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