The genetic mechanisms referred to by McGuffin *et al* may certainly explain a small percentage of cases of schizophrenia, but considerable room remains for positing an environmental influence in the aetiology of schizophrenia.

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SIR: Although many hypotheses are put forward by McGuffin *et al* to account for the 'non-genetic' causes of schizophrenia, they have possibly overlooked one significant cause of the new appearance of schizophrenia in a patient with no family history: ambiguous paternity.

Estimates of the incidence of non-paternity vary from 2.8% to 30%. Rates of non-paternity depend on the population being investigated. For instance, Macintyre & Sooman (1991) report that one study of the correlation between antibody formation in artificial insemination and blood group had to stop because it had revealed that in the population being surveyed, 30% of the children could not have been sired by their mothers' husbands.

Published data have revealed non-paternity rates of 5% on the basis of ABO and rhesus markers (Johnstone, 1957), and Bellis & Baker (1990) predict a non-paternity rate of 6.9–13.8%. Via DNA fingerprinting, Le Roux *et al* (1992) estimated that, in a population with genetic disease, the rate of children not sired by the declared father was 2.8%.

There is no reason not to expect a similar phenomenon in a psychiatric subpopulation, where the relationships may be even more unstable. However, neither standard textbooks nor a literature search revealed any reference to non-paternity when discussing the heredity of psychiatric illness. Although this phenomenon may not account for all, it can explain part of the discrepancy between the observed and expected heredity. Non-paternity warrants further investigation when studying family histories.

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Lithium prophylaxis in recurrent affective illness

SIR: Guscot & Taylor (*BJP*, May 1994, **164**, 741–746) draw attention to some of the reasons for non-compliance with lithium. I profoundly disagree with the concept of separate specialised clinics which the authors propose would lessen the gap between efficacy and efficiency. This philosophy reflects a general trend in the National Health Service away from the 'generalist' towards fragmentation of services and the deskilling of staff, leading to resentment and demoralisation.

Specialist clinics with research-orientated staff on short-term contracts may not serve the patients' need for personal doctoring: long-term relationships, based on trust and mutual respect, characterised by consultations with staff who have taken the patients through relapses, and have knowledge of the family and social network. There is a need for some specialist services, but surely affective illnesses are the bread and butter of general psychiatrists.

This leads to the conundrum of training psychiatrists. How can programmes that rotate every six months possibly serve patients with long-term illnesses? The problems lie at the root of medical education, which lays emphasis on the seductive rewards of treating acute illness using an authoritarian medical model. I propose that this is an important source of non-compliance which specialist clinics cannot even provide sticking plaster for.

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Dyskinesia and withdrawal from alcohol

SIR: Duke *et al* (*BJP*, May 1994, **164**, 630–636) found that tardive dyskinesia (TD) in schizophrenic

patients with a lifetime history of problem drinking was no more severe than in control patients. This is in contrast to another study (Dixon *et al*, 1992) where in recently admitted psychotic in-patients alcohol abusers had significantly higher TD scores than non-abusers. The following case report may help to explain these different results.

Case report. A 37-year-old woman with a 16-year history of psychiatric disorder diagnosed latterly as bipolar affective disorder was readmitted to Crichton Royal Hospital on a Sunday evening. She was expressing suicidal ideas, had auditory hallucinations, and had been drinking more heavily than usual for two to three weeks; she admitted to three to four litres of vodka per week. As she had run out of money she had no drink on the Saturday or Sunday. She had been receiving flupenthixol decanoate for three and a half years; the constant dose over the previous 12 months was 150 mg weekly. On the Sunday evening there was no evidence of dyskinesia. The following day there was gross orofacial, neck and upper limb dyskinesia, which if formally rated would certainly have been scored 'severe' on the AIMS global scale (US Department of Health, Education and Welfare, 1976). Gross dyskinesia persisted throughout her stay of eight weeks, during which time there was no evidence of alcohol consumption. The dose of flupenthixol decanoate was reduced to 120 mg weekly. The patient went home on pass for a weekend before discharge. Over the Saturday and Sunday she admitted to consuming over 10 pints of beer. She returned to hospital on the Monday morning; there was no evidence of dyskinesia. Because of disruptive behaviour, she was discharged the same day, but agreed to attend the day hospital. She is again abstaining from alcohol, and dyskinesia has reappeared.

Although Duke *et al* give detailed information, it seems that most of their patients were assessed outside hospital, and therefore had ready access to drink. Continuing alcohol consumption may have masked dyskinesia and contributed to lack of difference in severity of TD between problem drinkers and control patients. This is in contrast to the Dixon study, where patients were not assessed until towards the end of their hospital in-patient stay.

This case report suggests either that alcohol can diminish the severity of dyskinesia produced by antipsychotic medication, or that 'withdrawal' dyskinesia can occur with alcohol as with neuroleptic medication. The latter is perhaps more likely; dyskinesia is common in detoxified alcoholic patients (Lucey & Dinan, 1992).

DIXON, L., WUEIDEN, P. J., HAAS, G., *et al* (1992) Increased tardive dyskinesia in alcohol abusing schizophrenic patients. *Comprehensive Psychiatry*, **33**, 121–122.

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Cannabis consumption and schizophrenia

SIR: The May issue of the *BJP* contains two papers about schizophrenia, and substantial numbers of the patients studied in both were taking psychoactive substances, in one alcohol, and in the other cannabis and other drugs. The basis on which the diagnosis of schizophrenia was made is therefore called into question.

Duke *et al* (**164**, 630–636) identified 352 patients with schizophrenia, and of the 271 who completed the alcohol assessment, 22% were problem drinkers. The 81 patients who did not complete the assessment are likely to have included some problem drinkers, and the remainder will have included substantial drinkers, where the alcohol intake did not amount to a 'problem'. They used Feigner diagnostic criteria (although apparently not exclusively), and one of these is the "absence of alcoholism or drug abuse within one year of onset of psychosis". Although Feigner does not consider this an essential criterion, I believe that for anyone with psychosis taking a psychoactive substance, no diagnosis can be made until after they have been free of the substance for a significant time, in case of alcohol usually about two weeks. This may not be easy to ensure, and the authors should therefore be asked whether the diagnosis was made at a time when the subjects were free of alcohol and should state how they ensured this. Otherwise, the diagnosis of schizophrenia is untenable.

Martinez-Arevalo *et al* (**164**, 679–681) state that patients were included in their study if the diagnosis fulfilled DSM–III criteria, and they report that 30 of the 62 patients regularly consumed cannabis at the beginning of the study, while many others were taking other drugs or alcohol. DSM–III states that, "The diagnosis is made only when it cannot be established that an organic factor initiated and maintained the disturbance". The authors should be asked whether the diagnosis was made at a time when the subjects were free of cannabis and of other psychoactive substances, and should state how they ensured this. Otherwise, the diagnosis of schizophrenia is untenable. The same question could be asked of most of the papers they quote.