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Correspondence

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BIMODALITY AND THE NATURE OF DEPRESSION

DEAR SIR,

Some people may, I fear, be misled by Everitt's paper (*Journal*, April 1981, 336–339). He states "A number of studies, for example Kiloh and Garside (1963), and Pilowsky, Levine and Boulton (1969), indicate that they have evidence for the existence of two subtypes of depression, whilst others, for example, Kendell (1969) and Kendell and Gourlay (1970) argue for the existence of only a single type". The first part of this statement is correct, but the second part is not. Kendell (1972, p. 575), correcting a previous similar misunderstanding, wrote "In fact I have never at any stage said, or even believed, that there was only one kind of depressive illness, or that the differences between one patient and another were merely differences in severity. In my original monograph (Kendell, 1968a) I took some pains to emphasize my acceptance of the fact that there are important and fundamental differences between different depressions that are not simply differences in severity or chronicity and I did so again in a subsequent review (Kendell, 1968b)".

The basis of this regretfully common confusion is the failure to distinguish between (a) the classification of symptoms and (b) the classification of patients. The importance of this distinction was pointed out by Kendell (1969, p. 336) in his paper cited by Everitt. It has also been discussed by Eysenck (1970, p. 349): "There are two, not one, problems involved, relating to (a) the unitary or binary nature of depression, and (b) the categorical or dimensional nature of these illnesses. Factor analysis is relevant to (a), and conclusively favours the binary view; distribution of scores is relevant to (b)".

The fact is that while there is fairly general agreement that there are (at least) two kinds of depression, there is no agreement as to whether there are two (or more) diagnostic groups of patients which are relatively distinct from one another, in the sense described by Kendell and Brockington (1980). They, perhaps rather misleadingly, used the phrase 'disease entity' in this context as implying a "*natural boundary* or discontinuity between the condition in question and its neighbours". They went on to say that the most obvious way of demonstrating such a discontinuity is "to demonstrate, in a representative and unselected population, that patients exhibiting a mixture of the symptoms of the condition in question and those of neighbouring syndromes are relatively uncommon. The mixed forms, the greys, must be shown to be less common than the pure forms, the blacks and whites, which in mathematical terms involves demonstrating that a distribution of scores on a linear variable, derived from the relevant symptoms, is bimodal with a 'point of rarity' in the middle, rather than unimodal".

When Everitt (1981, p. 337) says "The mixing of two unimodal frequency curves produces a bimodal distribution only if the components are fairly widely separated, and it is the mixing *not* the bimodality which is fundamental" he is missing the point that it is precisely the bimodality that is important. For if the distribution of the dimension discriminating between the two depressive conditions is unimodal, there are no distinct diagnostic groups and most patients cannot then be diagnosed as suffering from either one or the other depressive illness; the greys are more common than either the blacks or the whites.

In view of the conflicting evidence regarding the bimodality of the differentiating dimension (Kendell and Brockington, 1980, p. 325), attention should now, perhaps, be focussed upon the distributions of psychotic (endogenous) and of neurotic (reactive) depressions separately, as was done by Fahy *et al*, 1969. They found that the distribution of their psychotic component was bimodal, but that of the reactive component was not. Kiloh and Garside (1977) using Lewis's original data, also found the psychotic component to be clearly bimodal, but they were unable to isolate a unitary condition of neurotic depression from the data. Moreover several cluster analytic studies have shown that there is a distinct group of patients suffering from psychotic depression, for example Pilowsky *et al* (1969), Everitt *et al* (1971), Matussek *et al* (1981) and Paykel (1971). But this does not seem to be true of neurotic depressions; Everitt *et al* (1971, p. 411) concluded "patients with depressive and other neuroses . . . showed no tendency to form distinct clusters". They suggested that the most useful form of classification will prove to be a combination of a dimensional system, in relation to the neuroses, with a typological (categorical) one in relation to

psychoses. Kiloh *et al* (1972) made a similar suggestion.

The distribution of the dimension discriminating between psychotic and neurotic depressions is a mixture of distributions along two, roughly orthogonal, separate, axes (Kendell, 1968a, p. 39; Kiloh *et al*, 1972, p. 189 and Matussek *et al*, 1981, p. 369): a psychotic (bimodal) one and a neurotic (unimodal) one. It is therefore not surprising that there is little agreement regarding the shape of the discriminating distribution (Garside and Roth, 1978, p. 62). Depression consists of two independent conditions that are not mutually exclusive.

To sum up: there is fairly general agreement, as Kendell (1976, p. 25) pointed out, (a) that there are two kinds of depression and (b) that there is a distinct group of patients suffering from the disease entity of psychotic depression. But there is no general agreement as to the distribution of neurotic depression; it is probably best regarded as continuous rather than bimodal, at least until shown to be otherwise.

It seems, therefore, that the attempt to arrive at a differential diagnosis in relation to depressed patients in inappropriate. Two separate questions should be asked, not one: first, is the patient suffering from psychotic depression or is he not and, second, to what extent is he suffering from neurotic depression.

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POST INFLUENZAL-DEPRESSION

DEAR SIR,

Sinanan and Hillary (*Journal*, February 1981, **138**, 131–3) state that there is no convincing evidence for the existence of post-influenzal depression. A good deal hinges on how the word 'convincing' should be interpreted, although I agree that no epidemiological study, so far, has demonstrated an association between influenza and depression.

On the other hand, I doubt whether their paper has proved that such an association does not exist. What they have shown is a lack of correlation between a rating scale for depression and levels of influenza antibodies in a group of patients suffering from a variety of psychiatric disorders. Such a conclusion does not invalidate the observation, based upon clinical experience, that "intractable depression may sometimes follow attacks of influenza". As such patients in all probability would be suffering from severe endogenous rather than neurotic or mixed depression, taking all these categories together might have obscured the presence of some patients in their series whose endogenous depression followed an attack of influenza. Unfortunately we are not given sufficient information about the actual antibody titres and it is impossible to deduce these from the mean rank figures given in Table I. With respect to the second Table, it is unclear from the text whether the figures apply only to depressed patients or to the whole group, including those with other diagnoses.