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Abnormal Seasonality of Schizophrenic Births

A Specific Finding?

H. Häfner, S. Haas*, M. Pfeifer-Kurda, S. Eichhorn, and S. Michitsuji**

Central Institute of Mental Health (CIMH), J5, P.O. Box 5970, D-6800 Mannheim 1, Federal Republic of Germany

Summary. The unusual finding of an abnormal seasonal distribution of schizophrenic births, showing an excess of 10% in the winter or spring months and an equal deficit in the summer or autumn months, cannot be explained by artefacts. It has not yet been established whether the finding is specific to schizophrenia. We observed an excess of schizophrenic births of some 10% in March to May, significant at the 5% level, and a deficit of approximately the same size in June to August on the birth data of first-admission patients with the clinical diagnosis of schizophrenia. The data, obtained from the Mannheim Psychiatric Case Register, were compared with those of the Mannheim population and a control group matched by birth year and sex. The total population of mentally retarded children aged 7 to 16 years from the Mannheim population showed an excess of some 20% in April to June and an equal deficit in the last two quarters of the year, compared with the Mannheim population of the same birth years. The finding was not significant, but allowance must be made for the low case number of 415. We also compared 3409 first-admission patients with depressive syndromes (ICD 296 and 300.4) and 5615 first-admission patients with the diagnosis of "neurosis and personality disorders" (ICD 300–302, except 300.4, and 305–309) from the Mannheim Case Register with a control population and a parallel control group. Depressed males showed an excess of births in March to May, which was significant at the 1% level; the birth peak for females was smaller and not significant. The same findings were obtained for the category of neurosis and personality disorders, i.e. an excess of about 10% in March to May for males, significant at the 1% level, and a non-significant excess for females. Our findings are awaiting replication. Causal explanations will be discussed with great reservation. The procreational hypothesis, assuming those factors that lead to an equidirectional seasonal pattern of births with a slight deviation from the average of a year in the general population, to be reinforced in the disease categories mentioned, is regarded as the most simple and plausible explanation. It is based on the assumption that some of the parents of individuals suffering from schizophrenia, mental retardation or probably also some other mental disorders running from generation to generation, have a higher threshold in partner-seeking behaviour, which is overcome more easily in the summer months with the consequence of increased pregnancies.

Key words: Schizophrenia – Epidemiology – Season of birth

* *Present address:* Eichberg Psychiatric Hospital in Eltville, Federal Republic of Germany

** *Present address:* Nagasaki University School of Medicine, Department of Neuropsychiatry, Nagasaki, Japan

Offprint requests to: H. Häfner

Introduction

Since Tramer (1929) reported a slight excess of birth dates of schizophrenic patients in the winter or spring months, about 40 studies in 14 countries have been published on this topic (for a review see Bradbury and Miller 1985; Boyd et al. 1986). The majority of the studies investigating the seasonality of schizophrenic births has revealed an excess of some 10% in the winter and spring months and, less consistently, an equal deficit in the summer and autumn months, i.e. an exaggeration of the seasonal pattern of births in the total population. The significant excess of birth dates of schizophrenic patients between December and May in the northern hemisphere seems to be corresponded by an equal excess between June and October in the southern hemisphere. This, however, has only been demonstrated in a few studies, all of which have shown some methodological shortcomings. The aetiological facts to explain this surprisingly consistent epidemiological finding are limited: if risk factors were transmitted by winter or spring birth, they would either affect only a small part of schizophrenics or account only for a small part of a multifactorial risk.

In their recent review Bradbury and Miller (1985) analysed 43 studies on this topic directly or indirectly. Only 11 of them were free from major methodological inadequacies; 10 of them showed a significant seasonal deviation, with 9 revealing an excess of the size mentioned in the winter or spring months. The authors discussed thoroughly the methological difficulties and shortcomings of the majority of the studies published on this topic. Boyd et al. (1986) analysed 30 of the studies discussed by Bradbury and Miller and found that apart from considerable methodological inadequacies, 19 showed an excess of births in the winter and spring months which was significant at least at the 5% level and 6 of the studies an excess that was significant at the 1% level.

In Fig. 1 we have listed all the studies investigating a sufficiently large number of cases (≥ 2000) to produce significant results with respect to the expected size of the deviation from

Investigator	Year of publication	n	Significance
Northern hemisphere			
Tramer	1929	3100	***
De Sauvage	1934/1951/1954	4679	***
Hare and Price	1968	3596	*
Dalen	1968	16238	***
Hare et al.	1974	5139	**
Ødegard	1974	19749	*
Videbech et al.	1974	7427	***
Parker and Balza	1977[a]	3508	***
Shimura et al.	1977	5431	**
Torrey et al.	1977	53584	*
O'Hare et al.	1980	4855	***
Watson et al.	1984	3246	**
Kendell and Kemp	1985	3224	*
Häfner et al.	1987	2020	*
Southern hemisphere			
Dalen	1975	2947	*
Parker and Neilson	1976	2256	**

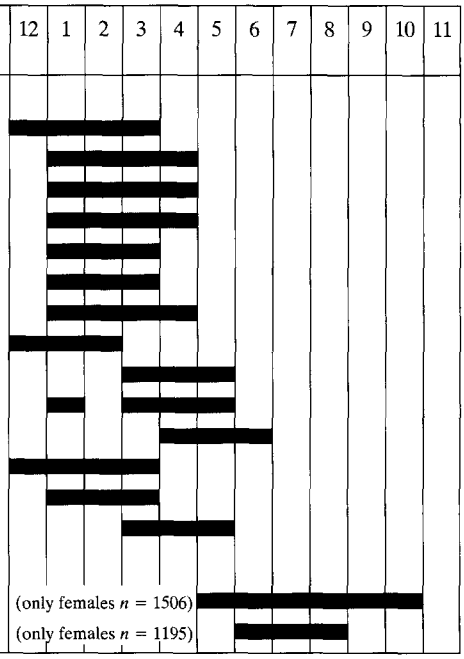

Fig. 1. Distribution of monthly births of schizophrenics in selected studies (only studies based on more than 2000 cases)

[a] Conducted in the equatorial zone and therefore not fully comparable with the studies from the northern hemisphere

* $P \leq 0.05$
** $P \leq 0.01$
*** $P \leq 0.001$

the normal seasonal distribution of births over 12 months. Unlike Bradbury and Miller, we only included studies that were directly available to us. Other methodological shortcomings were not taken into account. The months or quarters with a significant excess of schizophrenic births at least at the 5% level are depicted as black bars, the levels of significance by asterisks. The Figure shows that the studies conducted in the northern hemisphere were relatively well in agreement in showing a significant excess of schizophrenic births in the first 3 months of the year. Five studies showed an excess in the months of April and May and four in the month of December. Among the studies from the northern hemisphere we have listed Parker and Balza's (1977). It was conducted in the equatorial zone (the Philippines) and is therefore not fully comparable with the other studies in this category. The two studies from the southern hemisphere showed a significant deviation for females only. Thus, an analogous seasonal distribution of schizophrenic births has not yet been proven conclusively for this region.

The few studies conducted on the seasonality of births in other mental disorders have concentrated on affective, neurotic and personality disorders (Watson et al. 1984; Boyd et al. 1986). The results, however, are much less consistent than in schizophrenia. Some studies on affective disorders have shown a slight excess of spring births, with the majority yielding negative results. Watson et al. found that 6 studies out of 11 on manic depressives, 4 studies out of 6 on neurotics and 2 studies out of 6 on patients with personality disorders revealed a significant or insignificant excess of births somewhere between January and May. On the basis of their own data, which included non-schizophrenic patients from the diagnostic categories of neurosis, personality disorders and alcoholism, Watson et al. (1984) could not confirm the finding. Boyd et al. (1986) concluded that the inconsistent results in affective disorders had something to do with the non-specific diagnosis of manic depressive illness. With reference to Krauthammer and Klerman (1979) they stressed the fact that only 15% to 32% of the manic depressives showed a bipolar affective disorder with manifest manic episodes. They cited two studies (Hare et al. 1974; Hare 1975a) on manic or bipolar patients which had revealed a significant excess of births in the first quarter of the year. Parker and Neilson (1976), however, did not observe any significant difference in the seasonality of births between patients with bipolar disorders and a control population in Australia, and Dalen (1975) obtained different results for different years. Boyd et al. obviously followed the assumption that bipolar psychosis, as a severe and relapsing functional psychosis, which is to some degree genetically determined, could be more similar to schizophrenia in terms of birth seasonality than minor mental disorders like neurotic, personality and unipolar affective disorders. They rightly point out that "the evidence is not as strong as that for schizophrenics, but this may be due in part to the far smaller sample sizes in mania. If there is a winter excess of bipolar births, the magnitude of the excess could be as great as that for schizophrenia". In order to confirm these assumptions, additional studies of an exact methodological design are required on sufficiently large, if possible, unselected populations with bipolar affective disorders and other non-schizophrenic groups of disease.

Secular influences or birth cohort effects have not yet been studied systematically. Templer and Austin (1980) for example observed a falling trend of abnormal birth seasonality in schizophrenics born in Missouri between 1900 and 1960. Dalen (1975) found different seasonal patterns in manic, but also to a less extent in schizophrenic patients in different years or time periods.

Before commencing the difficult causal interpretation of these findings we have to ask whether they can be attributed

to an artefact, and whether they are specific to schizophrenia, bipolar affective disorders or other diseases as well.

Explanation with Reference to Artefacts

Age-Incidence and Age-Prevalence Effect

Hare (1975b) and Dalen (1975) were the first to draw attention to the fact that in the January of an admission year the January-born are almost 1 year older than the December-born of the same birth year. In the steeply increasing curve of age-related incidence rates the January-born of the younger age groups show higher incidence rates per admission year than those born in December, who are 11 months younger. In higher age groups, in which the age-related incidence rates fall, those who are 11 months older show lower incidence rates. Because of the considerably less steeply decreasing indicence figures after the third decennium of age, the descending and the ascending part of the age-incidence curve need bot be identical (Boyd et al. 1986).

The age-prevalence effect, first observed by Lewis and Griffin (1981) and discussed in detail by Templer (1982), Watson et al. (1982), Shur and Hare (1983), Pulver et al. (1983), Bradbury and Miller (1985) and Boyd et al. (1986), is related to the age-incidence effect. It proceeds from the fact that the cumulative risk for schizophrenia increases with age. Thus, as was the case with the age-incidence effect, the January-born display a higher risk for first-admission in a certain year than those born in December, even if a longer latency period between first-onset and first-admission were taken into account. Even if the birth dates of schizophrenics were distributed over the year analogously with those of the general population, those born at the beginning of a year should, according to Lewis and Griffin, be over-represented per year of admission, as their cumulative lifetime risk is higher than that of individuals born later in the same year.

Lewis and Griffin (1981) also criticised the statistical methods applied in earlier studies on this topic, especially for not taking into account changes in the year to year birth rates of the general population. To illustrate their criticism the authors compared the monthly birth dates of 10,363 schizophrenic patients of the mental hospitals in Missouri with those of the general population of the same federal state. By applying the usual analytical techniques they demonstrated a highly significant winter excess of schizophrenic births. After allowance was made for artefacts – to correct for the age-prevalence effect they used "a crude and not clearly defined method" (Boyd et al. 1986) – no significant season of birth effect emerged.

The discussion of artefacts, intensified by Lewis and Griffin, has been carried on by Watson et al. (1982), Templer (1982), Pulver et al. (1983), Shur and Hare (1983), Bradbury and Miller (1985) and Boyd et al. (1986). The arguments raised against the explanation of the abnormal seasonality of schizophrenic births by artefacts can be summed up as follows:

(1) The age-incidence and age-prevalence effect should lead to a maximum excess of schizophrenic births in the early months of the year and to a maximum deficit later in the year. By contrast, five studies conducted in the northern hemisphere (Fig. 1) revealed significant excesses in December. Also the maximum deficits did not fall in the last 4 months of the year, as expected on the basis of artefacts effects, but in the months of July and August. The studies conducted in the southern hemisphere also contradicted the assumption of artefact effects, if only their results, not yet sufficiently founded, could be replicated.

(2) The age-related risk effects should also be observable in other diseases with varying age of onset, or not, if the risk of falling ill was not related to age. The evidence available on the seasonality of births in neurotic and affective disorders seems to contradict rather than support the artefact hypothesis (Parker and Neilson, 1976; Watson et al. 1984).

Recently attempts have been made to adjust for the age-prevalence effect by statistical devices (Watson et al. 1982), and a significant season of birth effect persisted even after such an adjustment.

Age-incidence and age-prevalence effects can be eliminated by defining the patient populations studied by month of admission and month of age rather than by year of admission. Pulver et al. (1983) used standardised life table techniques for calculating the probability of receiving the diagnosis of schizophrenia month by month and still observed an excess of winter and spring births.

It can be concluded that the uneven distribution of schizophrenic births cannot be explained by age-incidence and age-prevalence effects or by erroneous statistical analysis. The discussion of artefacts promoted particularly by Lewis and Griffin and carried on by Bradbury and Miller has drawn attention to and helped to improve the methods of controlling for artefacts.

Problem Definition

Our study focussed on the question whether the abnormal seasonal distribution of births is in fact specific to schizophrenia, when methodological artefacts can be ruled out fairly reliably as a possible explanation. At the present state of knowledge the following five prerequisites should be taken into account to obtain valid results from comparisons of the seasonal distribution of births in certain patient groups and the population at large:

(1) case identification, i.e. the assignment of patients to the diagnostic category under study must be reliable enough;
(2) the study population, e.g. hospital admissions, must be more or less comprehensive or representative for all cases – treated or untreated – of the disease category studied in a defined population;
(3) the control group must be representative for the population to which the patient group studied belongs, as the seasonality of births varies over populations and geographic regions;
(4) comparisons of the control population and the patient sample must be based on birth years or groups of birth years, as the seasonality of births may also vary over birth years;
(5) artefacts that might result from age-related disease risks must be minimized or eliminated by adequate techniques.

The Present Study

One of the aims of the present study on the seasonality of schizophrenic births was to replicate the findings reported in the literature and to test whether our study population showed the same seasonal pattern of births.

Like Odegard (1974), Videbech et al. (1974) and Pulver et al. (1983) we drew our study group from a case register population comprising all the patients that had contacted the psychiatric inpatient and outpatient services, except private

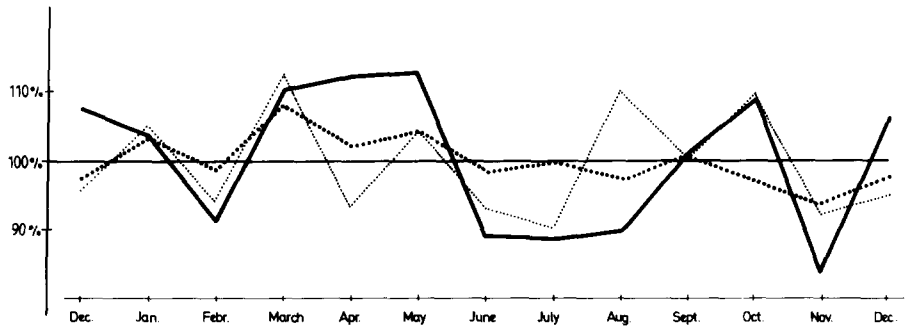

Fig. 2. Monthly distribution of schizophrenic births compared with births in the control population ≥15 years and a residence-, age- and sex-matched control group: (——) Schizophrenics ($n = 2{,}020$); (·····) Control population ($n = 220{,}700$); (·····) Control group ($n = 2{,}020$)

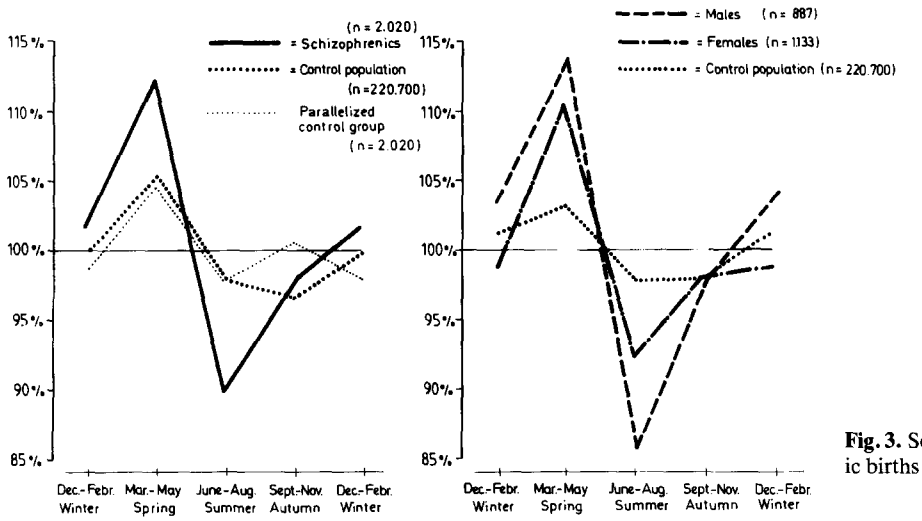

Fig. 3. Seasonal distribution of schizophrenic births for males and females

psychiatrists, from a defined population. The study group consisted of all the German inhabitants over 15 years of age in the city of Mannheim ($n = 200{,}700$) who in the period May 30, 1973 to December 31, 1980 were admitted to any of the psychiatric services with the diagnosis of schizophrenia (ICD No. 295.0–9, 297.0–9, 298.3) and, thus, recorded in the cumulative psychiatric case register of the Central Institute of Mental Health ($n = 2020$). The clinical diagnosis of these categories seems to be sufficiently stable. Klug (1983) studied the probability of changes in psychiatric diagnoses by using the same case register population. He found a relatively high stability of $P = 0.81$ for schizophrenia over two to nine admissions and various psychiatric institutions over a period of 4 years.

We compared the birth months of the 2020 schizophrenic patients over 15 years of age from the Mannheim population with the birth months of the Mannheim population and a control group of 2020 subjects from the same population, in parallel with the study group of schizophrenics by birth year and sex. Due to incompleteness of the data on birth place we were not able to make the comparison exactly on the basis of the same birth places. Well in to the 1970s Mannheim was a city with a stable population. The present seasonal pattern of birth dates of the Mannheim population corresponds almost exactly to the pattern expected on the basis of the birth rates of the country Baden-Württemberg. The error resulting from the unknown proportion of Mannheim inhabitants born elsewhere will thus hardly be relevant.

Figure 2 shows the monthly distribution of schizophrenic births for both sexes compared with that of the control population and the parallel control group. The average per year of all births, distributed over 12 months, was chosen as a baseline (= 100%). Somewhat later than in the average of the comparable studies from the northern hemisphere the trend of births in the total population showed a slight excess in the spring months of March, April and May and a slight deficit later in the year, i.e. June, August and November. Another small peak was found in January. Correspondingly, schizophrenic births peaked more clearly in the months of March to May and showed a marked deficit in the months of June, July and August. Both findings were significant at the 5% level. The trend of births in the control group did not show any clear difference to that of the total population, but more fluctuations due to its considerably smaller size.

Figure 3 depicts comparisons by birth quarters. This procedure has the advantage of eliminating age-related risk effects and levelling off monthly chance fluctuations in small populations. The Figure shows the annual trend of birth in the control group with an excess in the quarter of March to May and a deficit in the two successive quarters of June to August and September to November. In the group of schizophrenics this distribution pattern was clearly amplified. For males both the upward and the downward deviations were significant at the 5% level, in spite of the low case number, whereas for women they did not reach the threshold of significance.

In the autumn and winter months we did not observe any relevant deviations in the birth rates between the total population and the control group on the one hand and the schizophrenics on the other hand. The effect for males in our data cannot be interpreted, as the bulk of the studies examining sex

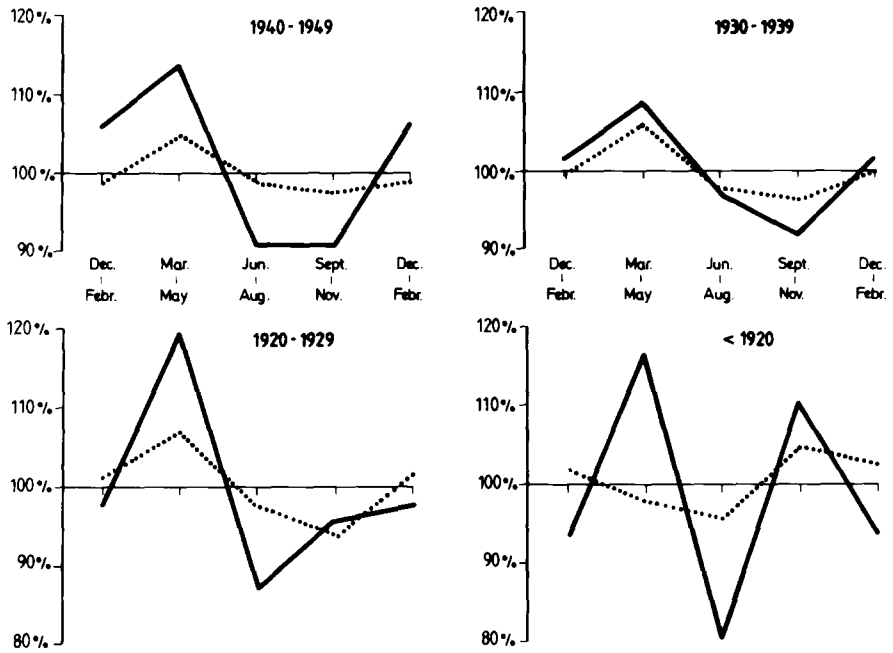

Fig. 4. Secular seasonal distribution of schizophrenic births compared with births in the control population: (———) Schizophrenics; (·····) Control population

differences has revealed either marked effects for females or no sex differences at all (Boyd et al. 1986).

To pay attention to the assumption that the risk mediated by a spring birth might be associated with a certain distinctive group of schizophrenics, we distinguished between chronic (ICD 295.0, 0.1, 0.5, 0.6; 297.0) and acute schizophrenia (ICD 295.2, 0.3, 0.4, 0.7, 0.8, 0.9; 297.1, 0.9; 298.3) despite the low case number. This distinction is imprecise. Unlike Pulver et al. (1983) we found practically identical seasonal patterns of births in both groups, a finding in agreement with that of the sophisticated Scottish case register study by Kendell and Kemp (1985). Nor did we observe any differences in the seasonality of births between the paranoid-hallucinatory subgroup and other groups of schizophrenics. We did not attempt to distinguish any subgroups of schizophrenics by genetic risk (Kinney and Jacobsen 1978; Shur 1982; Shensky and Shur 1982; Templer and Veleber 1982; Machon et al. 1983), because there are not yet sufficiently reliable markers of a differing genetic load in the population of schizophrenics available. The method of counting schizophrenics in the kin of index patients is not reliable enough.

Shimura et al. (1977), Templer and Austin (1980), Torrey et al. (1977), Torrey and Torrey (1979) and O'Hara et al. (1980) found marked differences in the seasonal distribution of schizophrenic births for various groups of birth years. The findings, however, did not indicate any clear-cut trend. We examined three birth decades from 1949 back to 1920 and included those born before 1920 as a fourth group (Fig. 4).

The four groups hardly differed from one another in terms of size, but in all four cases the numbers, ranging between 335 and 505, were too small to yield reliable results. Those born in the first decade up to 1929 and after 1939 showed, compared with the total group of schizophrenics and the respective control group, an equidirectional birth date pattern with a plus in the spring months of March to May and a minus of a comparable size in the summer quarter of June to August or in the summer and autumn quarter. Only in the decade of 1930 to 1939 did the birth date pattern of schizophrenics not differ from that of the control population. The few relevant characteristics in terms of which the period of 1930 to 1939 differed from the other three, were a steeply rising trend of births and marriages, boosted by the national socialist propaganda, and the absence of heavy warfare and its immediate consequences. The other two periods were characterized by the two world wars and the period of economic depression following World War 1. An interpretation of these time period-related effects is not justifiable at the present stage because of the low case number and lack of agreement between our findings and those of other studies. The results, however, encourage further research on period-related effects.

Mental Retardation

Encouraged by Dr. Norman Sartorius, with whom we discussed our research project, we studied the seasonal pattern of births of a more or less comprehensive population of 415 severely or moderately severely mentally retarded children ($IQ \leq 60$) aged 7 to 16 years of 14 birth years (1958–1971) from the Mannheim population[1] (Fig. 5). Due to the small size of the study population we compared it only with the control population comprising the Mannheim inhabitants of the same birth years on the basis of birth quarters.

The control population of the corresponding birth years showed the usual pattern of birth dates with a slight excess in the first two quarters and a slight deficit in the last two quarters compared with the annual average. Contrary to expectations the mentally retarded showed an excess of births of almost 20% in the second and a slight deficit in the third and first quarters of the year. Due to the low case number the deviation did not reach the level of significance. It should be noted that in mental retardation the quarter with the largest excess in births began 1 month later than in schizophrenia. It should also be noted that age-incidence and age-prevalence effects were hardly present in mental retardation, as the

[1] We thank Prof. B. Cooper, Mannheim, for providing us the data from an epidemiological study of mental retardation conducted in Mannheim.

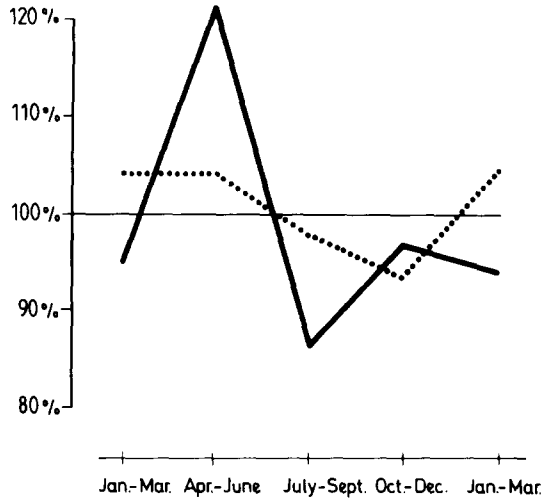

Fig. 5. Seasonal distribution of births of mentally retarded children. (———) Mentally retarded $n = 415$; (·····) Control population $n = 55{,}700$

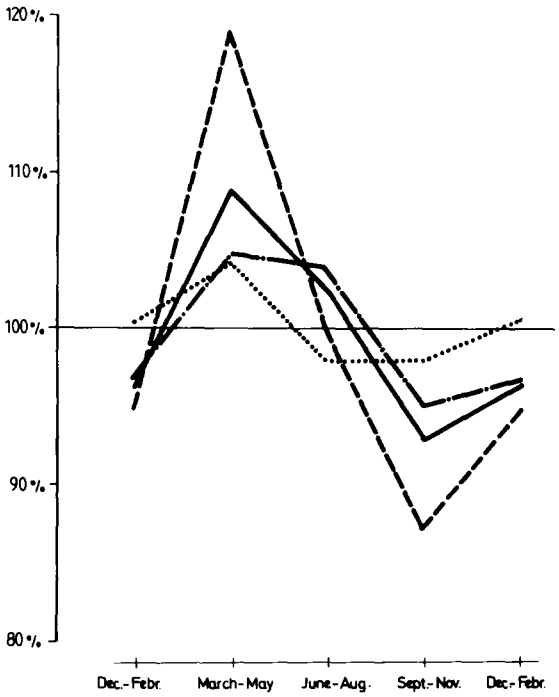

Fig. 6. Seasonal distribution of male and female births with depressive syndromes: (– – – –) Males ($n = 1{,}001$); (–·–·–·) females ($n = 2{,}408$); (———) males and females ($n = 3{,}409$); (·····) Control population ($n = 220{,}700$)

cumulative illness risk had already attained 100% at the age of the study population (= 7 years).

Depressive Syndromes

As it was thus probable that the amplification of the normal seasonal pattern of births is not specific to schizophrenia, we examined the seasonal distribution of the birth dates of 3,409 patients with depressive syndromes (ICD 296 and 300.4), who were first recorded in the Mannheim Case Register in 1973–1980 (Fig. 6). In contrast with the complete case finding under

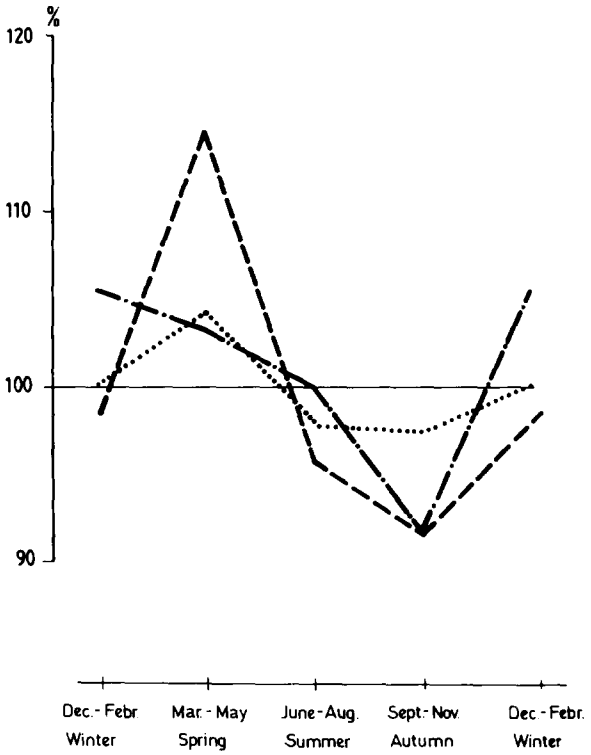

Fig. 7. Seasonal distribution of male and female births: Neuroses and personality disorders (from the Cumulative Psychiatric Case Register) (– – – –) Males ($n = 2{,}378$); (–·–·–·) Females ($n = 3{,}237$); (·····) Control population ($n = 220{,}700$)

the precise diagnosis of mental retardation this diagnostic group had several methodological flaws: the diagnosis was rather unreliable, especially in less severe states. The cases recorded in the case register comprised only a tiny proportion, some 10%, of the population with depressive disorders. The reason for this is that, unlike schizophrenics, only a minority of the persons suffering from depression, i.e. mainly the more severely depressed, contact specialist services and inpatient mental health services. It was not possible for us to heed the recommendations of Boyd et al.(1986) and to distinguish "bipolar affective disorders", as the numbers were too low for the purpose of studying the seasonal distribution of births.

As Fig. 6 shows, males had an excess of births in the months of March to May, which was significant at the 1% level, and a deficit in the months of September to November, which was significant at the 5% level, when compared with the birth dates of the Mannheim control population parallelled by birth year. This finding, however, cannot be generalized for the reasons mentioned above and because of insufficient agreement with the results of other comparable studies. It should, however, be stressed that in their evaluation of the studies on seasonal birth in bipolars, Boyd et al. (1986) tended to the assumption that patients with bipolar disorders have an excess of births in the months of January to March, probably also to April. However, the evidence in this illness category is not as strong as in schizophrenia.

Neurosis and Personality Disorders

We also examined all the patients ($n = 5615$) recorded for the first time with the diagnosis of neurosis or personality disorder (ICD 300–302, except 300.4, and 305–309) in the Mannheim

case register. With respect to these group, the same allowances were made as in the group of depressive syndromes: the diagnosis was not reliable enough and the study population represented only a small proportion of the actual morbidity in the population and was probably also exaggerated in terms of the degree of severity of the disorder. As Fig. 7 shows, in this group, too, the trend of the gerneral population to an excess of births in the months of March to May and to a deficit in the months of June to August and September, compared with the average of the year, is clearly amplified only for males. The excess of spring births for males, amounting to more than 10% was significant at the 1% level, compared with the parallell control population.

Given the inconsistencies between the results of the few studies conducted on comparable populations (Watson et al. 1984) and the methodological shortcomings discussed above, our findings cannot be interpreted as providing reliable evidence for a clear-cut excess of spring births in the category of neurotic and personality disorders. On the whole, our results do not proove the abnormality of the seasonal distribution of births in diagnostic groups other than schizophrenia. With respect to this disease category they are in agreement with the consistent results of the majority of the studies conducted on this topic. Nevertheless, they are of some value for the discussion of these questions in that they justify doubts about the specificity of the excess of spring births in schizophrenia and support the assumption that the normal seasonal distribution of births in the general population, showing a small excess of spring births and a small deficit of summer births, might be amplified in several severe mental disorders in an analogous, but non-specific way.

Discussion

We have to assume that the artefacts discussed do not provide an exhaustive explanation for the observed trend. In mental retardation age-related risk factors are irrelevant. In all other groups, irrespective of control of artefacts by basing comparisons on birth or age quarters, an increase in birth rates was observed only in the spring months, whereas in the months of January and February the age-related risk, contrary to expectations, did not show any relevant excesses.

Figure 8 illustrates the chronological order of season-related environmental factors that might contribute to the illness risks discussed, or in the case of the procreation hypothesis, the seasonally mediated influence on reproduction behaviour or frequency of conception. The seasonally mediated environmental influence should become active in the form of some kind of brain damage, which in turn has an influence upon the illness risks. In connection with this hypothesis brain damage could occur pre-natally, peri-natally or post-natally. The hypotheses range from extreme temperatures (Hare and Moran 1981), seasonal variation of nutritional practices (Torrey et al. 1977), vitamin deficencies (de Sauvage 1954), exposure to toxic insecticides (Gershon and Shaw 1961), peri-natal brain trauma, bacterial or viral infections (Torrey and Torrey 1979; Hare 1979; Tyrell et al. 1979; Taylor et al. 1982; Shimura and Miura 1980; Watson et al. 1984). In recent years the explanation has increasingly been sought in viral or retroviral hypotheses (Hare 1983; Machon et al. 1983; for review see Crow 1984). The arguments put forward in favour of these hypotheses include the seasonal incidence of measles, capable of causing fetal damage (Dudgeon et al. 1973; Peckham 1978) and congenital cytomegaloviral infections (Hanshaw 1971), also correlating with seasonal birth peaks. Watson et al. (1984) even observed elevated winter birth rates for schizophrenics in Minnesota predominantly for the unmarried – in 10 of a total of 45 years (1915–1959) preceded by summers with higher than average rates for bacterial and viral infections. In years followed by winters with lower rates for infectious diseases the seasonal trend of schizophrenic births differed less from that of the total population.

All these explanations referring to climate-related brain damage are speculative in nature. There are not yet any clear-cut aetiological findings on schizophrenics available which could be regarded as attributable to a viral infection, obstetric or para-natal complications, malnutrition, vitamin deficiencies etc. in association with a winter or spring birth. The amount of brain damage observed on CT of schizophrenics provides only a moderate indication, as the findings are not specific, nor have they been shown reliably to be associated with the illness risk. It cannot yet be ruled out that they result

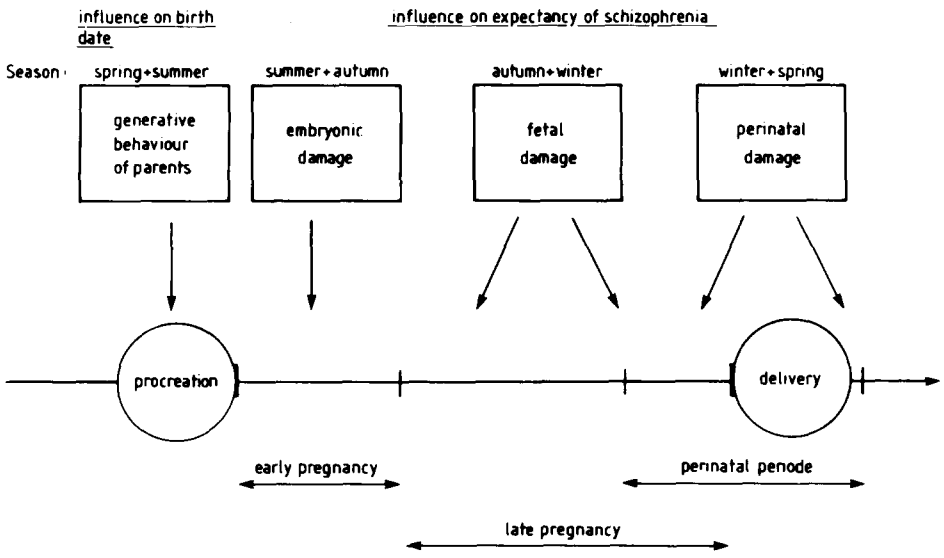

Fig. 8. Chronological order of explanatory hypotheses in the course of pregnancy

from a selection of schizophrenic patients with additional brain damage and, thus, an unfavourable prognosis from hospital admissions and readmissions. Nevertheless, it would be worth examining whether the seasonal excess of births is more pronounced in this group than among schizophrenics without brain damage, should CT findings on a fairly large and representative number of schizophrenic patients be some day available.

The assumption that the abnormal seasonality of schizophrenic births might be explained by the socioeconomic status of the parents of schizophrenics is a secondary hypothesis, as the effect on the seasonal distribution of births in the patient population should be mediated either by a higher risk of pregnancy complications and, thus, again by climate-related brain damage or deviant reproductive behaviour. Also the assumption that seasonal factors might cause damage to the CNS during the fetal or pre-natal period and thus lead to a correspondingly enlarged risk for schizophrenia, is anything but clear with regard to certain aetiological principles and on the whole seems very unlikely given the present state of knowledge. The assumption favoured by Boyd et al. (1986) and not confirmed with our data that there exists a seasonal type of schizophrenia that is accompanied by all the aetiological factors mediated by a winter or spring birth and differs from the remaining 90% of schizophrenics with a normal seasonal birth distribution is not very convincing either. No data are available that would support this assumption.

In their recent study Kendell and Kemp (1985) once again tested the hypothesis postulating differences in the seasonality of births between various schizophrenic subgroups by using data of 571 schizophrenic patients from the Edinburgh Psychiatric Case Register and of 2,653 patients from the psychiatric inpatient records of the Scottish Health Service. They found no significant differences between the schizophrenic subtypes or with regard to factors related to the disease course. For this reason and because of our doubts about the specificity of the abnormal seasonality of schizophrenic births we think it sensible to investigate the procreational habits hypothesis. According to it the parents of schizophrenics and probably also of patients with other severe mental disorders conceive more often in summer – in our sample primarily in the months of June to September – thus producing an excess of spring births, whereas in other studies they conceive more often in the spring and produce a winter excess of children with a higher risk of schizophrenia (Huntington 1938; Hare and Price 1968; Dawson 1978; Ödegard 1974, 1977). Two essential objections have been raised against this hypothesis. If it were correct, the siblings of schizophrenics should also show a comparable distribution of birth dates. Bradbury and Miller observed that four studies examining this hypothesis (Hare 1976; Watson et al. 1984; Buck and Simpson 1978; McNeil et al. 1976) produced only little evidence favouring a winter excess of births among the siblings of schizophrenics. The authors rightly point out that this cannot be regarded as contradictory to the hypothesis, as the birth patterns of the children with an increased risk of schizophrenia might be influenced by differential fertility of the parents (Kellett 1978), e.g. by the sizes of the schizophrenic families or by the order of birth within those families. It is not unlikely that schizophrenics, probably also individuals with severe mental retardation or other severe mental disorders, are predominantly first and only children. The corresponding findings, however, are inconsistent (Ernst and Angst 1983). Our own data were not complete enough to enable investigation of the pattern of births by taking birth order into account. We also tried to investigate the birth date patterns of the first versus the next born in the Mannheim population born between 1968 and 1983 (information was not available from earlier years) and found a slight, but not significant excess in the summer months for the first born. We were thus unable to find evidence in support of this particular hypothesis.

Bradbury and Miller argued against the procreational hypothesis (i.e. the assumption that the same processes to which the seasonality of births in the population at large are subject to, are at work to an increased degree in schizophrenics, Parker and Neilson 1976) that the factors responsible for the winter excess of births have not yet been identified.

Nevertheless, it is plausible to assume that the frequency of conception in the total population is higher in the warmer season and the holiday months usually coinciding with it, because there are more frequent and more intensive erotic stimuli and better opportunities for sexual contact and conception. In view of this assumption it is not difficult to imagine that a small proportion of the parents of schizophrenics and individuals suffering from mental retardation, other severe mental disorders or brain diseases is more handicapped in heterosexual mating behaviour than the average of the general population. This might be the case particularly with such diseases in which genetic factors play an important role. Among the parents of individuals suffering from such diseases there might for example be heterozygotics in whom mental disturbances below the threshold of mental disease or intelligence deficits are present. Such individuals, who have to overcome a higher threshold before engaging in hertosexual activities due to being handicapped in social, especially heterosexual, contact behaviour, might be in need of the more intensive erotic stimuli and the better opportunities for mating and conception in the spring and summer months more than the average of the general population.

The reproduction hypothesis is indeed the most simple explanation for the abnormal seasonality of schizophrenic births. It is not specific to schizophrenia, although it is very plausible that some of the parents of individuals suffering from the disease are handicapped in heterosexual mating behaviour. This explanation would also be applicable to severe mental retardation, bipolar affective disorders and other mental disorders, if the same pattern of birth seasonality could be proven conclusively in these disorders, too. The plausibility of the explanation rests primarily on the fact that the exaggeration of an analogous trend in the seasonal reproduction behaviour of the population in general is interpreted by an extention of the same principle of causal explanation applicable to the parents of individuals with these diseases.

The touchstone of this hypothesis is the proof of an amplified deviation of the seasonal pattern of births of the population in general in schizophrenic birth rates. The hypothesis has been confirmed by the majority of the studies conducted on large and representative populations, in particular by studies using case register populations (Dalen 1968; Hare et al. 1974; Ödegard 1974, 1977). In one of four studies conducted on very large samples (Torrey et al. 1977) and in three studies conducted on smaller samples (O'Hare et al. 1980; Parker and Balza 1977; Shimura and Miura 1980) the relation of the deviation between the birth pattern of schizophrenics and that of a control population was non-existent or very weak. Of these studies only that of O'Hare et al., conducted

in Ireland, can be regarded as valid without any greater reservations. Parker and Balza carried out their study in an equatorial region (Philippines) without any distinct seasonal variation in temperature. In the study of Torrey et al. objections may be raised against the representativeness of the group of schizophrenics studied, collected from the patient population of the Mental State Hospitals of 20 USA federal states. The same applies to the study by Shimura and Miura, based on the long-stay patients of a large mental hospital in Tokyo. Nevertheless, we must agree with Bradbury and Miller that the assumption of an amplified deviation, although our own data seems to confirm it, has not yet been proven conclusively.

The only reliable conclusion that we can at present draw after a careful examination of the facts is that the explanation of the deviant seasonal pattern of schizophrenic births by seasonaly deviant reproduction behaviour of the parents of schizophrenics sounds more plausible than the assumption that damage caused to the CNS by environmental factors mediated by winter or spring births increases the multifactorial illness risk in schizophrenia by 10% or contributes to the illness risk in a small number of schizophrenics. For further research into the question not only differential studies on schizophrenics but also carefully designed studies on the seasonality of births in comparable mental and physical disorders are required, in order to obtain valid evidence for the specificity of the seasonality of schizophrenic births, which our results fail to support.

References

Boyd JH, Pulver AE, Stewart W (1986) Season of birth: Schizophrenia and pipolar disorder. Schizophr Bull 12:173–185

Bradbury TN, Miller GA (1985) Season of birth in schizophrenia: A review of evidence, methodology and etiology. Psychol Bull 98: 569–594

Buck C, Simpson H (1978) Season of birth among the sibs of schizophrenics. Br J Psychiatry 132:358–360

Crow T (1984) A re-evaluation of the viral hypothesis: Is psychosis the result of retroviral integration at a site close to the cerebral dominance gene? Br J Psychiatry 145:243–253

Dalen P (1968) Month of birth and schizophrenia. Acta Psychiatr Scand 203:55–60

Dalen P (1975) Season of birth: A study of schizophrenia and other mental disorders. North-Holland, Amsterdam

Dawson DF (1978) An explanation for the seasonality of births in schizophrenia. Am J Psychiatry 135:1434–1435

de Sauvage Nolting WJJ (1934) The relation between schizophrenic and related disorders and the month of birth. Ned T Geneesk 79: 528

de Sauvage Nolting WJJ (1951) The relation between the month of birth and schizophrenic and manic-depressive mental disorders. Ned T Geneesk 4:3855–3864

de Sauvage Nolting WJJ (1954) Vitamin C and the schizophrenic syndrome. Folio Psychiatry Neerl 57:347–355

Dudgeon JA, Peckham CS, Marshall WC, Smithells RW, Sheppard S (1973) National congetial rubella surveillance programme. Health Trends 5:75–79

Ernst C, Angst J (1983) Birth order. Its influence on personality. Springer, Berlin, Heidelberg, New York

Gershon S, Shaw F (1961) Psychiatric sequelae of chronic exposure to organophospharous insecticides. Lancet I:1371–1374

Hanshaw JB (1971) Congenital cytomegalovirus infection: a fifteen year perspective. J Infect Dis 123:555–561

Hare EH (1975a) Manic-depressive psychosis and season of birth. Acta Psychiat Scand 52:69–79

Hare EH (1975b) Season of birth in schizophrenia and neurosis. Am J Psychiatry 132:1168–1171

Hare EH (1976) The season of birth of siblings of psychiatric patients. Br J Psychiatry 119:49–54

Hare EH (1979) Schizophrenia as an infectious disease. Br J Psychiatry 35:468–470

Hare EH (1983) Epidemiological evidence for a viral factor in the aetiology of the functional psychoses. Adv Biol Psychiatry 12:52–75

Hare EH, Moran P (1981) A relation between seasonal temperature and the birth rate of schizophrenic patients. Acta Psychiatr Scand 63:396–405

Hare EH, Price JS (1968) Mental disorder and season of birth: Comparison of psychoses with neuroses. Br J Psychiatry 115:533–540

Hare EH, Price JS, Slater E (1974) Mental disorder and season of birth: A national sample compared with the general population. Br J Psychiatry 124:81–86

Huntington E (1938) Season of birth: Its relation to human abilities. Wiley, New York

Kellett J (1978) Seasonality in schizophrenia. Lancet I:664

Kendell RE, Kemp IW (1985) Winter born v. summer born schizophrenics. Paper presented at the World Psychiatric Association Section Symposium "The future of psychiatric epidemiology", 23–26 September 1985, Edinburgh

Kinney DK, Jacobsen B (1978) Environmental factors in schizophrenia: New adoption study evidence. In: Wynne LC, Cromwell RL, Matthysse S (eds) The nature of schizophrenia: New approaches to research and treatment. Wiley, New York, pp 38–51

Klug J (1983) Psychiatrische Diagnosen: Einflußfaktoren auf die Stabilität im zeitlichen Verlauf. Beltz, Weinheim, Basel

Krauthammer C, Klerman GL (1979) The epidemiology of mania. In: Shopsin B (ed) Manic illness. Raven Press, New York, pp 11–28

Lewis M, Griffin T (1981) An explanation for the season of birth effect in schizophrenia and certain other diseases. Psychol Bull 89: 589–596

Machon R, Sarnoff A, Mednick S, Schulsinger F (1983) The interaction of seasonality, place of birth, genetic risk and subsequent schizophrenia in a high risk sample. Br J Psychiatry 143:383–388

McNeil T, Kaij L, Dzierzykray-Rogalska M (1976) Season of birth among siblings of schizophrenics. Acta Psychiatr Scand 54:267–274

Ödegard Ö (1974) Season of birth in the general population and in patients with mental disorder in Norway. Br J Psychiatry 125:397–405

Ödegard Ö (1977) Season of birth in the population of Norway, with particular reference to the September birth maximum. Br J Psychiatry 131:339–344

O'Hare A, Walsh D, Torrey F (1980) Seasonality of schizophrenic births in Ireland. Br J Psychiatry 137:74–77

Parker G, Balza B (1977) Season of birth and schizophrenia – an equatorial study. Acta Psychiatr Scand 56:143–146

Parker G, Neilson M (1976) Mental disorder and season of birth – a southern hemisphere study. Br J Psychiatry 129:355–361

Peckham CS (1978) Congenital rubella surveillance. J R Coll – Physicans Lond 12:250–255

Pulver AE, Stewart W, Carpenter WT, Childs B (1983) Risk factors in schizophrenia: Season of birth in Maryland, USA. Br J Psychiatry 143:389–396

Shensky T, Shur E (1982) Season of birth of schizophrenics: Cyclic trends and their analysis. Br J Psychiatry 141:316–317

Shimura M, Miura T (1980) Season of birth in mental disorders in Tokyo, Japan, by year of birth, year of admission, and age at admission. Acta Psychiatr Scand 61:21–28

Shimura M, Nakamura I, Miura T (1977) Season of birth of schizophrenia in Tokyo, Japan. Acta Psychiatr Scand 55:225–232

Shur E (1982) Season of birth in high and low genetic risk schizophrenics. Br J Psychiatry 140:410–415

Shur E, Hare E (1983) Age-prevalence and the season of birth effect in schizophrenia: A response to Lewis and Griffin. Psychol Bull 93:373–377

Taylor GR, Crow TJ, Ferrier IN, Johnstone EC, Parry RP, Tyrrell DAJ (1982) Virus-like agent in CSF in schizophrenia and some neurological disorders. Lancet II:1166–1167

Templer DI (1982) Comment on the Lewis and Griffin explanation for season of birth effect in schizophrenia. J Orthomolecular Psychiatry 11:219–221

Templer D, Austin R (1980) Decreasing seasonality of schizophrenic births. Arch Gen Psychiatry 37:959–960

Templer D, Veleber D (1982) Seasonality of schizophrenic births: Harmful effects or genetic morphism? Br J Psychiatry 140:323–324

Torrey EF, Torrey BB (1979) A shifting seasonality of schizophrenic births. Br J Psychiatry 134:183–186

Torrey EF, Torrey BB, Peterson MR (1977) Seasonality of schizophrenic births in the United States. Arch Gen Psychiatry 34: 1065–1070

Tramer M (1929) Über die biologische Bedeutung des Geburtsmonates, insbesondere für die Psychoseerkrankung. Schweiz Arch Neurol Psychiatr 24:17–24

Tyrrell DAJ, Crow TJ, Parry RP, Johnstone EC, Ferrier IN (1979) Possible virus in schizophrenia and some neurological disorders. Lancet I:839–841

Videbech T, Weeke A, Dupont A (1974) Endogenous psychoses and season of birth. Acta Psychiatr Scand 50:202–218

Watson CG (1985) Birth seasonality and schizophrenia. In reply. Arch Gen Psychiatry 42:107–108

Watson CG, Kucala T, Angulski G, Brunn C (1982) Season of birth and schizophrenia: a response to the Lewis and Griffin critique. J Abnorm Psychol 91:120–125

Watson CG, Kucala T, Tilleskjor C, Jacobs L (1984) Schizophrenic birth seasonality in relation to the incidence of infectious diseases and temperature extremes. Arch Gen Psychiatry 41:85–90

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