the induction of p450 enzymes by carbamazepine. This leads to a lowering of serum tricyclic concentrations when tricyclics are given with carbamazepine, thus reducing their antidepressant action (Leinonen *et al*, 1991; Preskorn, 1993). Such interactions altering the serum concentrations of tricyclic antidepressants are unlikely to be adequately appreciated until the measurement of serum tricyclic antidepressant concentrations becomes routine clinical practice, especially for cases that are difficult to treat.

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Cross-cultural studies

SIR: The success of the Bradford Somatic Inventory (BSI) in identifying minor psychiatric morbidity in a rural Pakistani district underlines the value of instruments which have been validated in ways that are language and culture sensitive (Mumford *et al*, 1996). The identification of these psychiatric cases however recalls the argument of the legitimacy of imposing Western psychiatric diagnoses in non-Western societies (Kleinman, 1977). In stating that to the casual Western visitor life in this remote district appears idyllic and contented suggests that the authors, while good intentioned, may have fallen into the trap of the ethnic psychiatrists of the past, who sought out psychopathology in exotic places while reinforcing colonial and neocolonial attitudes and behaviour (Bhugra, 1993). It is important to recognise that translation (as in establishing a diagnostic index in one setting and then translating it so that it can be used in another) does not ensure congruity of sociocultural perspectives. Translation only ensures that there is an approximate equivalence between categories whose origins lie in very different social contexts (Skultans, 1993).

It is therefore unfortunate that there is no account of how the villagers themselves interpret and articulate their distress as psychiatry itself is a Western construct and imposes its own sociocultural parameters when observations are made on its behalf. In addition, the lack of information about physical illness is a hindrance to the accurate interpretation of the findings since the correlation of deprivation with both physical and psychiatric morbidity is well recognised and the diagnosis of any psychiatric illness must be related to the presence of physical illness.

There is undoubted value in cross-cultural psychiatric research as mass migration is creating increasingly multicultural Western societies. However, when psychiatry co-opts non-Western experience in ways that would allow the distress that is derived from that experience to be interpreted and categorised in Western terms, the native interpretations are often undermined. What is also lost is the capacity to incorporate this perspective into Western formulations so that the delivery of care in multicultural Western societies can be more relevant and effective. A goal that continues to be elusive.

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Lithium and weight gain

SIR: Previous studies show that weight increases during the first two years of lithium therapy and then stabilises, that a quarter of patients do not put on weight at any stage (Vestergaard *et al*, 1990) and that a quarter become obese (Chen & Silverston, 1990). Consequently the BNF mentions weight gain as a problem. This was investigated in a lithium clinic where in 17 years no patient had dropped out because of weight gain.

Forty-two unipolar and bipolar patients on lithium for 1–25 years with or without concomitant antidepressants or neuroleptics were weighed in shoeless light attire and their height measured. Using the same balance arm weighing machine and measure, controls similarly attired from the same locality and not suffering from any known

untreated illness were matched from the general practice computer by sex, height within 2 cm, age within 2 years (except that a woman born in 1916 had a control born in 1912, and a man aged 75, height 1.66 m was paired with a man aged 74 of height 1.70 m). The first presented match was used, therefore being random in regard to weight.

First, 19 patients who had taken lithium regularly with or without antidepressants or neuroleptics for 10–24.5 years (average 15.5 years), had a combined weight of 1507.3 kilos compared with 1500.6 kilos of the matched controls.

Second, the 42 patients on lithium had a mean BMI of 27.7 with the control's mean BMI being 27.5. With a BMI of 30 or over, 10 lithium patients were obese as were 11 controls.

Third, paired t-tests, applied to patients and their matched controls, show that for 17 patients taking lithium only, $t=1.56$, $P=0.14$, and for 25 patients taking additional neuroleptics or antidepressants, $t=-0.5$, $P=0.81$.

Thus in 400.5 lithium-patient years, with or without tricyclic antidepressants or neuroleptics, weight gain was not significant. The same conclusion was reached at Epsom's Affective Disorder Clinic where patients were weighed regularly for 5 years after commencing lithium (Coppen, 1994).

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Influenza and schizophrenia

SIR: Cannon et al (1996) traced a small cohort of women known to have developed a flu-like illness in pregnancy at the time of the 1957 influenza epidemic, compared them with matched controls with no history of influenza and failed to find any higher incidence of schizophrenia in the children of the former than of the latter (2 v. 2). On this basis they concluded that "our results do not support the hypothesis that individuals exposed to influenza in prenatal life are at increased risk of schizophrenia". As Woodgate & Curtis (1996) have already observed, this is indeed true, but neither do their results threaten the hypothesis, for their cohort was far too small.

Ecological studies of the 1957 epidemic of A2 influenza in Finland, England, Scotland, Denmark and Queensland all found a significantly increased incidence of schizophrenia in the offspring of women exposed to that epidemic during the second trimester of pregnancy, but only in that trimester. The hypothesis to be tested, therefore, is whether or not maternal influenza in the second trimester of pregnancy is subsequently associated with an increased incidence of schizophrenia in the child. Unfortunately, only 80 of Cannon et al's 238 index mothers had had flu-like illnesses in the second trimester and to test the hypothesis they would have needed at least 2700.

Depending somewhat on the diagnostic criteria employed, the lifetime risk of schizophrenia is about 0.85%. For simplicity, let us assume a lifetime risk of 1%, and also ignore the fact that Cannon et al's subjects were only 33 or 34 years old at the time of study, and therefore not yet out of the risk period for schizophrenia, and that all of the flu-like illnesses recorded may not have been influenza. Let us also assume that maternal influenza in the second trimester of pregnancy doubles the child's lifetime risk of schizophrenia (or, alternatively, that exposure during an unidentified three week period in that trimester increases the risk 8-fold). If Cannon et al had wished to leave only a 25% risk of failing to detect a doubling of the incidence of schizophrenia in their subjects' offspring (Type II error) and only a 5% chance of obtaining, by chance, a spurious doubling of that risk (Type I error) the sample size they would have needed can be calculated by solving the two equations:

$$\alpha = \sum_{r=r_0}^{r=t} \text{Bin } (r; t; P)$$

$$\beta = \sum_{r=0}^{r=r_0-1} \text{Bin } (r; t; P)$$

where α is the value of the Type I error, β the value of the Type II error and Bin $(r; t; P)$ the binomial probability of r events in a sample size t when the probability is P. It is important to appreciate, though, that t refers to the number of schizophrenic offspring rather than to the number of mothers (i.e. for Cannon et al, $t=4$). If there is no difference between the number of schizophrenic offspring produced by the index and control mothers, $P=P_0=\frac{1}{2}$. If, as we hypothesise, the number of schizophrenic offspring is twice as high for the index mothers as it is for their controls, $P=P_1=\frac{2}{3}$.