Seasonal Changes in Mood and Behavior

The Role of Genetic Factors

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Background: Seasonal rhythms in mood and behavior (seasonality) have been reported to occur in the general population. Seasonal affective disorder, a clinically diagnosed syndrome, is believed to represent the morbid extreme of a spectrum of seasonality. Two types of seasonality have been clinically described: one characterized by a winter pattern and a second by a summer pattern of depressive mood disturbance.

Methods: By using methods of univariate and multivariate genetic analysis, we examined the relative contribution of genetic and environmental factors to the risk of seasonality symptoms that were assessed by a mailed questionnaire of 4639 adult twins from a volunteer-based registry in Australia.

Conclusions: There is a tendency for seasonal changes in mood and behavior to run in families, especially seasonality of the winter type, and this is largely due to a biological predisposition. These findings support continuing efforts to understand the role of seasonality in the development of mood disorders.

Results: Seasonality was associated with a winter rather than a summer pattern of mood and behavioral change. In each behavioral domain (ie, mood, energy, social activity, sleep, appetite, and weight), a significant genetic influence on the reporting of seasonal changes was found. Consistent with the hypothesis of a seasonal syndrome, genetic effects were found to exert a global influence across all behavioral changes, accounting for at least 29% of the variance in seasonality in men and women.

Seasonal influence on the incidence of mood disorders has been recognized for centuries, and individual cases of seasonally recurring depressions and manias have been documented since classical times. In recent years, syndromes of recurrent seasonal depression have been more completely described, including recurrent winter depression (which is also known as seasonal affective disorder [SAD]), summer depression, and subsyndromal winter depression. The term "seasonal pattern" was incorporated into DSM-III-R and DSM-IV to describe any forms of recurrent mood disorders that show regular seasonal variations.

Patients with winter-SAD typically report that they oversleep, overeat, and gain weight during their depressive periods, while patients with summer depression are more likely to report that they lose sleep and weight and have a decreased appetite. Both patient groups report that they experience decreased energy, decreased socialization, and sadness during their depressions. There is evidence to suggest that winter-SAD is induced in vulnerable individuals by the decreased ambient light that occurs during the winter, whereas summer-SAD may be induced by high temperatures. These associations have led researchers to treat patients who have winter-SAD with enhanced environmental lighting with some success. Less is known about the therapeutic potential of decreasing environmental temperature or environmental light in patients with summer-SAD.

Clinical researchers who have worked on seasonality independently and in separate geographical regions have documented similar symptoms in patients who were recruited for SAD. Boyce and Parker found evidence of summer and winter forms of SAD in the southern hemisphere that corresponded to the symptom profiles described by clinical researchers who worked in the northern hemisphere.
PATIENTS AND METHODS

SAMPLE

The sample was drawn from an adult twin registry maintained by the Australian National Health and Medical Research Council, Brisbane. The Australian National Health and Medical Research Council Twin Registry is a volunteer twin panel that was first surveyed in 1980-1981.28,30 In spring 1988 (November in Australia), a mailed questionnaire was sent to the 3808 twin pairs who had both responded to the original 1980-1981 mailing, with remailings and telephone follow-up of nonrespondents. Twins who did not return a mailed questionnaire were asked to complete an abbreviated telephone interview. Mailed questionnaire or telephone interview data were obtained from both members of 2997 twin pairs (79% pairwise response rate) and from one twin only from 334 pairs (83% individual response rate). However, since the abbreviated telephone interview did not include the Seasonal Pattern Assessment Questionnaire (SPAQ),11 the final sample for the purposes of this article included 2487 twin pairs in which both members participated and 687 twin pairs in which only one twin responded to the questionnaire items on seasonality (65% pairwise response rate and 74% individual response rate). The zygosity assigned to the twin pairs was that determined by questionnaire in the original 1980-1981 study.28,29

The same questionnaire was remailed in 1990 to a subsample of 500 female and 500 male respondents to the 1988 mailing, yielding 2-year test-retest data from 430 female and 410 male respondents. The mean (±SD) age for the 1988 sample was 41.3±12.7 years, and the age range of the twins was 25 to 89 years.

MEASUREMENTS

The SPAQ is a self-administered, retrospective questionnaire.31 Based on behaviors determined to be abnormal in patients with SAD, this inventory inquires about the pattern and degree of seasonal changes in behavior, and the extent to which such seasonal changes are experienced as a problem by the respondent. This instrument has been widely used in epidemiological studies that were designed to determine the prevalence of SAD and of subclinical levels of seasonality in the general population.5,14,17,32,33 Two sections from the SPAQ were used to answer the questions addressed in this study: six seasonal “change” items and 120 “calendar” items. By using five-point scales (range, 0 to 4), the SPAQ change section asks respondents to rate the degree of seasonal change that they experience across six behaviors: sleep length, social activity, mood, weight, appetite, and energy level. The calendar items ask the respondent to denote the months that are typically affected for each of five of the above-mentioned behaviors (excluding changes in energy level) and the direction of the changes (ie, best or most vs worst or least).

DATA ANALYSIS

Before attempting a genetic analysis, the data set was evaluated for the representativeness of the sampling. The assumption of equal environments between monozygotic (MZ) and dizygotic (DZ) twins was tested; the effects of age24,35 and the effects of twins who were living in dissimilar seasonal environments36 were examined. Important violations of the assumptions of the twin method were not found (data available on request).

Factor Analysis

Traditional factor analyses were performed separately for each sex, ignoring the twin structure of the data, to explore whether a seasonality construct (phenotype) could be observed in these data. More specifically, we examined the following issues: (1) whether sensitivity to changes in season, as measured by responses to the six change items on the SPAQ, could be explained by a single seasonality factor; (2) whether there is evidence for different seasonal patterns (eg, summer or winter), as indicated by relatively separate patterns of seasonal changes in mood and behavior on the 120 calendar items; and if so, (3) whether such patterns show differential associations with the degree of sensitivity to seasonal changes (as measured by the six change items on the SPAQ).

Three matrices of product-moment correlations among the six change and the 120 calendar variables were factor-analyzed37: (1) a 6×6 correlation matrix of the change items, (2) a 120×120 correlation matrix of the calendar items, and (3) a 126×126 correlation matrix of the full set of variables from both categories of data. An oblique rotation (Promax) was used to examine the degree of correlation among factors. In each analysis, the number of factors retained was determined by the number with eigenvalues greater than unity and the consistency of the solution across sexes. When too few factors are extracted, sex differences in the ordering of factors by magnitude may misleadingly result in very different factor solutions for males and females.38

Genetic Item Analysis

Vulnerability to seasonality was evaluated by performing genetic analyses using responses to the six seasonal change questions. The genetic analysis was begun at the level of the individual item.39 Polychoric correlations for each item were estimated by the method of maximum likelihood40 separately for each of the five zygosity groups. Polychoric correlations were also estimated for the test-retest data separately for men and women: these provide estimates of the proportion of variation in each seasonality item that is stable sensitive to limitations in the representativeness of the sample. Subjects for SAD research have been predominantly recruited by referral; hence, their reports are subject to bias owing to information on SAD that is disseminated through public

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Their findings are consistent with the hypothesis that seasonal changes in mood and behavior are related to changes in the physical environment and inconsistent with the argument that temporal patterns in symptoms can be explained as anniversary or adjustment reactions to the holiday season. As with any finding, the clinical description of SAD is
over time (for a comparison, see the study by Kendler et al\(^{29}\)). Estimation of a polychoric correlation implies the assumption that a normal distribution of liability to seasonality underlies the observed categorical response distribution (a plausible assumption for a behavior that may be influenced by multiple genetic and environmental factors) and that the joint distribution of twin pair members for liability to seasonality is bivariate normal.

Univariate genetic analyses of the symptom scores were carried out under a multiple threshold model by the method of maximum likelihood.\(^{29,40}\) Thresholds were allowed to vary as a function of sex, but were constrained to be the same in twins of the same sex from MZ, DZ like-sex, and DZ opposite-sex pairs. A series of standard genetic and environmental models were fitted to the data: models that tested for additive and nonadditive genetic effects and environmental effects (either shared or unshared with other family members) and models that allowed for gender differences in the relative magnitude of genetic and environmental effects. Environmental and additive genetic submodels were compared with the full model by the likelihood-ratio chi squared test.\(^{29,35}\)

**Multivariate Genetic Analyses**

Multivariate genetic factor analyses\(^{35,36}\) were performed to test hypotheses about the number of genetic or environmental factors that were needed to explain the correlations observed between the seasonality symptoms. Multivariate genetic analysis is a generalization of factor analysis that permits estimation of separate genetic and shared and nonshared environmental common and specific factors. Common-factor effects influence two or more symptoms and will give rise to symptom clustering. Specific-factor effects influence only one particular symptom. We tested whether the genetic contribution to vulnerability to seasonal changes reflects a single dimension of genetic variation (or a single “genetic factor”), affecting a broad range of symptoms, or two or more genetic factors, perhaps associated with different symptom constellations.

We also examined whether the genetic factor structure closely paralleled the environmental factor structure (ie, the “common-pathway” model), or whether it was necessary to allow for different genetic vs environmental factor structures (ie, the “independent-pathway” model).\(^{30,41}\) The independent-pathway models attribute the spectrum of seasonality symptoms to one or more underlying liability factors in which environmental and genetic factors are assumed to affect each symptom in qualitatively different ways. Statistically (see diagram of a one-factor model in the Figure, top), separate sets of environmental factor loadings and genetic factor loadings are estimated for the seasonality symptoms. This allows for the possibility that there may be distinct “genetic” and “environmental” clusters of symptoms, with certain symptoms occurring together because of a genetic predisposition, and other symptoms occurring because of environmental effects on behavior (eg, colder temperatures and shorter days leading to changes in sleep patterns, as well as providing fewer opportunities to socialize with friends, neighbors, and relatives).

Compared with the independent-pathway model, a common-pathway model hypothesizes that genetic and environmental effects on symptoms are mediated through one or more intervening biological final common pathways, and it is therefore more supportive of a biological explanation for symptom development. This is illustrated in the one-factor model in the Figure, bottom. The genetic factor loadings for each item are estimated as a scalar multiple (“h”) of the corresponding environmental loadings with the heritability of the latent factor (ie, the proportion of the variance in the factor attributable to genetic effects) estimated as \(h^2/(1 + h^2).\)\(^{35}\) We also examined the effects of estimating a mixed “common-pathway+independent-pathway” model, where genetic and environmental loadings on the first factor only were constrained to follow the common-pathway model. We might expect this mixed model to fit the data if the SPAQ is indeed providing a valid measure of seasonality, but additional genetic or environmental influences, unrelated to seasonality, are also contributing to correlations between the items of the SPAQ. The common-pathway model is a submodel of the independent-pathway model; hence, models of each type that estimated the same number of common factors were directly compared by using the likelihood-ratio chi squared test.

The same genetic and environmental risk factors were assumed to affect the vulnerability to seasonal changes in men and women, but the magnitude of their effects were allowed to vary by gender in all models, except where indicated otherwise. Thus, correlations between additive genetic effects in unlike-sex pairs were fixed to .5, and correlations between shared environmental effects were fixed to unity.\(^{35}\)

The multivariate genetic analyses were performed by using a structural equation–modeling package (LISREL).\(^{35,36,42}\) The raw data were summarized by polychoric correlations that were estimated by using a commercially available software package (PRELIS).\(^{39}\) One- and two-factor models were fitted by asymptotic weighted least squares.\(^{39,43}\) Beginning with simple one-factor environmental and genetic factor models, we systematically added genetic or environmental factors in an effort to select the simplest model, consistent with the observed data, to explain the determinants of seasonality. All models also allowed for environmental and additive and nonadditive genetic influences that were symptom-specific. Models that gave a poor fit to the data \((P<.05)\) were rejected by the chi squared goodness-of-fit test. Models that gave an adequate fit to the data by the goodness-of-fit test were compared by using the likelihood-ratio chi squared test.

and professional channels, to traits associated with clinical research volunteerism (eg, severity of illness\(^{29}\) or help-seeking behavior), and to criteria imposed by the researchers. Consequently, diagnostic criteria for seasonal mood disorders, based on such clinical descriptions, may be too broad or too narrow. Patients who are defined as suffering from SAD may actually constitute a heterogeneous group with different conditions;\(^{29}\) or alternatively, the two types of SAD that have already been defined may merely represent a subset of a variety of pathological seasonal disorders that are present in the general population. This issue can be addressed by turning from clinical to community samples, which have not been selected for problem behavior or illness.
Seasonality has been reported as a problem by a substantial proportion of the general population. When surveyed by mailed questionnaires, 25% of a randomly selected sample in New York City\(^6\) and 27% of a Maryland sample\(^7\) complained that seasonally experienced changes were a problem. Some 5% of the Maryland respondents\(^8\) reported a degree of change and impairment that resembled the questionnaire response of patients who were clinically diagnosed as having SAD. Consistent with findings in clinical research, many fewer individuals report a summer vs a winter pattern of seasonal changes by mailed questionnaire. Only about 10% to 12% of the adults who participated in these surveys\(^6,37\) reported that they felt worst during the summer compared with 43% to 50% of respondents who reported a winter pattern of behavioral changes.

This project was designed to evaluate the validity of the seasonality construct through the analysis of data from a volunteer sample of twin pairs who were not selected for problem behavior or illness. Our present approach differs from those of previous survey studies in that we formulate a description for symptoms of seasonality based on patterns of family resemblance that were observed in the data. If there are genetic influences on seasonality, we would expect to observe different degrees of similarity between identical vs fraternal twin pairs; hence, we have been able to differentiate combinations of symptoms that may share a biological (or environmental) predisposition without replicating the bias of self-selection that has confounded many laboratory and treatment studies on seasonality.

The literature suggests that the relatives of patients with SAD may have a higher prevalence of mood disorders in general and SAD in particular than the general population\(^4,14\). To date, however, no formal family studies of SAD have been conducted. It would be useful to know whether SAD as a disorder and seasonality as a trait are familialy transmitted, and whether any such familial transmission has a biological basis. In the present study, we have set out to address two key hypotheses: (1) genetic vulnerability to seasonality is manifested by a seasonal change in a broad spectrum of behaviors that include mood, sleep pattern, social activity, weight, appetite, and energy; and (2) there exist genetic differences in vulnerability to seasonality that are manifested by summer and winter patterns of disturbance.

### RESULTS

The twins in this survey were located throughout the Australian continent: about 40% of the sample were from the state of Victoria (the region most similar in seasonal change in sunlight exposure to Montgomery County, Maryland, where research on SAD has been pioneered\(^4,30\)); 30% were from New South Wales; 10% were from Queensland; 9% were from South Australia; 5% were from Tasmania; 5% were from Western Australia; 3% were from Australian Capital Territory; and 2% were from the Northern Territory or overseas. Thirteen percent of our sample complained that seasonality was a personal problem, 17% reported that they felt worst during the winter, and 8% reported that they experienced a summer pattern of worsening in mood. Only 2% of our sample reported a degree of seasonality that resembled the response of patients who were clinically diagnosed as having SAD.\(^6\) Of individual twins who reported behavioral changes during the winter, 28% to 33% reported increases in sleep, food intake, and weight (at most, 5% of winter-affected individuals reported decreases in these behaviors); by contrast, 26% to 31% reported decreases in weight, food intake, and sleep during the summer (9% to 12% reported increases in these behaviors).

### FACTOR STRUCTURE

When a conventional factor analysis was performed, a one-factor solution was found to describe adequately the pattern of covariation of the six seasonal change items. All six items (sleep length, social activity, mood, weight, appetite, and energy level change) exhibited high loadings (in the range of .55 to .78), and a similar factor pattern was defined for both sexes. The analysis was consistent with the assumption of a single global seasonality factor.

Factor analysis of the 120 calendar items identified four oblique seasonal factors (two winter and two summer factors) that replicated across sexes. One factor had high loadings on items that specified a worsening of mood and a decrease in social activity during the winter; a second winter factor had high loadings on items that indicated an increase in weight, appetite, and sleep. The interfactor correlation between these two winter factors was .4 in both sexes. A third factor had high loadings on items that indicated a decrease in mood and social activity dur-
In the joint-factor analysis of the six seasonal change items and 120 calendar items, in women, the change items had positive loadings only on the winter factors, with no item loading higher than .04 on either summer factor. A similar trend was observed in the men (highest-loading item, .25). Sensitivity to changes in season, as measured by the six change items, seemed to be associated with winter rather than summer patterns of mood and behavioral changes, especially in women.

GENETIC ANALYSIS: INDIVIDUAL SEASONALITY ITEMS

Table 1 summarizes twin correlations for each zygosity group, separately for each seasonal change item. Two-year test-retest correlations are shown in the last two rows of Table 1. The MZ correlations are about one half of the reliability estimates, suggesting that about one half of the longitudinally stable variance is owing to factors responsible for family resemblance (ie, either genetic or environmental influences that are shared between a twin and co-twin). For most items, the DZ correlation was no more than one half the MZ correlation, implying an influence of genetic effects, especially for women. An analysis of variance was performed to test the effect of season of return for the mailed questionnaire on the report of seasonal changes in behavior (summarized by the sum of the respondent’s answers to the seasonal change items). No significant differences in the total score as a function of the season when the questionnaire was completed were found (men: \( F=0.21, df=3, 1979 \), and \( P=.89 \); women: \( F=1.17, df=3, 3673 \), and \( P=.31 \)).

Table 2 summarizes the results of fitting genetic models to the data for each seasonal change item. The left-hand column on Table 2 shows, for each item, the \( \chi^2 \) test of goodness of fit for the full model (ACE), allowing for both genetic and shared and unshared environmental effects. For two items, namely, sleep length and appetite, the goodness of fit of the best-fitting model was somewhat marginal (\( P=.02 \) and \( P=.04 \), respectively), and for the mood change item, the goodness of fit was poor (\( P=.002 \)). Relaxing the constraint that threshold values be the same in MZ and DZ pairs led to an adequate fit for the latter item (\( \chi^2=54.46, df=40, P=.06 \)), but not for the former two items (sleep length: \( \chi^2=57.56, df=40, P=.04 \); appetite: \( \chi^2=36.96, df=40, P=.04 \)). Allowing threshold values to differ between zygosity groups produced a negligible change in the estimate of the twin polygenic correlations and, hence, did not have any appreciable effect in the model-fitting analyses.

The next two columns give the likelihood-ratio \( \chi^2 \) tests of submodels that assume either no shared environmental effects (AE) or no genetic effects (CE). The three right-hand-most columns of Table 2 give the parameter estimates under the best-fitting model. Parameter estimates have not been corrected for measurement error. There was no evidence of heterogeneity of genetic parameters across sex (ie, no genotype×sex interaction) for any symptom, except appetite, where models allowing for sex dependence of parameters are reported. In all cases, the environmental model gave a worse fit than the full model. This indicated a genetic influence on the vulnerability to seasonal changes. For the mood item only, the data were best explained by a model that incorporated nonadditive genetic as well as additive genetic effects, and the maximum likelihood estimate of the additive genetic variance was negative. (In twin studies, estimates of the additive and nonadditive genetic variances are strongly negatively correlated, making it difficult to obtain accurate simultaneous estimates of additive and nonadditive genetic components.) Estimates of the broad heritability of the seasonal change items (ie, the proportion of the variance in liability accounted for by additive genetic plus nonadditive genetic factors) were in the range of 20% to 35%, uncorrected for reliability of measurement. This range increases to 39% to 73% if we take into account test-retest reliability and consider instead the proportion of the temporally stable variation explained by genetic factors.

**Table 1. Sample Sizes and Twin Pair and Test-Retest Polychoric Correlations for Seasonality Items**

<table>
<thead>
<tr>
<th>Group</th>
<th>No.†</th>
<th>Sleep Length</th>
<th>Social Activity</th>
<th>Mood</th>
<th>Weight</th>
<th>Appetite</th>
<th>Energy</th>
</tr>
</thead>
<tbody>
<tr>
<td>MZ females</td>
<td>854</td>
<td>.23</td>
<td>.29</td>
<td>.34</td>
<td>.34</td>
<td>.35</td>
<td>.31</td>
</tr>
<tr>
<td>DZ females</td>
<td>492</td>
<td>.15</td>
<td>.13</td>
<td>.04</td>
<td>.10</td>
<td>.21</td>
<td>.15</td>
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<tr>
<td>MZ males</td>
<td>390</td>
<td>.18</td>
<td>.26</td>
<td>.38</td>
<td>.31</td>
<td>.23</td>
<td>.26</td>
</tr>
<tr>
<td>DZ males</td>
<td>215</td>
<td>.15</td>
<td>.27</td>
<td>.14</td>
<td>.10</td>
<td>.12</td>
<td>.06</td>
</tr>
<tr>
<td>DZ opposite-sex</td>
<td>536</td>
<td>.09</td>
<td>.07</td>
<td>.10</td>
<td>.10</td>
<td>.08</td>
<td>.16</td>
</tr>
<tr>
<td>Retest sample</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Females</td>
<td>430</td>
<td>.45</td>
<td>.53</td>
<td>.48</td>
<td>.60</td>
<td>.51</td>
<td>.57</td>
</tr>
<tr>
<td>Males</td>
<td>410</td>
<td>.53</td>
<td>.52</td>
<td>.58</td>
<td>.61</td>
<td>.61</td>
<td>.54</td>
</tr>
</tbody>
</table>

* MZ indicates monozygotic; DZ, dizygotic
† Number of pairs for twin groups, but number of individuals for retest sample.
Table 2. Univariate Genetic Analyses of Seasonality Items

<table>
<thead>
<tr>
<th>‘Change’ Item</th>
<th>Goodness-of-Fit Tests Against Full Model</th>
<th>Parameter Estimates Under Best-Fitting Model*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Full Model (ACE), χ² (df=27)</td>
<td>Genetic, (AE), χ² (df=1)</td>
</tr>
<tr>
<td>Sleep length</td>
<td>92.00</td>
<td>1.50</td>
</tr>
<tr>
<td>Social activity</td>
<td>85.95</td>
<td>2.35</td>
</tr>
<tr>
<td>Mood§</td>
<td>105.89</td>
<td>1.55</td>
</tr>
<tr>
<td>Weight</td>
<td>74.92</td>
<td>3.73</td>
</tr>
<tr>
<td>Appetite¶</td>
<td>87.06</td>
<td>1.83</td>
</tr>
<tr>
<td>Energy level</td>
<td>77.79</td>
<td>1.61</td>
</tr>
</tbody>
</table>

*Parameter estimates are uncorrected for reliability of measurement.  
†P<.05.  
‡P<.001  
§For “Mood,” the full model estimated additive genetic plus dominance genetic effects (ADE).  
¶For “Appetite,” models reported allow for sex-dependent genetic or environmental effects (A_m, C_m, E_m, and a correlation for between shared environmental effects in males and females, r_c). Degrees of freedom for these models were 3 (environmental, genetic models) and 64 (full model).  
#For males.  
**For females.

MULTIVARIATE GENETIC ANALYSIS

Results of fitting multivariate genetic analyses are presented in Table 3. Each model tested a different hypothesis about common-factor effects, that is, about the number of genetic or environmental common factors that contribute to the correlations between the six seasonal change measures of seasonality in the SPAQ. Two hypotheses were rejected by an overall χ² test of goodness of fit (P<.05, results not shown): (1) the hypothesis that the SPAQ measures only a single dimension of liability to seasonality (ie, all models that estimated only one common factor [genetic or environmental] were rejected); and (2) the hypothesis that sensitivity to changes in season can be explained entirely by environmental (ie, shared and nonshared environmental) influences.

Of all the independent-pathway models, models with two nonshared environmental common factors, one shared environmental common factor, and either two additive genetic (model 5) or one additive genetic and one nonadditive genetic factor (model 6) were the simplest models consistent with the observed data.

The common-pathway models 10 and 11 gave a significantly worse fit than the more general independent-pathway models by the likelihood-ratio test (models 11 vs 6, P<.004; models 10 vs 5, P=.04). The simplest model to explain the observed data was a mixed model (model 13), with a common-pathway structure specified for the first factor and an independent-pathway structure specified for the second genetic and environmental factors. Under this model, additive genetic estimates were constrained to be the same across sex for the common-pathway factor. For the independent-pathway factors, the magnitude of the genetic estimates were allowed to dif-
The spectrum consists of subsyndromal-SAD: individuals who express asymptomatic seasonal behavioral changes and those who report no seasonal changes whatsoever.

Our results suggest that seasonality is inheritable, with at least 29% of the variance in risk of seasonality due to genetic influences in men and women. Supportive of the hypothesis of a seasonal syndrome, we found genetic effects to exert a global influence across seasonal changes in eating, sleeping, weight change, socializing, energy level, and mood. Interestingly, genetic and environmental determinants of seasonality appeared to be mediated through a single biological pathway. Consequently, one would predict individuals who present with seasonality to include complaints on changes in mood, sleep length, social activity, weight, appetite, and energy level with a high degree of consistency across cases. These findings are consistent with those of Young et al,18 that atypical symptoms of depression (ie, fatigue, hypersomnia, and increased weight and appetite) in patients with winter-SAD demonstrate a pattern of onset and course that is substantially different from patterns observed for other complaints that are commonly reported by these patients (ie, decreases in self-esteem, mood, interest, ability to concentrate, and problems with social withdrawal).

Consistent with problems that are commonly reported by individuals with winter-SAD, genetic predisposition to seasonality (as measured by the SPAQ) appears to be manifested by complaints associated with the atypical vegetative symptoms of depression (ie, increases in sleep, eating, and weight gain) and decreases in socialization, energy, and mood. This finding is compatible with the results from treatment outcome studies, which also have found that complaints of hypersomnia, overeating, and weight gain are predictors of a good response to phototherapy in patients who are diagnosed as having winter-SAD.32,53

Consistent with the results of Rosen and Rosenthal,34 who used data collected in the northern hemisphere, we found patterns of response in our data to support the presence of summer and winter patterns of behavioral changes. However, our results indicate that genetic susceptibility for sensitivity to changes in season is only associated with winter forms of mood and behavioral changes. If a genetic predisposition to a summer form of seasonality exists, the prevalence of this condition must be too low in the general population of Australia to be detectable by using the sample sizes of the present study.

In interpreting our findings, several important limi-

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Table 4. Factor Loadings and Heritability Estimates Under Best-Fitting Multivariate Genetic Model

| 'Change' Item | Females | | Males | | |
|---------------|--------|--------|--------|--------|
| | Common-Pathway Factor | Independent-Pathway Factor | Common-Pathway Factor | Independent-Pathway Factor |
| | Additive Genetic | Nonshared Environment | Additive Genetic | Nonshared Environment | Additive Genetic | Nonshared Environment | Additive Genetic | Nonshared Environment |
| Sleep length | .30 | .47 | .11 | .28 | .10 | .29 | .46 | -.09 | .28 | .10 |
| Social activity | .32 | .50 | .17 | .23 | .00 | .31 | .48 | -.40 | .23 | .00 |
| Mood | .44 | .70 | .30 | .04 | -.09 | .45 | .71 | -.14 | .04 | -.10 |
| Weight | .24 | .38 | .36 | .16 | .46 | .26 | .41 | -.07 | .16 | .46 |
| Appetite | .25 | .40 | .50 | .24 | .66 | .32 | .50 | .01 | .24 | .66 |
| Energy level | .40 | .63 | .23 | .13 | .18 | .43 | .68 | .00 | .13 | .18 |

*Table 4 does not include specific-factor loadings; hence, percentage of phenotypic variance that is explained by these common-factor effects will be less than 100%. The percentage of explained phenotypic variance was as follows: for females—common-pathway factor, 39%; independent-pathway factor, 9%; (additive genetic), 4% (shared environment), and 12% (nonshared environment); for males—common-pathway factor, 43%; independent-pathway factor, 3% (additive genetic), 4% (shared environment), and 12% (nonshared environment).
tations should be borne in mind. First, assessments of seasonality in the present study were carried out by a retrospective self-report questionnaire, and our findings were limited by the ability of the SPAQ to discriminate those individuals who were affected with seasonality from the unaffected persons in the general population. Our demonstration of significant familial aggregation of seasonality, and the fact that this familial aggregation is genetically determined, is another step in the ongoing evaluation of the validity and specificity of this questionnaire.

Second, our ability to detect a contribution to the risk of seasonality of environmental factor(s) shared by family members (eg, seasonal changes in the surrounding environment) may have been limited by characteristics of our sample. There is an overrepresentation of twins from southeastern Australia (40% of twins live in the state of Victoria). Assuming that latitude is a reasonable indicator of exposure to seasonal changes, an evaluation of the significance of the shared environmental contribution to the risk of seasonality ideally would require a more balanced sample in the distribution by geographical location. Sample sizes significantly larger than those used to test the hypotheses of this project are required to detect modest shared environmental influences against a background of genetic effects when MZ twin correlations are as low as those observed in the present study.44

Last, the low prevalence of clinical levels of seasonality (only 2%) limits the interpretation of our results to seasonality within the subclinical or normal range, and it precludes statements about genetic influences on SAD per se.

To conclude, there is a tendency for seasonality to run in families, and this is largely owing to a biological predisposition. These findings support continuing efforts to determine the role of seasonality in the development of affective disorder. Two questions remain: (1) Do the same genetic and environmental risk factors that determine the risk of seasonality, as we have found here, also predispose to clinically significant seasonal changes in mood? (2) Is the tendency to experience seasonal changes in mood and behavior transmitted along with a vulnerability to a mood disorder, or are these separate heritable factors?

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Reprint requests to Department of Psychiatry, Washington University School of Medicine, Box 8134, 4940 Children’s PI, St Louis, MO 63110 (Dr Madden).

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