chronic schizophrenia (the Type 2 syndrome of Crow, or classical Kraepelinian schizophrenia) may be due to cerebral dopaminergic underactivity. Crow, however, thinks that the Type 2 syndrome is more likely to be due to "cell loss and structural changes in the brain". Whatever the cause of chronic Kraepelinian schizophrenia, both authors supported the concept of a 'dopamine hypothesis' to explain the positive symptoms of acute Schneiderian schizophrenia. I believe that such a view of the role of cerebral dopamine in psychiatric illness is too restricted.

The 'dopamine hypothesis' of schizophrenia obviously was a simplistic notion from its birth. The discovery that the neuroleptic drugs used to control acute positive Schneiderian symptoms of schizophrenia all possessed the ability to antagonize cerebral dopamine receptor action led to the concept that over-activity of cerebral dopamine mechanisms might cause schizophrenia. As both Mackay and Crow pointed out, these drugs only control the positive symptoms such as florid delusions, hallucinations and thought disorder of schizophrenia. However, neuroleptic drugs also control similar symptoms occurring in many other types of illness. Neuroleptic drugs frequently are used to control mania. Casualty officers, general physicians, psychiatrists and neurologists have for many years employed neuroleptic drugs to calm patients with acute toxic confusional states (delirium) due to drugs, fever, metabolic encephalopathy, encephalitis, stroke, brain tumour, and so on. Indeed, neurologists sometimes are forced to employ neuroleptics to control the acute toxic confusional state provoked by levodopa in patients with Parkinson's disease. Levodopa also may provoke florid delusions and visual hallucinations in the setting of clear consciousness and preserved insight in some patients with Parkinson's disease, and such symptoms also respond to the administration of a neuroleptic drug. The other plank of the 'dopamine hypothesis' was the discovery that amphetamine could provoke a syndrome akin to schizophrenia in some individuals, but amphetamines also can cause a typical acute toxic confusional state.

Such clinical observations suggest that the current focus of attention on dopamine in schizophrenia is too narrow a view. I suggest that the 'dopamine hypothesis' for schizophrenia should originally have been constructed in a wider context as a 'dopamine hypothesis' to explain the positive symptoms of acute psychotic illness, whatever its cause.

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ANOREXIA NERVOSA IN A DEVELOPING COUNTRY

DEAR SIR,

Anorexia nervosa is rare in Africans and in those of African extraction. The first case in a black child was described by Warren and Vande Wiele (1973). A further case is now reported.

The patient was a 22-year-old Nigerian girl admitted to the University Teaching Hospital of Lagos. Her father, to whom she was very close, had separated from her mother. She blamed the latter for the separation. She had an elder sister. During adolescence she was teased by her peers on account of her precocious physical development. She had married after becoming pregnant while still at school but had separated before the birth of her child. At the time of admission she was living with her mother.

The history began during her pregnancy, six years prior to admission. She attempted to lose weight by reducing her food intake and using purgatives. For the last five months she had refused all food and had induced vomiting after being forced to eat. Her menstruation returned after parturition but then ceased five months before admission. Her normal weight was 56 kilos but on admission was only 30 kilos.

On examination she was mute and emaciated. Lanugo hair was prominent on her back.

The initial management included intravenous fluid replacement and tube-feeding. This was subsequently changed to a 4,800 calorie diet. She reacted to weight gain by self-induced vomiting. Chlorpromazine and cyproheptidine were prescribed to stimulate her appetite. She developed a severe depressive condition which responded to six applications of electroplexy. After an admission of five months she was discharged but was readmitted one month later with further food refusal. During this second admission of one month hypnotherapy was employed. Her final weight was 56 kilos and her menstruation returned.

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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