Season-of-birth and "age incidence" effects

SIR: Eagles *et al* (1995) report an increase over the period 1900 to 1969 in the ratio of winter/spring to summer/autumn births of males (but not females) who were later admitted with a diagnosis of schizophrenia. These authors attribute the increase to seasonal environmental factors such as infection and malnutrition.

An entirely different explanation is suggested by Lewis & Griffin (1981) and Lewis (1989) who drew attention to the "age incidence" effect – the fact that for a disease risk which increases with age those born in the earlier months of each year have a greater risk than those born in the later months (simply because they are older) of becoming ill in a succeeding (within a lifetime) year of recorded admissions. As Lewis (1989) points out, the effect will be greater (because the size of the effect is inversely related to age) with earlier onsets of illness than with later onsets. In Eagles *et al*'s study those who were born in the decade 1900–9 are obviously likely to have a later mean age of onset than those born in 1960–9 (who were aged between 18 and 27 years when the period of investigation ended in 1987). The age incidence effect therefore appears to account for Eagles *et al*'s main finding. Moreover since the female:male ratio in schizophrenic illnesses is linearly related to age (early onsets are commoner in males, and late onsets in females) a sex difference in the findings is also predicted.

According to this interpretation Eagles *et al*'s findings can be accounted for without recourse to hypotheses of an environmental influence.

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LEWIS, M. S. (1989) Age incidence and schizophrenia: I. The season of birth controversy. *Schizophrenia Bulletin*, **15**, 59–73.

— & GRIFFIN, T. A. (1981) An explanation for the season of birth effect in schizophrenia and certain other diseases. *Psychological Bulletin*, **89**, 589–596.

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AUTHORS' REPLY: We were intrigued by Crow's hypothesis, but feel that he overplays the magnitude of any possible effect of the "age-incidence artifact". That this artifact may exist in schizophrenic birthdates is very likely, but it should exist to a similar degree in other conditions, notably those with an early age at onset (Dalen, 1990). Lewis (1989) himself can cite few instances of the "artifact" in other conditions. Furthermore, as Torrey & Bowler (1990) have argued, if Lewis were correct about the importance of the artifact, then January should have the highest rate of schizophrenic births while December the least, and southern hemisphere findings should mirror those in the northern hemisphere. The data do not bear out the hypothesis. Furthermore, studies which have corrected for age incidence (Watson, 1990) continue to find a season-of-birth effect in schizophrenia.

It is worth noting that, in our own paper, April birthdates were most common while January and December birthdates were fifth and sixth commonest respectively (p. 470).

We feel that Crow's attempts to explain the gender differences in our study again suffers from the difficulty of taking a factor of small magnitude to attempt to explain a large effect. Mean age at onset is about four years younger for men than it is for women (Goldstein *et al*, 1989), and our study spanned a 70-year period of birthdates.

DALEN, P. (1990) Does age incidence explain all season-of-birth effects in the literature? *Schizophrenia Bulletin*, **16**, 11–12.

GOLDSTEIN, J. M., TSUANG, M. T. & FAROANE, S. V. (1989) Gender and schizophrenia: implications for understanding the heterogeneity of the illness. *Psychiatry Research*, **28**, 243–253.

LEWIS, M. S. (1989) Age incidence and schizophrenia: part I. The season of birth controversy. *Schizophrenia Bulletin*, **15**, 59–73.

TORREY, E. F. & BOWLER, A. E. (1990) The seasonality of schizophrenic births: a reply to Marc S. Lewis. *Schizophrenia Bulletin*, **16**, 1–3.

WATSON, C. G. (1990) Schizophrenia birth seasonality and the age-incidence artifact. *Schizophrenia Bulletin*, **16**, 5–10.

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Anorexia nervosa rates – conclusions for the wrong reasons

SIR: We read with interest the review paper on rates of anorexia nervosa by Fombonne (1995). We completely concur with him that "there is no evidence of a secular increase in its incidence". However, we feel that Fombonne's conclusions, albeit based on an ambitious attempt to cover the literature, are not well supported by the evidence he cites.

The main reason why there is no evidence for an increase in the rate of anorexia nervosa is that there are so few community based studies that a proper meta-analysis taking account of secular trends cannot be undertaken. Fombonne fails to discuss this state of affairs and instead concentrates on a whole

host of clinical studies which he mixes up (like apples and pears) with the handful of community based studies that exists. One of the few methodologically acceptable community based studies of anorexia nervosa (see Treasure, 1990; Patton & King, 1991) – the one performed in Göteborg, Sweden in the 1980s (Råstam *et al*, 1989) – was excluded from Fombonne's analysis for "obvious reasons". It seems these "obvious reasons" were (1) that the material of the Göteborg study was presented in sufficient detail to allow specific analysis of whether DSM–III or DSM–III–R criteria applied, (2) that partial syndromes – later meeting full DSM–III–R criteria (Gillberg *et al*, 1994) – were included as a separate group in the original study, and (3) that the birth-cohort was followed up for a few years leading to the appearance of new cases. The findings were presented in a way which has made it possible for Fombonne to calculate all sorts of rates needed for a thorough review. He himself complains that several studies have not provided enough information about the diagnostic criteria used, and that few authors have looked at cohorts in a longitudinal fashion, so we had some difficulty understanding what was so "obvious" about the reasons for excluding this study.

The prevalence rate of anorexia nervosa in the community based studies was considerably higher than the median rate calculated by Fombonne. Again, this should not be taken as evidence that there has been an increase in prevalence rate over the years. However, it is essential that conclusions be based on the most reasonable data sets rather than those that, according to the standards set out by the author of a review/meta-analysis, are less than adequate.

FOMBONNE, E. (1995) Anorexia nervosa, No evidence of an increase. *British Journal of Psychiatry*, **166**, 462–471.

GILLBERG, I. C., RÅSTAM, M. & GILLBERG, C. (1994) Anorexia nervosa outcome: six-year controlled longitudinal study of 51 cases including a population cohort. *Journal of the American Academy of Child and Adolescent Psychiatry*, **33**, 729–739.

PATTON, G. C. & KING, M. B. (1991) Epidemiological study of eating disorders: time for a change of emphasis. *Psychological Medicine*, **21**, 287–291.

RÅSTAM, M., GILLBERG, C. & GARTON, M. (1989) Anorexia nervosa in a Swedish urban region: a population-based study. *British Journal of Psychiatry*, **155**, 642–646.

TREASURE, J. (1990) Anorexia nervosa and bulimia nervosa. *Current Opinion in Psychiatry*, **3**, 211–214.

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Periodic psychosis of puberty

SIR: The article by Abe & Ohta (1995) regarding adolescent onset brief periodic psychoses raises a number of important issues which must be clarified before this condition can be so confidently defined. It is not clear from the report whether the cases they describe met criteria (ICD–10 or DSM–IV) for other psychiatric diagnoses. Certainly, if the subjects cross-sectionally met criteria for depression or mania, it should come as no surprise that these disorders would recur.

Without a description of family history, natural course, comorbid symptoms/diagnoses, or other external validators, it is difficult to assign any diagnostic validity to the concept of a "periodic psychosis of puberty". The reported relationship of worsening psychotic symptoms associated with menses is well recognised in adolescent in-patient units in which severely ill teenage girls with mania or depression are treated. This phenomenon does not of itself qualify for a unique diagnostic label.

In ten consecutive years of adolescent in-patient practice, we have only ever seen one teenager (white, male, age 16) with a non-substance induced, non-affective or non-schizophreniform brief psychosis. This is one of about 1000 admissions. Perhaps there is a cultural diversion to this type of presentation in psychiatrically disturbed teenagers that differentiates Canadian from Japanese youth.

ABE, K. & OHTA, M. (1995) Recurrent brief episodes with psychotic features in adolescence: periodic psychosis of puberty revisited. *British Journal of Psychiatry*, **167**, 507–513.

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Treatment of PTSD

SIR: Busuttil *et al* (1995) suggest that their case series strongly endorses the use of psychological debriefing (PD) in the treatment of PTSD. However, the 63 hours of "formal work sessions" in a residential setting described in their paper seems excessive and difficult to justify for PTSD sufferers who have not first tried out-patient treatment. Briefer therapies can work. For example, Foa *et al* (1991) described a randomised controlled trial in which PTSD sufferers experienced a marked reduction in symptoms after nine 90 minute exposure therapy sessions at three and a half month follow-up.