Season of Birth Effects in Autism*

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ABSTRACT

This study examined a sample of preschool-age children with autism in an attempt to identify patterns of birth dates that deviated from expected frequencies by month or season. Birth dates of children with autism and those of a non-autistic sibling control group were compared to the number of total live births gathered from U.S. Census data. Analyses included two types of chi-square analyses and a seasonal harmonic trend analysis. Previously unmentioned in the literature is a seasonal effect finding for females within the entire sample, and both a seasonal and monthly effect for children classified as socially Passive by the Wing system. A significant elevation was also found in March within the Boston sub-sample (n = 37). This sample largely comprised low-functioning boys with autism, a finding consistent with previous findings in the literature. Peri-natal complications and early life development of the subjects from the Boston site are detailed.

Analyses of birth season seek to identify deviations from the expected pattern of births with the hope that such deviations will point to etiologic factors. This type of analysis has been conducted in a variety of psychopathological conditions with suspected biological origin. Variations from the usual population birth-date pattern have been reported in autism, schizophrenia, affective disorders, personality disorders, Alzheimer’s Disease, and Parkinson’s Disease (Fossey & Shapiro, 1992).

Central to the purpose of conducting season-of-birth studies is the usefulness of the findings in determining etiology. This has important benefits with regard to diagnosis, research, and clinical treatment of the disorders under investigation. One popular hypothesis suggests that these conditions could be the result of environmental insult to neural development and maturation. This could take place in a developing fetus or neonate in the form of temperature, nutritional deficiency, obstetric complication, or by viral or bacterial infection. An ideal finding is a peak of births that can be readily explained by an environmental factor. If the factor is common to a sizable number of the sample within that peak month, then it represents strong evidence for an environmental cause for the disorder. Links between autism and environmental factors like these have been discovered for cytomegalovirus (Bolton et al., 1992), congenital rubella (Chess, 1977), and pneumonia and bronchiolitis (Tanne, Oda, Asano, & Kawashima, 1988). Unfortunately, collateral evidence of environmental risk factors is scarce and hard to find uniformly across a given sample. The lack of a seasonal finding usually is taken as evidence for a genetic determinant for the disorder.

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Many studies seeking birth effects in autism have been published in recent years. For a good review of the findings, refer to Bolton et al. (1992). In general, the findings conflict. Many studies (Atlas, 1989; Barak et al., 1995; Bartlik, 1981; Gillberg, 1990; Konstantareas, 1986; Mouridsen et al., 1994) uncover peaks in the incidence of March births in their samples. Some of them also implicate the month of August. Speculation in an early study (Konstantareas, 1986) that the effect applied primarily to low-functioning, nonverbal male children with autism has been difficult to replicate or to unequivocably disprove. Another handful of studies refutes the conclusion that children with autism have a disproportionate excess of March birthdays. Tanoue et al. (1988) found peaks in other months (April, May, and June). Other studies find no indications of seasonal trends at all (Bolton et al., 1992; Fombonne, 1989). Weighing all of the findings, one realizes that the mixed evidence does not lead to any easy conclusions at this time.

Critics of this research approach cite these mixed findings as evidence that a seasonal birth effect does not exist. However, it is wise to recognize that there are important differences among the studies that greatly limit their comparability. These limitations include the use of variable diagnostic criteria when sampling. The method of inquiry has been criticized on theoretical grounds (see Bolton et al., 1992 for a review of how the “age-incidence” and “age-prevalence” effects are successfully addressed in season-of-birth research for autism). Criticism of the statistical methodology used to detect trends in birth frequency data is also seen in literature on seasonal birth effects. Goodness-of-fit chi-square tests can be difficult to interpret because of the arbitrary designation of months to season and their lack of power relative to other statistical tests. An alternative method of data analysis is to test birth data against a sinusoidal function (Edwards, 1961; Jones et al., 1987). Bolton et al. (1992) discuss these limitations and point out that any one method of data analysis will likely be insufficient if used alone.

METHOD

The present study analyzed the frequency of birth dates from a large cohort of subjects with idiopathic autism as defined by a recent diagnostic system. It further examined site differences and a select number of theoretically-supported subgroups which were defined using standardized measures. The sample of children with autism was compared to the number of total live births gathered from U.S. Census data and also to a non-autistic sibling control group. Statistical analyses included two types of chi-square analyses and a seasonal harmonic trend analysis (Jones et al., 1987). Significant birth effects were determined only when two redundant analyses confirmed the findings.

Selection of Children with Autism

The sample of children with autism was collected for use in the Autism and Language Disorders Nosology Project, a longitudinal study of autism, language disorder, and mental retardation. Data collection began in 1983 and lasted 8 years. Children were recruited by clinical referral for communication disorders and through participation of schools and programs for children with special needs in several sites across the eastern United States. These included Boston, Massachusetts; Bronx, New York; Cleveland, Ohio; Long Island, New York; and Trenton, New Jersey. The sites provided different numbers of autistic children for the study. These children differed by socioeconomic factors from site to site. All children were screened using the Wing Autistic Disorder Interview Checklist (WADIC, Wing, 1985) to confirm the presence of social impairment, prior to diagnostic interview with psychiatrists. Child psychiatrists completed a comprehensive evaluation that included a DSM-III (American Psychiatric Association, 1980) diagnosis, a DSM-III-R (American Psychiatric Association, 1987) diagnosis, and the completion of a 21-item Social Abnormalities Scale (Rapin, 1996). Children were screened prior to final diagnosis for medical conditions known to produce autistic symptomatology in order to ensure that the participants were representative of idiopathic autism. In addition, every participant underwent a standard neurological examination and parents and teachers were asked to complete questionnaires for information about each child’s behavior and skills at home and in school. For more detailed information on the sampling procedure, please refer to the monograph by Rapin (1996).
Autistic Sample Composition

The sample used in this analysis is based on DSM III-R criteria, as it was the diagnostic system in use during data collection. Because of the low base rate of autism, as many cases as possible were gathered from every site. Therefore, the sample may not be as strictly representative of the autistic population as a randomly selected sample. One participant in the final database did not have birth date information and was excluded from subsequent analyses. The 175 children used in this analysis who formed the autistic group were comprised of 146 boys and 29 girls. At the time of data collection, these children were between 36 and 83 months old. They had no hearing deficits, no gross sensori-motor deficits, no frequent seizures, and were not on high doses of anticonvulsant or psychotropic medications. All subjects had English as their primary language. The children’s birth dates ranged from August 1977 to November 1985. Approximately 43% were high-functioning and 37% were nonverbal as determined by standardized measures detailed below.

Control Participants

A sample of sibling birth data was generated from the family history of the participants included in the sample. Adopted and step-siblings were excluded from this group. The final control sample consisted of 100 full siblings; 51 boys and 48 girls (the gender classification of one sibling control was missing from the database). Another group was created that added 23 half-siblings to form a second control group of 123 children. These were the control groups used for comparison with the autistic sample.

Measures Used

The study provided detailed data on every individual, including family, medical, and developmental history, neurological and medical examination, psychiatric inventory and evaluation, adaptive developmental functioning, cognitive functioning, language development, play development, and socioeconomic status of the family (Rapin, 1996). Nonverbal IQ was measured primarily by the Stanford-Binet Test of Intellectual Ability (Thorndike et al., 1986) Abstract/Visual Reasoning subtest. Because many individuals were untestable using the Stanford-Binet, it was necessary to assess some low-functioning children with the Bayley Scales of Infant Development (Bayley, 1969) Mental Scale which also provided an estimate of nonverbal intelligence. Based on the assumption that both tests used visual/spatial items assessing similar constructs, both sets of scores were converted into comparable nonverbal mental age scores (Rapin, 1996). High-versus low-functioning was distinguished by a cutoff score of 65 nonverbal IQ ratio. This ratio is the nonverbal IQ divided by the child’s chronological age. The nonverbal IQ ratio ranged from 0 (or untestable) to 167.

Communication status was determined by a rating of language by the psychological examiner. In this study, verbal versus nonverbal communication status was defined by those children in the autistic group who had from 1 to 25 utterances or were fully verbal versus those who were mute, could vocalize, or whose speech was unintelligible. This is a gross distinction that is different from language classification used in some previous studies of autism. It is based merely on the presence or absence of language use, rather than its complexity or development. Other factors measured included a rating of social functioning based on a questionnaire developed by Wing (1985). The questionnaire yielded ratings of interaction in three groups. These were defined as Aloof, Passive, and Active-but-Odd. Social rating information was available for 131 of the 175 individuals included in this study. Socioeconomic status was measured using the Hollingshead (1975) SES classification scale.

Data Analysis

First, the frequencies of birth dates were aggregated across years and sites in order to test the entire sample for the presence of seasonal effects. Seasons were delineated by groups of months. Winter was defined as January through March; spring was April through June; summer was July through September; and fall was October through December. The number of live births nationally was aggregated by month from the U.S. Census for 1977 to 1985. These figures were averaged to create a total-per-month of children born in each month. Using this data, an expected value was calculated for the number of autistic births per month in this time frame. Chi-square analyses used critical values of the chi-square distribution determined by Pearson (1958) to assess statistically significant difference. In order to have a redundant statistical procedure, the aggregated sample was then analyzed using a program provided by Jones et al. (1987). This analysis models the data as a short Fourier series including up to three harmonics or sine peaks. This program compares the autism birth data to the Census control group using a goodness-of-fit index of the data fitted to a sine wave pattern. This analysis procedure was also applied to certain selected subgroups of the data – notably a distinction between genders, high versus
low functioning, verbal versus nonverbal, and levels of social interaction style.

Because it was suspected that an aggregated sample might not reflect site-to-site differences, individual sites were also tested. As the expected values derived from U.S. birth data was no longer applicable to individual sites, another form of chi-square analysis was employed that assessed whether the pattern of births differed from a random – or equal – distribution of births. As the Cleveland and Long Island sites contributed a total of only 8 cases together, they were excluded from the individual site analyses. The site data from Boston, the Bronx, and Trenton were also tested using the periodic regression program. Again, however, individual sites used an equal distribution of expected births rather than U.S. Census data for detection of seasonal trends.

Subsequent descriptive post hoc analyses were then performed on groups that showed statistically significant effects in order to describe these participants’ characteristics.

RESULTS

Significance Testing
There was no seasonal or monthly birth effect found for the aggregated sample. No significant effects were found for high-functioning, low-functioning, verbal, nonverbal, or male subgroups. However, the seasonal birth pattern for females in the total sample was found to be significantly different than expected by population value \( \chi^2(3, N = 29) = 8.10, p < .05 \) (Figure 1). This effect was due to the increased frequency of female births seen in winter.

A significant monthly effect was also found for one of the groups formed by Wing’s (1985) social functioning classification. The effect was found for the Passive social interaction, \( \chi^2(11, N = 37) = 23.95, p < .025 \). The frequency distribution of seasons of birth indicates that there is an excess of births of Passive children with autism in winter, with a relative deficit in spring and summer. In addition, this group of Passive children with autism displayed a significant monthly birth effect (p = .018). The results of this analysis are displayed in Figure 2. This effect can be attributed to peaks of births in the months of March and November.

For children from the Boston, MA site there was a peak in the number of autistic births for the month of March. The equal distribution comparison was significant, \( \chi^2(11, N = 37) = 21.05 \ p < .05 \). This suggests that the peak of births seen in March (Figure 3) is significantly

![Graph](image-url)
different than expected in the general population.

Additionally, periodic regression indicated an effect for the Trenton, NJ site. The Trenton site displays a peak around December with an apparent trough in early Spring months--March, April, and May. However, because this finding was not confirmed by both chi-squares, further analyses were not pursued.

In order to control for the arbitrary assignment of months to seasons, a statistical criticism of seasonal effect research, the seasons were shifted forward and also backward by one month. The data were re-analyzed using these two new seasonal groupings. No differences in the findings were discovered. All analyses were re-done using periodic regression, which produced the same effects as the chi-square analyses.

Analysis of the full-sibling control group produced no significant seasonal effects as compared to an equal distribution of births in the population. This was true for month of birth as well as season of birth. Seasonal criteria were shifted forward and back as was done in the analysis of the autistic group without producing a statistically significant effect. No effects were seen for the full- and half-sibling control groups when compared to the autistic group.

Post Hoc Analyses
As there were no significant main effect findings for the aggregated sample, no further analyses were conducted for this group. The subgroup analyses from the aggregated sample are detailed above, which leaves only the site-by-site data for further descriptive analysis. The group of individuals from Boston, MA who comprised the peak (those who were born in the month of March) was examined. This group’s characteristics are contrasted with the aggregated sample in Table 1. As can be seen by these comparisons, this subgroup of March-born subjects have markedly different characteristics from the rest of the sample. Although both groups are primarily male, the Boston-March subgroup comprises more low-functioning individuals, evenly split on their verbal status. Two-thirds of them also have a Passive social interaction style using Wing’s (1985) criteria. A review of peri-natal
Table 1. Comparison of March-born Autistic Subjects from Boston with the Remainder of Aggregated Sample.

<table>
<thead>
<tr>
<th>Subject Characteristics</th>
<th>Boston March-Born (n = 10)</th>
<th>Remainder of Aggregated Sample (n = 166)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>90%</td>
<td>83%</td>
</tr>
<tr>
<td>Verbal</td>
<td>50%</td>
<td>37%</td>
</tr>
<tr>
<td>Low-Functioning (Nonverbal IQ ratio &lt; 65)</td>
<td>80%</td>
<td>56%</td>
</tr>
<tr>
<td>Socially Passive (Wing, 1985)</td>
<td>70%</td>
<td>27%</td>
</tr>
<tr>
<td>Perinatal Complications</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Born head first</td>
<td>50%</td>
<td>67%</td>
</tr>
<tr>
<td>Born with umbilical cord around neck</td>
<td>20%</td>
<td>6%</td>
</tr>
<tr>
<td>Born stiff upon delivery</td>
<td>50%</td>
<td>3%</td>
</tr>
<tr>
<td>Early Life History</td>
<td></td>
<td></td>
</tr>
<tr>
<td>History of encephalitis</td>
<td>10%</td>
<td>1%</td>
</tr>
<tr>
<td>Generalized seizures</td>
<td>30%</td>
<td>7%</td>
</tr>
<tr>
<td>Infantile spasms</td>
<td>20%</td>
<td>4%</td>
</tr>
<tr>
<td>Difficulty falling asleep</td>
<td>70%</td>
<td>30%</td>
</tr>
<tr>
<td>Difficulty rising</td>
<td>70%</td>
<td>28%</td>
</tr>
</tbody>
</table>

Fig. 3. Proportion of birth dates per month in the Boston sample.
history reveals that they although they had a similar rate of head-first births, the Boston-March group had greater incidence of other obstetric complications. Early life history reveals increased prevalence of viral insult and manifestation of neurological signs in the early development of the Boston-March subgroup that also reach statistical significance.

**SUMMARY AND DISCUSSION**

The current findings support the existence of a season-of-birth effect for children with autistic symptoms. Because 13 different analyses were performed on this sample, one has to consider the possibility that the significant findings in this paper are merely due to random effects. However, the findings point to both winter and to the month of March as the peak times of autistic births. This concordance with other studies (Atlas, 1989; Barak et al., 1995; Bartlik, 1981; Gillberg, 1990; Konstantareas, 1986; Mouridsen et al., 1994) also strongly suggests that this is not a random finding. We assert that the finding represents evidence in support of seasonal effects for children with autism, at least as defined by DSM-III-R criteria. As the definition of Autistic Disorder has been refined (i.e., DSM-IV; American Psychiatric Association, 1994), it is not possible to generalize these findings to DSM-IV defined samples given the differences in composition between groups defined by each of the two systems (Rutter & Schopler, 1992; Volkmar, Cicchetti, Bregman, & Cohen, 1992; Waterhouse et al., 1996). However, these findings can be compared directly to other studies using the same diagnostic system. Only one other study in the literature on seasonal effects in autism has used DSM III-R criteria for diagnosis. Gillberg (1990) found a peak of March births in his sample that did not reach statistical significance ($p < .10$). This autistic group was primarily male, but split roughly equally between high and low functioning. This further supports the veracity of the current findings.

The study failed to detect a seasonal or monthly birth effect for the aggregated sample or the control group. Subgroup analyses do show some interesting findings that have not been reported previously. One such finding is a significant seasonal effect for females for the aggregated sample. The predominant communication status of the girls who comprised the peak was mixed; approximately 36% of them have some preserved language capacity, ranging from a very limited vocabulary to intact language. Another finding not seen previously in the literature was both significant seasonal and monthly birth effects for children with autism classified as Passive by Wing’s (1985) social functioning scale. Although it is too early to conclude, this finding may suggest that passive interaction style in middle childhood might be indicative of an environmentally-mediated etiology for certain autistic children.

Specifically, it was found that there was an over-representation of March births for the Boston, MA site. The finding suggests that environmental factors specific to Boston during the years of data collection may play a greater role in the etiology of autistic symptoms for these children than for those gathered from other sites. However, the consistency with many previous findings lends support to the position that the etiological factor likely is not specific to the geographic location or time of data collection, but rather is a general factor common to various regions across years. An analysis of the mostly male participants from the Boston site reveals that over 70% have a Nonverbal IQ ratio below 65 and 58% are nonverbal. This reflects a trend similar to Konstantareas’ 1986 conclusion that the seasonal effects in her sample were due to low-functioning, nonverbal boys. Socioeconomic indicators of the families of these children show that they come from middle to lower-middle class environments.

This research identifies factors as possibly relating to a suspected environmentally-related autistic disorder. Several of these, stiffness upon delivery, generalized seizures and infantile spasms, and sleep disturbance might be further investigated as early risk signs. Another factor common to these children – the early life history of encephalitis – is less easy to understand. An encephalitis epidemic that affected young children with weak immune systems might plausibly
explain later development of autistic symptoms. However, it does not readily explain the concordance of March birthdays. Despite these perplexing questions, we are able to emerge from this study with a loosely-sketched possible phenotype that describes this suspected environmental-epidemiology subgroup. They appear to be low-functioning and socially-passive, which is in accord with previous findings. Second, these results support the need for future study of autistic birth dates with detailed pre- and peri-natal information that may lead to discovering specific risk-factors for autism in select groups.

REFERENCES


