dominance gene which itself is highly variable between normal individuals.

A model for this last feature is the mouse LINE element described by Loeb *et al* (1986) that includes a variable number of tandem repeats. These authors consider that the tandem repeat sequence may regulate expression. Ono *et al* (1987) have reported a human retroviral sequence with similar repeats.

In addition to the above three postulates, I suggest that the new mutations required by the theory occur specifically in the course of male gametogenesis and are season-dependent (Crow, 1987*c*). In this way an environmental influence on the structure of the gene, and thereby on the occurrence of psychosis, is introduced.

The case that the psychosis gene indeed is closely related to the cerebral dominance gene has been strengthened by the finding that temporal horn enlargement in schizophrenia, by contrast with that in Alzheimer-type dementia, is highly selective to the left hemisphere (Crow *et al*, 1988*a*). A locus in the pseudoautosomal region of the sex chromosomes has been suggested (Crow, 1987*d*) and is supported by the observation that in a series of pairs of siblings with psychosis, concordance by sex is inherited from the paternal and not the maternal side (Crow *et al*, 1988*b*). These findings may facilitate a molecular approach to the nature of the psychosis gene.

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Capgras Syndrome and the Amygdala

SIR: The patient described by Lipkin (*Journal*, July 1988, **153**, 117–118) demonstrates, as do several other cases reported by Dr Lipkin, that the Capgras syndrome may be an early symptom of dementia. This case report continues the debate as to whether this syndrome has an organic basis. It does occur in organic conditions with diffuse and focal cerebral lesions, but precise anatomical localisation of the lesion is not available. The localisation of lesions giving rise to an inability to distinguish one face from another, proposagnosia, is better established and seems to involve an area of the cerebral cortex at the occipito-temporal junction (Meadows, 1974).

Electrophysiological work in monkeys has shown that there are at least two areas of the brain in which neurones are selectively responsive to faces. One of these is a cortical area, the superior temporal sulcus (Bayliss *et al*, 1985). Neurones here respond with a shorter latency than in the other area, the amygdala (Leonard *et al*, 1985). This finding has been interpreted as indicating additional processing of sensory information, which, in view of the role of the amygdala in the regulation of social and emotional behaviour (Thompson *et al*, 1977), probably involves the incorporation of social and emotional cues necessary to identify a particular face as that of a genuine close relative. It is this function which appears lost in Capgras syndrome.

There is an increasing body of evidence that the amygdala may be subject to damage in the early stages of the commonest cause of dementia, Alzheimer's disease. The pathological changes are most severe in the hippocampus and amygdala, and these regions appear to be affected early in the course of the disease (Brun, 1985). Moreover, we have described a number of cases of Alzheimer's disease in which there were marked reductions of the cholinergic innervation of the amygdala in the absence of the characteristic loss of such innervation of the cerebral cortex (Palmer *et al*, 1986). While it is difficult to extrapolate the results of discrete brain lesions to patients with diffuse brain damage, the case reported by Dr Lipkin and other cases reviewed by him may reflect early damage to the amygdala in dementia.

This syndrome illustrates also that the symptoms of dementia which are most difficult to deal with, both for the patient's relatives and health care professionals, are not necessarily the cognitive impairments, but the deterioration in social behaviours. At least some of these may be a consequence of subcortical disease, and it is now important to investigate these behaviours in dementia and to follow this by pathological and neurochemical studies.

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Dysmorphophobic Avoidance

SIR: I found the behavioural therapy described by Marks & Mishan (*Journal*, May 1988, **152**, 674–678) fascinating. While the results of systematic exposure therapy in their series of five patients were encouraging, the inclusion of two patients who were deluded that they smelled challenges the definition of the term dysmorphophobia. This has usually been reserved to describe patients who complain of a subjective feeling of disfigurement in the absence of any objective abnormality (Hay, 1970; Thomas, 1984; American Psychiatric Association, 1987).

It has been argued that the condition may be primary or secondary to an underlying psychiatric illness (Thomas, 1984). While the authors state that "it feels rather Procrustean to force a diagnosis of what was 'primary' ", none of their patients could be considered to be suffering from a primary diagnosis of dysmorphophobia by virtue of the presence of delusions (Thomas, 1984; American Psychiatric Association, 1987).

I was intrigued by the percentage scoring of delusional conviction, and should be interested to know how the score was determined. There is an implication (in case 1) that a score of 50% constitutes an overvalued idea. If that is so, how should the score of 80% (where 100% = total conviction and 0 = none) in case 2 be interpreted, and can this scoring system aid in distinguishing beliefs such as primary delusions from delusion-like ideas or overvalued ideas (Jaspers, 1946)?

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