many aspects of the enigmatic aetiology of the condition".

However, the most direct test of this hypothesis – whether mothers who suffer from influenza during pregnancy actually have children who are at greater risk of schizophrenia – does not support it. Of the 945 mothers who had influenza in the second trimester of pregnancy, no more than the expected three children had by the age of 28 years developed schizophrenia by broad criteria (Crow *et al*, 1991). This leads one to inquire more closely about the somewhat indirect correlational approach that leads Dr Sham *et al* to their conclusions.

The first sentence of the results section states that "The average response curve [Fig. 2(a) of their paper] shows an obvious increase in the average number of schizophrenic births after the start of influenza epidemics" but what this actually shows (since epidemics generally start in the late autumn) is the well-known season-of-birth effect. Years are defined as from November to October, a definition that maximises the impression that there is a relationship between the season of birth effect (whatever this is due to) and the timing of influenza epidemics. Dr Sham *et al* counter this objection by Fig. 2(b) which shows that the "average season of birth" curve for high influenza years differs from that of low influenza years but no *P* value is given for the example of comparison between 'high' and 'low' influenza years.

If there is indeed a significant difference here one might ask how far this is dependent on the particular analysis adopted? For example, if the authors had matched schizophrenic births following each high influenza year with those in the succeeding low influenza year defined from the month in which influenza deaths exceeded 100, would the conclusions have been the same?

A curious feature of the findings reported in Fig. 1(b) is the absence of an excess of influenza deaths for the year 1957. The authors have previously presented the claim (O'Callaghan *et al*, 1991) that there was an 80% increase in schizophrenic births in the general population following the 1957 epidemic of Asian influenza. But if 1957 was a low influenza year the increase in schizophrenic births following high influenza years should have been correspondingly greater. But careful scrutiny of Fig. 1(a) (the only raw data that the reader is permitted to glimpse) reveals no evidence that this is the case.

The reader searches for, but does not find, a critical examination of the hypothesis to see whether it holds up in the face of different assumptions. The data on which the authors have based their analysis have not been presented in a way which others can examine and reanalyse them.

CROW, T. J., DONE, D. J. & JOHNSTONE, E. C. (1991) Schizophrenia and influenza. *Lancet*, **338**, 116–118.

O'CALLAGHAN, E., SHAM, P. C., TAKEI, N., *et al* (1991) Schizophrenia after prenatal exposure to 1957 A2 influenza epidemic. *Lancet*, **337**, 1248–1250.

SHAM, P. C., O'CALLAGHAN, E., TAKEI, N., *et al* (1992) Schizophrenia following pre-natal exposure to influenza epidemics between 1939 and 1960. *British Journal of Psychiatry*, **160**, 461–466.

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AUTHOR'S REPLY: Dr Crow is right to be sceptical about the maternal viral infection hypothesis, for several recent hypotheses about the aetiology of schizophrenia have proved to be false (e.g. Crow & Done, 1986). However, his critique of our paper (*Journal*, April 1992, **160**, 461–466) is uncharitable, for we made clear in our final paragraph that the hypothesis was not yet firmly established, and that further research is necessary. What Dr Crow quoted was our attempt to state concisely the findings of the current analysis, according to the commonly accepted criteria for statistical significance. It is obvious that a degree of uncertainty is involved in the results of any single study. This applies also to the negative conclusion of Dr Crow's own study, the limitations of which have been discussed (e.g. Knight *et al*, 1990). Indeed, Mednick (unpublished) has presented findings which contradict those of Dr Crow's study. Dr Mednick and colleagues examined the antenatal records of all those schizophrenics born in Helsinki in Spring 1958, i.e. those who were exposed to the Asian influenza epidemic in the second trimester of gestation and who experienced an increased risk of schizophrenia (Mednick *et al*, 1988); the records showed that 87% of the mothers of these schizophrenics had suffered influenza during pregnancy, compared with 20% of controls. These data are likely to be more accurate than those quoted by Dr Crow, as they were recorded at the time of the infection, not several months later.

The statistical issues we encountered in our study were complex. Two previous investigations of the same question (Torrey *et al*, 1988; Barr *et al*, 1990) have employed two different approaches. Our preference was to combine graphical and modelling methods. An advantage of statistical modelling is the ability to examine several relationships simultaneously, while allowing for random fluctuations. However, the results of such a model, in terms of

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parameter estimates and significance tests, are often difficult for non-staticians to interpret. For this reason, graphical methods should be employed, to present the data in such a way that highlights and summarises the features relevant to the relationships in question. It is rare, and usually unhelpful, to present the 'raw data' of the study, especially when a large quantity of data is involved.

The graphs of our paper were presented in this spirit. The year was defined from November to October for the simple reason that a flu epidemic in November and December can only be related to a spring excess of schizophrenic births in the next, but not in the same, calendar year. This problem is highlighted by Dr Crow's erroneous comment about Fig. 1(b) and the flu epidemic of 1957. In fact, the Asian flu epidemic peaked in Autumn of 1957, and is therefore of potential relevance to the risk of schizophrenia in individuals born in the early months of 1958. Careful inspection of our Fig. 1(b) will reveal that the early months of 1957 did indeed experience a low rate of influenza deaths, but this was not so far for the later months of 1957. Indeed, the A2 influenza epidemic of that autumn was the most widespread epidemic to have occurred in the UK in the post-war period. The main reason that more influenza-related deaths were not recorded was that a similar strain of influenza had been common in the early years of the century, so that many older people, who are most likely to die following influenza, were immune to that strain of the virus.

No *P* value was presented for Fig. 2(b) because the issue of statistical significance was dealt with by statistical modelling, not graphical methods.

Our paper addressed a single, clear hypothesis, and we believe that it proceeded to examine the hypothesis in a reasonable and statistically correct manner. We are content to await the results of other epidemiological and experimental studies examining the prenatal influenza hypothesis, to see whether or not they confirm our findings.

BARR, C. E., MEDNICK, S. A. & MUNK-JORGENSEN, P. (1990) Exposure to influenza epidemics during gestation and adult schizophrenia: a 40-year study. *Archives of General Psychiatry*, **47**, 869–874.

CROW, T. J. & DONE, D. J. (1986) Age at onset of schizophrenia in siblings: a test of the contagion hypothesis. *Psychiatry Research*, **18**, 107–117.

KNIGHT, J. (1991) Schizophrenia and influenza. *Lancet*, **338**, 390.

MEDNICK, S. A., MACHON, R. A., HUTTENEN, M. O., *et al* (1988) Adult schizophrenia following prenatal exposure to an influenza epidemic. *Archives of General Psychiatry*, **45**, 189–192.

TORREY, E. F. RAWLINGS, R. & WALDMAN, I. N. (1988) Schizophrenic births and viral diseases in two states. *Schizophrenia Research*, **1**, 73–77.

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Preconscious perceptual processing

SIR: Klemperer (*Journal*, September 1992, **161**, 420) suggests that a failure of preconscious processing underlies *all* perceptual experiences in which the subject makes a faulty interpretation of an external stimulus.

In my paper I alluded to the possibility that the model of a cycle of false belief and misperception reinforcing one another, which I use to explain delusional misidentification, may be relevant to the development of some delusional disorders (see section Seeing and believing in the delusional disorders). However, this model is not able to explain all perceptual distortions; for example, it cannot explain the unusually vivid visual experience of a hypomanic patient.

I agree with Dr Klemperer that better understanding of the interpretative aspects of preconscious processing of perceptions is essential for our understanding of the psychopathology of perceptions.

It is possible that such processes may also be relevant to the development of hallucinations, and help to explain the temporal coincidence of the development of delusions and hallucinations, which appear otherwise to be quite independent of one another, which is commonly observed in psychotic relapses. The reasoning processes that underlie normal belief formation (Alloy & Tabachnik, 1984) and those that are involved in the interpretive aspects of preconscious perceptual processing are similar; there is a trade-off between prior expectation and the quality of current situational information or sense data. A single reasoning fault might result in interpretations being uncoupled from the evidence on which they should be based, to produce delusions if the evidence is current situational information, or hallucinations if the evidence is sense data.

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