findings depends on our design having had sufficient power to detect these effects if they exist. With 76 data points, there was a power (\( p < .05 \), two-tailed) of .76 to find a correlation of .30 (Cohen, 1988). On the other hand, given the limited sample size in each year, it is possible that the measurement error for onset risk was too large for us to have detected relationships. The ranges of our environmental variables covered all values that occurred naturally over a 7-year period. However, the environmental variables may not reflect exactly the stimulus that was actually received by each participant. There is some variability in weather across the Chicago metropolitan area, and we could not control when, or for how long, participants were outdoors (Eastman, 1990; Wehr, Giesen, Moul, Turner, & Schwartz, 1995). There is some evidence that awareness of weather, despite being indoors, may affect the symptomatology of SAD (Bouhuys, Meesters, Jansen, & Bloem, 1994).

If variations in sunshine, temperature, and solar radiation are not causally related to SAD onset, then it would be a mistake to attribute particularly early or late onsets noted by patients to these environmental factors. Similar misattributions have been reported. For example, Hagglund, Deuser, Buckelew, Hewett, and Kay (1994) found that the severity of fibromyalgia symptoms was correlated to beliefs about the effect of weather on symptoms but not to actual weather conditions. Alternatively, it is possible that these attributions are correct in some individual cases. This would be consistent with the finding of idiosyncratic effects of weather on energy level by Albert and colleagues (1991), although these effects were uncommon in the fall and winter. Also, it is possible that environmental conditions affect the severity of symptoms but not their onset. For example, during an episode of depression, a person may feel worse on cloudy days, although cloudiness had nothing to do with the initiation of the episode. It is important to distinguish among vulnerability factors (diatheses), triggers of onset (stresses), and moderators of severity and course for factors in the physical environment, just as is done for factors in the cognitive and social–environment realms.

Conclusions

SAD received its name because investigators believe that it is triggered by environmental factors associated with the seasons. Such an environmental factor should be able to account for variations in the risk of onset that occur across time and space. We found that photoperiod could account for these variations across latitude and months of the year. It is important to note that photoperiod is almost completely confounded with times of sunrise and sunset. Therefore, although investigators have focused on photoperiod, it is possible that SAD onset risk is related to the earlier sunsets or later sunrises that occur in the fall.

If an environmental factor that varies from year to year is causally related to SAD, it should produce predictable differences in onset from year to year. We failed to find evidence of this effect for hours of sunshine, mean temperature, or total daily radiation. Particularly high (or low) environment values were not associated with particularly high (or low) onset risk. Photoperiod (or sunrise or sunset) cannot account for annual differences in onset risk because it does not vary from year to year. Thus, other factors may be responsible for annual variations in onset risk either directly or by moderating the effect of photoperiod. These factors include (a) other annually varying climate variables; (b) annually varying factors within the individual that exhibit annual rhythms (e.g., platelet serotonin uptake; Wirz-Justice & Richter, 1979); and (c) randomly varying factors whose impact may differ by season, such as stressful events.

Previous research in this area has relied on self-reports of typical onset times. This study improved on this method by assessing specific episodes and using procedures designed to increase the accuracy of recall. Additional methodological refinements can contribute to future research on the etiology of SAD. Using prospective weekly assessments can potentially increase the validity of onset measurements. Following the same participants over multiple years would have the benefits of within-subject comparisons of onset differences across years. Conducting replication studies at multiple locations would increase the range of environmental variables and the manner in which they are interrelated. Ultimately, designs will combine, in a single model, environmental factors and variables associated with an individual’s vulnerability to them.

References


ONSET OF SEASONAL AFFECTIVE DISORDER


(Appendix follows)
Appendix

Symptoms Assessed to Determine Episode Onset

Felt depressed, sad, blue, down in the dumps, or empty.
Had less interest in, or got less pleasure from, the things that I usually like.
Was more impatient, easily annoyed, or angered.
Had trouble concentrating or making everyday decisions.
Had less to do with people than usual.
Felt more hungry than usual or gained weight.
Had less physical energy or was more tired than usual.
Had difficulty doing my usual activities: work, household duties, school, etc.
Slept more than usual.
Was slowed down: could not move or speak as quickly as normal.
Felt inadequate, worthless, or like a failure.
Blamed myself or felt guilty for things I did or didn't do.
Worried about my physical health.
Had thoughts about death or killing myself.
Felt anxious, frightened, scared, or apprehensive.
Felt less hungry than usual or lost weight.
Had difficulty with breathing, headaches, sweating, tension, dizziness, chest pain, or trembling.

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AMERICAN PSYCHOLOGICAL ASSOCIATION

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