virus would act in a positive manner, one would expect that its influence would decline rather than increase with time.

Where does this leave the retroviral/transposon theory of psychosis? Confusion has resulted because it has grown to become so complex – more a synthesis of several theories with a common thread. Perhaps it is time for Dr Crow to discard the parts which are not useful, thereby cutting a knot which both psychiatrists and geneticists find hard to unravel.

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Dr Crow's detailed response will be published next month.

SIR: What else can be explained by this 'schizophrenic-mutagenic-virogene'? Dr Crow's account of schizophrenic aetiology via a virogene associated with a high rate of mutation (*Journal*, October 1987, **151**, 460–465) seems to resolve much of the contradictory data surrounding this baffling disease. Evidence such as the discordance in monozygotic pairs, age of onset uninfluenced by environment, seasonality of birth, adoption away from schizophrenic relatives not reducing risk of disease, relationship with paternal age, the apparent continuum of psychosis, constant incidence rates across populations, and even the increasing incidence in the 19th century can all, it seems, be drawn together with one explanation (Crow, 1987).

However, many of these features which are accounted for remain themselves controversial, and not everyone accepts them as part of the description of schizophrenia. What incontrovertible aspect of schizophrenia does this virogene hypothesis explain? There is some data that is accepted universally which may have been inadequately explained until now.

The evidence on life-time expectancy for schizophrenia in relatives of schizophrenic patients is well established. If one parent is affected, the average risk for a child is 12%. However, if a child is affected, the risk for parents is only 5%. This relatively low risk has been explained until now by the suggestion it is the more healthy parents who tend to reproduce.

A rapidly mutating gene might also predict this and perhaps account for the phenomenon more satisfactorily.

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Psoriasis and Lithium

SIR: Humphreys & Waddell (*Journal*, March 1988, **152**, 437–438) suggest that lithium therapy led to an improvement in their patient's psoriasis. As their references reveal, the literature tends to show that lithium exacerbates psoriasis. There may be possibly an alternative explanation for this dermatological improvement, the clue to which lies in their patient's heavy drinking history.

Vincenti & Blunden (1987) reported a small series of regular alcohol abusers who had found a striking association between stopping drinking and improvement of their psoriasis. A much larger earlier study (Chaput *et al*, 1985) found a significant association, independent of alcohol liver damage, between alcohol abuse and psoriasis at the level of $P < 0.001$.

It would be interesting to ascertain whether the patient reported by the authors had successfully reduced his alcohol intake around the time he started lithium therapy.

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VINCENTI, G. E. & BLUNDEN, S. M. (1987) Psoriasis and alcohol abuse. *Journal of the Royal Army Medical Corps*, **133**, 77–78.

SIR: We are grateful to Dr Vincenti for his remarks with regard to our report, the intention of which was

not simply to suggest a connection between lithium and remission of psoriasis, but to invite comment on this interesting case with its many paradoxical aspects.

Chaput *et al* (1985) showed psoriasis to be more common among a sample drinking more than an average of 50 g alcohol a day compared with those consuming less than that amount. They point out that this is not synonymous with a causative relation – high alcohol intake may precede psoriasis or psoriasis may enhance the onset of alcoholism. In the three cases reported by Vincenti & Blunden (1987) there was rapid remission of psoriasis during detoxification intimately related to reduction in alcohol intake. In the case we reported, following initial referral the patient became abstinent with only minor early relapses, and has remained so since. It was not until two years later that lithium treatment was started and improvement in his psoriasis was noted. Prior to this his psoriasis had remained active, and had been present for many years before his drink problem. In this case, reduction in alcohol consumption did not relate to improvement in psoriasis.

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General Practice Patients on Long-term Psychotropic Drugs

SIR: I would like to make several points about the study by Catalan *et al* (*Journal*, March 1988, **152**, 399–405). Firstly, the authors did not explicitly blame the GPs for the poor prescribing practices described but that was the general impression that I gained from the paper. This impression may or may not be valid. As their data was obtained from FP10 prescriptions, I wonder how many of these prescriptions were initiated, maintained, or advised by psychiatrists.

The authors state that 24% of index patients had received psychiatric out-patient treatment, and 10% had received in-patient treatment, at any time before the 12-month period of the study. Even assuming no overlap of these two groups, we would be left with 24% of the patients on long-term psychotropic medication who had been seen by a psychiatrist in the past. It may have been pertinent to ascertain how many of the index group had been assessed by a psychiatrist in the recent past, for example in the 12 months before the study period. It would then be interesting to clarify the contribution (if any) the psychiatrists had made to reviewing these prescriptions.

Secondly, the authors suggest that practices which have links with visiting psychiatrists could review patients on long-term psychotropic medication. The number of patients (318 out of a practice of 8842) would represent a major undertaking for a psychiatrist working in a 'traditional' hospital-based service. This study I feel highlights the need for psychiatrists to work at least partly in primary health care settings as described by Mitchell (1985).

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MITCHELL, A. R. K. (1985) Psychiatrists in primary health care settings. *British Journal of Psychiatry*, **147**, 371–379.

SIR: As Dr Donnelly points out, we did not explicitly blame the GPs for the prolonged prescribing of psychotropic drugs. We would like to stress that we certainly had no intention of blaming them implicitly. When the patients in our study were started and maintained on their drugs, the climate of opinion among doctors and people in general was in favour of such prescribing. Recently, this climate of opinion has changed with the increasing concern about the efficacy and side-effects of many psychotropic drugs prescribed in general practice.

Our findings suggested that psychiatrists had played a small part in initiating prescribing for the patients: 3% of those on anxiolytics, 5% of those on antidepressants, and 9% of those on major tranquillisers. The GP had initiated prescribing for most of the patients: 50% of those on anxiolytics, 60% of those on antidepressants, and 42% of those on major tranquillisers. Among the remaining patients, prescribing had been initiated by a hospital physician or the patient's former general practitioner. Although nearly a quarter of the patients in our study had a history of past psychiatric consultation, no patient had consulted a psychiatrist in the 12 months before the study period.