Age Incidence and Schizophrenia: Part I. The Season of Birth Controversy

by Marc S. Lewis

Abstract

Distortions in incidence data are probably responsible for the apparent excess of schizophrenia among people born in winter. The widely held belief that the excess reflects a winter component in the etiology of schizophrenia is based on errors in the design and interpretation of seasonal studies.

In 1929, when schizophrenia was still commonly known as dementia praecox, when the predominant theories of etiology were toxic endocrine poisons and poor mental hygiene, when genetic involvement was still just a suspicion and the primary treatment was occupational therapy—in that distant year, Tramer first reported that an excess number of future schizophrenic patients appeared to be born in winter. It was a promising find in an era of little promise. Seasonality was one of Hippocrates’ great clues to the etiology of disease, and no one familiar with the history of medicine would have been surprised if within a year or two of Tramer’s report, someone had turned up a cold weather variable responsible for some forms of schizophrenia.

The importance of seasonality can be illustrated by another finding of the same era. Maxcy’s (1926) report that typhus fever had different peak seasons in the United States and Europe led to the unexpected discovery that two diseases with identical symptoms were involved. Within 5 years the seasonal vector for American typhus was identified, and control of the disease was established (Dyer et al. 1931).

Schizophrenia has not yielded to seasonal study as gracefully as typhus. Sixty years after Tramer, we know only that although there is disagreement, the obvious winter variables (e.g., temperature) do not appear to be responsible and that a confusing array of other variables do. The situation is illustrated in table 1, which lists some recent findings. The theory with the most explanatory power attributes seasonality to harmful environmental agents, but no theory explains even half of the results, and there is no consistent pattern across studies (compare, e.g., Kendler [1982] with Shensky and Shur [1982] and Corgiat et al. [1983]; Bradbury and Miller [1985] with Boyd et al. [1986]; Gallagher et al. [1983] with Bradbury and Miller [1986]; Templer and Veleber [1982] with Nasrallah and McCalley-Whitters [1984]; or Hafner [1987] with Baron and Gruen [1988]). These are just 6 years of results, and they represent only a fraction of the diversity found across the entire literature.

How could the seasonality of schizophrenic births have so many unrelated origins? Lewis and Griffin (1981) recently suggested that the answer could be found in an artifact called age incidence (Dalen 1975). A significant result caused by that artifact would be mistakenly attributed to the variable under investigation, and eventually the literature would be filled with a wide variety of unrelated findings.

Age incidence works as follows. If the incidence of a disease increases with age, 40-year-olds will produce more cases than 20-year-olds, and to a lesser extent 31-year-olds will produce more cases than 30-year-olds. Dalen’s contribution was the observation that this principle applies within years as well as...
Table 1. Results from recent studies of seasonality in schizophrenia

<table>
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<tr>
<th>Study</th>
<th>Major result</th>
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<tr>
<td>Kendler (1982)</td>
<td>No season of birth effect in 536 twin pairs (including monozygotic, dizygotic, concordant, and disconcordant subanalyses). Mild evidence against prenatal/birth insult explanation</td>
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<td>Shur (1982)</td>
<td>Nonsignificant deficit of high-risk cases born in winter</td>
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<td>Shensky &amp; Shur (1982)</td>
<td>Deficit of high-risk cases born in winter. Supports prenatal/birth insult explanation</td>
</tr>
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<td>Templer &amp; Veleber (1982)</td>
<td>Greater seasonality in paranoid than catatonic and hebephrenic patients</td>
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<tr>
<td>Watson et al. (1982)</td>
<td>Correcting for age incidence does not eliminate seasonal effects</td>
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<td>Corgiat et al. (1983)</td>
<td>Seasonality is strongest in late-onset cases. Supports explanations based on harmful effects with delayed action (e.g., slow viruses)</td>
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<tr>
<td>Gallagher et al. (1983)</td>
<td>Winter excess limited to blacks. Supports socioeconomic explanations (e.g., nutritional deficit)</td>
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<tr>
<td>Machon et al. (1983)</td>
<td>Seasonality only in urban-born, high-risk schizophrenics. Supports viral hypothesis</td>
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<tr>
<td>Pulver et al. (1983)</td>
<td>Seasonality limited to patients hospitalized &lt; 180 days</td>
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<tr>
<td>Shur &amp; Hare (1983)</td>
<td>Correcting for age incidence does not remove seasonal effects</td>
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<td>Gallagher et al. (1984)</td>
<td>Winter excess limited to blacks</td>
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<tr>
<td>Nasrallah &amp; McCalley-Whitters (1984)</td>
<td>Seasonality in nonparanoid females, trend in paranoid males. Schizophrenia gene has protective effect in males; seasonality in females caused by harmful outside effects</td>
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<td>Opler et al. (1984)</td>
<td>Deficit in winter-born, positive-syndrome schizophrenic patients; none in negative-syndrome schizophrenic patients</td>
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<tr>
<td>Watson et al. (1984)</td>
<td>Seasonality in unmarried schizophrenic patients born in years following outbreaks of infectious disease (especially bacterial). Seasonality unrelated to temperature</td>
</tr>
<tr>
<td>Bradbury &amp; Miller (1985)</td>
<td>Review: Existence of seasonality established, but no overwhelming evidence for any proposed explanation</td>
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<td>Lo (1985)</td>
<td>Seasonality in schizophrenic patients with positive family history</td>
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<tr>
<td>Boyd et al. (1986)</td>
<td>Review: Seasonality associated with early onset, low risk, and favorable prognosis</td>
</tr>
<tr>
<td>Bradbury &amp; Miller (1986)</td>
<td>Gallagher et al. (1983, 1984) result due to white deficit in winter births, not to black excess</td>
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<tr>
<td>Pasamanick (1986)</td>
<td>Comment: Winter seasonality due to the effect of hot summers on gestation</td>
</tr>
<tr>
<td>Machon et al. (1987)</td>
<td>Seasonality in high-risk, urban-born schizophrenic patients with prenatal/birth complications</td>
</tr>
<tr>
<td>Watson et al. (1987)</td>
<td>Schizophrenic patients born after winters with elevated incidence of infectious disease tend to be seasonal, reactive (vs. process), and high in anhedonia</td>
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<tr>
<td>Hähnner (1987)</td>
<td>Excess incidence of schizophrenia among people born in March and April; similar excesses in neurosis and personality disorder. Results plus review of past studies support the nonspecific harmful agent, biological, and procreational theories, but not the diathesis-stress theory</td>
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The Nature of Age Incidence

Dalen (1975) dealt age incidence a serious blow in the same paragraph in which he proposed it:

First admissions with schizophrenia show a rising incidence with age in young people. The chances of being included in the sample are therefore slightly higher for a person born in January than for one born in December of the same year but differences between years of birth are eliminated when cases are summed over years ("circular time"). The maximum bias is therefore proportional to the rise (or fall) in incidence rates within one year of age, and this is usually a small quantity. [p. 65]

No seasonal birth differences between schizophrenic patients with vs. without positive family history or between cases grouped by sex, birth order, age of onset, or subtype. Winter-spring excess for patients with broad spectrum disorders vs. first-degree relatives. Support for stress-diathesis model

Baron & Gruen (1988)

between them. People born in January, February, and March of a given year are older than people born in later months, and therefore they should produce more new cases. An investigator seeing this excess incidence might mistakenly attribute it to winter.

From the start, nobody—not even Dalen—believed that age incidence was responsible for the season of birth effect in schizophrenia. This doubt was so widespread that only one investigator of the time (Hare 1975a, 1975b) ever really studied age incidence, and he found what everyone already knew; its effects were not strong enough to cause false season of birth results. A few years ago, however, Lewis and Griffin (1981) presented evidence that an especially strong form of age incidence (which we called age prevalence) could create false seasonal results. Since then, several articles (Templer 1982; Watson et al. 1982; Pulver et al. 1983; Shur and Hare 1983) have argued against the existence of this strong form of age incidence.

Although the question remains undecided, most investigators believe that season of birth effects are not caused by age incidence. The reasons for that opinion, summarized by Bradbury and Miller (1985), are as follows: (1) Age incidence is not strong enough to account for season of birth effects. (2) The strong form of age incidence (i.e., age prevalence) does not exist because several studies have failed to find it. (3) Studies in the Southern Hemisphere report that the season of birth effect is reversed to correspond with their winter. (4) Season of birth effects are commonly found in December, a result that cannot be explained by age incidence because people born in that month are younger, not older, than people born in other months. Taken at face value these are strong arguments. The purpose of the present article is to show that they are incorrect. In particular, I will show that age incidence is strong enough to produce spurious seasonal age incidence effects; that the failure of some investigators to find the strong form of age incidence in schizophrenia is due to problems in their analyses; that studies in the Southern Hemisphere do not support the seasonal hypothesis; and that December season of birth effects are not common in well-controlled studies. We begin with one of the primary unanswered questions: Can age incidence produce false season of birth effects?

Circular time refers to the fact that in a sample spanning several years, people born in January of one year are followed by an older group born in December of the next year, who in turn are followed by an even older January-born group, and so on. In this way, each subsequent older group dilutes the age incidence effect of the younger group that preceded it.

Because of Dalen's comment, it is widely believed that age incidence cannot produce false season of birth effects in any disease, including schizophrenia. Sometimes, however, widely held beliefs do not survive close analysis, and the present section shows that the small quantity of bias that Dalen ascribed to age incidence may not be so small after all.

Consider first the χ² test for seasonality, which asks whether the number of schizophrenic persons born in a given month differs from chance. For example, if 1/12 of the population was born in January, a sample of 4,800 schizophrenic persons should contain about (1/12 of 4,800) = 400 January-born cases. The current view is that if the sample contains far more than the expected number of January-born cases, season of birth effects are responsible. The question to be considered here is whether such excesses can also be caused by age incidence. That question is not difficult to answer. As shown in the appendix, if the rate of a disease changes in a regular way, it is possible to calculate observed and expected incidences for each month and to inspect them for false sea-

No seasonal birth differences between schizophrenic patients with vs. without positive family history or between cases grouped by sex, birth order, age of onset, or subtype. Winter-spring excess for patients with broad spectrum disorders vs. first-degree relatives. Support for stress-diathesis model

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sonal effects. For example, the appendix shows that a disease whose risk rises linearly with age (e.g., lung cancer) can create age incidence effects strong enough to produce false season of birth results. An investigator who was unaware of that artifact would find a false seasonal excess for January about 48 percent of the time and an overall winter excess about 72 percent of the time. The important point, then, is this: age incidence can produce spurious winter season of birth effects which are not eliminated by the circularity of the calendar year.

One of the most surprising findings of the appendix is that age incidence is not the straightforward influence that it is thought to be; rather, its behavior depends upon the type of disease under study. For example, if the risk of a disease changes exponentially with age, the rate of that change, an unimportant variable in the linear model, suddenly becomes very important. As in the linear model, large age incidence effects are possible.

Both situations considered thus far involve diseases whose risk increases with age. Some researchers believe that if the risk of a disease decreases with age, a negative age incidence effect will occur because people born in January, being older than people born in December, will have a lower incidence rate. That belief is incorrect. Season of birth studies are based on cumulative incidence, and people born in January of a given year will always have a cumulative incidence at least equal to that of people born in December.

Although many of the principles that govern age incidence change from situation to situation, there is one consistency that is both interesting and important; the effect is strong in young groups and weak in old ones. This result predicts that almost no season of birth effect will be found among older schizophrenic patients and it explains a finding that has long been considered a mystery. Although there are exceptions, most investigators of older populations (e.g., Dalen 1975; Shimura et al. 1977) have reported a surprising absence of seasonal effects among people born around 1900. What those authors have overlooked is that the season of birth effect was first discovered in Tramer’s subjects, all of whom were born around 1900. Seasonal results continued to be reported for that birth group by the investigators who followed Tramer in the 1930’s and 1940’s; then suddenly in recent years the result disappeared. In other words, when people born in the early 1900’s were young, they produced an apparent winter excess, but as they grew older, that excess disappeared. This result cannot be explained by seasonality, but it can be explained by age incidence. Age incidence also predicts that if the researchers of Tramer’s time had looked at older populations, they would have found no season of birth effect among people born in the 1800’s. That is exactly what happened. Early investigators were as convinced that the season of birth effect did not exist before 1880, as present investigators are that it did not exist before 1900. For example, Huntington (1938) found a strong season of birth effect among schizophrenic patients born in 1914–24, a weaker effect among those born in 1885–1914, and the same mysterious absence of a winter excess among people born before 1885.1 Again, these findings are paradoxical when interpreted by the seasonality hypothesis, but they are easily accounted for as a natural consequence of age incidence.

An Impossible Result

Once we know the properties of age incidence, it is simple to find them in real situations provided that we choose a sample with friendly parameters. To avoid the type of controversy that has arisen concerning age incidence in schizophrenia, let us choose a situation in which seasonality is unlikely to be important. If we find an apparent season of birth effect where none should be present, the most reasonable explanation is that the result is due to age incidence. An interesting choice is mortality where the question is, are people born in winter more likely to die than people born in other months? This is such an unlikely hypothesis that no one has cared to study it, but age incidence predicts that mortality data will contain just such a false seasonal result.

Table 2 shows mortality data for 5,134 people born in Texas between 1933 and 1941 and who died in 1983. I have analyzed those data using the standard \( \chi^2 \) test for seasonality. The top two rows of the table show observed and expected data may have been misinterpreted. Those data, presented in a very small graph, appear to have been read from hashmarks on the abscissa. When read in this manner, his figures do not add to 100 percent, they do not match Huntington’s description of his results, and they do not correspond to the places where the slope of his graph changes direction. It appears that his data should be read at the midpoint between hashmarks on the abscissa, directly under the letter used to designate each month of birth. This possible misreading has no effect on the present discussion.

Observed  487  443  441  402  383  413  442  436  424  433  390  440

Expected  453.0  408.6  426.8  377.0  366.6  379.0  446.7  480.0  464.6  455.4  431.1  445.1

$\chi^2$  2.55†  2.90†  0.47†  1.66†  0.73†  3.05†  0.05†  4.03†  3.55†  1.10†  3.92†  0.06†

$\chi^2 = 24.07; df = 11, 5134; p < .02$. 

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case of schizophrenia is the next topic.

**Age Incidence and Schizophrenia: The Evidence**

Tests for Age Incidence. The Texas mortality data presented in Table 2 were easy to interpret because age incidence was a reasonable hypothesis and seasonality was not. Schizophrenia is a tougher problem because age incidence and seasonality are both reasonable hypotheses and the $\chi^2$ test does not distinguish between them. In the Lewis and Griffin (1981) article, however, I suggested a method that could distinguish between the two hypotheses. That approach, which I will call the correction test, involves treating data in a way designed to negate age incidence effects. Expected values are obtained by multiplying the number of people born in each month by the length of time that they have been at risk. When Griffin and I applied that test to data for 10,363 schizophrenic patients, the seasonal effect vanished. Two years later, Shur and Hare (1983) reported the opposite result for a correction test of schizophrenic patients in England and Wales. When their results are considered in detail, however, a different picture emerges.

First, there is no question that the uncorrected Shur and Hare data contained significant monthly incidences for January, February, and June and significant deficits for August, September, and December. The June excess occurs in several of their analyses, and it is predicted by neither theory; the December deficit is predicted by age incidence but not by seasonality, and all other results are predicted by both theories. After Shur and Hare applied the correction test, many of these seemingly seasonal results disappeared; the correction removed all winter results, leaving only a significant June excess and an August deficit. Of particular note is the fact that the December data behaved exactly as predicted by age incidence, showing a deficit in the initial analysis and no deficit after correction. The seasonal hypothesis predicted neither finding.

A second correction test, using schizophrenic patients under 21 years old, was described by Shur and Hare (1983) as the crucial test of age incidence. The bottom row of Table 3 contains the $\chi^2$ values from that analysis as reported on p. 375 of the Shur and Hare article. Here it can be seen that the corrected data did not show the winter excess that was claimed; the only notable result is an excess in June. From this result Shur and Hare (p. 375) concluded that correcting for age incidence "leaves a chi-square value that is still significant at the 5% level. . . ." This statement implies that their result supported seasonality. It did not. In fact, as the authors later state, the June excess is probably due to random variation and, therefore, their result supports age incidence.

To test the hypothesis that schizophrenia is caused by seasonality, Shur and Hare (1983) applied a second way to distinguish age incidence from seasonality which I will call the displacement test. The test uses a sample based on people born during a July–June year rather than the traditional January–December year. For example, if the traditional test is based on incidence among people born between January 1950 and December 1959, the displacement test would be based on incidence among people born between July 1950 and June 1959.

In effect, the test displaces the calendar year, making people born in July the oldest group in the sample. The data are then analyzed twice, once using expected values based on a traditional birth year and once using values based on a displaced birth year. If a monthly excess is caused by seasonality, making people born in July the oldest group should have no effect; both the standard and displacement tests should show high incidence rates for winter months and low rates for nonwinter months. But if a result is caused by age incidence, making July the oldest group should cause the age incidence effect to shift 6 months. In other words, the standard test for sea-

**Table 3. Data from several seasonal analyses reported by Shur & Hare (1983)**

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<td>1074</td>
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<td>1.3</td>
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<td>0.4</td>
<td>1.5</td>
<td>0</td>
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<td>2.0</td>
<td>0.7</td>
<td>0.3</td>
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*Note.—Top row—observed values from the standard test; middle row—observed values from the displacement test; bottom row—$\chi^2$ values for a sample of young schizophrenic patients.

$p < .01$. 
The fact that age incidence effects of table 3 show observed values (Watson et al. 1982). Rows 1 and 2 beginning to appear in other dis-
seasonal hypothesis. It is not evidence for surprising, and it is not evidence for
the displacement test is smaller than the number reported in the standard test (e.g., 1,074 vs. 1,024 for January). This reduction is a natural consequence of the displacement test. The test requires the investigator to remove all cases that occurred among people born between January and June of the oldest birth year in the sample. When Shur and Hare did so, observed values for those months decreased. Remember, however, that the displacement test also requires the investigator to remove all cases that occurred among people born between July and December of the youngest birth year in the sample. Apparently this group produced no cases, because as table 3 shows, eliminating those data had no effect on observed values; observed incidences for July through December should have been smaller in the displacement test than in the standard test, and they were not. Thus, for example, the table reports 898 first admis-
sions for people born in December in both the standard and displacement tests. This situation reflects a misunderstanding about the proce-
dure of the displacement test. A similar situation in Watson et al. (1982) suggests the following comments.

When a sample is collected over several years, each of those years is a separate set of data and each must be displaced. For example, the Watson et al. data contained schizo-
phrenic patients born from 1910 to 1959 and discharged during a 1963-78 observation period. To apply the displacement test, it is necessary to remove birth data for January–June 1910 and July–December 1959. Observed values are then similarly displaced producing 15 different periods of data collection (i.e., July 1963–June 1964 through July 1977–June 1978). The displaced birth data are then used to calculate a set of expected values for each of the 15 displaced observation years. Observed and expected values are then summed across observation years and analyzed as usual. If Watson et al. or Shur and Hare did not follow that procedure, their samples were displaced by only a small percent-
age of the degree to which they should have been.

While on this subject, let me comment on a related error. In con-
ducting a displacement test, there is no point in truncating data for groups that have not yet entered the risk period, or for those that have passed through it entirely. That is, all truncations should remove at least one case. The need for this precaution is demonstrated by an extreme situation. Removing data for people ages 1 and 100 is not likely to affect observed values, but it is likely to affect expected values because birth rates have increased in the last 100 years.

A different type of truncation error appears in the attempt of Pulver et al. (1983) to remove age incidence from their data. Those authors reasoned that although people born in January are older than those born in December, both groups have passed through a common age period in the past. If onset rates are compared over that common period (age 17–23 in the Pulver study), there should be no differ-
ces in time at risk and hence no age incidence effect.

This idea is so good that it is hard to understand how the Pulver group was able to introduce a mis-
take that could negate its value. After selecting a sample of schizo-
phrenic patients born between 1942 and 1946, they abandoned their plan to include everyone diagnosed between ages 17 and 23 and instead...
added a new requirement: anyone not diagnosed between January 1963 and December 1965 was eliminated from the sample.

The effect of the new requirement can be seen in the period of greatest risk (i.e., age 23). Among people born in January, the only group to pass through that risk period is the one born in 1942 and observed during 1966; among people born in December, no group passes through that risk period. In fact, at the end of the study the oldest December group, the one born in 1942 and observed during 1965, had spent less than 1 month in the age 23 risk period compared to the more than 11 months spent by the oldest January group. In addition, because of the new requirement, people born in January were observed almost 1 year longer than people born in December. The reason is that people born in December 1946 were only 16 years old in 1963, and they did not meet the study’s age 17–23 requirement for inclusion in the sample. By contrast, all January groups met that requirement. Knowing that groups born in the early months of the year were observed during a greater period of risk and for a longer time than groups born in the late months of the year, one has little difficulty in explaining the January, February, and March excess that Pulver et al. reported.

To avoid the misunderstandings that have arisen in distinguishing age incidence from seasonality, I have considered several alternative ways to analyze seasonal data. The one that appears superior involves the creation of “long years,” which are generated by expanding the calendar year to 24 months. For example, to apply this test to the Missouri schizophrenic patients in the Lewis and Griffin (1981) study, we joined data for people born in, say, 1942 with that for people born in 1941 to create a single year that is 24 months long. Similarly, we joined 1940 with 1939 and so on for all pairs of years in the study. The result is a sequence of long years, each of which contained two Januarys (the 1st and 13th month), two Februaries, and so on. We next constructed a table similar to Table 2 except that there were now 24 cells instead of 12. This table allowed us to compare the result for a given month in two different positions of the year. If a month showed excess admissions in its early position but not in the late position, we concluded that age incidence effects were responsible; if it showed excess admissions in both positions, we concluded that seasonality was responsible.

When the long-year test was applied to the youngest age groups in the Missouri data (n = 3,687), the result supported age incidence. Table 4 shows results for the months of greatest interest with subscripts indicating each month’s position in the long year. The winter months show a season of birth effect only when they occur in the early part of the long year. For example, the first February of the year (Feb₁) shows an apparent season of birth effect, but the second February (Feb₂) does not. Note that the result for December also supports age incidence. This result is included because of the common belief that season of birth effects occur in that month. Actually, December effects are not common, as I will demonstrate later.

In general, Table 4 indicates that when the winter months occur in the middle of the year, the season of birth effect disappears. I can think of no explanation for this result other than age incidence.

Southern Hemisphere Studies. A convincing argument for seasonality comes from studies in the Southern Hemisphere where the excess in schizophrenic births is reported to shift 6 months to coincide with their winter. These studies are commonly cited as if they provided a unitary mass of evidence, and they are considered the single strongest source of support for the seasonality hypothesis. Closer inspection reveals otherwise.

For example, Parker and Nielson (1976) initially failed to find a winter excess in schizophrenic births in New South Wales. When they analyzed their data by sex, however, they found that schizophrenic births were seasonal, but only among females. The agent that affected females in New South Wales appears to have operated in...
reverse in Australia where Jones and Frei (1979) found a seasonal excess limited to males. Another Australian study (Syme and Illingworth 1978) failed to resolve the issue. Those authors found an enormous excess of male schizophrenic patients born in May and a correspondingly large deficit of female schizophrenic patients born in June. A large excess of female schizophrenic births was found in September. In every case, the excesses and deficits were confined to a single month; no trends were found in adjoining months. It is difficult to imagine a causal variable that behaves so circumspectively.

The two other Southern Hemisphere studies are even less encouraging. Krupinski et al. (1976) found no winter excess in Australia, but they did report a nonsignificant trend toward an excess of births in two new seasons: summer and autumn. Similarly, Dalen (1975) failed to find a seasonal excess in South African schizophrenic patients. However, after dividing his data by sex, length of hospitalization, and decade of birth, he found a trend toward a May–October excess among schizophrenic patients hospitalized less than 3 years. This trend reached statistical significance only in the group born between 1940 and 1949 where a May–July excess was found for female patients and an October excess for males. Additionally, the number of schizophrenic patients born in September was unaccountably smaller than expected for both sexes. These latter results were based on data for 530 schizophrenic patients out of the original sample of 2,947.

Dalen’s South African study illustrates two problems that run throughout Southern Hemisphere studies. First, in many instances, small samples make it difficult to be confident of the results. Second, in both hemispheres, a large number of statistical tests are routinely conducted; as many as 40 tests have been attempted in some studies, with as few as 2 of them being found statistically significant and taken as indicative of seasonality. It is not difficult to see that under such conditions, results of one type or another will be found. If those results formed a consistent pattern, we might have something of interest, but the fact is that they do not.

Parker and Balza (1977) are sometimes cited as supporting Southern Hemisphere results. For example, Bradbury and Miller’s (1985) review of the Southern Hemisphere literature reported it as the only study to find a statistically significant season of birth effect. As it happens, however, Parker and Balza conducted their study in the Philippines, which is located on the Equator, not in the Southern Hemisphere. Not only does that area have no true winter (the yearly temperature varies from 25 to 27 °C, i.e., 77 to 81 °F), but in addition, the season of birth effect was not reversed there. In fact, Parker and Balza found the same January–March effect commonly reported in the Northern Hemisphere. Thus, not only was this not a Southern Hemisphere study, but the Southern Hemisphere season of birth effect was not found.

These studies represent the entire basis for claiming a winter reversal in the Southern Hemisphere. On inspection, they do not show a strong reversal of Northern Hemisphere findings. At best they show that when data are subdivided by sex and decade of birth, significant results are found in some studies in some seasons. However, because the pattern of results is not very consistent and because that pattern changes dramatically from study to study, a reasonable alternative interpretation is that the results are due to overanalysis. Despite the spotty nature of the Southern Hemisphere data, they continue to be regarded as strong evidence for the existence of seasonality in schizophrenia. The foregoing review shows that regard to be unfounded.

They also reported a large December excess which, as I will show later, is not a common finding in Northern Hemisphere studies.

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<th>Table 4. A long-year test for seasonality</th>
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Note.—Subscripts refer to each month’s position in the long year.

$^{1}p < .05.$
The Case for December. Several early studies of seasonality found an excess of schizophrenic births in December. For example, Bradbury and Miller's (1985) review of the literature lists December excesses in four of seven studies conducted before 1960 (Tramer 1929; de Sauvage Nolting 1934; Petersen 1934; Pile 1951). Because people born in that month are the youngest members of the calendar year, the finding cannot be attributed to age incidence and, therefore, it is sometimes cited as evidence for seasonality. The results of early seasonal studies are, however, considered inaccurate because of contaminated samples, controls, or analyses (Bradbury and Miller 1985). For example, about one-half of Tramer's original sample were not schizophrenic births; Pile's (1951) data were grouped into seasons making analyses of individual months impossible. Moreover, Pile found no seasonal effects in his data; the result attributed to his study came from a reanalysis by Barry and Barry (1969) based on a single comparison of grouped winter months with grouped summer months.

When improved designs were introduced in the 1960's, December results dropped sharply. In fact, the dozen or so studies published between Pile (1951) and Diebold (1975) contain no significant December excesses. This sudden disappearance would have posed a serious problem for the seasonal hypothesis were it not for the fact that it happened in an era in which the December result was already believed to have been firmly established. As it was, the absence went unnoticed, and belief in its frequency was unaffected.

Today, December excesses continue to be cited as evidence for seasonality. Because there is no agreement about what constitutes a December result, these citations sometimes include studies that reported excesses only for grouped winter months, studies with a significant overall result and any December excess no matter how small, and occasionally even a study with no December excess whatsoever. None of these practices are common, but the fact that they occur at all suggests that any criterion used to assess the December issue may meet with disagreement.

In the past few years, however, two reviews of the literature (Bradbury and Miller 1985; Boyd et al. 1986) have contained tabulated results for a large number of studies. Because both articles conclude that the evidence from those studies supports seasonality, I will use their criteria instead of my own to judge the frequency of significant December results.

Bradbury and Miller's (1985) careful review cites December results in 5 of 30 modern studies in the Northern Hemisphere. This box score approach is admittedly crude—for example, it combines studies of individual months with those of groups, it includes multiple reports from one population, and so on—but even so, it is far smaller than the same count of significant January, February, or March results (17, 15, and 13, respectively). The Boyd et al. (1986) box score is even smaller, with only 2 December excesses in 23 modern Northern Hemisphere studies. This ratio is comparable to the finding of Torrey et al. (1977) of 3 December results in 19 original analyses of hospital data from different States.

The December evidence is so thin that it is likely that it would have been interpreted as chance were it not for the foundation laid by the seasonal hypothesis that it in turn supports. In fairness, it should be noted that although the December results are too small to support seasonality, they are also too small to support age incidence (which, you may recall, predicts a December deficit). The explanation for the apparent neutrality of that month is complex enough to require an article of its own, but having learned to leave no seasonal stone unturned, I will address it briefly.

Many sources of incidence data (especially hospital admissions) are not collected over a January–December year, but rather over some other 12-month period—say, July–June. Data collected in that way resemble the truncated data of a displacement study. The effect on the analysis, however, will not be the clean shift of the displacement test because expected values are not calculated over birth years that have been similarly truncated. A variation of this type of error occurs in the Watson et al. (1982) and (1984) studies. In both studies, the schizophrenic sample was based on people born during a standard January–December year, but expected values were calculated using displaced birth years (July–June in the 1982 study, August–July in the 1984 study). Thus, for example, in the 1982 study, people born in January–June of the oldest birth year contributed to the observed number of schizophrenic patients, but not to the expected number, and the same is true of people born in July–December of the youngest birth year. Given the rise in birth rates over this century, this procedure caused expected values for January–June to be larger than they should have been, and it caused expected values for July–December to be smaller. The effect of this error is not always predictable, but it usu-
ally creates the backward drift of age incidence effects seen in the Watson et al. studies. For example, in the 1982 study, the shift increased the observed/expected ratio for December by about 2 percent. This is a small change, but the case for December is built on just such small differences. Again, in fairness to the Watson et al. (1982) study, it should be mentioned that even without this truncation error, their December data contained a nonsignificant excess. On the other hand, even with the error, the excess was not large enough to reach statistical significance, and the direction and the magnitude of the effect correspond to the predictions of age incidence.

In pointing out the influence of truncation errors on such seasonal studies, I do not mean to detract from the efforts of Watson et al. The displacement test is new and, with new procedures, it is not always possible to forsee all of the precautions needed to apply it correctly. In fact, there are many other potentially dangerous variables whose influence has not yet been assessed. The need for such assessment is, however, paramount because almost every error that can occur in a displacement study can also occur by accident in a traditional study. Potential influences include the number of birth years in the study, the increased availability of psychiatric hospital beds during this century, monthly variations in hospital admissions, the rate at which birth rates change relative to incidence rates for a given period, whether the data represent incidence or prevalence, increases in life expectancy, decreases in infant mortality, and unsuspected differences in sample and control group birth patterns. (For example, the schizophrenic patients in both Watson et al. samples came entirely from VA hospital discharges, while expected values came from State birth rates. Differences between VA and State birth patterns might occur for many reasons—for example, because of age incidence, a mid-year military emergency might raise conscription rates among people born in that month of the year.)

There are more potential influences of this type than I care to mention, and their effects on seasonal studies are unknown. Until those effects can be evaluated, it would be wise to construct a standard set of birth and observation years to be followed as closely as possible in all seasonal studies. In particular, the practice of reporting two studies of the same data using a July–June displacement in one and an August–July displacement in the other, or of using different birth years as a comparison group, is a deadly invitation for type II errors. More generally, a study that departs from normal procedures and discovers an abnormal result should be interpreted cautiously.

**Conclusion**

Overall, the evidence suggests that the season of birth effect is a superfluous explanation for results reported in seasonal studies of schizophrenia. We have seen that age incidence occurs in diseases whose risk changes over time, and that schizophrenia is such a disease. In addition, age incidence accounts for the results of the displacement and long-year tests, it explains the failure to find season of birth effects in old schizophrenic patients, it explains why seasonal effects were found when those schizophrenic patients were young, and it predicts the otherwise unusual excess of deaths among people born in winter that was found in the Texas mortality data and several other results. The season of birth hypothesis neither predicts nor explains any of these peculiarities. One may draw whatever conclusions seem reasonable from these results, but the history of such matters suggests that the simplest explanation that accounts for all of the data is usually the most profitable. In the present case, age incidence seems the most parsimonious of the two hypotheses, and the burden of proof should now shift to those who favor seasonality.

**References**


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Shur, E. Season of birth in high and low genetic risk schizophrenics.


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Appendix

Overview

The purpose of this appendix is to determine whether age incidence can produce spurious season of birth effects. To that end, I will explore two models based on incidence data from equations rather than from actual observations. Because these equations contain no seasonal variable, it is a statistically significant result that false season of birth effects can occur when seasonality is not present.

The approach used to generate the data for the studies was as follows. In a standard season of birth study, incidences among people born in successive years (e.g., 1900-50) are recorded over one or more calendar years (e.g., 1965). Now if the risk period for a disease begins at, say, age 15, people born in January 1950 will not have entered the risk period at the start of the study, and therefore they will have been at risk for 12 months. If the rate at which new cases arise at any given set of data.

Similarly, the cumulative incidence for people born in November will be \( f(t)dt \), and, in general, the total incidence for people born in any month will be \( \int f(t)dt \), where \( M \) is a number assigned to each month such that January = 12, February = 11 ... December = 1; that is, \( M \) represents age differences among people born in different months of the same year.

In a population of cases, the proportion born in a particular month will be:

\[
\int_0^{12X+M} f(t)dt
\]

\[
c = \frac{12 \int_0^{12X+M} f(t)dt}{M = 1 \sum_{M=1}^{12} \int_0^{12X+M} f(t)dt}
\]

It follows that samples of size \( N \), drawn from that population of cases, will contain, on average, about \( Nc \) cases born in the month of interest. In a standard season of birth study, however, an investigator would expect the sample to contain, on average, about \( Np \) cases, where \( p \) is the proportion of the general population born in the month under study. For simplicity, we will assume that in all analyses \( p = 1/12 \). Thus, for example, if \( N = 4,800 \), \( c = 1/10 \), and \( p = 1/12 \), the average number of cases observed in a sample will be \((1/12)(4,800) = 480\) but the investigator will expect an average of \((1/10)(4,800) = 400\). As will be shown, knowing \( c, p, \) and \( N \), we can calculate the probability of finding a false season of birth effect in any given set of data.

Models

Suppose that the instantaneous incidence of a disease changes linearly over time such that \( \frac{df}{dt} = rt \). This equation can be rewritten as \( dl = rt \) and integrating both sides we have \( l = \int_0^l rt \) dt. Thus,

\[
\int 12X + M \int_0 x dx
\]

\[
c = \frac{12 \int_0^{12X+M} f(t)dt}{M = 1 \sum_{M=1}^{12} \int_0^{12X+M} f(t)dt}
\]

where \( X \) is the number of years that the oldest group in the sample was at risk at the start of the study. If we let \( X = 10 \) and \( M = 12 \), we find that in a sample of \( N = 4,800 \) the observed number of cases among people born in January will, on average, be \( Nc = 0.09(4,800) = 436.8 \). Within any given sample, however, the observed number of cases will vary. What we want to know is the probability that \( Nc \) will exceed \( Np \) by a margin large enough to produce a statistically significant \( \chi^2 \) test. That probability can be determined via the non-central \( \chi^2 \) distribution (Patnaik 1949). To use that distribution, we first compute \( \chi^2 \) in the standard way, using \( Nc \) as the observed value for each cell and \( Np \) as the expected value. The resulting \( \chi^2 \) and its degrees of freedom are the two parameters needed for entering a table of the noncentral \( \chi^2 \) distribution (e.g., Haynam et al. 1970). The tabled value is the probability that \( Nc \) will exceed \( Np \) by a margin large enough to produce statistical significance. For example, in our present analysis we have two cells: one containing cases born in January for which \( Nc = 436.8 \) and \( Np = 400 \); and one containing cases born in all other months for which \( Nc = 4,363.2 \) and \( Np = 4,400 \). The overall
\( \chi^2 \) is 3.69, \( df = 1 \), and the Haynam et al. (1970) table of the noncentral \( \chi^2 \) tells us that a statistically significant result will be found just under half (48 percent) of the time. When all 12 months in the linear model are considered individually, \( \chi^2 = 14.90 \), \( df = 11 \), and the noncentral \( \chi^2 \) shows that a false season of birth effect will be found about 72 percent of the time.

One of the most striking features of equation 1 is that it does not contain the term that we most expect to see. Specifically, Dalen (1975) believed that age incidence effects were proportional to the rate at which incidence changed with time. That rate appears as the constant, \( r \), in the linear model, but during the course of the analysis it cancels. The point here is not that \( r \) has no influence on age incidence—in fact, in the next model we will see that it is influential—rather, the point is that the properties of age incidence are less straightforward than currently believed, and those properties change across situations.

One unexpected finding that grew from exploration of the linear model is that the design of a season of birth study has a marked effect on the result. If, for example, the study does not include people in their first year at risk, the value of \( c \) in the linear model changes to:

\[
\begin{align*}
\int_{X_1}^{X_2} + M &= \int_{X_1}^{X_2} + M \\
M &= \int_{X_1}^{X_2} + M \\
J &= \int_{X_1}^{X_2} + M \\
72(12X + 12X) + 78
\end{align*}
\]

where \( X_1 \) and \( X_2 \) are the number of years that the youngest and oldest groups in the sample were at risk at the start of the study, and all other variables are as defined previously. Similarly, it can be shown that adding additional observation years (a common practice in season of birth studies) changes the value of \( c \) in a way that intensifies age incidence effects. Thus, small differences in design can produce unexpected differences in results, even when the data are almost identical.

To change the linear model into an exponential model, we use:

\[
I = \int_0^{12X + M} e^{rt} dt; \text{ this also changes the properties of age incidence. Under this exponentially increasing model,}
\]

\[
\begin{align*}
\frac{e^{12X + M}r - 1}{e^{12X + M}[1 - e^{12r}] - 12(1 - e^r)}
\end{align*}
\]

Interpretation of this model parallels that of the linear model with the exception that the variable \( r \) (the rate that incidence changes with time at risk) assumes an importance not seen previously.