others all showed personality disorder of various kinds. Almost all had a previous history of self-mutilation, slashing of the wrists, or previous suicide attempts. The largest group, four patients, consisted of long-standing transsexuals whose disturbed personalities had possibly suggested a poor prognosis for sex reassignment surgery, and who consequently had been refused such surgery by the various specialists they had encountered. Of interest in a generally young population is the higher than average age of the group. While the high proportion of personality disorders may simply reflect the catchment population, which may also account in part for the low number of psychotic patients, this report serves to emphasise that such behaviour is not necessarily, nor particularly often, associated with paranoid schizophrenia. The most common associations seem to be a disturbance of sexual identity, a previous history of self-mutilation, and personality disorder.

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Ethnic labels in South Africa

SIR: We refer to the correspondence regarding the use of South African Population Registration Act categories as a basis for psychiatric research (Sashidharan & Lipsedge, *Journal,* April 1986, **148,** 484; Teggin *et al, Journal,* November 1986, **149,** 667–668; Graham, *Journal,* November 1986, **149,** 669). During the time that we were working at the MRC/University of Cape Town Clinical Psychiatry Research Unit this was a major issue of debate. The labels "Black", "Coloured", "Indian" and "White" as used in South Africa are fundamentally political, and do not refer in any scientific sense to discrete ethnic or cultural groups (Sharp, 1980). The important way in which these labels are "real" is that they dictate vastly differing access to resources of all kinds, including housing, education, employment, and health care.

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The Continuum of Psychosis and the Gene

SIR: Crow attempts to replace the Kraepelinian dichotomy between manic depressive psychosis and schizophrenia with a continuum of psychosis (*Journal,* October 1986, **149,** 419–429). We agree that data such as those obtained by Kendell *et al* support the conclusion that a symptomatic continuum exists. However, the evidence for a genetic continuum of the form envisaged by Crow is less persuasive.

Crow's model is based on several studies from the older literature reporting an excess of individuals with schizophrenia among the offspring of patients with affective disorder while failing to observe the converse. None of these studies employed modern diagnostic criteria, and it is possible that misclassification occurred. Indeed, studies (quoted by Crow) using modern methods have failed to show an increase in schizophrenia among the relatives of probands with affective disorder. Crow also argues that similarities in season of birth effects point to an underlying commonality of genetic mechanisms. However, the season of birth effect in schizophrenia is associated with an absence of a manifest genetic predisposition, suggesting a relationship with environmental factors (Boyd *et al,* 1986).

In contrast, evidence that schizophrenia and affective disorder are based on two independent genetic diatheses is more compelling. Firstly, as Crow acknowledges, the two major functional psychoses by and large breed true. Secondly, bipolar affective disorder appears to have a stronger genetic component than schizophrenia. Crow's hypothesis would appear to predict the converse. Thirdly, Elsässer (1952) found an equal prevalence of the two psychoses in the children of marriages between an affective and a schizophrenic individual. A greater prevalence of schizophrenia than affective disorder is predicted by Crow's hypothesis. Finally, Crow's model also fails to take into account the existence of "schizophrenia spectrum disorders", or the evidence that minor depression and depressive and cyclothymic personality disorder are genetically related to affective disorder. These findings suggest that there are separate phenotypic spectra related to the schizophrenic and the affective genotypes which are orthogonal to the phenomenological continuum that exists between the two disorders.

Given this evidence for independent genotypes, where does this leave the nosological status of schizoaffective disorder which occupies the intermediate position in the symptomatic continuum? One possibility is that it consists of both affective and schizophrenic types of illness. However, as Crow reminds us, the concept arose out of the failure

to demonstrate such a division. Secondly, it might be a genuine interform between schizophrenia and affective disorder (Kendell, 1983). This implies that both illnesses are polygenic, with the presence of some elements from each genotype predisposing to schizoaffective disorder. A third possibility, which does not imply polygenic inheritance of both Kraepelinian psychoses, is that it represents the presence of both the schizophrenic and the affective genotypes in the same patient: it occurs in individuals who, in genetic terms, have both illnesses. It has been argued by Kendell (1983) that this is unlikely because schizoaffective disorder is more common than would be expected given that "the chance coincidence of two illnesses each affecting around one person in a hundred is one in ten thousand".

We disagree with such a conclusion on several grounds. Firstly, there is no reason to suppose, as Kendell does, that for schizoaffective disorder to occur a genetic diathesis to schizophrenia would have to exist with one for bipolar rather than unipolar affective disorder. The morbid risk for all types of affective disorder combined is much greater than 1%, and therefore Kendell's expected figure would be larger. Secondly, assortative mating might take place to increase the likelihood of both genotypes being present in combination. Thirdly, Kendell assumes that possession of one genotype will not affect expression of the other. On the contrary, it seems likely that schizophrenic symptoms will trigger the onset of affective illness in a predisposed individual and vice versa. Moreover, it is quite possible that *subclinical* expression of one genotype increases the likelihood of the other being expressed in a predisposed individual. For example, possession of an affective diathesis might render an individual with a schizophrenic genotype more likely to develop a psychotic reaction to stressful life events. Such a psychosis might be expected to show both schizophrenic and affective features.

In summary, we suggest that the symptomatic continuum reflects phenotypic interaction rather than the relationship proposed by Crow.

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Procyclidine Abuse

SIR: In reporting five more cases of procyclidine abuse, Fenech & Khoosal (*Journal*, October 1986, **149**, 524) pointed out that the latest edition of the *British National Formulary* (BNF) did not mention the potential for abuse of that drug. Pullen *et al* (1984*a*) drew attention to the fact that the BNF omitted reference to the abuse of any anticholinergic.

At last the BNF (Number 11, 1986) does include one sentence on abuse in its section on the use of antipsychotic drugs (p. 139), although there is still no mention of this danger in the main section on anticholinergics (pp. 182–184). These preparations continue to be freely prescribed, and there still seems to be continuing ignorance about these significant drugs of abuse (Pullen *et al*, 1984*b*).

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Neurological Factors in Obsessive-Compulsive Disorder

SIR: In their article on obsessive-compulsive disorder (OCD) (*Journal*, September 1986, **149**, 315–319), Kettl & Marks emphasise the case for organic precipitants, probably operating 'downstream' from the primary mechanisms initiating the condition. Whether cognitive abnormalities in OCD attributed to these organic precipitants could be intrinsic or secondary features of the condition is not assessed. This remains unclear in the recent studies quoted (Behar *et al*, 1984; Flament & Rapoport, 1984), which showed computerised tomogram and cognitive abnormalities in adolescents with OCD. Brain injury was not used as an exclusion criterion in the group concerned. Some patients had histories of head trauma and birth injury, and one is mentioned as having tardive dyskinesia.

I have studied 19 DSM-III-diagnosed obsessive-compulsive patients, none of whom had neurological