funds were not available from the catering budget in the financial year.

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A Freudian lacuna?

SIR: In his article on the psychoanalytical aspects of morbid jealousy in women (*Journal*, January 1990, **156**, 68–72), Freeman seems to be saying that "the phallic sexual organisation" of his two neurotic female patients was instrumental in their downfall (figuratively speaking). Does he mean that possession of such an apparatus would therefore also be aetiologically significant in the development of morbid jealousy in male patients, since they are ordinarily so endowed by nature? Or is his assertion rather, that the two women have a confused sexual identity – the evidence for which is, that although "feminine and attractive in appearance", they have a phallic sexuality, and that this somehow gives rise to their need for triumphant retribution against those who are more naturally endowed in this way? Or does a phallic sexuality only become sadistic and exhibitionistic in the wrong hands, so to speak?

Without putting too fine a point on it, Dr Freeman has succeeded in spinning a great yarn about the origins of morbid jealousy in these women with precious little evidence that their jealousy was 'morbid'. What he described are two chaotic and unhappy marital relationships (marriage being an arrangement between two people, unless I am mistaken) where sexuality has become the bargaining chip and weapon, as it not uncommonly does for damaged and powerless people of both sexes.

Finally, it must be the women in the textbooks of psychiatry (or psychoanalysis) for whom the absence of vaginal orgasm is pathological, the onset of menstruation was not a shock (for those over 30), and the admiration of breasts bigger than one's own is a homosexual urge.

Freud was a man of undisputed genius, but he did not know (as distinct from presume) much about women – how could he? (See Peter Gay's new biography: *Freud–A Life for Our Time*.) I hope those who purport to heal psyches in the name of psychoanalysis are not following in his footsteps.

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Genetic origins of psychosis

SIR: Crow recently discussed the genetic origins of psychosis (*Journal*, June 1990, **156**, 788–797). We recently completed a retroactive case study of 574 patients suffering from schizophrenic psychosis. Our results showed that the incidence of schizophrenia increases with progressively increasing paternal age. The association between parental age and mental illness has often been reported in the past. Such reports have come from different parts of the world and have involved thousands of patients. Kinnell (1983) wrote: "There are few areas of schizophrenic research where one finds such impressive concordance of results from different studies as that of parental age". Our results showed that the age of the father is the operant factor. The association between paternal age and at least 11 genetic diseases or recent mutational origin has been documented. Evans (1988) wrote: "There is considerable and long-standing evidence that mutation frequencies for a variety of different human genes causing disease increase with increasing paternal age". Vogel & Rathenberg (1975) reported that the special sensitivity of the male germ cells for mutation increases with age.

It can be concluded that increased mutation rate in the germ cells of the aged father could play a role in the causation of schizophrenia in some cases. Schizophrenic individuals born to older fathers could constitute an aetiologically distinct subgroup of the schizophrenic population.

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SIR: Crow (*Journal*, June 1990, **156**, 788–797) discusses the hypothesis that schizophrenia results from a disorder in the gene which determines cerebral dominance. In conceptual form the theory is supported by analysis of the unifying feature of the first-rank symptoms. These refer to the reception of vocal input ascribed to an external source, passivity phenomena in which a controlling external agency is implicated in thought insertion or withdrawal, or the imposition of motor or sensory data. In thought broadcast, thoughts are felt to be available to another externally. The subjective experience of the non-dominant hemisphere in its interaction with a dominant vocal hemisphere explains the above if the unifying feature is the awareness of the involvement of an external agency. These symptoms are understandable when considered in systems control theory as the experience of a processing unit which achieves a relative independence and seeks to understand its controller (e.g. reception of information by the right hemisphere from the left would be seen as thought insertion and vice versa as thought withdrawal). The above system would conform to the requirements for a neuropsychology of schizophrenia described by Frith & Done (1988) if the monitor and plan-evolving centre with willed intention represent dominant hemispherical functioning, while synthesis of perception, stimulus intention and resultant action represent non-dominant functioning. Support for the above possibility is mentioned in Birchwood *et al* (1988), where an account is given of the absence of first-rank symptoms in split-brain patients, post-hemispherectomy or in those with agenesis of the corpus callosum.

While there is conceptual support for Dr Crow's hypothesis, in consideration of the phenomenology of schizophrenia, the ascription of the abnormality of cerebral dominance to a single genetic abnormality is questionable. The single strongest point of opposition is Badian's (1983) paper (quoted by Crow, 1986) concerning the seasonal incidence of left-handedness. The results showed an excess of left-handedness in males born between September and February, with a high statistical significance. This pattern of increased left-handedness correlates well with the observed excess of winter births in schizophrenia. However, for cerebral dominance to interact so closely with an environmental variable any genetic theory of dominance must invoke an intermediate stage in development which is subject to the effects of an environmental variable. I am presently researching the possibility that the ratio of light:dark in the first few months following birth affects the left hemisphere to a greater extent than the right, and that genetic sensitivity to its effect would explain the majority of the above findings. In the first few months following birth, with the four-fold increase in brain mass, there would be a particular neurodevelopmental susceptibility to the action of an environmental variable.

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Post-traumatic stress disorder

SIR: Kennedy (*Journal*, January 1990, **156**, 129) wrote questioning whether the data in my recent studies about Australian volunteer fire fighters is capable of generalisation to post-traumatic stress disorder (PTSD) as a whole. He suggests that this may not be the case because this group was trained, well motivated and had previous experience. He goes on to state that the event lacked the element of surprise and unfamiliarity experienced by other PTSD groups examined.

These are important issues to consider. However, the experience of these fire fighters in the Ash Wednesday disaster was far beyond their most extreme expectations. The intensity of the heat in these fires was very great, with five metres of fire-front generating as much energy as a large power station (Webster, 1986). Most of these volunteers were also members of the local community and many had their homes damaged or destroyed. As well, while they were fighting the blaze, most knew that their families were also facing considerable risk. This was particularly difficult for them because they were not in a position to protect their families. Furthermore, often the radio communications used by the fire networks were completely ineffective because the intensity of the heat ionised the atmosphere. This created an atmosphere of isolation and lack of direction. Moreover, much of the fire-fighting equipment

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