among British Afro-Caribbeans (*Journal*, December 1991, **159,** 783–789). He omits to suggest a role for cannabis use, for which there is some evidence. Cannabis use among psychotic Afro-Caribbeans in Nottingham was much higher than among white patients (Harrison *et al*, 1989). Frequent use among young men is followed by a six-fold increase in admissions for schizophrenia (Andreasson *et al*, 1987). Interestingly, sharp excesses are seen for schizophrenia in young Afro-Caribbean men in the UK (Glover, 1989) and in Jamaica (Burke, 1974).

Data from a follow-up study I have completed (Sugarman, 1992) produced two incidental findings. A history of cannabis use had been recorded in the case notes significantly more often for UK-born young black men with schizophrenia than for matched white patients ($P<0.005$). While age, age of onset, length of illness, sex, family history, and presence of first-rank symptoms did not predict outcome, Afro-Caribbeans noted to take cannabis had a better total outcome score than other Afro-Caribbeans ($P<0.05$). Stepwise multiple regression did not suggest that this was attributable to the age and sex distribution of cannabis use.

It may be, therefore, that there is a subpopulation of Afro-Caribbean schizophrenics, mainly young men, with a good prognosis illness precipitated by cannabis. Such cases may be part of the explanation for high rates of illness in this ethnic group.

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Anorexia nervosa and XY gonadal dysgenesis

SIR: I read with great interest the report by McCluskey & Lacey (*Journal*, January 1992, **160,** 114–116) which demonstrated how anorexic psychopathology might result from XY gonadal dysgenesis, its adverse psychosocial effects and medical treatment. While I do not debate that the patient exhibited low self-esteem, psychosexual identity problems, and family conflicts, as well as certain clinical features which may be found in female patients with anorexia nervosa, and was likely to benefit from psychological help, the case raises interesting questions about the diagnosis of anorexia nervosa in subjects with XY gonadal dysgenesis which the authors did not discuss. They mentioned that the patient fulfilled the DSM–III–R criteria for anorexia nervosa which essentially required: (a) weight loss of 15% from standard body weight; (b) amenorrhoea; (c) an intense fear of obesity or body-image distortion.

One problem in diagnosis may arise because subjects with XY gonadal dysgenesis, as the authors indicated, are tall and with a eunuchoid habitus. Unlike the much more frequently reported association of anorexia nervosa with Turner syndrome in which affected subjects are typically short, this body build will naturally lead to a low body mass index (BMI) to start with. Hence, it is doubtful whether one can estimate weight loss for these subjects by making reference to standard weight charts of normal females. Surprisingly, the authors did not mention the original body weight or BMI of the patient before oestrogen therapy. Nonetheless, even when she was happiest during her 'only sexual relationship', she was noted to weigh only 47 kg at a height of 1.74 m. This gives a BMI of only 15.5 kg/m^2 which, according to the ICD–10 proposed criteria (specifically BMI $\leqslant$ 16 kg/m^2) for the diagnosis of anorexia nervosa (World Health Organization, 1987), is still in the anorexic range. It is therefore possible that the patient could have had an 'anorexic body shape' even before the development of her psychopathology. If this premorbid body weight was used as reference, the patient would have a weight loss of only 12.8% (6 kg) which was below the DSM–III–R requirement. Thus, the patient might fulfil DSM–III–R criteria only if her body weight achieved with oestrogen therapy was used as her 'original' body weight in the estimation of weight loss. However, this medically induced 'normal' body weight, which she seemed ill prepared for, was paradoxically a contributory factor to her eating disorder, as the authors pointed out.

The above diagnostic problem is compounded by two other issues. Firstly, as subjects with XY gonadal dysgenesis by their very nature suffer from primary amenorrhoea, this diagnostic criterion, being automatically fulfilled, will become less useful clinically. Thus, I have encountered Hong Kong Chinese girls (including two medical students recently) who have a BMI of 16 kg/m^2 and a certain fear of obesity, but

whose perfectly normal menstrual history practically excludes anorexia nervosa. The same diagnostic reasoning, obviously, cannot be used in thin subjects who have primary amenorrhoea of whatever cause, including XY gonadal dysgenesis.

Secondly, as 'body image distortion' is non-specific, poorly defined and found in a significant proportion of normal Western females too (Hsu & Sobkiewicz, 1991), the significance that can be attached to it, in the absence of other convincing anorexic features, is meagre as in the case reported. Besides, other anorexic behaviours such as binge-eating, self-induced vomiting or laxative abuse were absent in the patient.

In so far as the exact aetiology of anorexia nervosa remains unclear and its diagnosis thus depends on the constancy of association of a particular constellation of clinical features, the above three factors may make the diagnosis of anorexia nervosa an enigmatic one in subjects with XY gonadal dysgenesis and psychopathology. Therefore, while agreeing with the authors that anorexia nervosa, in the synergistic presence of other psychopathology, may stem from the biological and psychological confusion caused by XY gonadal dysgenesis, I would also like to add a further source of confusion to this potential association, namely, diagnostic confusion. Prudence, especially among those of us inclined to unearth anorexia nervosa, is suggested.

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Methylfolate and psychiatric illness

SIR: We would like to echo the comments of Anderson *et al* (*Journal*, January 1992, **160**, 130) on the interesting paper by Godfrey *et al* (1990) concerning the enhancement of recovery from psychiatric illness by methylfolate.

Apart from the lack of sufficient information regarding changes on the Hamilton Rating Scale for Depression, it should be noted that the use of this scale and the Beck Depression Inventory as a measure of changes in schizophrenic patients (41% of the total sample) was inappropriate. The lack of correlation was therefore expected. We wondered why more suitable and even simpler scales for this group of patients, such as the Brief Psychiatric Rating Scale, were not used. Instead, only a crude four-point clinical outcome scale was employed. It is quite difficult, for example, to differentiate between a score of '2' (residual symptoms/socially recovered) and '3' (pronounced impairment without full social recovery). Furthermore, the baseline ratings of the two groups at month zero were not reported and the question of their comparability remained. Thus, the difference between the methylfolate and placebo groups at one month, as measured by the clinical outcome scale, may partly be due to selection bias, or the initial clinical effect of methylfolate. Finally, the authors did not clearly state whether there was any change in dosage and regime of medication or the use of non-pharmacological therapy throughout the trial. This is noteworthy because the clinical outcome scores of the placebo group, for unclear reasons, showed no overall improvement after six months of psychiatric treatment. Instead, both depressed and schizophrenic patients on placebo remained markedly impaired (bordering on score '3').

Despite their potential importance, folate studies among non-Western populations are virtually non-existent. Recently, we measured the folate level of 46 Chinese out-patients on chronic lithium treatment, and found that practically none of them had low serum or erythrocyte folate (Lee *et al*, 1992). Yet, within the normal range of serum folate (7–39 nmol/l), patients ($n = 14$) with a higher serum folate level (33.4 nmol/l) were found to have a lower one-year affective morbidity than those ($n = 32$) with a lower folate (26.0 nmol/l). This is the first Chinese study to strengthen the suggestion that it may be a high rather than normal folate level which is associated with lower psychiatric morbidity (Coppen *et al*, 1986). In the study of Godfrey *et al*, the significant difference in outcome between the folate and placebo groups at six months, despite the already normalised red cell folate level of the latter, gives further support to this possibility.

The folate debate will continue but appears a promising one. The current research findings, as Procter (1991) put it, "should surely warrant the attention of all practising psychiatrists and if confirmed may deserve wider general application". A controlled therapeutic trial, for example, is valuable for finding out whether Chinese patients with normal folate status may also benefit from folate pharmacotherapy. If it works, it may not really matter much, as in the case of psychotropics generally, to find out why it does so.