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HYSTERIA AND URBANIZATION

DEAR SIR,

It is interesting that Fukuda *et al* (*Journal*, September 1980, **137**, 300–301) have reported a decline in the incidence of hysteria in Japan over a period of two decades. Whether this is a real decline is a matter of debate, and the finding may, in fact, represent a change in diagnostic fashions or criteria. Further, the speculation by Fukuda *et al* that the risk for hysteria may be related to the changes in life-style attendant upon urbanization, is refuted by epidemiological reports from India (Dube, 1968, 1970) where hysteria has been found to show the highest prevalence in *rural* areas. Dube (1970) also found that ". . . The usual setting for hysteria is a joint family . . ." which contradicts Fukuda *et al's* conjecture that the loss of traditional sociocultural ties may predispose to hysteria. On the contrary the disorder seems to be more common in families which adhere to orthodox modes of functioning, even while existing in a changing world.

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GLUCOSE-6-PHOSPHATE DEHYDROGENASE AND MANIC-DEPRESSIVE PSYCHOSIS

DEAR SIR,

Professor Mendlewicz and colleagues (*Journal*, October, 1980, **137**, 337–42) find a close genetic linkage between manic-depressive psychosis and glucose-6-phosphate dehydrogenase (G6PD) deficiency. They conclude that their results strengthen . . . "the hypothesis of X-linkage in a subgroup of manic-depressive psychosis" and they add that their findings . . . "cannot be generalized to all cases of manic-depressive psychosis because instances of father-to-son transmission . . . have been observed". Such instances, they state, are "clearly inconsistent with X-linkage". This latter view would be justified if an X-linked gene were the the sole predisposing factor and if the mother of a male proband were not a carrier of the predisposing gene.

From an analysis of the age pattern of onset of manic-depressive psychosis in New York State (Malzberg, 1955), and from the familial studies of Kallmann (1953, 1959), I concluded that predisposition to the disorder was polygenic, entailing an autosomal (dominant effect) gene (AM) together with an X-linked (dominant effect) gene (XM) (Burch, 1964). The frequency in New York State of AM was estimated to be about 1 per cent and of XM to be around 30 per cent; we can be virtually certain that predisposition is not determined by an X-linked gene alone.

Given that the frequency of XM is approximately 0.3, about 50 per cent of women will carry XM; nearly 10 per cent will be homozygous and about 40 per cent of women will be heterozygous. Thus, if a father (heterozygous for AM) has manic-depressive psychosis the chance of transmitting AM to his son will be 0.5 and the chance of XM being transmitted from the mother to the son will, for random mating, be about 0.3. Therefore, apparent 'father-to-son transmission' of predisposition can be expected to occur in about 15 per cent of instances. On these grounds, therefore, Mendlewicz *et al* (1980) have no need to postulate genetic heterogeneity. Nevertheless, we cannot eliminate the possibility that additional genes, within the AM/XM genotype, might help to determine distinctive types of manic-depressive psychosis.

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PHENOTHIAZINE WITHDRAWAL IN SCHIZOPHRENICS IN A HOSTEL

DEAR SIR,

Oral phenothiazine medication continues to be used widely for the maintenance of chronic psychiatric patients. At the same time increasing attention is being

paid to hostel or 'half-way house' care as an alternative to hospitalization. While a number of studies have looked at the effects of drug withdrawal from hospitalized patients (Andrews *et al*, 1976), no British studies of drug withdrawal have been carried out in a hostel setting.

We report the results of a controlled trial of phenothiazine withdrawal with a group of chronic schizophrenic residents in a residential home. The home is a large country house with relatively low staff levels. The 24 residents who were confirmed as chronic schizophrenic and who were stabilized on oral chlorpromazine over a six-month period entered the trial. The residents were divided into three matched groups (mean age 54.7 years, mean length of hospitalization 26.7 years) and after a baseline assessment period each group entered the trial at successive one-monthly intervals. Residents were randomly allocated to active or placebo medication on entering the trial, and after 6 months medication was crossed over. The allocation of residents to medication was carried out by the hospital pharmacy, and residents received medication solely from their individually marked bottles.

Determination of relapse was initiated by the trained hostel staff: the research doctor was called in every case, and gave 50 mg chlorpromazine IM following discussion with ward staff. If no change occurred the next day, the patient was considered relapsed. The majority of relapses involved the recurrence of symptomatic behaviour. Two residents were withdrawn from the trial because of physical illness. Of the remaining 22 residents, eleven (50 per cent) relapsed, 10 of whom were on placebo at the time of relapse, although one of these had changed from active to placebo medication only 2 days before relapse. Urine tests for the presence of phenothiazine were carried out immediately before the cross-over and immediately before the end of the trial, and in every case the test was consistent with the result expected from the patient's trial drug state. Relapse occurred between 2 days and 4 months (mean 8 weeks) after placebo medication was started for the residents who relapsed on placebo, and 10 weeks after cross-over for the single active-drug relapser. There was no difference between the relapsing and non-relapsing residents in terms of level of medication, age, or length of stay.

An unusual aspect of the results was the occurrence of 6 of the relapses (all on placebo) within a period of 5 weeks during the total 16 months of the trial. Immediately before this series of relapses one of the residents died by drowning in a nearby river, an open verdict being returned at the inquest, and at the same time another trial resident threatened a nurse on night duty. This high proportion of relapses within such a short period is highly unlikely by chance ($\chi^2 = 27.5$; $P < .01$), and the rate of relapse before this 5-week period was exactly the same as that in the period following.

The temporal relationship of these relapses to the two presumably stressful 'life-events' for both residents and staff may not be causal. During this period there was pressure from staff for the trial to end: the staff may have altered their threshold of acceptability of disturbed behaviour, or may have responded to the presence of minor side-effects. It is of interest that with one exception, only residents on placebo were identified as relapsers, and accordingly the active drug may have protected residents during a period of significant social trauma. The exact criteria used by ward staff to determine relapse are unclear, but Lehmann (1975) has pointed out that nurses often assume they know the type and dosage of medication to maintain patients at an acceptable level, and Hamilton *et al* (1979) have commented in their carefully designed study on the anxieties which drug withdrawal may create in nursing staff. It would be of value to examine in some detail exactly how direct-care staff judge the appropriate level of medication for chronic patients, and how they determine when medication needs changing.

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