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

space. Indeed, he admits this himself when he states 'The somewhat bimodal distributions of the Newcastle workers may be interpreted as supporting, though rather weakly, a categorical model . . .' Of course, the argument about distributions is weak, and he is quite right in drawing attention to the effects of selection of cases, which can profoundly affect the correlations, factors and distributions. The problem of selection has been sadly ignored in the literature.

He blames the confusion which exists in the controversy on to the paper by Dr. White and myself, but the confusion does not lie where he suggests it is. In our paper, we extracted four factors and we suggested that the first could be named 'endogenous depression' and the second 'reactive depression'. This was a mistake, for careful examination of the factor loadings indicates that the first would be better regarded as a general factor of severity, and the second regarded as a bipolar factor of endogenous versus reactive (to use his terms, which I dislike because we are considering only symptoms, not aetiology). These two factors are (more or less) the two sloping axes in his Fig. I. I pointed out this mistake in *naming* in my paper (Hamilton 1967). The 'confusion' would not have arisen if the data in the Hamilton and White paper had been examined carefully. What was said in that paper was therefore appropriate, even if the terminology dealt with the factors as if they had been rotated factors (Eysenck's ordinate and abscissa), which they were not.

Professor Eysenck is correct in pointing out that in a two-dimensional surface each patient requires to be identified by two scores. Indeed, in my 1967 paper I pointed out that he should be identified by as many scores as there are significant factors in the matrix of correlations, and I found six. May I add here that I have examined the distribution of scores (using a much larger number of cases than in my 1959 paper) of the cases reported on in my 1967 paper, and have found, in both rotated and unrotated factors, that these distributions did not differ significantly from normal.

Again, Professor Eysenck is correct when he says 'Factor-space and person space are two different conceptions, and should not be used interchangeably', but the difference between them is not all that great. If we return to the original data plotted in multidimensional space, then a simple transformation will convert one into the other, as Godfrey Thomson pointed out (Thomson, 1940). This point is relevant to Dr. Kendell's Fig. II, in which is plotted the vectors representing the items in a space determined by the two factors of endogenous and reactive depression. An attempt is made here to demonstrate that the items fall into two clusters, and it would appear that 'the fundamental fallacy in Kendell's thinking' is simply that he is following the example of Thurstone (1947, pp. 126 and 185). I am not convinced by the diagram that the items do fall into two clusters, but had they done so it would have been legitimate to conclude that there are two factors, because such clusters do define factors. They are the rotated correlated factors which are so popular with the American workers in factor analysis.

To sum up, it is always worth while to look at the distribution of scores on an appropriate dimension to see if there is evidence of bimodality. If none is found then the case is 'not proven'; if it is found then it is necessary to consider the problems raised by selection. The argument concerning distributions is therefore a weak one, but in the absence of a better it is worth considering. This applies to all conclusions based on factor analysis.

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DEAR SIR,

Kendell and Gourlay in their article of this issue 'The Clinical Distinction between Psychotic and Neurotic Depression', pp. 257–66, found no distinction between depressive neurosis and depressive psychosis, as defined by the British Glossary, when they applied discriminate function analysis to data collected by several psychiatrists using a standardized technique.

Using a slightly different approach as the preliminary stage to another study, I have been able to confirm their findings. A consecutive series of 94 depressed in-patients was interviewed personally with the same standardized technique. Unlike Kendell and Gourlay, only mental state items were used (36 in all); historical items were omitted. The criteria of the British Glossary were not used in reaching a diagnosis because these descriptions presuppose certain points under investigation in the main study. Instead, descriptions were based on mental state items traditionally believed to distinguish between the two types of depression. The British Glossary description of depressive neurosis is anyway vague and unsatisfactory.