SIR: One reason why Dr Bristow is baffled by my contribution may be that he is not familiar with the relevant literature.

The presence of transposable elements has been demonstrated in mammals in general (Singer & Skowronski, 1985; Deininger & Daniels, 1986) and specifically in man. Several distinct classes of endogenous retroviral sequence (Benveniste & Todaro, 1976; Martin *et al*, 1981; Bonner *et al*, 1982; Mager & Henthorn, 1984; Callahan *et al*, 1985), a novel transposable element (Paulson *et al*, 1985), and repetitive sequences of the LINE (Musich & Dykes, 1986) and other (Kiyama *et al*, 1986) classes with features of mobile elements have been described in the human genome. The problem for the hypothesis is not, as Dr Bristow supposes, that transposons are not present in vertebrates, but that a large number of virogene candidates has accumulated.

Dr Bristow is in further error in stating that Alu elements have not so far been associated with any illness. Exon-Alu recombination deleting 5 kb from the low-density lipoprotein receptor gene has been described as a cause of hypercholesterolaemia (Lehrman *et al*, 1986). Whether other instances of disease caused by Alu or other mobile elements will be discovered in due course cannot be prejudged.

When he refers to "polygenic" theories, it appears that Dr Bristow means theories that allow for environmental as well as genetic aetiological contributions (i.e. 'multifactor' rather than 'polygenic'). Almost certainly, he is in a large majority in believing that there must be environmental factors and that observation of discordance in monozygotic (MZ) twin pairs demonstrates the insufficiency of genetic factors. I believe, on the contrary, that there is no compelling evidence for an environmental contribution (at least in the postnatal period), and that for the following reasons no such contribution may exist:

(a) age of onset is apparently uninfluenced by the environment (Crow & Done, 1986)
(b) adoption away from a schizophrenic relative does not reduce risk of the disease (Karlsson, 1970)
(c) incidence rates are approximately constant across such populations as have been well studied (Sartorius *et al*, 1986).

For these and other reasons I have recently argued (Crow, 1987*a*,*b*) that the possibility that Dr Bristow dismisses – that psychosis is associated with a high rate of mutation, and that mutation rather than differences in life experience between the twins accounts for discordance in MZ pairs – deserves serious consideration. The reason why this gene is associated with a particularly high rate of mutation I suggest is that it is a late and perhaps characteristically human evolutionary development. Specifically, I argue that it is the gene which determines lateralisation in the brain (Crow, 1986).

What I claim for this theory is that it generates predictions concerning the genetic contribution which are more precise than those of other current theories (particularly those of the multifactor and polygene type) and that, in principle at least, it is testable. I concede to Dr Bristow that the theory needs to be made yet more precise, and perhaps expounded with greater clarity.

Dr Lo's explanation of seasonality of birth is different from, and I think simpler than, my own. It has the merit of providing a unitary explanation of seasonality of both birth and onset of psychosis. However, as far as I can see the hypothesis predicts sex differences in both seasonal trends. Such data as are available in the literature (Hare & Walter, 1978; Bradbury & Miller, 1985) suggest that these trends apply to both sexes. Thus, while brain growth in songbirds is an attractive (and lateralised) paradigm of the normal mechanisms which we may suppose are disturbed in psychosis, I am not yet persuaded that gonadal hormones play a critical role in onset of the disease.

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Barking Mad

SIR: In reply to Dr Buchanan's letter (*Journal*, October 1987, **151**, 562–563) we report two further cases of the 'barking mad'.

Case reports: (i) A 61-year-old German divorcee was admitted after having taken an overdose of a tricyclic antidepressant. She claimed it was a "cry for help", as she could no longer cope with her "mission". She described herself as being a dog (a German Shepherd), having known this since she barked instead of coughing 6 years previously following a cold. She showed her hand as proof of canine appearance, and also claimed that the transparent plaster over her CVP insertion site was a sign of moulting.

(ii) A 24-year-old unemployed man was admitted under Section 2 of the Mental Health Act. Six months prior to admission it was noted he was becoming increasingly withdrawn, self-neglectful and uncommunicative. On presentation he was mute, apart from turning his head from side to side and making clearly recognisable barking noises. His symptoms remitted within a month of treatment with anti-psychotic medication.

Unlike Dr Buchanan, we are unable to comment on the impact of such symptoms on the diplomatic service, but wonder whether, given the over-representation of this fairly unusual syndrome in our hospital, it could possibly be explained by the proximity of the Newham Health District to Barking and the Isle of Dogs.

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SIR: When I was a resident in psychiatry in 1974–75 at the Leeds General Infirmary, I was called late one evening by a rather dominant sister to see a "dog" in casualty. I told her I had no veterinary qualifications, but being an animal lover I made my way to casualty in a perplexed mood. I was pointed in the direction of a room in the casualty department that normally lacked any furniture, but had a mattress where, traditionally, alcoholics and psychiatric patients were examined. I saw a young man, properly dressed, between 18–22 years of age, on the mattress on his knees and elbows. When I started my usual psychiatric interview he continuously barked loudly, just like a dog, with no other types of noises. When I persisted on questioning, he suddenly said "I am a dog" and then barked again. Obviously I could not obtain further history. As psychiatric patients were on neurology wards in Leeds General Infirmary in those days, it was difficult to admit very disturbed patients. I avoided ringing Professor Hamilton at home and managed to admit the patient to High Royds Hospital.

My impression was that he was under some kind of family conflict and stress, and the intensity and frequency of barking was reduced when he knew he was going to be admitted. I later discovered that he had no previous history and settled rapidly without much pharmacological intervention and with psychosocial support.

Barking has been described as a part of a clinical picture in patients with Tardive Tourette syndrome (Stahl, 1980), and in Gilles de la Tourette's syndrome (Abuzzahah, 1982). Involuntary noises are not uncommonly heard in patients with severe tardive dyskinesia due to the involvement of muscles of expiration. It is also not uncommon for schizophrenic and severely agitated depressives to make some strange noises. Pure barking as a clinical symptom in the absence of any other associated illness is extremely uncommon, and is most probably stress-related and under voluntary control.

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