severe trauma only within a window of vulnerability ending in early adolescence.

If anyone in Britain, South Africa, or any other country outside North America would like to conduct such a study, I can be contacted at the address below. Once such studies had been conducted, we could then begin a scientific discussion.

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Life events and management in schizophrenia

SIR: We have previously reported (Tarrier *et al*, *Journal*, October 1988, **153**, 532–542) on the success of a behavioural intervention with families to reduce relapse rates in schizophrenic patients living with high expressed emotion (EE) families compared with a short educational intervention and routine treatment. We also found that in families receiving the family intervention there were significantly greater changes in the relatives' ratings of EE from high EE to low EE over the nine month follow-up. Hence there appears to be an association between relapse rates and change in the relatives' EE ratings. It could be hypothesised that reduction in the relatives' EE resulted in reduced relapses, although these data do not provide unequivocal support for this hypothesis. We were interested in examining alternative hypotheses for the different relapse rates. It could be possible that the family intervention resulted in the patient having greater contact with the psychiatric services in general, or receiving higher doses of medication or showing greater medication compliance. However, we could find no evidence to support these alternative hypotheses.

A further possibility that we did not examine at the time, although the data were collected, is that patients in the high-EE education and routine treatment groups experienced more independent life events than patients in the family intervention groups, the occurrence of major life events being associated with relapse. Data on life events over the nine month follow-up period was collected on 77 patients. Of these, 50 (65%) did not experience a life event. Three patients (16%) from the low-EE groups, six (20%) from the high-EE education and routine treatment groups, and 17 (61%) from the behavioural intervention group experienced at least one life event. A Kruskel-Wallis one-way ANOVA demonstrated that this difference was highly significant ($\chi^2 = 19.02$, $P < 0.001$) due to the very high frequency of life events in the behavioural intervention group. The hypothesis that a higher frequency of life events would be associated with the higher relapse rates in the high-EE education and routine treatment group was not confirmed. In fact, patients in the behavioural intervention group experienced more frequent life events and showed a decreased frequency of relapses. Similar results have been reported by Falloon and his colleagues in their intervention study (Hardestry *et al*, 1985).

These results suggest that family interventions designed to improve the family members' ability to cope with stress are successful in reducing the negative effects of major life events. This is evidenced by one patient in the family intervention group who had a 25-year history of schizophrenia involving 13 hospital admissions. She experienced seven independent life events over the 9 month post-discharge period, but remained symptom free over this period and was still well at two-year follow-up.

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Pimozide in pathological jealousy

SIR: It was with considerable dismay that I read Cohen's scathing attack (*Journal*, November 1988, **155**, 714) upon our brief report (*Journal*, August 1988, **155**, 249–251). Our suggestion that pimozide

improved our patient's morbid jealousy did not convince him, and indeed, he seems to believe that we actually missed an alcoholic in the process. The facts of the matter are these: the patient was not an alcoholic, although he did take alcohol at least once per week, and he would have been treated as such were such a condition present. The patient responded well to pimozide, at the dosage stated, and remained symptom-free for 5 months. At that time, he chose to discontinue his pimozide therapy, and the pathological jealousy re-emerged. Pimozide was once again used, with an excellent response again being noted. The patient had taken no alcohol for several months at that time, and there was no question of ethanol being involved in the syndrome. These details might serve to convince Dr Cohen of the efficacy of this preparation in the treatment of this syndrome.

Lamentably, alcoholism is all too frequently seen in our country, and had it been present in this situation, the problem would have been addressed. Dr Cohen is correct in assuming that drinking is discouraged among alcoholics on admission to our unit – but this patient's symptoms could not be attributed to that cause, and he was not managed along the lines suggested, for this very reason.

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Zinc taste test and postnatal depression

SIR: Bryce-Smith & Simpson (1984) have reported isolated patients with anorexia nervosa and "other depressive or neurotic states" being unable to taste a dilute zinc solution and responding to subsequent treatment with zinc sulphate, resulting in improved taste and mood. Additionally, there have been suggestions that postnatal depression may be linked to low levels of zinc caused by the demands of pregnancy, childbirth (with loss of placental zinc), and lactation, exacerbated by low dietary intake of zinc and by stress (Meadows *et al*, 1981; Ministry of Agriculture, Fisheries and Food, 1981; Cousins, 1986). Inability to taste a very dilute solution of zinc sulphate (the zinc taste test) has been proposed as a screening tool for identifying zinc deficiency.

We would like to report two investigations which question the association between postnatal depression and failure of the zinc taste test.

The first was in 12 patients being treated in a psychiatric unit for various forms of severe postnatal depression who were given the zinc taste test as described by Bryce-Smith & Simpson at between 2 and 30 weeks after delivery (Mean = 11 weeks). Seven reported no or a weak taste and five a positive or strong taste. According to Bryce-Smith & Simpson's (1984) criteria, no taste or a weak aftertaste would indicate a poor zinc status. In practice, it is very difficult to differentiate between "an aftertaste with no taste immediately apparent" and a "definite taste which intensifies with time". Many of the mothers found it difficult to describe the taste sensation. Only one of the patients reporting a weak taste (different from water) said that she could not taste anything initially, and it is notable that only one patient showed a complete inability to taste.

The second investigation was a prospective population study starting in mid-pregnancy, and involved less severe forms of postnatal depression. Mothers returned a questionnaire, some weeks after delivery, containing the Irritability, Depression, Anxiety Scale (Snaith *et al*, 1978), the subscales of which were combined to give a total depression score. Seventy-six per cent of these were returned between 9 and 19 weeks postpartum.

Of all the patients, 20% scored over 16 on the depression scale and were judged to be showing signs of mild to moderate postnatal depression. Higher scores were equated with increased severity of depression, and 9% scored over 20.

A total of 337 of these mothers had had the zinc taste test administered within 48 hours of delivery. The taste test was carried out as before, with the responses divided into 'tasters' and 'non-tasters'. Two hundred and ninety-seven patients who could taste the zinc solution returned depression scores with a mean of 11.33 ± 6.7, and 40 non-tasters returned a lower but not significantly different mean score of 10.75 ± 5.6.

We conclude, from unselected population data, that the zinc taste test is not useful as a predictive test for mothers suffering from post-natal depression. Zinc solutions are being marketed with claims of both diagnostic and therapeutic value. We feel that there is no evidence at present to support this practice in relation to postnatal depression.

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