or England and Wales cannot be generalised to other parts of the world such as India. India is the second most populous country in the world and approximately 80% of the population are farmers and poorly educated. Unfortunately, even after more than 50 years of Indian independence, farmers, who are perceived to be the backbone of the nation, are neglected in many ways. Because of this, last year, in the northern part of Karnataka State (south India), many districts like Bidar, Gulbarga, Raichur and Bijapur experienced not less than 100 suicides of farmers within three months (March–May) ("Another farmer commits suicide", "Money-lenders blamed for farmers' suicide", "Bellary farmers end life over crop loss", *Deccan Herald*, 15 March, 3 April and 11 May 1998). Today, the suicide rate in farmers in India is still not known. Malmberg *et al* discussed the lacunae in understanding of farmers' suicide problems and this is an area for more research.

In contrast to the Hawton *et al* study, farmers' suicides in India may be associated with different problems such as harassment by money-lenders, inability to repay debts following crop loss, inability to get medical treatment for the family, etc.

In developing countries like India globalisation and industrialisation are prominent, with multi-national companies competing in the industrial and agricultural sectors. This adds, directly or indirectly, to numerous problems such as supply of low-quality seeds to farmers, sales of sub-standard alcoholic drinks, sub-standard pesticide production and frequent power cuts and irregular power supplies. The lack of positive and cooperative support from banks and too many restrictions make problems worse. Unless there is a supportive government policy for safeguarding farmers during inclement weather or market fluctuations, and until banking systems are put in order, the plight of farmers will remain the same.

Furthermore, suicide in farmers could be controlled by effective and efficient community care, which has been a cornerstone of policy for mental health services for the past half-century (Tyrer, 1998). Policy concerns and research efforts should focus on reducing suicide in farmers.

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Fever and acute brief psychosis in developing countries

Sir: The study by Collins *et al* (1999) represents a significant advance in addressing the aetiological factors underlying acute brief psychosis, a common problem in developing countries but with very little systematic data available. Although the authors have only considered the aetiological significance of viral infections such as influenza and herpes simplex, the real difference between developing and developed countries lies in the prevalence of bacterial and protozoal infections. It appears that the authors have overlooked the significance of these commonly occurring infections and the drugs used to treat these in the aetiology of acute brief psychosis.

Two common causes of fever in developing countries are malaria and typhoid fever. These infections, as well as their treatments, may have direct aetiological implications for acute brief psychosis. Apart from the indirect effects of malaria and its complications on the activation of psychotic symptoms, cerebral malaria may have similar direct effect. Cerebral malaria is not uncommon in these settings. It has been reported in 20–40% of patients admitted with fever and altered consciousness (Durrani *et al*, 1997). Psychosis is also considered one of the side-effects of the most commonly employed treatment for malaria, chloroquine.

A toxic confusional state characterised by disorientation, delirium and restlessness, is characteristic of late-stage typhoid but, occasionally, these and other neuropsychiatric features may dominate the clinical picture from an early stage (Osuntoken *et al*, 1972; Breaky & Kala, 1977). Moreover, it is well known that the quinolones, now the most effective and widely used group of antibiotics in typhoid, can cause psychotic symptoms such as hallucinations.

These points serve to highlight the interplay of various biological factors in the aetiology and perhaps in the manifestations of acute brief psychosis in developing countries. There is a dire need for similar studies on the subject in these settings, which may also help in a search for finding the aetiology of psychosis in general.

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People at risk of schizophrenia and other psychoses: comments on the Edinburgh High-Risk Study

Sir: Hodges *et al* (1999) present a research design to study the process of transition to psychosis. Our purpose is to make some constructive comments on the paper and to describe an alternative research strategy which has similar aims.

Although the paper attracted criticism, mainly because of views about premature publication, we believe the study has merit. The problems of the study, however, relate more to design issues. The authors highlight the failure of the traditional high-risk strategy to realise its potential, and propose a partial solution. This failure is partly a result of the long latency to expression of risk in most (but not all) of these studies, and partly is due to the sole reliance on family history as a risk marker. The Edinburgh study appears at first glance to address the first weakness, yet this turns out to be a limited solution. The second weakness remains. The sample is defined as high-risk on genetic grounds only, a necessary consequence of restricting the sample to 'well' or pre-symptomatic individuals. This ultimately extends the latency of the expression of risk and hence the length of the follow-up period and limits the generalisability of the findings to psychosis or schizophrenia as a whole, since a small minority only of cases have this pattern of family history. The advantage is that the patients who develop psychosis will have been assessed from the asymptomatic

stage through to full expression of the disorder. A problem here may be that the snapshots are too widely spaced at 18 months. In addition, the estimated ultimate rate of transition is still quite low, around 15%. The costs for the design, and hence the funders, the subjects and the research team themselves, are that a large sample size and an extensive follow-up period are required. The study is, therefore, expensive and labour-intensive. Farmer (1999), in her commentary, estimates that only half of those making the transition will have done so within five years and Johnstone (1999) acknowledges that her co-workers may not be around for long enough to reap the fruits of their labours.

Hence, the main strength of the study is the modern assessment using imaging and other methodologies from asymptomatic stage through to full syndrome, a latter-day, enhanced version of earlier high-risk studies. The most interesting finding in the study to date is the high functioning observed in a subset of the young people at risk. A final quibble is the notion expressed in the author's response (Johnstone, 1999) that those ultimately expressing the disorder were 'destined' to do so. This is too deterministic. We think that the expression of risk is more dynamic and that the group who ultimately express the phenotype is not fixed at the start of a prospective study like this. It may be more like 'musical chairs' with risk factors such as substance use and stress operating to select the final sample.

An alternative design which addresses these problems, known as the 'close-in' strategy (Bell, 1992), has been applied by our group (Yung *et al*, 1996, 1998*a*) to the challenge of predicting, clarifying and trying to delay or prevent the transition to psychosis in a high-risk sample. Building on the concept of indicated prevention (Mrazek & Haggerty, 1994), we focus on cases with early clinical features which are associated with a high risk (approximately 41%) of transition to psychosis within 12 months (Yung *et al*, 1998*b*). In a proportion of these, there is a first-degree relative with a psychotic disorder, while others are defined on subthreshold or attenuated psychotic symptoms alone. It is important to emphasise that over half of these patients do not develop psychosis, though they do have other axis I disorders. This design does not allow study of the process of transition from as early as the asymptomatic period, but it does seem to address the two key weaknesses of the traditional high-risk approach.

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Comparing ICD–10 and DSM–IV

Sir: We wish to comment on First & Pincus's (1999) editorial, which itself is in part a response to the earlier editorial by Andrews *et al* (1999) on the comparability of ICD–10 and DSM–IV classifications. First & Pincus make much of the fact that Andrews *et al* have not compared DSM–IV to the 1992 clinical guidelines version of ICD–10, which influenced the development of DSM–IV. Instead, Andrews *et al* compared ICD–10 to the Diagnostic Criteria for Research version published in 1993 (World Health Organization, 1993). First & Pincus point out that there are subtle differences between the 1992 clinical guidelines and 1993 diagnostic criteria for research versions of ICD–10. Although this is true, comparing DSM–IV to the 1992 clinical guidelines version of ICD–10 would not be appropriate, and is probably not possible, since the clinical guidelines are descriptive only and are not in the same operationalised format as DSM–IV. In order to compare like with like, the 1993 operational definitions version of ICD–10 must be used.

We disagree that in "the world of research, the DSM system of specified diagnostic criteria is the *de facto* standard". Although the DSM system from the third edition onward has had an important influence in improving the reliability of psychiatric diagnosis, it remains a *national* system. Despite the aims of its authors to be 'atheoretical' the DSM must inevitably reflect the current culture and ideology of North American rather than world psychiatry. On the other hand, the ICD–10 classification has been derived on the basis of field trials and debate internationally, and is the official classification for many countries. ICD–10 can arguably be considered to have greater universality in terms of its international acceptance and use.

We accept that the literature review process and reliability studies that accompanied the most recent revision of the DSM system probably represent certain advantages over the field trial method undertaken for ICD–10. However, the latter were carried out in the late-1980s (our own in Cardiff was undertaken in 1988), whereas DSM–IV was published nearly a decade later. One would hope for an improvement in methodology over such time. Also, it is considerably easier to arrange detailed and costly studies of a nosology in a single rich country, than to undertake such testing in many countries, with associated differences in economy, language, custom and religion.

Finally, a point we have reiterated many times, is that no classification as yet has proven validity, since the causes of most mental illnesses remain uncertain. Only when the aetiology of psychiatric disorders is properly understood, will it be possible to identify the most valid classification. Until then all classifications must be considered as working hypotheses. Thus, there is more than a hint of diagnostic imperialism in First & Pincus's assertion that DSM–IV should be the accepted standard, and that "the introduction of the ICD–10 criteria is the main source of confusion among researchers . . . [and that] many (if not most) of the differences seem to exist for no good reason". Others besides ourselves have written about the comparability and differences between operational criteria (Farmer *et al*, 1991*a*,*b*). The current arguments relating to whether DSM–IV is 'better' than ICD–10 merely induce a state of *déjà vu*, and hopefully will not need to be