The need to research this field is paramount in view of increasing reports of 'perceived' or 'real' unmet needs of the mental health of Asians in the UK.

We at Leicester would be keen to set up a joint (bi-centre) pilot study with the author to investigate the 'pathways to psychiatric care for Asian patients'. Of the population of the city of Leicester, 25% is Asian; moreover, a comparison of the first and second generation Asian immigrants would be a useful study to understand the process of acculturation, for which Leicester is such a fertile experimental ground.

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Eating disorders among Asian girls in Britain

SIR: Arya (*Journal*, January 1992, **160**, 131–132) attempts to refute the hypothesis that intergenerational conflict contributes to the development of eating disorders among British-born Asian girls in Britain. It is surprising that he does not discuss the evidence presented in our epidemiological study in Bradford (Mumford *et al*, 1991). At the outset, we had assumed (like Arya) that the most 'Westernised' Asian girls would be at highest risk of developing an eating disorder: the adoption of western cultural values might make them more vulnerable.

However, in our study we found that, on the contrary, it was the Asian girls from most *traditional* families (as determined by Asian language and dress) who had the highest scores on the Eating Attitudes Test and Body Shape Questionnaire, and who were subsequently diagnosed at interview as having a DSM–III–R eating disorder. The degree of Westernisation (as measured by the use of the English language at home and eating western food) was not related to questionnaire scores nor to the likelihood of receiving an eating-disorder diagnosis.

These findings require an explanation. We have argued that all Asian girls growing up in Britain and attending school here are heavily influenced by prevailing majority cultural values. The girls who experience the most *conflict* are those who come from the most traditional families, not those from families which have already adopted Western values and outlook.

The specific cultural indicators of intergenerational conflict are likely to vary with different populations. We have repeated our Bradford study in English-medium schools in Lahore, Pakistan (Mumford *et al*, 1992). Here it was girls who were most 'Westernised' (as measured by the use of the English language at home and a Western diet) who had the highest scores on the Eating Attitudes Test and Body Shape Questionnaire, and who were subsequently diagnosed at interview as having a DSM–III–R eating disorder. Unsurprisingly, responses to items measuring traditional orientation (Asian language and dress) were not related to questionnaire scores nor to the likelihood of receiving an eating-disorder diagnosis, since these are prevailing cultural norms in Lahore.

The hypothesis of intergenerational conflict finds support in studies of adolescents in Kuwait, a conservative Muslim society which was experiencing rapid social change by exposure to Western values, even before the Gulf War. El-Islam *et al* (1986) found that adolescents' scores on the General Health Questionnaire were associated with greater *difference* in cultural attitudes between parents and children; there was no relationship between symptoms and 'liberal' attitudes as such.

Intergenerational conflict is widely accepted as a causative factor in many anorexics in Western culture. I regard the tensions which arise from cultural discrepancies between Asian parents and children as merely one specific instance of such conflict. It is highly likely that intergenerational conflict plays a major role in the development of eating disorders among Asian girls in Britain.

EL-ISLAM, M. F., ABU-DAGGA, S. I., MALASI, T. H., *et al* (1986) Intergenerational conflict and psychiatric symptoms. *British Journal of Psychiatry*, **149**, 300–306.

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—, — & CHOUDRY, I. V. (1992) A survey of eating disorders in English-medium schools in Lahore (Pakistan). *International Journal of Eating Disorders* (in press).

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Seasonal risk factors

SIR: Pulver *et al*, in their re-examination of the evidence for a seasonal risk factor (*Journal*, January 1992, **160**, 65–71, 71–75) in the development of schizophrenia, have neglected to mention several other possible explanations for the trend towards winter/early spring birth dates. Many other conditions whose aetiology is at present unknown have also been recognised as having a similar seasonal

variation. While those observations provide a clue to aetiology, their interpretation is problematic. Many potentially pathological influences vary with the season. Seasons influence the plant and animal environment with changes in the availability of food and in the vectors and hosts of infectious agents. There are also seasonal changes in many other aspects of human behaviour such as occupation and recreation. Some of these changes are reflected in physiological changes in the body. Serum calcium levels, for example, tend to be lower in the winter and early spring than at other times.

A similar excess of winter/early spring birth dates has been recognised in congenital dislocation of the hip (Record & Edwards, 1958) and spina bifida (Leck, 1972). In the former condition, swaddling infants with the hips in extension as seen in Laps and American Indians is a causative factor. As in the previous work of Pulver *et al* (1990) this risk factor is correlated with gender. Females are less prone to this seasonal influence, presumably because other aetiologies such as shallow acetabulae and temporary hormonal joint laxity predominate in female infants. In spina bifida the observation of a seasonal variation in birth incidence has led most investigators to investigate a number of possible causative 'summer teratogenic' factors.

In some conditions, careful data collection has revealed changes in the seasonal influence with time. Anencephaly, as a cause of stillbirth, for example, appeared to lose a previously well recognised seasonal variation (Leck & Record, 1966), without any reduction in incidence. Future investigators into the seasonal trend factor in schizophrenia need to be mindful of the large variety of possible influences at work.

LECK, I. (1972) The etiology of human malformations: insights from epidemiology. *Teratology*, **5**, 303–314.
— & RECORD, R. G. (1966) Seasonal incidence of anencephalic stillbirths in Scotland by quinquennia. *British Journal of Preventive and Social Medicine*, **20**, 67.
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Peripheral audiosensitivity

SIR: Bohman *et al* (*Journal*, December 1991, **159**, 860–863) described a subjective test for assessing intolerance for graded loud sounds. They seem unaware that there is a large literature on the Loudness Discomfort Level (LDL) Test (Dix, 1974), a standard quick procedure in clinical audiology for measuring loudness recruitment. Patients with cochlear deafness do not hear quiet sounds, but their LDLs are normal or only slightly raised. Working in an ENT clinic, I have not found the LDL test very useful. Objective acoustic stapedial reflex thresholds give the same information about recruitment and loudness registration, and LDLs correlate poorly with patients' complaints of loudness intolerance. If one is going to rely on subjective measures, there is no substitute for a detailed history.

Dr Bohman *et al* highlight the terminological confusion between lowered tolerance of higher intensities and hypersensitivity for threshold levels. Actually, the situation is more complex, at least in the audiological literature, since a third phenomenon, recruitment, has been confounded. I have tried (Gordon, 1986) to clarify this issue by restricting the term recruitment to a property of a damaged cochlea, whereby low-intensity loudness receptors are destroyed, middle-intensity ones impaired and high-intensity ones remain. On the basis of a careful history, audiosensitivity was defined as aversion to the output of TVs, radios or record players at levels tolerated by normal listeners. I restricted the term hyperacusis to better than normal pure-tone thresholds. Recruitment can only occur in patients with raised pure-tone thresholds or cochlear lesions, while audiosensitivity can occur in any patient, often with normal thresholds (e.g. autistic children, neurotics, Williams' syndrome, etc.). The relationship between audiosensitivity and hyperacusis is unclear since clinically they may occur independently, yet both are associated with incipient Menier's disease, lumbar puncture, etc. (Gordon, 1986, 1991). Audiosensitivity has a clear physiological basis, correlating with reversed or hyperactive stapedial reflexes (Gordon, 1986). Curiously, I could find no aural correlate of intolerance to loud noises other than those from audio equipment, i.e. audiosensitivity.

Dr Bohman *et al* assume that the sensory changes after head injury must be cortically mediated, and they implicate the highest level, the frontal area. They do not consider any lower level dysfunction. However, there is much clinical and anatomical evidence showing the post-concussional syndrome to be of labyrinthine origin (Gordon, 1989; Grimm *et al*, 1989).

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— (1989) Post-concussion syndrome. *Australian & New Zealand Journal of Psychiatry*, **23**, 154–155.