concerned with the structure of depression but simply with whether there was any evidence for clinical differences between 'psychotic' and 'neurotic' depressives. Not only did we fail to find anything more than a severity difference between them but there is no reliable evidence in the literature that 'neurotic' depressives have *any* symptoms distinguishing them from 'psychotic' depressives: they appear simply to have fewer of the same set of symptoms. However, many studies do not provide the data that are basic for establishing group differences—viz. a tabulation of the frequencies of individual symptoms in each of the groups in question. Were the Newcastle group to publish this information there would be a firmer base for discussion and further inquiry.

Finally, the notion that a bimodal score distribution (and it seems that any scores will do) necessarily implies the existence of 'two (or more) conditions' at least one of which is 'categorical' is mistaken. We have shown that bimodal and unimodal distributions of discriminant scores can be obtained at will from the same groups of patients by choosing variables appropriately. As an absolute criterion bimodality is clearly useless. The many mechanisms which may generate spurious bimodal distributions are discussed and amply illustrated by Murphy (1964).

References

CATTELL, R. B. (1965) Factor analysis: An introduction to essentials. II. *Biometrics*, **21**, 405–35.

MURPHY, E. A. (1964) One cause? Many causes? The argument from the bimodal distribution. *Journal of Chronic Diseases*, **17**, 301–24.

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Dr Ní Bhrolcháin appears to believe that useful data relating to the classification of affective disorders cannot be placed on record unless investigators allow themselves to be guided by the six models set out in her paper. She states that the Newcastle studies of depressive and anxiety states made the unvalidated assumption that either Models 1 or 4 are "actually true". The significance of these findings has nothing to do with the truth or otherwise of 1, 4 or any of the other models she lays down as a necessary starting point for all investigators. Their validity has been substantiated by the results of the independent follow-up studies to which we referred in our previous letter (July, 1979). It is in the light of such findings rather than the inferences Dr Ní Bhrolcháin deduces from her special models that the validity of the tentative classification which we proposed has to be assessed.

Neither in the studies of depressive illness (Carney *et al*, 1965) nor those relating to depressive and anxiety states was it concluded that the syndromes differentiated were homogeneous. Indeed, there is good evidence that there are two genetically different varieties of endogenous depression, unipolar and bipolar; whilst Kiloh *et al* (1972) have drawn attention to the heterogeneity of the group of patients diagnosed as suffering from 'neurotic depression'.

Dr Ní Bhrolcháin is in error in stating that the results of Carney *et al* (1965) have not been corroborated. Findings recorded by Sandifer *et al* (1966) Garside *et al* (1971) and by Kiloh and Garside (1977), derived from the original data of Lewis, all provided supporting evidence.

The conclusion reached by Ní Bhrolcháin *et al* (*Journal*, **134**, 94–107) that psychotic and neurotic depressives differ primarily in severity is refuted by three main lines of evidence. To regard an item such as 'retardation' as primarily a measure of severity is debatable. Secondly, 'psychotic-endogenous' disorders, ostensibly the more severely ill group, enjoy a more favourable course and outcome than neurotic cases. Thirdly, recent biochemical evidence strongly supports the qualitative nature of the difference between the two groups of disorders (Carroll *et al*, 1976 (a) and (b); Checkley, 1979). Finally, even if the bimodal distributions Ní Bhrolcháin *et al* derived from their maximizing analysis could be regarded as a 'severity' dimension (and this view is open to question), their conclusion that the result was compatible with the continuity of the psychotic and neurotic patient groups in their material would be erroneous.

Ní Bhrolcháin is incorrect in claiming that Cattell (1965) regards it as inappropriate to apply factor analysis to data from diverse groups. What he does say is that in applying factor analysis "one is forced to begin with an actual group of obscurely intermixed subspecies". Also (p 421) "factor analysis is best preceded, *or accompanied as suggested below*, where there is any doubt about homogeneity of population, by an examination for the existence and nature of

https://doi.org/10.1192/S0007125000022029 Published online by Cambridge University Press

possible type-structure in the form of subspecies". She quotes this passage in her letter but omits the important phrase we have italicized.

In our papers cited by her, we have done just what Cattell suggests: we have carried out component analysis (which is similar to factor analysis) and also discriminant function analysis.

Ní Bhrolcháin says that there is no reliable evidence in the literature of symptomatic differences between neurotic and psychotic depression. But she is discounting the views of others. Kendell (1968, p 71) has stated that "there is a wealth of evidence that there are important and fundamental differences between different depressions that are not simply differences in severity or chronicity", and Eysenck (1970) (p 249) "There are two, not one, problems involved, relating (a) to the unitary or binary nature of the depression, and (b) to the categorical or dimensional nature of these illnesses. Factor analysis is relevant to (a), and conclusively favours the binary view: . . .".

Carney *et al* did not tabulate symptom frequencies for the two depressive groups, but they did publish (their Table IV) correlations of each feature with diagnosis. Some of these were negative and others positive thus indicating that the two diagnostic groups differed in relation to *pattern of features*. These correlations are very similar to the bipolar factor loadings which indicate a *qualitative* difference between the two depressions.

It is true, as Murphy (1964) points out, that spurious bimodal distributions can occur in some circumstances. Only two of those mentioned by Murphy apply to our data cited by Ní Bhrolcháin. These are non-independence of items and the inequality of scores. But the average intercorrelation of each set of our data was below 0.2, which is not high enough to generate bimodality (Guilford, 1956, p 451) and no single item had a score (or weighting coefficient) "large compared with those for all the others" (Murphy, p 304). The bimodality obtained by us was therefore a valid finding.

Finally, she says that "We have shown that bimodal and unimodal distributions of discriminant scores can be obtained at will from the same groups of patients by choosing variables appropriately". It is true that if irrelevant uncorrelated variables are considered, unimodal distributions will be obtained. But discriminating variables will generate bimodal distributions.

References

CARNEY, M. W. P., ROTH, M. & GARSIDE, R. F. (1965) The diagnosis of depressive syndromes and the prediction of ECT response. *British Journal of Psychiatry*, **111**, 659–74.

CARROLL, B. J., CURTIS, G. C. & MENDELS, J. (1976a) Neuroendocrine regulation in depression. I. Limbic system-adrenocortical dysfunction. *Archives of General Psychiatry*, **33**, 1039–44.

—— —— —— (1976b) Neuroendocrine regulation in depression. II. Discrimination of depressed from non-depressed patients. *Archives of General Psychiatry*, **33**, 1051–8.

CATTELL, R. B. (1965) The role of factor analysis in research. *Biometrics*, **21**, 405–35.

CHECKLEY, S. A. (1979) Corticosteroid and growth hormone responses to methylamphetamine in depressive illness. *Pscyhological Medicine*, **9**, 107–15.

EYSENCK, H. J. (1970) The classification of depressive illnesses. *British Journal of Psychiatry*, **117**, 241–50.

GARSIDE, R. F., KAY, D. W. K., WILSON, I. C., DEATON, I. D. & ROTH, M. (1971) Depressive syndromes and the classification of patients. *Psychological Medicine*, **1**, 333–8.

GUILFORD, J. P. (1956) *Fundamental Statistics in Psychology and Education*. New York: McGraw-Hill.

KENDELL, R. E. (1968) *The Classification of Depressive Illnesses*. Oxford University Press.

KILOH, L. G. & GARSIDE, R. F. (1977) Depression: a multivariate study of Sir Aubrey Lewis's data on melancholia. *Australian and New Zealand Journal of Psychiatry*, **11**, 149–56.

MURPHY, E. A. (1964) One cause? The argument from the bimodal distribution. *Journal of Chronic Diseases*, **17**, 301–24.

SANDIFER, M. G., WILSON, I. C. & GREEN, L. (1966) The two-type thesis of depressive disorders. *American Journal of Psychiatry*, **123**, 93–7.

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https://doi.org/10.1192/S0007125000022029 Published online by Cambridge University Press