venture that they are themselves mistaken and that the evidence cited above effectively undermines the case that prenatal exposure to influenza is a cause of schizophrenia. I would be more cautious in expressing this opinion were I not also convinced that the ecological evidence that they cite is fraught with inconsistencies and contradictions (Crow, 1994).

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REPLY: In our recent letter (Adams & Kendell 1996) about a paper by Cannon *et al* (1996) we made a passing reference to an earlier study by Crow & Done (1992). Crow now claims that our argument involved two errors, "the first trivial ... the second crucial".

The first is easily disposed of. It arises out of our statement about Crow & Done's study that "even though they had 945 mothers with a history of flu-like illness in the second trimester of pregnancy they only had 7 schizophrenic offspring". The disputed figure 7 refers to the total number of offspring with a diagnosis of schizophrenia (broad diagnostic criteria) produced by the 1851 women describing a flu-like illness during pregnancy (0+3+4) in Crow & Done's Table 2; that is, "they" in the sentence above refers to Crow & Done, not to the 945 mothers, and we apologise for the ambiguity.

We do not accept, however, that our argument was fallacious. On the contrary, we believe that there is a serious error in Crow & Done's arithmetic, which Crow repeats in his present letter. With the data available to them Crow & Done could have used either or both of two arguments to justify their claim to have established that exposure to influenza in the second trimester of pregnancy is not associated with any increased risk of schizophrenia in the child. They could have argued that there was no significantly increased risk of schizophrenia after exposure to influenza in the second compared with the first and third trimesters of pregnancy. The point of our passing reference was that the total number of cases of schizophrenia (7) identified after maternal influenza at any stage of pregnancy was far too small for this to be practicable. Instead, Crow & Done chose to compare the risk of schizophrenia in the offspring of the 945 mothers describing flu-like illness in the second trimester of pregnancy with the risk in all 16 268 mothers in the NCDS cohort, 88.6% of whom had not described a flu-like illness at any stage of pregnancy, and set out to test the hypothesis that the risk of schizophrenia after maternal influenza in the second trimester of pregnancy was increased by a factor of 1.87 (very similar to our hypothesis that the risk is doubled). They then suggested that this hypothesis required the excess number of schizophrenic births (broad diagnostic criteria) to "be 26.5 ($57 \times 0.87/1.87$)" and the observed number to be 29.5 rather than 3.

This is, quite simply, wrong. Using broad diagnostic criteria, 57 schizophrenics were identified by Crow & Done among the index children of the 16 268 mothers in the NCDS cohort. This represents one schizophrenic child for every 285.4 mothers, and one schizophrenic child for every 283.8 mothers among the 15 323 who did not report a flu-like illness in the second trimester of pregnancy. This rather low yield is presumably attributable to the imperfect means of case ascertainment employed. None the less, it represents the base rate in the cohort. One would therefore expect the 945 mothers who described a flu-like illness in the second trimester of pregnancy to yield $945/283.8 = 3.33$ schizophrenic offspring, if there was no increased risk involved, and $(945/283.8) \times 1.87 = 6.23$, if there was an 87% increase in risk. The difference between the expected number of 6.23 and the observed number (3) could easily be a chance finding (an exact test gives a probability of 0.15, or 0.12 if the hypothesis is a 100% rather than an 87% increase in risk), confirming our previous claim that Crow & Done had too few schizophrenic subjects to test their hypothesis adequately. Equally importantly, the figure of 6.23 bears no relationship to the expected figure of 29.5 (26.5+3) calculated by Crow & Done.

Exactly similar arguments would apply had narrow diagnostic criteria been used. Indeed, the numbers, both observed and expected, would have been even smaller. We therefore hold to our previous view that Crow & Done (1992) were not

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justified in concluding that their evidence refuted the hypothesis that exposure to influenza in the second trimester of pregnancy was associated with an increased risk of schizophrenia. Their numbers were too small and their arithmetic faulty.

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The uses and limitations of mental health service needs indices

SIR: While it is now taken as axiomatic that local mental health services should be planned in relation to needs, the definition of need, the distinction between population and individual levels of need, and the ways in which need can be operationalised all remain to be clarified. The paper by Smith *et al* (1996) adds further quantitative evidence to support the population-level estimates of need, which have been described elsewhere (Thornicroft *et al*, 1992). The paper concludes that the index of needs produced by Smith *et al* shows that the expected rates of local service use may vary by a factor of more than three times between Huntingdon and Central Manchester, the least and most morbid areas included in the study. At first sight this is a remarkable finding given that actual National Health Service expenditure on mental health services varies by a factor of about 1.5 in practice. Nevertheless, the findings of Smith *et al* may still be an underestimate. The recent household National Psychiatric Morbidity Survey, conducted by the Office of Population Censuses and Surveys (1995), revealed a range of annual period prevalence for psychotic disorders between 0.2 and 0.9% of the total population, that is a 4.5-fold variation. Similarly, the MINI computerised program by Glover (1996) also finds approximately four-fold variations in actual utilisation of psychiatric in-patient beds. There does seem to be steadily accumulating evidence that the most and least socially deprived parts of Britain will have, approximately, morbidity levels double and half the national average, respectively; that is a four-fold variation.

Despite the value of the paper by Smith *et al*, and the parallel work in producing the MINI index, these calculations are still, at this stage, of somewhat academic interest. Such formulae guide the allocation of monies from the NHS Executive to purchasing authorities. Purchasing authorities have very wide degrees of discretion about how their total budget may be spent for their varying categories of health care. There is no overall discernible pattern of actual expenditure at the provider level, which varies in relation to any assessment of population need. If there is an overall national pattern, it reflects somewhat higher expenditure in districts which did have long-stay institutions and which appear to have retained some budgetary advantage. Two issues arise: first, in the funding distribution formula adopted by the Department of Health, which is a variation on the proposals put forward by the York Group two years ago, there remains a 20% component of the total budget which is not currently weighted. It is therefore open to discretion of the NHS Executive to re-weight this in line with emerging evidence of variations in mental health care needs that are greater than previously expected. Second, it is becoming clearer that it will be necessary for the NHS Executive to provide clearer guidance to purchasers on the expected proportion of their total budget which should be spent for mental health services in each local area.

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Treatment of severe personality disorders

SIR: As a member of a community mental health team, that in our case includes the local in-patient unit, I found the paper by Norton & Hinshelwood (1996) on the treatment of severe personality disorders troubling in many respects. It reads like a sales pitch for a particular type of facility, particularly given the obvious commitment of the authors

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