with 20 mg of intravenous diazepam. Because catatonia was required for these symptoms, there was no NMS; rather, there was a combination of definite catatonia and neuroleptic effects. In order to demonstrate NMS the authors would have had to observe drug-induced symptoms in the absence of catatonia, which they did not; their assertion of separate episodes of both catatonia and NMS is not only unproven, it is untested. The care of catatonic patients has always been risky, and the clinical state of this patient may have been as unstable as NMS, but that does not make it NMS.

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'Mabi bark tea' consumption and psychosis?

SIR: Hassiotis *et al* (*Journal*, September 1992, **161**, 404–407) suggest that the use of mabi bark may be the precipitating cause of a psychotic illness in a 23-year-old West Indian woman. They cite the temporal link between ingestion of the mabi bark drink and the onset of her psychosis, the lack of a previous personal or family history of mental illness, an identical twin not developing a similar illness, and a biochemical basis that mabi bark causes central dopamine release.

We find the hypothesis untenable. Firstly, our observations in Trinidad and Tobago are not consistent with this. Mabi bark drink (mauby) is very widely used by the Trinidad and Tobago population and has not been found to contribute to the onset of psychotic illness. In a recent analysis of 634 schizophrenic patients, the period prevalence rate was found to be 5.0/1000 population with the disorder being three times more common in African Trinidadians than Indian Trinidadians. The use of mauby was investigated and was not found to be a contributory factor in a single case. Mauby has been drunk here for over a century and continues to be a very popular drink. Their patient's consumption of mauby was described as high but two-thirds of a pint daily for a week can hardly be described as excessive. In addition, for many years it has been served as a cold drink on the psychiatric wards and to date, no reports of its association with psychosis have been made.

In considering the above, it is clear that the association between mabi bark and this girl's psychotic illness is a spurious one and more than likely to be a chance association. There are too many uncontrolled confounding variables. Why implicate mauby? Why not coffee or tea?

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Simultaneous kidney disease and manic–depressive psychosis

SIR: We would like to report the first recorded case of simultaneous transmission of autosomal dominant polycystic kidney disease (ADPKD) and manic–depressive psychosis (MDP).

Case reports. Mrs F (aged 51) had her first episode of brief mental disorder at the age of 25 after the birth of her first child. After the birth of her second child five years later she had a further depressive illness which was treated with medication and electroconvulsive therapy (ECT). At the same time she was diagnosed as suffering from chronic renal failure due to ADPKD. In 1976 she had a very prolonged episode of treatment-resistant depressive illness. She was very depressed in mood, psychotic, and almost stuporose. However, after a long period of illness she recovered completely. She has had several further episodes of a prolonged psychotic depressive illness, particularly in 1989 when she was admitted to hospital for over six months – receiving antidepressants, *L*-tryptophan, lithium, and 24 applications of ECT. She has had lithium augmentation for treatment of her depressive illness since 1978. Since 1985 she has been on peritoneal dialysis. Her maternal grandmother had some kind of kidney disease but died in her 70s of an unknown cause. Her husband is well. They have two children.

Her elder son has had an episode of severe depression of similar magnitude to that experienced by his mother. He first presented at the age of 25 with a six-week history of lethargy and depressed mood. He was treated with fluvoxamine without response. He then became suicidal, taking several overdoses and cutting his wrists. On the day of admission he had been found by his flatmate trying to electrocute himself using wires from an electric lamp. He improved slowly with antidepressants and was transferred to Day Care, without regaining his premorbid state. He relapsed, was readmitted and responded very well to ECT.

He has been well or even slightly hypomanic since. Ultrasound scan of his kidneys showed a number of small cysts in the right kidney and a single large cyst in the left kidney, but his renal function is normal. He has a younger brother who is well psychiatrically.

Unfortunately, there are insufficient surviving members of Mrs F's family to allow gene linkage studies and there was a resistance within the family for further ultrasound scanning.

ADPKD is the most common genetic disease (Gabow, 1990). The clinical phenotype can result from one of two gene defects. One of these is located on the short arm of chromosome 16, while some 4% of families with the disorder have an unknown mutation elsewhere in the genome (Parfrey *et al*, 1990). There is a suggestion that the q21–22 region of chromosome 11 may be a promising area to examine for genes predisposing to major mental illness (St Clair *et al*, 1990) and particularly that bipolar affective disorders may be linked to DNA markers on chromosome 11 (Egeland *et al*, 1987).

Despite the difficulty in completing investigations of our patients it is a rare combination of circumstances and we felt that the case should be reported so that if any other clinicians have a similar family it may be possible to take the issue further.

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Down's syndrome, longevity, and Alzheimer's disease

SIR: Prasher (*Journal*, November 1992, **161**, 722) comments on the rare elderly Down's syndrome individuals who have not developed Alzheimer's disease, either clinically or neuropathologically. Dr Prasher is right to suggest that these cases are of interest, since they may also shed light on the role of the β-amyloid precursor protein (APP) gene in both disorders.

There is now strong evidence that abnormalities in the APP gene and its products are a central molecular event in the aetiology and pathogenesis of Alzheimer's disease. The APP gene is located on chromosome 21. In Alzheimer's disease associated with Down's syndrome, the cause is thought to be overexpression of the APP gene concomitant with the extra copy of genes on chromosome 21. It is therefore of significance that a small number of Down's syndrome cases are not due to complete trisomy of chromosome 21, but merely part of it; crucially, this 'obligatory' Down's syndrome region does not include the APP gene locus (Korenberg *et al*, 1990). Thus, the Down's syndrome individuals who die without Alzheimer-type pathology may be those without triplication of the APP gene. This could be tested by a combination of cytogenetics and subsequent neuropathological analysis.

KORENBERG, J. R., KAWASHIMA, H., PULST, S.-M., *et al* (1990) Molecular definition of a region of chromosome 21 that causes features of the Down syndrome phenotype. *American Journal of Human Genetics*, **47**, 236–246.

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Eating disorders in Hong Kong

SIR: Lee *et al*'s report on bulimia nervosa in Hong Kong (*Journal*, October 1992, **161**, 545–551) and their earlier report on anorexia nervosa in Hong Kong (1989), raise the interesting question of the extent of sociocultural factors in the aetiology of anorexia nervosa. This disorder, once seen primarily among the affluent white populations, has now been widely reported in developing countries and Eastern cultures.

Our study in West Malaysia, a multiracial developing country, supports Lee's studies that eating disorders are rare. We examined the ward registers of admissions to male and female psychiatric wards in the University of Malaya from 1970 to 1988. No male cases were detected in approximately 8000 male psychiatric admissions. Out of over 9000 female admissions, 15 cases fulfilled two or three of the criteria for anorexia nervosa (weight loss, amenorrhoea, and characteristic psychopathology). There were 13 Chinese, one Malay and one Indian. One had a history of bingeing. There was only one case of bulimia nervosa. She was a 24-year-old girl who had a four-year history of binge eating. Lee pointed out that body-image disturbance has not been found among anorexia nervosa subjects in India and that the diagnostic criteria may need to be modified in