

Correspondence

THE CLASSIFICATION OF DEPRESSIVE ILLNESSES

DEAR SIR,

Thank you for inviting me to comment on Professor Eysenck's 'brief note' (Pp. 241-50).

As I understand it, Eysenck's argument proceeds in four stages. First he points out, I think wisely, that quite distinct issues are involved: (a) whether the system is categorical or dimensional in nature, and (b) how many units (categories or dimensions) there are. He starts with the second of these two (which he refers to as the unitary v. binary issue, ignoring other possibilities) and claims that factor-analytic studies have proved that there are two units, which conveniently correspond with his own concepts of psychoticism and neuroticism. Having decided this question 'once and for all' he then goes back to question (a), discusses the different statistical properties of two-category and two-dimension systems, and concludes that the evidence strongly favours the latter. Finally, and this appears to be the main purpose of his essay, he criticizes Hamilton's work and mine, and to some extent that of the Newcastle group also, maintaining that the dimensions we studied were meaningless, that the score distributions we plotted were irrelevant to the unitary v. binary issue. In particular, he objects to my (single) continuum model of depression as being 'inadequate statistically and irrelevant psychologically', though he appears to concede that it is more useful than the traditional categorical classification.

Eysenck is quite right in stating that most of my work was not relevant to the unitary v. binary issue, but it was never meant to be. I was more concerned with the other and more fundamental of the two issues. I was trying to find out whether or not any justification could be found for the categorical (disease entity) classification traditionally used by psychiatrists, and I stated this clearly at the outset (Kendell, 1968): 'The purpose of this investigation is to determine whether or not any justification can be found for the division of depressions into two or three distinct entities.' For this reason I used discriminant function analysis as my main statistical tool, and only resorted to factor analysis secondarily in an attempt to cope with the problems raised by inconsistent diagnostic criteria. I failed to find any evidence to support the existing categorical system, and so committed myself to providing a dimensional alter-

native to it. I used a single dimension because this was the simplest, because I already possessed a means of identifying the positions of individual patients on this dimension, and because I was in a position to demonstrate its practical superiority over the traditional classification into three distinct diseases.

Eysenck objects to my doing this on the grounds that factor-analytic studies have already proved that there are two, and only two, dimensions involved. But this assumption, the cornerstone of his argument, is unjustified. There are nearly a score of factor-analytic studies of the symptomatology of depressions in the literature. The number of significant factors obtained and their clinical identity vary greatly from one study to another, and the authors of these studies have placed very variable interpretations on their findings. The only reasonably consistent finding is that if unrotated factors are used the loadings of one of the early factors usually correspond to a greater or lesser extent with the clinical picture of endogenous or retarded depression. Doubtless many of the differences between one study and another are due to variations in the items chosen for analysis, in the patient populations, and in the precise statistical procedures employed. But the fact remains that these studies do not provide a consistent picture, and the claim that they demonstrate conclusively that there are two, and only two, dimensions involved cannot be taken seriously, particularly when it is presented *ex cathedra*, unsupported by objective assessment or even by the opinions of other workers. Eysenck quotes my factor-analytic studies in support of his position, but they could equally well be used to prove that there are twelve, or four, dimensions involved.

Even if the findings of factor-analytic studies were in agreement with one another, of their nature they could never tell us how many dimensions of symptomatology there are; they could only suggest the most appropriate number to use in a representational model. Other considerations are also involved. The main purpose of any classification of illness is to enable clinicians to communicate accurately with one another and to make the most effective choice amongst the therapeutic tools at their disposal. For this reason a classification which clinicians will not use has little value, no matter how strong its aesthetic appeal to academicians. Psychiatrists are so accustomed to their familiar disease entity classification that they will only be persuaded to change to a dimensional

system if that system is simple to use and has obvious and immediate practical advantages. A two-dimensional model of depressions would be capable of conveying more information than a uni-dimensional model, and I have myself suggested using two dimensions rather than one (Kendell, 1969); but the advantage of additional information has to be weighed against the disadvantage of increased complexity, and the same consideration will apply when someone suggests the addition of a third or fourth dimension. There is also another important consideration. Almost all those who have taken an interest in this field, Eysenck and myself included, have confined their attention to depressive illnesses, tacitly assuming that these could be considered in isolation. This was not an unreasonable approach while there was still some hope that we were dealing with a categorical system, but once we have decided to use a dimensional system we can hardly assume a discontinuity between depressions and other surrounding areas of symptomatology. It follows that we would be unwise to make firm decisions about the number of dimensions we need before we have included these adjacent areas in our analyses.

For these reasons the appropriate number of dimensions is for me still an open question. I would not claim that the single dimensional system I have advocated is necessarily the best, though it is the simplest. The important thing is for us to agree on the inadequacy of our existing classification and on the necessity for replacing it with a dimensional system.

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DEAR SIR,

The entry of such a formidable controversialist as Professor Eysenck into the discussion on the classification of the Depressions should at least convince outsiders that it is not a frog-and-mouse battle. His contribution is to be welcomed, since it makes many of the points clear in a way that has not been done previously. As he points out, the resolution of the matrix of intercorrelations of the symptoms into at least two factors demonstrates that the notion that the difference between the two 'types' of depressive syndrome can be interpreted as signifying merely the

difference between severe and mild symptoms is untenable.

His account of the difference between the dimensional and categorical classifications perhaps needs expanding. If we plot the position of persons suffering from a particular illness in the multidimensional space defined by their symptoms we obtain a cloud of points which represents their distribution in that space. Patients suffering from another illness could also be plotted in that space, provided that they also have those symptoms. In general, patients suffering from two different illnesses will have symptoms which are not common to the two conditions, and it is the symptoms which are not common that differentiate the two disorders. This is not always so, for what differentiates paratyphoid A, B and C is not the difference in symptoms but the difference in immunological characteristics. If the two types of depressive illness should ever be shown to have different biochemical or genetic bases this will settle the question, regardless of the symptoms or distribution of symptoms. There is one particular case where two disorders would be differentiated even if all the symptoms were common and there were no external criterion to distinguish them, and that is the case where the two clouds of points were quite distinct in the multidimensional space. Even if there were some overlap, the difference could be accepted if a statistical test were to demonstrate that the hypothesis of a common distribution was untenable. The categorical and dimensional models are therefore not as different as Professor Eysenck suggests.

Professor Eysenck agrees with this when he states that the conditions for such a situation would be met if the points representing the persons were to cluster round the two axes of endogenous and reactive depression. If we examine his Fig. I, these two patterns of symptoms form the ordinate and abscissa of his diagram, and we can imagine a cloud of dots surrounding these two axes in his diagram. In this diagram he also provides two other axes, the one labelled 'Kendell's continuum' and a line at right angles to it. The latter, he points out, would represent a general factor of 'severity of illness'. It would not be reasonable to postulate that each cloud of dots was in the form of a normal distribution, though it doesn't really matter. If we now project these distributions on to the 'Kendell's continuum' we would find two normal distributions overlapping to some extent. Thus the model which he states would confirm the 'categorical' hypothesis will show itself as a bimodal distribution on the bipolar factor 'endogenous versus reactive'. It is therefore not illegitimate to look for a bimodal distribution on some appropriate dimension in the multi-dimensional