Season of Birth: Schizophrenia and Bipolar Disorder

by Jeffrey H. Boyd, Ann E. Pulver, and Walter Stewart

Abstract

Studies investigating the association between the risk of schizophrenia and season of birth are reviewed and the association clearly established. This association cannot be explained on the basis of age-incidence or age-prevalence artifacts. Other studies suggest there may be an association between bipolar disorder and season of birth. The leading theory in explaining the season of birth phenomenon is that a seasonal factor (such as viral infection, malnutrition, vitamin deficiency, prenatal or obstetrical complications, or ambient temperature) can damage an infant's brain and thereby predispose the child to later development of psychosis. Evidence suggests that the seasonal effect is associated with a subgroup of schizophrenics who have early onset of psychosis, less genetic loading than other schizophrenics, and better prognosis. Case-control studies are needed comparing winter-born to nonwinter-born schizophrenics.

Since 1929, it has been reported that more schizophrenics are born during the winter and early spring than during other seasons (Tramer 1929). Although the basis for this finding is unknown, it has been suggested that it may be a clue to the identification of a subtype of schizophrenia (Dalen 1975). Some studies also indicate that this is true for patients with bipolar affective disorder (mania) (Hare, Price, and Slater 1974; Dalen 1975; Hare 1975a).

The purpose of this paper is fivefold: (1) to review the literature on the relationship between season of birth and schizophrenia; (2) to discuss whether this association is valid or due to artifact; (3) to review the relationship between season of birth and bipolar affective disorder; (4) to review the evidence that season of birth distinguishes a subtype of schizophrenia; and (5) to review the theories offered to explain the basis for the association and to suggest ways to test them.

Investigations of the Relationship Between the Risk for Schizophrenia and Season of Birth

Table 1 provides a summary of reports in English of investigations of the seasonal distribution of births of schizophrenic patients. Season of birth studies have been done in 14 different countries. Despite the variety of study methods and possible differences in diagnostic criteria, strikingly consistent results have been found.

The studies with a large n (over 1,000) are based on case registries of a State or nation, with the exception of Koechler and Jacoby's study (1976). Case registry data may be considered a representative sample of hospitalized schizophrenic patients.

In all but two of the studies (Jones and Frei 1979; Pulver, Sawyer, and Childs 1981), the seasonal distribution of births of schizophrenics has been compared with that of births in the general population; a goodness of fit test is then applied. Pulver, Sawyer, and Childs (1981) calculated the probability for being diagnosed as schizophrenic using the census population for the denominators of the rates. Jones and Frei (1979) used matched controls.

Several sources of error should be considered in evaluating these...
Table 1. Schizophrenia season of birth

<table>
<thead>
<tr>
<th>Place, time of study</th>
<th>Sample size</th>
<th>Adequate controls</th>
<th>Adjustment for age-incidence effect?</th>
<th>Months of excess</th>
<th>Percent excess</th>
<th>Statistical significance</th>
<th>Comment</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>USA 1917-1980</td>
<td>536 twins</td>
<td>no</td>
<td>no</td>
<td>Jan-Mar</td>
<td>4</td>
<td>N.S.</td>
<td></td>
<td>Kendler 1982</td>
</tr>
<tr>
<td>Minnesota, USA 1963-1978</td>
<td>3,556</td>
<td>yes</td>
<td>yes</td>
<td>Dec-Mar</td>
<td>7</td>
<td>&lt;.001</td>
<td></td>
<td>Watson et al. 1982</td>
</tr>
<tr>
<td>Philippines 1975</td>
<td>3,508</td>
<td>no</td>
<td>no</td>
<td>Dec-Feb</td>
<td>15</td>
<td>&lt;.01</td>
<td></td>
<td>Parker &amp; Balza 1977</td>
</tr>
<tr>
<td>Ireland 1972-1975</td>
<td>4,855</td>
<td>yes</td>
<td>no</td>
<td>Apr-June</td>
<td>11</td>
<td>&lt;.001</td>
<td></td>
<td>O'Hare, Walsh, &amp; Torrey 1980</td>
</tr>
<tr>
<td>Missouri, USA 1966-1975</td>
<td>10,363</td>
<td>yes</td>
<td>yes</td>
<td>Dec, Feb</td>
<td>9</td>
<td>&lt;.01</td>
<td></td>
<td>Lewis &amp; Griffin 1981</td>
</tr>
<tr>
<td>Australia 1974</td>
<td>467</td>
<td>no</td>
<td>no</td>
<td>Winter¹</td>
<td>—</td>
<td>&lt;.01</td>
<td>Males</td>
<td>Jones &amp; Frei 1979</td>
</tr>
<tr>
<td>Australia 1974</td>
<td>448</td>
<td>no</td>
<td>no</td>
<td>Winter¹</td>
<td>—</td>
<td>N.S.</td>
<td>Females</td>
<td>Jones &amp; Frei 1979</td>
</tr>
<tr>
<td>Australia 1970-1974</td>
<td>1,061</td>
<td>no</td>
<td>no</td>
<td>Winter¹</td>
<td>—</td>
<td>N.S.</td>
<td>Males</td>
<td>Parker &amp; Nielson 1976</td>
</tr>
<tr>
<td>Australia 1970-1974</td>
<td>1,195</td>
<td>no</td>
<td>no</td>
<td>Winter¹</td>
<td>12</td>
<td>&lt;.01</td>
<td>Females</td>
<td>Parker &amp; Nielson 1976</td>
</tr>
<tr>
<td>USA 1973-1974</td>
<td>53,584</td>
<td>yes</td>
<td>no</td>
<td>Jan, Mar, Apr, May</td>
<td>—</td>
<td>&lt;.05</td>
<td></td>
<td>Torrey, Torrey, &amp; Peterson 1977</td>
</tr>
<tr>
<td>Denmark 1972-1974</td>
<td>207</td>
<td>no</td>
<td>no</td>
<td>Jan-Mar</td>
<td>15</td>
<td>&lt;.03 Urban, high genetic risk</td>
<td></td>
<td>Machon, Mednick, &amp; Schulsinger 1983</td>
</tr>
<tr>
<td>Japan 1987-1973</td>
<td>5,431</td>
<td>yes</td>
<td>no</td>
<td>Apr</td>
<td>12</td>
<td>&lt;.05 Born &gt; 1900</td>
<td></td>
<td>Shimura &amp; Miura 1980</td>
</tr>
<tr>
<td>Japan 1987-1973</td>
<td>2,529</td>
<td>yes</td>
<td>no</td>
<td>May</td>
<td>20</td>
<td>&lt;.01 Born &lt; 1900</td>
<td></td>
<td>Shimura &amp; Miura 1980</td>
</tr>
<tr>
<td>England 1970-1973</td>
<td>9,760</td>
<td>yes</td>
<td>yes</td>
<td>Jan-Mar</td>
<td>7</td>
<td>&lt;.001</td>
<td></td>
<td>Hare 1975; Hare 1975b</td>
</tr>
<tr>
<td>West Germany 1962-1972</td>
<td>1,576</td>
<td>yes</td>
<td>no</td>
<td>—</td>
<td>—</td>
<td>N.S.</td>
<td></td>
<td>Koehler &amp; Jacoby 1976</td>
</tr>
<tr>
<td>Region</td>
<td>Total</td>
<td>Case Control</td>
<td>Season</td>
<td>Month Range</td>
<td>Sample Size</td>
<td>Significance</td>
<td>Authors and Year(s)</td>
<td></td>
</tr>
<tr>
<td>-------------------------</td>
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<td>---------------------</td>
<td></td>
</tr>
<tr>
<td>England</td>
<td>5,139</td>
<td>yes</td>
<td>no</td>
<td>Jan-Mar</td>
<td>7</td>
<td>&lt;.01</td>
<td>Hare, Price, &amp; Slater 1974</td>
<td></td>
</tr>
<tr>
<td>South Africa 1971</td>
<td>1,713</td>
<td>yes</td>
<td>no</td>
<td>Winter³</td>
<td>6</td>
<td>&lt;.05</td>
<td>Not chronic</td>
<td></td>
</tr>
<tr>
<td>South Africa 1971</td>
<td>1,234</td>
<td>yes</td>
<td>no</td>
<td>Winter³</td>
<td>—</td>
<td>N.S.</td>
<td>Chronic</td>
<td></td>
</tr>
<tr>
<td>Australia 1961-1971</td>
<td>6,291</td>
<td>no</td>
<td>no</td>
<td>—</td>
<td>—</td>
<td>N.S.</td>
<td>Females</td>
<td></td>
</tr>
<tr>
<td>Norway 1970</td>
<td>19,740</td>
<td>yes</td>
<td>no</td>
<td>Jan-Mar</td>
<td>20</td>
<td>&lt;.001</td>
<td>Ødegård 1974</td>
<td></td>
</tr>
<tr>
<td>Maryland, USA 1963-1968</td>
<td>1,455</td>
<td>yes</td>
<td>yes</td>
<td>Jan-Apr</td>
<td>8</td>
<td>&lt;.01</td>
<td>Pulver, et al. 1983</td>
<td></td>
</tr>
<tr>
<td>Sweden 1962-1968</td>
<td>19,820</td>
<td>yes</td>
<td>no</td>
<td>Jan-Apr</td>
<td>0-10</td>
<td>&lt;.01</td>
<td>Dalen 1975</td>
<td></td>
</tr>
<tr>
<td>Missouri, USA 1967</td>
<td>22</td>
<td>no</td>
<td>no</td>
<td>—</td>
<td>—</td>
<td>N.S.</td>
<td>Woodruff, Guze, &amp; Clayton 1974</td>
<td></td>
</tr>
<tr>
<td>Maryland, USA 1944-1961</td>
<td>94</td>
<td>no</td>
<td>no</td>
<td>Jan-Mar</td>
<td>35</td>
<td>&lt;.05</td>
<td>Blacks only</td>
<td></td>
</tr>
<tr>
<td>Canada 1959</td>
<td>3,617</td>
<td>yes</td>
<td>no</td>
<td>Jan, Apr, May</td>
<td>—</td>
<td>&lt;.05</td>
<td>Norris &amp; Chowning 1962</td>
<td></td>
</tr>
<tr>
<td>Sweden 1949</td>
<td>2,232</td>
<td>no</td>
<td>no</td>
<td>—</td>
<td>—</td>
<td>N.S.</td>
<td>Dalen 1975</td>
<td></td>
</tr>
<tr>
<td>Virginia, USA 1949</td>
<td>1,469</td>
<td>no</td>
<td>no</td>
<td>—</td>
<td>—</td>
<td>N.S.</td>
<td>(Laestadius)</td>
<td></td>
</tr>
<tr>
<td>Massachusetts USA, 1933-1945</td>
<td>1,453</td>
<td>yes</td>
<td>no</td>
<td>Jan-Apr</td>
<td>—</td>
<td>&lt;.01</td>
<td>Barry &amp; Barry 1961</td>
<td></td>
</tr>
<tr>
<td>USA 1938</td>
<td>10,000</td>
<td>no</td>
<td>no</td>
<td>Jan-Mar</td>
<td>—</td>
<td>—</td>
<td>Graph only</td>
<td></td>
</tr>
</tbody>
</table>

**Significance Levels:**
- <.01: Significant
- <.05: Marginally significant
- N.S.: Not significant
- o: Other
- z: Zero

**Other Notes:**
- 'Jan-Mar': January to March
- 'Jan-Apr': January to April
- 'Winter': Winter
- 'Jan, Apr, May': January, April, May
- 'High genetic risk': High genetic risk
- 'Low genetic risk': Low genetic risk
- 'Blacks only': Blacks only
- 'Graph only': Graph only
- 'Hare, Price, & Slater 1974': Hare, Price, & Slater 1974
- 'Dalen 1975': Dalen 1975
- 'Krupinski, Stoller, & King 1976': Krupinski, Stoller, & King 1976
- 'Ødegård 1974': Ødegård 1974
- 'Shur 1980': Shur 1980
- 'Dalen 1975': Dalen 1975
- 'Hare & Price 1968': Hare & Price 1968
- 'Gallagher, McFalls, & Jones 1983': Gallagher, McFalls, & Jones 1983
- 'Norris & Chowning 1962': Norris & Chowning 1962
- 'Pile 1951': Pile 1951
- 'Dalen 1975 (Laestadius)': Dalen 1975 (Laestadius)
- 'Dalen 1975 (Huntington)': Dalen 1975 (Huntington)
Since the seasonal distribution of births in the general population may vary from year to year and from region to region, it is necessary to choose controls whose place and time of birth match those of the cases. Furthermore, since infants who do not survive cannot have schizophrenia, the seasonal effects of infant mortality on the distribution of live births must be considered.

To increase the power of the analysis, many investigators grouped the dates of birth into quarters of the year (e.g., January through March, April through June). This could mask a true seasonal pattern; more schizophrenics than expected are likely to be born in December, but if October and November lack an excess, the fourth quarter may fail to show any excess.

The season of excess risk is shown in the fifth column of table 1. Studies in both the northern and southern hemispheres show an excess risk for the winter months. This is especially significant in terms of statistical artifacts, such as the age-incidence and age-prevalence effects, which result from the use of calendar time and not age of risk. In studies with a sample size over 1,500, the excess ranges from 0 to 20 percent, with a mean of about 10 percent.

Of the 30 studies, 19 showed differences that were significant at a level of .05. Six of these studies are significant at a level of .001. The only two studies that had a control group matched to patients on year of birth and geographical region of birth, and that also controlled for the age-incidence effect, found significant results at the .01 level (Hare 1975a, 1975b; Lewis and Griffin 1981).

In summary, table 1 shows that a majority of studies have found an association between the risk for schizophrenia and birth in the winter

<table>
<thead>
<tr>
<th>Place, time of study</th>
<th>Sample size</th>
<th>Adequate controls</th>
<th>Adjustment for age-incidence effect?</th>
<th>Percent excess</th>
<th>Months of excess</th>
<th>Statistical significance</th>
<th>Comment</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Holland 1934</td>
<td>2,589</td>
<td>no</td>
<td>no</td>
<td>no</td>
<td>Nov-Mar</td>
<td>&lt; .001</td>
<td>Graph</td>
<td>Dalen 1975 (Lang)</td>
</tr>
<tr>
<td>Illinois USA 1934</td>
<td>3,467</td>
<td>no</td>
<td>no</td>
<td>no</td>
<td>Dec-Jan</td>
<td>N.S.</td>
<td>Dalen 1975 (Nolling)</td>
<td></td>
</tr>
<tr>
<td>Germany 1931</td>
<td>3,976</td>
<td>no</td>
<td>no</td>
<td>no</td>
<td>Dec-Jan</td>
<td>N.S.</td>
<td>Graph</td>
<td>Dalen 1975 (Petersen)</td>
</tr>
<tr>
<td>Connecticut USA 1907-1931</td>
<td>6,751</td>
<td>no</td>
<td>no</td>
<td>no</td>
<td>Dec-Mar</td>
<td>&lt; .001</td>
<td>Upper class patients</td>
<td>Barry &amp; Barry 1964</td>
</tr>
<tr>
<td>Switzerland 1916-1927</td>
<td>3,100</td>
<td>no</td>
<td>no</td>
<td>no</td>
<td>Dec-Mar</td>
<td>15</td>
<td>Psychosis Dalen 1975 (Tanner)</td>
<td></td>
</tr>
</tbody>
</table>
months. This is true in populations in both the northern and southern hemispheres. Although this finding has not been universal (Pile 1951; Barry and Barry 1964; Dalen 1975; Koehler and Jacoby 1976; Krupinski, Stoller, and King 1976; Jones and Frei 1976; Kendler 1982), there is a remarkable degree of consensus given the wide variety of study settings and methods.

This difference in the cumulative risk for relatively small differences in the age period of risk is the source of the age-incidence and age-prevalence effects.

The problem arises because calendar time, not age, is the basis for deriving cumulative measures of risk. For those people born in a given year (Y1) and first hospitalized in another year (Y2), those born in January of year Y1 will be 11 months older by the end of year Y2 than those born in December of year Y1. This age difference, depending on month of birth, is the basis of both the age-incidence effect (Dalen 1975; Hare 1978; Pulver, Sawyer, and Childs 1981) and the age-prevalence effect (Lewis and Griffin 1981). Hypothetical age-incidence and age-prevalence curves are shown in figure 1.

Validity of the Association

Several investigators have suggested that the apparent association may be due to artifact (Hare, Price, and Slater 1974; Dalen 1975; Lewis and Griffin 1981). In more recent studies, however, artifacts have been disposed of by appropriate statistical techniques; the resulting pattern is often more significant (Hare, Price, and Slater 1974; Hare 1978; Pulver, Sawyer, and Childs 1981; Watson et al. 1982; Pulver et al. 1983; Shur and Hare 1983).

The most recent challenge is that of Lewis and Griffin (1981). They propose that the increased risk is due to a statistical artifact that they call the "age-prevalence effect," a measure that is the sum (or integral) of risk a defined population will experience over a given period of time. The older a person is, the greater is the cumulative risk of disease. The cumulative risk of schizophrenia is relatively high for the age period 17 to 24—so high, in fact, that there are differences in the cumulative risk for small differences in age. For example, a cohort of 20-year-olds born in January will have a significantly higher cumulative risk of schizophrenia than a cohort of 20-year-olds born in June when the cumulative risk is integrated to the same calendar time for both cohorts.

Figure 1. Age-incidence curve and cumulative risk curve

<table>
<thead>
<tr>
<th>Incidence per 100,000 per year</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
</tr>
<tr>
<td>0</td>
</tr>
<tr>
<td>1</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Cumulative risk in percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
</tr>
<tr>
<td>0</td>
</tr>
<tr>
<td>0.5</td>
</tr>
</tbody>
</table>

Note.—Hypothetical data.
Age-Incidence Effect. On the ascending side of the age-incidence curve (top of figure 1), those who are 11 months older will have a higher incidence rate. On the descending side of the age-incidence curve, those who are 11 months older will have a lower incidence rate. Therefore, the ascending and descending sides of the age-incidence curve produce opposite (but not equal) effects.

Age-Prevalence Effect. The cumulative risk curve (lower part of figure 1) ascends but never descends. Therefore, those who are 11 months older will have a higher cumulative risk for the onset of schizophrenia.

The age-prevalence effect (or cumulative-risk effect) has a simple mathematical relationship to the age-incidence effect: at a given age it represents the area under the age-incidence curve. This relationship can be expressed as follows: If age is \( a \), incidence rate for that age is \( I(a) \), and the cumulative risk for that age is \( R(a) \) then

\[
R(a) = \int_0^a I(a) \, da.
\]

At any given age the cumulative-risk that a person will have developed schizophrenia by that age is the lifetime prevalence of schizophrenia for that age group. This is why Lewis and Griffin referred to the cumulative-risk effect as an age-prevalence effect. The cumulative-risk curve plateaus at a line that we have marked "B" on the bottom of figure 1. This point, "B", represents the lifetime risk that a person will develop schizophrenia.

Correction factors have been suggested to adjust for both the age-incidence effect (Hare 1978; Pulver, Sawyer, and Childs 1981) and the cumulative-risk effect (Lewis and Griffin 1981; Pulver, Sawyer, and Childs 1981–1983; Watson et al. 1982; Shur and Hare 1983). Lewis and Griffin (1981) introduced a crude and not clearly defined method to correct for the difference in exposure to the period of risk, depending on month of birth: they found that the season of birth phenomenon was explainable from their data. They note, however, that the age-prevalence effect cannot account for the presence of the season of birth phenomenon in the southern hemisphere. Since the calendar year in the southern hemisphere is also divided at the December 31-January 1 point, according to their argument the excess risk of schizophrenia should occur for cohorts born between January through March, and not July through September. In fact, the excess births of schizophrenics in the southern hemisphere occur in the winter months, which in the southern hemisphere are July through September.

Similarly, the cumulative-risk effect could not explain an increased risk for schizophrenia for individuals in the northern hemisphere born in December (Pulver, Sawyer, and Childs 1981), the month of birth cohort that—by Lewis and Griffin’s 1981 definition—should have the lowest expected risk (Shensky and Shur 1982; Watson et al. 1982). A recent study has employed Lewis and Griffin’s statistical adjustment for the age-prevalence effect, and the results show that a significant season of birth effect remains (Watson et al. 1982). Another study shows that the season of birth effect persists even when a calendar year defined as July to June (Shur and Hare 1983).

One could completely eliminate both the age-incidence effect and the age-prevalence effect by defining the study population in a different way. This could be accomplished by defining the study population on the basis of month of age at admission to a hospital, rather than as all patients admitted to a psychiatric facility during a specific calendar time. Pulver et al. (1983) have reported results of an investigation in which the study population was defined by month of age at admission. Using standard life table techniques, the probability of being diagnosed as schizophrenic over the same specified age period for individuals born in each month was calculated. There was an increased risk for winter-born individuals to be diagnosed as schizophrenic.

Investigations of the Relationship Between the Risk for Bipolar Disorder and Season of Birth

The question of a season of birth effect for affective disorders has been examined by a number of investigators (Barry and Barry 1961; Hare and Price 1968; Hare, Price, and Slater 1974; Odegård 1974; Videbech, Weeke, and Dupont 1974; Woodruff, Guze, and Clayton 1974; Dalen 1975; Hare 1975a; Parker and Nielson 1976). The data do not suggest a seasonal effect in affective disorders.

There is reason to reexamine this question vis-a-vis bipolar disorder (Hare, Price, and Slater 1974; Dalen 1975; Hare 1975a). Most of the studies have focused on manic depressive illness rather than bipolar disorder. These diagnoses have quite different meanings; it has been reported that between 15 and 32 percent of manic depressives have had a manic episode (Krauthammer and Klerman 1979) while the remainder...
have had depression without mania. Since the winter excess of births for depressives is either nonexistent (Barry and Barry 1961; Ødegärd 1974; Videbech, Weeke, and Dupont 1974; Parker and Nielson 1976) or muted relative to that of manics (Hare, Price, and Slater 1974; Dalen 1975; Parker and Nielson 1976), the effect of studying manic depressive illness has been to obscure the question of whether manics (bipolars) have a distinctive seasonal birth pattern.

Table 2 shows the results of studies of the season of birth in bipolars. In the Australian study, no significant excess of manic patients was born in the winter—June, July, August—(Parker and Nielson 1976). A significant excess of manic patients born in the first quarter of the year was found in two studies with overlapping populations reported by Hare and others (Hare, Price, and Slater 1974; Hare 1975a). The other reports in table 2 are different aspects of one study conducted by Dalen in Sweden (1975). He found that there was a seasonal pattern to manic patients born in some years but not in others. The years 1921 to 1930 and 1942 to 1950 did show this seasonal phenomenon at the .01 level of statistical significance. The other years between 1901 and 1940 showed an excess, but it did not reach statistical significance.

In summary, there may be a seasonal excess of risk for bipolar disorder for those born between January and March and possibly in April. The evidence is not as strong as that for schizophrenia, but this may be due in part to the far smaller sample sizes for mania. If there is a winter excess of bipolar births, the magnitude of the excess could be as great as that for schizophrenia: table 2 shows over 10 percent excess winter births.
One plausible explanation of the seasonal pattern in both psychoses is that those born in the winter months may represent a subtype of patients who may be diagnosed either as manic or schizophrenic. Perhaps winter birth predisposes to an atypical psychosis that sometimes mimics schizophrenia and sometimes mimics bipolar disorder. A second possibility is that these seasonal patients are given both diagnoses at different times. In a followup of the Iowa 500 patients, 3 percent of those originally diagnosed as schizophrenic were rediagnosed as bipolar 30 to 40 years later, and 13 percent of those originally diagnosed bipolar were later rediagnosed as schizophrenic (Tsuang et al. 1981). A third possibility is that the season of birth phenomenon represents a necessary but not sufficient cause for both these disorders.

Evidence Suggesting That Season of Birth May Be a Clue to a Distinctive Group of Schizophrenic Patients

It has been proposed that schizophrenia is a heterogeneous disorder analogous to mental retardation. Accordingly, even without knowing the biological meaning of the season of birth effect, the idea that it is a clue to the identification of a distinct group of schizophrenics should be tested. Some evidence favoring this hypothesis has already been reported.

Sex Differences. Seven investigators subdivided their samples of patients by sex (Roche and Dalen 1974; Dalen 1975; Parker and Nielson 1976; Parker and Balza 1977; Jones and Frei 1979; Pulver, Sawyer, and Childs 1981; Pulver et al. 1983). Five reported a sex difference in the season of birth effects; four of the five found the seasonal effects to be more marked among the females.

Chronicity. There is some evidence that the winter-born form of schizophrenia is a less chronic form of the disease. Dalen noted a more marked seasonal effect for patients hospitalized less than 3 years in his Swedish sample but not in a sample of South African patients (Dalen 1975). Pulver et al. (1983) divided their population according to duration of first psychiatric hospitalization (more than 180 days and 180 days or less). Patients hospitalized 180 days or less were more likely to be born in the winter; patients hospitalized more than 180 days were not. Unfortunately, the sample size for those hospitalized more than 180 days was small. Watson et al. (1984), however, have shown that the season of birth effect is more pronounced among unmarried, presumably severe schizophrenics.

Genetic Risk. There is some evidence that season of birth is important to the etiology of a subgroup of schizophrenic patients who do not have a family history of schizophrenia. Kinney and Jacobsen (1978) divided their sample of 34 schizophrenic patients into (1) patients with a family history of schizophrenia or a history of postnatal brain damage and (2) patients with neither of these factors. The patients with neither risk factor were more likely to be born during the winter months. Shur (1982) divided his sample of 973 schizophrenic patients according to family history of psychiatric illness. The patients with a family history of either severe psychiatric illness or schizophrenia were born less often in the first quarter of the year. Other studies also provide supporting evidence that the season of birth effect plays a larger role in persons who have lesser genetic predisposition (Shensky and Shur 1982; Templer and Veleber 1982).

Machon, Mednick, and Schulsinger (1983) assume that there is a positive interaction between genetic predisposition and the season of birth effect. According to their hypothesis, perinatal viral infections specifically attack the nervous systems of genetically vulnerable fetuses. They do not, however, present data to show that the season of birth effect is higher in children at high genetic risk than in children at low genetic risk. In other words, they have not yet presented data to support this assumption. Their assumption of a gene-viral interaction contradicts the weight of published data from studies that have examined this issue (Kinney and Jacobsen 1978; Shensky and Shur 1982; Shur 1982; Templer and Veleber 1982).

Age of Onset. There is some evidence that early onset is associated with winter birth. Pulver, Sawyer, and Childs (1981) provide evidence suggesting that age of onset of schizophrenia (age at first hospitalization) may be used to identify a group of schizophrenic patients who are more likely to have an excess of winter births. Hare (1978) had previously concluded that either age of onset or year of birth may affect the seasonal distribution of birthdates of schizophrenic patients.

The Meaning of the Season of Birth Phenomena

Two theories have been offered to explain the season of birth phenomenon: (1) Some seasonably varying factor occurring during intrauterine life or during the first postnatal months alters the central
nervous system, in some way increasing the risk for schizophrenia; or (2) parents of schizophrenics are more likely to conceive in the spring or early summer.

Theory 1: Seasonally Varying Factor Alters the Central Nervous System

The season of birth phenomenon has been of interest primarily because it has been assumed to be evidence that the etiology of some as yet unidentified subtype of schizophrenia is due in part to a seasonal factor, such as viral infection, obstetrical or perinatal complications, malnutrition, vitamin deficiency, or temperature (Dalen 1975; Hare 1979).

If this theory is true, then there should be at least two types of schizophrenia: one type that is related to this seasonal etiologic factor ("seasonal type") and all others ("nonseasonal types"). The same may be true of bipolar affective disorder. The proportion of schizophrenics who are the seasonal type is unknown. The relationship of the seasonal to nonseasonal types may be as illustrated in figure 2. In the figure the seasonal type schizophrenics are shown to be about 5 to 10 percent of all schizophrenics; however, they entirely account for the season of birth phenomenon. The nonseasonal types of schizophrenia, by definition, have the same season of birth distribution as the general population.

Figure 2. Winter-born schizophrenics compared with nonwinter-born schizophrenics

![Diagram showing the percentage of schizophrenics born in winter versus nonwinter](image)

<table>
<thead>
<tr>
<th>Percent of schizophrenics</th>
<th>Winter born</th>
<th>Nonwinter born</th>
</tr>
</thead>
<tbody>
<tr>
<td>100</td>
<td>80</td>
<td>60</td>
</tr>
<tr>
<td>80</td>
<td>60</td>
<td>40</td>
</tr>
<tr>
<td>60</td>
<td>40</td>
<td>20</td>
</tr>
<tr>
<td>40</td>
<td>20</td>
<td>0</td>
</tr>
</tbody>
</table>

Note — Hypothetical data.

One way to discover which schizophrenics are the seasonal type is to compare those born in the winter with those born in other times of the year, looking for qualities that distinguish a segment of the winter-born schizophrenics from schizophrenics born at other times. Characteristics that might be studied this way are discussed next.

Brain Damage. All the agents that have been suggested to cause the seasonal type of schizophrenia are thought to do so through some sort of damage to the brain. There is likely to be a spectrum of degrees of brain damage. Some of this damage may be too subtle for detection by ordinary means, but some of it may be discovered by neurological exam, electroencephalogram, or computed axial tomography. Although these procedures have been done on many schizophrenics, we know of no studies in which an attempt has been made to correlate abnormal neurological results with season of birth.

Socioeconomic Status of the Parents at the Time of the Patient's Birth. It is important to stratify schizophrenics on the basis of parental socioeconomic status at the time of the patient's birth, because pregnant women and newborn infants of lower socioeconomic status are disproportionately exposed to almost all the etiologic factors of interest. They have higher risk pregnancies, more obstetrical and perinatal complications, more exposure to viral infection, poorer nutrition, more vitamin deficiency, and more exposure to seasonal temperatures (Bierman et al. 1965; Pasamanick and Knobloch 1971; Frederick and Adelstein 1978; Eisner et al. 1979; Gaziano, Freeman, and Allen 1981). If any of these factors are associated with etiology of the seasonal subtype...
of schizophrenia, then one might find schizophrenics born to parents of the lower socioeconomic groups to be overrepresented among the winter born. In the general population, socioeconomic status is not associated with an excess of winter births (Dalen 1975), and the socioeconomic status of parents of schizophrenics does not differ from that of the general population. In summary, we are suggesting that although parental socioeconomic status is not clearly associated with the risk for schizophrenia, except perhaps in large cities (Kohn 1973), nevertheless it may be associated with the risk for the seasonal subtype of schizophrenia.

Gallagher, McFalls, and Jones (1983) present data to show that the season of birth effect is found among black rather than white schizophrenics. This is taken as evidence of a socioeconomic status effect (Gallagher, McFalls, and Jones 1983).

Thus, it will be possible to investigate whether the season of birth phenomenon is associated, not with DSM-III schizophrenia, but with these other diagnoses. No one has examined this question. Furthermore, DSM-III provides data on premorbidity functioning in Axis V. Strauss and Carpenter (1974, 1977) have shown that the level of premorbidity functioning is the best indicator of prognosis. The question is whether schizophrenics with a higher level of premorbidity functioning are more likely to have been born in the winter than those with a lower level of premorbidity functioning. It is a question that could be investigated in a large data set that includes data of birth, DSM-III diagnosis, and Axis V information.

Good Prognosis vs. Poor Prognosis Schizophrenia. As noted above, there is some evidence that winter-born schizophrenics have shorter first hospitalizations (Dalen 1968; Dalen 1975; Pulver et al. 1983). This relationship should be verified in other samples. It will be possible to examine the issue in the United States when data from the third edition of the Diagnostic and Statistical Manual (DSM-III) (American Psychiatric Association 1980) are available in State and national registries. In DSM-III "schizophrenia" is a diagnosis that should be used only for patients with at least a 6-month duration of that disorder. Thus, many of those previously given this diagnosis in the United States will no longer qualify. Those with a disorder of shorter duration will receive a DSM-III diagnosis of "schizophreniform" or "atypical psychosis."

This theory suggests that the parents of schizophrenics and patients with bipolar affective disorder are more likely to conceive in the spring or early summer (Barry and Barry 1961; Hare and Price 1968; Dawson 1978). If that is true, the siblings of schizophrenics and patients with bipolar affective disorders should show the same surplus of winter births (Hare 1976; McNeil, Kajj, and Dzierzyk-ray-Rogalska 1976; Buck and Simpson 1978).

Table 3 shows the results of studies that have tested this hypothesis. Hare found a significant excess of first-quarter births among the siblings of manic-depressive probands (Hare 1976). Hare also found a nonsignificant excess of first-quarter births among the siblings of schizophrenics. This may be due to the small sample size. In a larger sample, however, Buck found a nonsignificant deficit in first-quarter births for the siblings of schizophrenics (Buck and Simpson 1978). Not shown on table 3 is a study by Lang that compared season of birth of manic depressive patients with their siblings and found no difference (Hare, Price, and Slater 1974).

The results of these studies are ambiguous but certainly do not prove a seasonal birth pattern in the siblings of schizophrenics. The largest study found negative results (Buck and Simpson 1978). Even if siblings were found to have a winter excess of births, two conceptual problems must be considered:

1. The relationship of season of birth between siblings in the general population must be examined. Let us suppose, for example, that there were always a significant tendency for one sibling to be born in the same season as the other sibling, unrelated to psychiatric diagnosis. This would indicate a seasonal mating pattern for all couples, but it would not provide any evidence for why schizophrenics should have a different season of birth distribution than other people. If schizophrenics have an excess of winter births, based on, for example, viral infections during pregnancy, then one would expect that their siblings would likewise have an excess of winter births by virtue of the seasonal mating pattern of all couples. This line of hypothetical reasoning would lead to the possibility that the siblings of schizophrenics have an excess of winter births, yet the season phenomenon is still due to viral infection in the womb.

2. It would be unclear whether the seasonal births were due to their status as siblings, or whether a small subgroup of siblings is destined later in life to become schizophrenics. The siblings who will eventually develop schizophrenia would be expected to
Table 3. Season of birth in siblings

<table>
<thead>
<tr>
<th>Place, time of study</th>
<th>Number of probands</th>
<th>Number of siblings</th>
<th>Control group</th>
<th>Findings</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maudsley Hospital, London, 1967-1971</td>
<td>Not stated</td>
<td>451</td>
<td>Nonpsychotic patients</td>
<td>Siblings of manic depressives have a significant (p &lt; .01) excess of births in the first quarter of the year, 20 percent more than expected.1</td>
<td>Hare 1976</td>
</tr>
<tr>
<td>Maudsley Hospital, London, 1967-1971</td>
<td>Not stated</td>
<td>219</td>
<td>Nonpsychotic patients</td>
<td>Siblings of schizophrenics have 13 percent more births in the first quarter than expected, but the sample is too small to reach statistical significance.1</td>
<td>Hare 1976</td>
</tr>
<tr>
<td>Sweden 1973</td>
<td></td>
<td>91</td>
<td>General population</td>
<td>Siblings of schizophrenics have a nonsignificant excess of births in the first quarter.</td>
<td>McNeil, Kajl, &amp; Dzierzykay-Rogalska 1976</td>
</tr>
<tr>
<td>Ontario, Canada 1975</td>
<td></td>
<td>210</td>
<td>General population</td>
<td>Siblings of schizophrenics have a nonsignificant deficit (− 7 percent) of births in the first quarter.</td>
<td>Buck &amp; Simpson 1978</td>
</tr>
</tbody>
</table>

1The present authors recalculated the data, using a chi square with 1 degree of freedom.

have a winter excess of births, because when they later become schizophrenics, they will have a winter excess of births. In this case a winter excess of births among siblings could indicate that some of them had been exposed to a seasonal factor that damaged their brains and predisposed them to schizophrenia.

Thus, even if a winter excess of births were found among the siblings of schizophrenics, it would not necessarily imply that the season of birth phenomenon is an artifact attributable to a tendency by the parents of schizophrenics to conceive in the spring.

Conclusion

There is substantial evidence that schizophrenics are more likely to be born in the winter than other seasons, even in the southern hemisphere, where the winter months are June, July, and August. Attempts to explain this apparent phenomenon as an artifact have been refuted. When various artifacts (such as the age-incidence or age-prevalence effects) are eliminated or statistically controlled, the excess risk for winter borns remains. There is evidence to suggest that the same winter excess of births may be found in bipolar affective disorder, although more studies are needed to verify the season of birth phenomenon for the bipolar disorder.

If a seasonal factor damages the central nervous system during fetal or perinatal development and thereby predisposes the fetus to later developments of schizophrenia, one might expect to find that the subgroup of schizophrenics exposed to that seasonal factor differs from other schizophrenics. There have been few studies investigating the characteristics that distinguish winter-born schizophrenics from nonwinter-born schizophrenics. There is nevertheless some evidence to suggest that winter-born schizophrenics are more likely to have a better prognosis, less genetic risk for schizophrenia, and an earlier age of onset of the disorder. Although early age of onset is associated with poor prognosis when all schizophrenics are considered together, this relationship does not necessarily hold for all subtypes of schizophrenics. In fact, the seasonal subtype appears paradoxically to have both early onset and a good prognosis, in contrast to the majority of schizophrenics. We know of no studies that have investigated neuro-
logic or radiographic evidence of central nervous system damage or that have investigated parental socio-economic status in relationship to season of birth; however, these areas need investigation.

The only other theory that has been proposed to explain this phenomenon is that the parents of schizophrenics are more likely to conceive in the spring or early summer. This theory has been investigated by studying the season of birth of siblings of schizophrenics, with ambiguous results. The largest study of siblings found a deficit of births in the winter months. At the present time, there is little evidence to support the theory of increased spring conception by parents of schizophrenics.

References


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The Authors

Jeffrey H. Boyd, M.D., M.P.H., is Assistant Chief, Epidemiology and Psychopathology Research Branch, Division of Clinical Research, National Institute of Mental Health, Rockville, MD. Ann E. Pulver, Sc.D., is Assistant Professor of Psychiatry and Epidemiology, Maryland Psychiatric Research Center, University of Maryland School of Medicine, Baltimore, MD. Walter Stewart, Ph.D., is Assistant Professor of Epidemiology, Johns Hopkins University, School of Hygiene and Public Health, Baltimore, MD.

Videotapes on Schizophrenia Available

The Video Center of the George Warren Brown School of Social Work, in cooperation with several community and mental health organizations, has produced four videotapes on the following topics relating to survival issues for chronically mentally ill persons and their families in the community.

Coping With a Chronically Mentally Ill Relative in the Community—The two videotapes on this topic were produced in cooperation with the Alliance for the Mentally Ill, St. Louis Chapter. Each videotape presents the experiences of a family which has had some success surviving the multiple problems arising from caring for a mentally ill relative in the community. The videotapes are intended for an audience of parents and relatives of chronically mentally ill persons who could benefit from a vicarious sharing of experiences with the families on the videotapes.

Psychosocial Rehabilitation: Two Agencies Based on the Fountain House Model—These two videotapes were produced in cooperation with the Missouri Department of Mental Health, Independence Center, and Places for People, St. Louis, MO. Each videotape presents a psychosocial rehabilitation agency from the point of view of its members. The tapes are intended for professional audiences as well as for families and mentally ill persons who could benefit from knowing what it's like to experience psychosocial rehabilitation "from the inside.”

For more information about the rental or purchase of these videotapes, please contact: Dr. David Katz, Video Center, Box 1196, Washington University, St. Louis, MO 63130.