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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