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# **Season of Birth in Psychiatry**

A Review

## **Key Words**

Season Birth Psychiatry

## Abstract

Numerous studies suggest that seasonal birth may play a pathogenic role in the development of mental disorders. A birth excess of 10% during winter and spring has been shown in schizophrenia. The few studies carried out on affective disorders revealed a significant increase of births in the first quarter of the year in bipolar disorders and major depressive disorder. Subjects with seasonal affective disorder show a peak of births in May. Data on personality, eating and 'neurotic' disorders are less consistent. At the moment there are no data in the literature about anxiety disorders.

#### Introduction

It has been reported that season of birth can influence human physiological developmental processes – such as birth weight and age at menarche [1]. Some studies have also shown that the month of birth may be a predisposing factor for several diseases, raising the possibility that very early environmental influences are involved. A seasonal pattern of birth has been suggested for glaucoma [2], allergic sensitization [3], asthma (August–January) [4], allergic rhinitis (November–May) [4], menstrual disorders [5], acute leukemia in infancy (winter) [6] and laterality of breast cancer [7].

The significant association between season of birth and the occurrence of some diseases suggests a periodicity of an environmental etiologic agent, perhaps acting in concert with endogenous rhythmicities in susceptibility to that agent. A season of birth tendency has been shown also for several psychiatric disorders (table 1). The seasonally mediated environmental influence is assumed to result in some kind of brain damage, which in turn has an influence upon the illness risks. The brain damage could occur prenatally, perinatally or postnatally, in accordance with the well-known central nervous system (CNS) plasticity in the first stages of life.

## Schizophrenia

The first author to report on season of birth in schizophrenia appears to have been Tramer (1929), who among 2,100 cases of schizophrenia found an excess of births from December to March [8].

Subsequently more than 100 studies have been published on this topic, showing an excess of some 10% in the

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Table 1. Season of birth in psychiatric and neurological illness

Diagnoses	Studies	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
Psychiatric disorders													
Schizophrenia	>100		_										
Bipolar disorder	>10					_							
Seasonal affective disorder	1				_								
Autistic disorder	10									_			
Schizoaffective disorder	2				_								
Neuroses	6						-						
Personality disorders	2						_						
Alcohol abuse	2									_			
Eating disorder	. 1						-						
Neurological illness													
Alzheimer's disease	1												
Parkinson's disease	1												
Motor neuron disease	1												
Multiple sclerosis	2									=			
Down's syndrome	5							-					
Mental retardation	1									-			

winter or spring months and an equal deficit in the summer or autumn months [for a review see 9–15].

The significant excess of births of schizophrenic infants between December and May in the northern hemisphere seems to correspond to an equal excess between June and October in the southern hemisphere [8, 16].

It has been proposed that infants born in the winter/spring months are more likely to be exposed to adverse environmental or obstetrical complications [17], extreme temperature [18], seasonal variation of nutritional practices [19], vitamin deficiencies [20] and toxic insecticides [21]. The most plausible hypothesis however is that winter-born children are prone to perinatal bacterial and viral (or retroviral) infections, which may damage the CNS and facilitate the manifestation of a functional psychosis in those genetically at risk [17, 22–24].

As confirmation of this hypothesis, the seasonal incidence of measles and cytomegalovirus infections, capable of causing fetal damage, is correlated with seasonal birth peaks [14, 25–27]. A study indicating that there are more urban infants born during the winter supports the viral hypothesis, as one would expect a greater risk of contracting an infection in a more populated area [14, 28]. Finally, elevated winter birth rates for schizophrenics have been observed in Minnesota in the years preceded by a summer with higher than average rates of bacterial and viral infections [29].

In accordance with the viral hypothesis, winter-born patients with schizophrenia have lower skin conductance levels and fewer skin conductance responses, while non-winter-born subjects do not differ from normal ones [30]. Some authors hypothesize that a viral infection, or some other perinatal complication associated with winter and early spring births, leads to temporal lobe damage and consequent dysregulation of electrodermal activity in patients with schizophrenia.

It has been reported that schizophrenics at low genetic risk are more likely than those at high genetic risk to be born in winter/spring; hence the winter increase in the birth rate might be confined to the former group [14, 31, 32]. The patients without a positive family history, on the other hand, are more likely to have had birth complications [33] and show more evidence of ventricular and cortical sulcal enlargement as assessed by computerized tomography [33–35]. There is a correlation therefore between low genetic risk, perinatal infections, ventricular enlargement and the likelihood of winter birth. The winter-birth patients, moreover, appear to have a prevalence of negative symptoms [36] and an early onset of the disease [37].

The pattern of schizophrenic births might be explained by the lower socioeconomic status of patients, given that the low social classes have a higher risk of pregnancy complications and deviant reproductive behavior [9]. The season of birth varies with social class [38]; the relevant factor nevertheless is not the social class of the patients at the time of their admission, but of their parents at the time of their birth [39], and the distribution of parental social class in schizophrenia does not differ from that of the general population [40].

The procreational habits hypothesis is based on the assumption that some of the parents of schizophrenic, bipolar and mentally retarded patients are inhibited in heterosexual mating behavior: they have a higher threshold in partner-seeking behavior, which is overcome more easily in spring/summer and thus they conceive more often in these months [40–45]. Nevertheless there is little evidence of a winter excess of births among the siblings of schizophrenics [29, 46, 47]. The factors responsible for the procreational habits hypothesis have not yet been identified [48].

Dalen [17] did not find any sex difference in the season of birth distribution, even though on the hypothesis of constitutional damage we might suppose that males, being generally more vulnerable to early adverse environmental influences, might show a greater excess of winter births than females. Recently, a study carried out in the Netherlands confirmed the lack of differences in the season of birth for men and women [13].

No significant differences in birth pattern have been found between the schizophrenic subtypes (paranoid, simple, catatonic, hebephrenic and chronic undifferentiated) [22].

## **Affective Disorders**

Many studies have been published on the birth seasonality of patients with bipolar disorder. Despite methodological problems, they consistently show a winter/spring excess of births and a deficit in the months of September to November [9, 11, 14, 15, 45, 49].

The spring birth peak seems to be smaller in females than in males [45], while the risk of depressive illness is significantly decreased for women born in autumn (August-November), but not for men [13].

As for schizophrenia, the procreational habits hypothesis maintains that the patients' parents have traits of sexual behavior leading to an abnormal seasonal distribution in the births of their children [8].

Selten et al. [13] found a significantly decreased risk of affective illness for subjects born in autumn (August–November). Parents of patients might be more vulnerable to winter depression than the general population. If winter depression in these parents occurs somewhat earlier

than in the general population, seasonal fluctuations in their sexual behavior might explain these findings [13].

As for schizophrenia, the most plausible hypothesis is that winter-born children are prone to nutritional deficiencies or infections, which may damage the constitution of those genetically at risk [14, 45].

The finding that there was a significant birth excess in the first quarter of the year for patients diagnosed psychotic depression compared with those diagnosed neurotic depression is taken as evidence for a real difference between these two types of depressive illness [45].

Moreover the bipolar patients with mood-incongruent psychotic features appear to have an April peak of births that is more significant than for patients without those symptoms [50].

Nevertheless a few authors have found no significant differences in birth seasonality between manic-depressive patients and the general population [29, 51, 52].

Fossey and Shapiro [53] have suggested that some patients may have been misdiagnosed in previous studies. Tsuang et al. [54] reported that 13% of previously diagnosed bipolar patients and 3% of schizophrenics were later rediagnosed as schizophrenic and bipolar, respectively.

In the seasonal affective disorder (SAD) a seasonal birth pattern has also been found, with a peak in May and a decrease from August to December [55]. The authors hypothesize that parents of patients might be at risk of having SAD; thus winter depressive episodes might cause seasonal fluctuations in their sexual behavior.

# **Autism and Other Types of Childhood Psychoses**

Since the early 1980s, several studies have addressed the question of a connection between season of birth and autistic disorder. Bartlik [56] found a significantly higher rate of births of autistic children in March and August compared with the distribution of births in the general population. The results of a second study, by Konstantareas et al. [57] also indicated a significant increase of births of autistic children during the spring and summer, especially in March, May and June, and an autumn/winter deficit. Attempts to isolate specific characteristics associated with this pattern have identified a subgroup of nonverbal, lower functioning children. There is evidence that highly susceptible fetuses (as a result of prenatal damage), which are due to be born during the high-risk winter months but die just before or at birth, may produce the deficit in autistic births at this time of year [58]. However, the spring/summer peak may be a result of a group who survive the period of prenatal risk and are born alive, but more severely affected, during the lower risk summer months [57]. Tanoue et al. [59] examined both season and year of birth in an epidemiological sample of native-born children in Japan and found an increased rate of births in the spring quarter (April, May, June).

A different seasonal pattern of births in autistic children emerged from two subsequent studies. Fombonne [60] reported that winter birth was a risk factor for childhood psychosis. His finding, like that of Konstantareas, suggested that the association was strongest in the subsample of low-functioning autistic people. The same was found by Garalda and Watt [61] for adolescent psychosis, suggesting a common etiological link between adult-onset and earlier-onset psychoses.

Gilberg [62] compared people with cryptogenic autism to 'autistic-like' and age-matched children. A higher rate of March births was found in the autistic group. In the study of Bolton et al. [63], a UK sample of autistic patients, compared with the general population, showed significant deviations from the expected monthly birth rates with peaks in December/January, June/July and October.

The study of Mouridsen et al. [64] encompassed all children with infantile autism and other childhood psychoses born in Denmark between 1946 and 1980; an excess of March-born boys with infantile autism was found. There was a peak in November for boys with autistic-like disorders, while no seasonality was observed for a borderline sample.

Barak et al. [65] compared data for autistic patients registered in Israel during the years 1964–1986 with data on the monthly distribution of live births in Israel for the corresponding period; they observed a significant increase of autistic children born in March and August.

Even though carried out in different climatic areas, most studies confirm the earlier findings that March and August births are a risk factor for the development of autistic disorder. This raises several questions about gestation at certain times of the year and exposure to damaging factors present during certain seasons – even in different continents.

Winter is associated with viral infections and with nutritional and vitamin deficiencies, which may have an adverse effect on a group of highly vulnerable fetuses, either during intrauterine life or around the time of birth or during the first months of life. Several studies have revealed the higher incidence of autistic conditions following viral exposure, or clinical illness, particularly rubella [66, 67].

#### Schizoaffective Disorder

An excess of births in the first quarter of the year has also been reported for patients with schizoaffective disorder [50]. This is not surprising since this illness belongs to the schizophrenic spectrum.

#### Neurosis

Early studies of 'neurotics' revealed an excess of births between January and May [9, 17]. Hare [16] found two birth peaks, in June and August. Hafner et al. [45] reported an excess of births from March to May and a deficit from June to September for male neurotic patients, but not for females. The procreational habits hypothesis maintains that the summer months could enable an otherwise inhibited population to overcome the social barriers of their handicap and to take advantage of the greater opportunity for social contacts. Nevertheless all these studies suffer from several methodological problems, including unreliable diagnoses.

## **Personality Disorders**

The first study on season of birth in personality disorders reported a birth peak in June and August [16].

However Boyd et al. [11] found an excess of births from March to May and a deficit from June to September.

Hafner et al. [45] confirmed this pattern and reported a deficit from June to September for male patients with personality disorders, but not for females. The procreational habits hypothesis has also been proposed for personality disorders.

#### **Alcohol Abuse**

The few studies available on alcoholic patients report an excess of births from March to July [15, 29].

#### **Eating Disorders**

Nielsen [68] found in the Danish population that anorexic patients born after 1963 had a birth excess from February to May, just a little later than the birth excess found in psychoses in the northern hemisphere. No season of birth variation was found in those born before 1963. In 1996, Rezaul et al. [69] found a season of birth variation in a sample of patients with eating disorders, with a peak season of birth occurring in May. However, among patients born after 1963 the peak season of birth is in March, which is significantly different from the season of birth cycle of the general population. This finding may imply links between the etiology of earlier-onset eating disorders and the psychoses; similar first-quarter birth peaks have been found in schizophrenic and affective psychoses, i.e. in January and February. In contrast, for the neuroses and personality disorders, birth peaks have been found in June and August, similar to the June birth peak found in this study for later-onset eating disorders, which though was not significantly different from the season of birth peaks expected from the general population.

On the other hand it may be that a factor specific to eating disorders is not responsible for this season of birth finding. Genetic epidemiology studies find that families in which anorexic probands also exhibit clinically prominent features of affective disorder resemble pure affective disorder families more than do families of nondepressed anorexics, as far as loading for major affective disorder is concerned [70]. The increased risk of affective disorder among relatives of anorexic probands is accounted for largely by the subset of anorexics with coexisting depressive disorder [70], and the winter season of birth effect found here may be accounted for by this subset of anorexics. If it was possible to identify and remove this subset, the majority of eating disorder patients with no coexisting depressive disorder might not show this winter season of birth effect. Moreover, the evidence that 5-HT is involved in a variety of psychiatric disorders and that the processes involved may be influenced by a photoperiod suggests that such seasonal patterns of birth in several psychiatric conditions share a common neurophysiological substrate [12].

The season of birth peak in the early months of the year could differentiate anorexics from bulimics.

#### **Neurological Diseases**

A seasonal pattern of births has been reported also in several neurological disorders.

Philpot et al. [71] demonstrated a birth peak in the first quarter of the year for patients with Alzheimer's disease without a family history of dementia. The environmental factors that may be implicated are, as in schizophrenia, infectious agents.

A seasonal birth pattern, with a peak in May and a trough in July, exists also in idiopathic Parkinson's disease [72].

An increase of births during the spring and summer months and a decrease in winter has been reported for motor neuron disease [73].

Templer et al. [74] reported that Danish patients with multiple sclerosis were born more often in March, April, May and June, and less often in the other months. The authors suggested that infection has a role in the etiology of the disease. However, the monthly birth distribution of multiple sclerosis patients did not differ significantly from that of the general population in a Canadian study [75]. There was only a nonsignificant excess of multiple sclerosis births in March, April, May and June as compared with the other months.

Some studies have found a summer peak in the birth of individuals with Down's syndrome [76]. Births during January and February, which concerned only 6% of patients, were associated with females.

There appears to be a trend for subjects born from December to March not to develop epilepsy [76].

Finally, in the study of Hafner et al. [45] the mentally retarded showed an excess of births in the second quarter and a slight deficit in the first and third quarters of the year. The authors proposed the procreational hypothesis also for mental retardation.

#### Discussion

In several psychiatric and neurological disorders, patients seem to exhibit a seasonal pattern of birth date. The hypotheses proposed by various authors concern especially the gestational or peripartun influence of environmental factors, infectious or not, on seasonal variations.

Differences in the exposure to external stimuli could be important for the early development of the brain. Several authors have reported the birth date effect in sport, university recruitment, and scientific innovation [77], suggesting that particular birth months confer advantages. Various causes of the birth month effect are possible, apart from the effect of climate and environmental factors during pregnancy and the first months of life: for example, the length of schooling effect arising from the varying date of entry into the first school [77] and each individual's position within the peer group. The climatic explanation is disproved because international comparisons reveal that birth months conferring advantages vary according to the following academic year in countries with dif-

fering school/college starting dates [78]. Studies on academic success of medical students have reported a 'spring or summer advantage' [79], showing a significant difference in the average ratios between groups born during the late spring/summer and winter. The hypothesis proposed by Gotoda [79] is that subjects born in summer spend their first 2 months of life unrestrained by heavy clothes and illness. They also have the opportunity to stay outdoors, which increases their physical movement and openness to external stimuli, both probably important for early development of the brain.

It is evident that any environmental factor could influence brain development, especially in the period of greatest plasticity.

In the course of a few months after gestation, the human central nervous system changes from a microscopic band of embryonic neuroblasts to a 350 gram mass with more than 109 million interconnected highly differentiated neurons in the cortex alone. By age 5, the human brain reaches 90% of its adult weight (1.3 kg) and the density of synapses in the cortex has already peaked and is beginning to decline. Moreover in the first period of life there is an extraordinary growth and intricate connectivity which results in sensorimotor, cognitive, affective and behavioral development [80].

Each factor that might influence the growth process could lead to irreversible modifications of the CNS as of other organs.

In agreement with this hypothesis is the observation of a season of birth effect on cortical grey matter thickness [81].

Abnormalities of neural migration or differentiation might underlie various behavioral disorders, offering the hope that developmental approaches might prove important for an understanding of psychopathology and could offer new strategies for treatment.

#### Conclusions

Season of birth patterns have been documented in certain psychiatric conditions. Hypotheses regarding the cause of the individual trends have been numerous, but none has proved conclusive.

Further research should investigate the possible presence of seasonality of births in other disorders. Indeed the attempt to explain seasonal birth patterns in psychiatric illnesses could serve to clarify the etiological bases of such disorders.

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