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Can Categorical and Dimensional Views of Psychiatric Illness be Distinguished?

D. A. GRAYSON

Bimodality in a distribution of symptoms is often claimed to be convincing evidence that a disorder is categorical, a discrete disease entity, rather than the extreme on a continuous dimension. However, using concepts from contemporary psychometric theory it is shown that bimodality can arise from the dimensional viewpoint. In fact, contrary to the usual belief, bimodality would be expected to occur in many research contexts if the dimensional alternative were correct.

In recent psychiatric research, debate has arisen as to whether certain disorders are categorical or dimensional. For example, in depression there is debate about the binary or bipolar nature of the illness (Garside & Roth, 1978; Kendell & Gourlay, 1970*a*). One unchallenged principle is that the existence of discrete disease entities is confirmed when a linear function of scores on a symptom inventory shows bimodality. Moreover, such data are believed to be inconsistent with the opposing dimensional view (Moran, 1966). Indeed, the point is made by Fleiss (1972), Everitt (1981) and Garside & Roth (1978) that such bimodality is a sufficient, but not necessary, indicator of disease categories. The implication of this is that the demonstration of mild bimodality, or even just skew, is enough for an underlying categorical illness to be inferred. For instance, Cloninger *et al* (1985) cite, as evidence in favour of categories, the results of 'admixture analysis' showing that two normals are better than one in fitting a discriminant function distribution.

Few would argue with the description given by Kendell (1975) of the conceptual linkage from a categorical illness to bimodality. But before one makes the reverse inference from bimodality to a categorical illness, there is a fundamental obligation to show that such bimodality cannot arise from a dimensional illness. Ruling out this latter alternative has not received appropriate attention.

Everitt (1981) states what is probably a widely held belief:

> Now for a *population* frequency curve, bimodality is (except in pathological cases) a sufficient (although not a necessary) condition for the presence of subtypes and certainly if, in a fairly large sample, bimodality appeared no matter how the data were arranged, it would be pedantic to insist it might be an artefact.

Garside & Roth (1978) provide a more substantial argument for believing bimodality is inconsistent with a dimensional illness. In discussing bimodality of a linear discriminant function score, they state:

> If a significantly non-unimodal distribution is then obtained, it seems safe to reject the hypothesis that the population distribution is unimodal, and to conclude that distinct groups of patients exist.

This conclusion is supported by an earlier statement to the effect that the central limit theorem entails unimodality in the sum of a number of (almost) uncorrelated (0, 1) variables. However, to infer distinct groups, and not a dimensional illness, requires the truth of a premiss suppressed in their reasoning: it must be true that a dimensional illness will yield data wherein pairs of symptoms show little or no correlation.

This premiss is in fact false, and the main thesis of this paper is that symptom–symptom correlations yielding bimodality can arise from a dimensional illness, and that such a state of affairs is far from being a 'pathological' case, as has hitherto been assumed.

The paper is divided into three major sections. The first demonstrates how bimodality can arise from a dimensional illness and exhibits similarities in the way symptom inventory data may arise from either dimensional or categorical illnesses. The second section discusses research biases which tend to ensure that dimensional and categorical illness will be indistinguishable. The final section deals with the implications of these issues.

From here on it is assumed that the bimodality under discussion arises from a symptom inventory of dichotomous symptoms scored 1 or Yes in the direction of illness. It is further assumed that the

score at issue is the simple sum of endorsed symptoms. These simplifications are made in the interest of clarity, and the points made below readily generalise to weighted symptoms sums and finer symptom scoring than (0, 1).

Bimodality with dimensional illnesses

This section explains Latent Trait models (contemporary psychometric accounts of questionnaire data assuming a latent dimension) and presents distributions of the symptom sum that explore the conditions under which a dimensional illness will yield bimodality. The close conceptual similarity between such dimensional models and disease entity models is examined.

Latent Trait model

The main applications of the Latent Trait theory are in aptitude-testing (Rasch, 1960; Birnbaum, 1968; Lord, 1980), and the theory seeks to account for questionnaire data in terms of a single latent dimension and aspects of the individual items. If one equates the items with symptoms and regards the questionnaire as an inventory relating to an underlying dimension of illness severity, then the theory is quite appropriate in psychiatric contexts. Such applications are now appearing in the literature (Gibbons *et al*, 1985; Duncan-Jones *et al*, 1986; Grayson, 1986).

The model usually assumes that the population takes a standard normal distribution (mean zero, unit variance) on the underlying illness dimension. Those with positive scores are 'sicker' than those with negative scores. The remaining feature of the model attempts to capture how the individual symptoms relate to this latent illness dimension. Generally it is assumed, for each symptom, that probability of endorsement is an increasing function of illness severity only. The higher a subject is on the illness dimension, the greater his probability of endorsing a given symptom. This assumption is very general, and in applications the symptom functions are given explicit functional form.

Figure 1 represents such a structure. On the x-axis the value on the latent dimension is plotted. The lower graph shows, inverted, the normal distribution of subjects on this dimension, and the upper graph shows, for a given symptom, the function relating endorsement probability to the value on the latent dimension. Subjects higher on the dimension ('sicker') have higher probability of symptom endorsement. This particular symptom function has the 2-parameter logistic representation. If $P_j(z)$ is the probability of symptom j endorsement for a subject of illness dimension value z, then

$$P_j(z) = \frac{\exp\,[s_j(z-t_j)]}{1+\exp\,[s_j(z-t_j)]}$$

or

$$\log\left(\frac{P_j(z)}{1-P_j(z)}\right) = s_j(z-t_j)$$

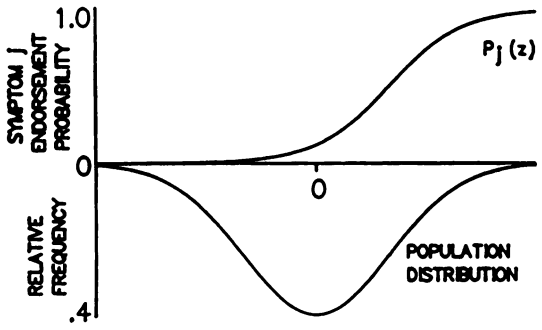

FIG. 1 Symptom function for a dimensional illness.

The expression is characterised by two parameters, s_j and t_j. When $z=t_j$, $P_j(z)=0.5$. So t_j is the value of the illness dimension that yields a 50% chance of endorsing symptom j. It is called the 'threshold' of the symptom, and serves to locate the symptom at a particular place on the illness dimension. This logistic curve rises slowly from the left, has maximum slope at $z=t_j$, and flattens out above this value. The slope at $z=t_j$ is $s_j/4$, *and the* s_j parameter is called the 'slope'. The larger the s_j value, the better the symptom is discriminating at threshold, where it must discriminate maximally. The symptom in Fig. 1 has a threshold at $t_j=1.0$ and a slope of $s_j=2.0$, indicating that it is discriminating well among moderately sick (positive z) subjects, but not among the well or the extremely sick.

Dynamics of dimensional bimodality

By choosing a number of symptoms with particular (s_j, t_j) combinations, it is possible to generate the data arising from application of a symptom inventory to a dimensional illness, and thus to investigate the conditions under which bimodality occurs. Such theoretical distributions are presented in Tables I–III.

Table I shows the distributions of this number-endorsed score for a variety of inventories applied to a normally distributed latent dimensional illness. In each inventory all symptoms are identical, with $t_j=0$ and s_j as indicated in the table. The number of symptoms in the inventory is also specified ($n=6$ or $n=23$). The slopes range from 16 to 1.9.

In all these examples, distinct bimodality occurs. The dynamics of this effect seem clear. All symptoms are located in the middle of the population ($t_j=0$). Thus, the bulk of the subjects low on the dimension rarely endorse many symptoms, and the bulk of those high on the dimension rarely fail to endorse many symptoms. This is more so for steeper slopes. Thus over intermediate patterns of symptoms there is a preponderance of all-zero or all-one patterns of responses, and hence bimodality in the distribution. So two conditions jointly yielding dimensional bimodality are coincident symptom thresholds and moderate to steep slopes. In other words, the symptoms must be discriminating well at a similar level of illness severity.

Examples in Table II show that the bimodality is surprisingly robust. The twelve-symptom inventory consists

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TABLE I
Bimodal distributions from dimensional models; 1000 subjects; $t_j = 0$

		Distribution of number of endorsed symptoms												
Slope (s_j)	*Inventory size (n)*	*0*	*1*	*2*	*3*	*4*	*5*	*6*	*7*	*8*	*9*	*10*	*11*	*12*
16	6	443	30	19	17	19	20	443	—	—	—	—	—	—
16	12	425	27	15	11	9	8	8	8	9	11	15	27	425
7.94	6	389	58	37	33	37	58	389	—	—	—	—	—	—
7.94	12	354	51	29	22	19	17	17	17	19	22	29	51	354
3.95	6	293	102	72	66	72	102	293	—	—	—	—	—	—
3.95	12	235	82	54	42	37	34	33	34	37	42	54	82	235
2.60	6	218	130	103	97	103	130	218	—	—	—	—	—	—
2.60	12	149	92	70	60	54	51	50	51	54	60	70	92	149
1.90	6	161	144	131	127	131	144	161	—	—	—	—	—	—
1.90	12	91	87	79	73	70	67	67	67	70	73	79	87	91

TABLE II
Distributions from dimensional models; 1000 subjects

	Distribution of number of endorsed symptoms												
Inventory size (n)	*0*	*1*	*2*	*3*	*4*	*5*	*6*	*7*	*8*	*9*	*10*	*11*	*12*
6	140	200	112	96	112	200	140	—	—	—	—	—	—
12	70	114	112	73	57	51	49	51	57	73	112	114	70

TABLE III
Distributions from dimensional models with no extreme symptoms; 1000 subjects; $s_j = 3.0$

		Distribution of number of endorsed symptoms												
Thresholds	*Inventory size (n)*	*0*	*1*	*2*	*3*	*4*	*5*	*6*	*7*	*8*	*9*	*10*	*11*	*12*
0, 0.2, 0.5, 0, −0.2, −0.5	6	213	130	107	101	107	130	213	—	—	—	—	—	—
	12	149	88	69	60	55	52	52	52	55	60	69	88	149
0.1, 0.3, 0.6, −0.1, −0.3, −0.6	6	197	132	115	111	115	132	197	—	—	—	—	—	—
	12	136	86	70	63	59	57	57	57	59	63	70	86	136

of two six-symptom inventories combined, and both these inventories consist of symptoms with (s_j, t_j) combinations (4, 0), (4, 0), (4, 0.2), (4, −0.2), (1.7, 1.2), (1.7, −1.2). The total proportions endorsing the symptoms are 0.5, 0.5, 0.43, 0.57, 0.2 and 0.8, respectively. The first, third and fifth symptoms have thresholds lying at the 50th, 58th and 88th population percentiles, respectively. The fifth and sixth symptoms, at either extreme, have the effect of ensuring that few subjects at the well extreme and few subjects at the sick extreme obtain inventory patterns of all zeros or all ones respectively. Thus the distribution is bimodal with two 'humps' rather than U-shaped.

All the distributions in Table III were generated with symptoms having a common slope of 3.0. The six-symptom inventories were constructed from three such symptoms with given thresholds and three with the same, but negative thresholds. The twelve-symptom inventories, again, are two six-symptom inventories combined. Removing the extreme items restores the more extreme U-shape bimodality.

All these examples demonstrate that a dimensional illness can produce bimodality, and together they provide insight into how this occurs. The conditions seem to be that the symptoms all perform their best discrimination round about the same place on the latent dimension. In terms of the

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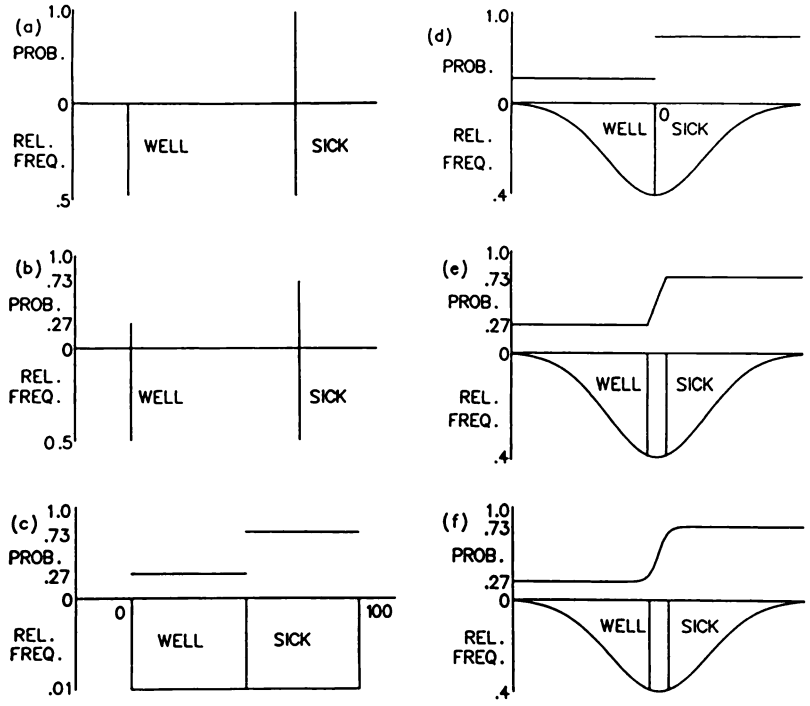

FIG. 2 Symptom function for a categorical illness.

symptom functions used here, the thresholds of a majority of symptoms must cluster about a common region on the illness dimension, making the population appear as two groups in the inventory data.

Similarity of data from dimensional and categorical illnesses

The data from a symptom inventory consist of frequencies of the possible patterns of ones and zeros across the inventory. These in turn depend on the relationship between each symptom and the underlying illness. If the responses to a given symptom can be shown, in principle, to be produced in much the same way from either type of illness, then it is, in principle, impossible to discriminate categorical from dimensional illness using data from a symptom inventory.

Part (a) of Fig. 2 represents the ideal relationship between a symptom and a categorical illness (with prevalence of 0.5 in the study population). Sick subjects, and only sick subjects, endorse the symptom. This is ideal, and an inventory of such symptoms would yield response patterns consisting of all zeros (the well group) and all ones (the sick), and nothing intermediate.

Two ways in which this unrealistic ideal might be realistically degraded are as follows. The first is by allowing the symptoms to be less than perfectly reliable. The second is by allowing the illness to be less than perfectly categorical; that is, having some subjects intermediate between well and sick. In accordance with the concepts of Kendell (1975), such subjects should be relatively infrequent.

Part (b) of Fig. 2 shows a symptom which is not perfectly reliable. Sick subjects have 0.73 probability of endorsing the symptom, well subjects 0.27. Part (c) is another way of representing Part (b), but along an artificially constructed dimension instead of illness categories. The well group is aligned fully to the left of the sick group. Within each group the subjects are ordered randomly. To each subject one can assign a percentile score reflecting the percentage of the total population to the left of that subject. Thus, well subjects will have percentile scores of 50 or less, and the sick will have greater than 50. A given subject still retains the same symptom probability as in part (b). Expressing the mechanism giving rise to symptom data in this way is theoretically fully equivalent to that in part (b), even if artificial.

Part (d) of the figure takes the representation one step further, but is still equivalent to the two earlier representations. To each subject is assigned the z-score associated with that subject's arbitrary percentile score (obtained by using tables of a standard normal variate). The symptom's relationship to the two categories is now shown as a symptom function over a score which is normally distributed in the study population, albeit a 'score' with no empirical interpretation.

Next, the assumption of less than perfectly distinct categories is incorporated by imposing a small 'grey area'

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between the sick and well groups. Sick subjects still have 0.73 probability of symptom endorsement, well subjects 0.27, and intermediate subjects have intermediate symptom-endorsment probability, as in part (e) of the figure.

Finally, if we allow the reasonable possibility that within either the well or sick groups, symptom-endorsement probability is not totally homogeneous, part (f) results.

Comparison of part (f) with Fig. 1 reveals the close conceptual similarity between the mechanisms generating symptom inventory data in categorical and dimensional models of illness. It is this close similarity which, in principle, ensures that discrimination between the two views, using inventory data, will be difficult.

A categorical illness differs, in general, from a dimensional one for two reasons:

(a) If the subjects in the 'grey area' are infrequent, then the symptom functions must rise steeply above the corresponding region (the functions must have steep slopes).

(b) Each symptom function must change above the same region (the thresholds must cluster).

These are precisely the conditions under which a dimensional illness will yield bimodality, and it is argued in the next section that psychiatric research on this issue has systematic biases which tend to ensure that these conditions are met when an illness is dimensional.

Influence of bias on dimensional illness

Types of bias

Most researchers are aware of 'observer bias', outlined by Kendell (1968). Given a particular inventory, a researcher predisposed to a categorical view of the illness simultaneously classifies patients and completes the diagnostic inventory in accordance with his or her predisposition.

Symptom selection bias

The same prejudgement can operate, however, in the initial selection of the symptoms to be included in the inventory, even were it to be subsequently administered by an incorruptible computer. If an illness truly is dimensional and a researcher is predisposed to the (erroneous) categorical view, the researcher's belief or intuition must be based on observation of some sort, and presumably those extreme on the dimension will share characteristics that vaguely define the researcher's mistaken concept of a sick group. Consequently, it is to be expected that the symptoms chosen will be none other than the characteristics discriminating the dimensionally extreme from the rest. That is, the symptoms likely to be included in the inventory will be a biased selection from the possible universe of symptoms, and will have thresholds constrained to cluster about the region on the dimension corresponding to the researcher's mistaken perception of the categories' boundary.

Such a bias, if operative, is compounded by the common advice to exclude items whose overall endorsement probabilities are far from 50% (outside the 20–80% range), at least in study populations constrained to have near-equal representation of the putative sick and well groups.

Subject selection bias

In most applications of the bimodality approach, if not most psychiatric research, the study population is not the community population. Even when the community population is distributed normally along an illness dimension, if study populations overemphasise one or both extremes of this continuum, then they must artificially conform to a disease entity situation. For example, Cloninger *et al* (1985) selected hospitalised schizophrenics to compare with others, and search for bimodality. If, in reality, the illness is dimensional, then such a selected population will not include moderate schizophrenics who fail to appear at hospital, thus creating genuine distinct groups in the artificial study population.

Figure 3 represents the action of these biases. Three symptoms with slopes of 2.0 are shown in part (a). They have thresholds of 0.7, 1.0 and 1.3, indicating the operation of symptom selection bias. The population is the community population. Part (b) shows the effect of overemphasising the extreme 15% of the community population so that they

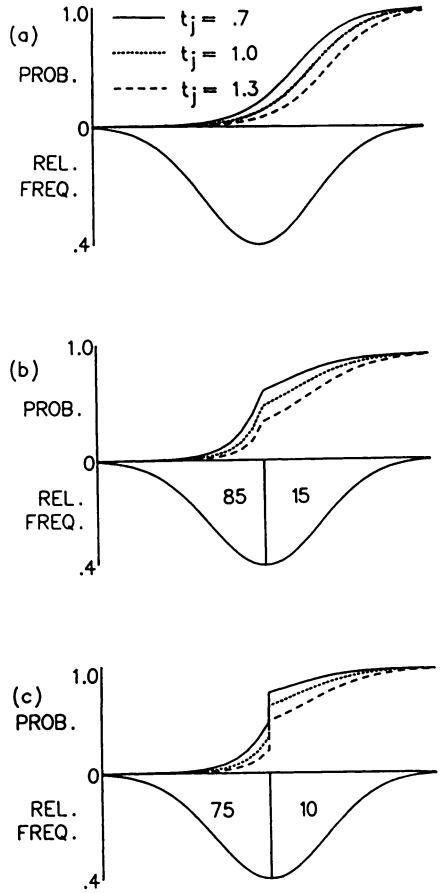

FIG. 3 Influence of biases on a dimensional illness.

become 50% in the study population. Part (c) describes the situation where the lower 75% and the upper 10% are chosen to form the artificial study population. These biases serve to ensure that the conditions required for categorical and dimensional illnesses to manifest identically are in fact met; that is, guarantee that the clustering of thresholds and steepening of slopes required for dimensional bimodality will arise.

Illnesses with two subtypes

The same problems arise in the search for binary disease entities, such as reactive and endogenous depression, although the interplay is more subtle. The study population is usually taken from treated series in hospital or clinics; for example, from hospitalised depressives, in the search for reactive and endogenous categories. As Eysenck (1970) says:

> It seems likely that selection for hospitalization is more in terms of severity . . . than in terms of endogenous or reactive . . . This might easily lead to a predominance of patients combining symptoms of endogenous and reactive depression in hospital, thus . . . disguising any bimodality that might actually exist. Conversely, it is possible that the Newcastle workers have selected patients for inclusion on the basis of extreme scores on Kendell's continuum, thus affecting the shape of the distribution in the opposite direction.

The appearance of bimodality is also related to the selection of symptoms. If the symptoms chosen are those that discriminate endogenous depressives (be they from a category or a dimensional extreme) from normals, and reactive depressives from normals, then they will not discriminate ideally between the two depressions. For this latter task, symptoms are required which are simultaneously endogenous and not reactive (or vice versa). On the other hand, symptoms which discriminate between the two groups may do so on bases other than endogenous versus reactive depression. Two groups of people will differ not only on the criterion used to segregate them, but on a variety of correlated criteria. Such other criteria, each separately correlated with the segregation criterion, need not intercorrelate among themselves, and thus will not produce bimodality, as the following example demonstrates.

Consider a two-symptom inventory applied to two groups of 16 subjects each, classified as endogenous and reactive depressives. Suppose the response patterns (1, 1), (1, 0), (0, 1) and (0, 0) occur with frequencies 8, 4, 4, 0 respectively among the reactives, and with frequencies 0, 4, 4, 8 among the endogenous depressives. For either symptom, the association with the depressive categories is given by the 2×2 table

	Symptom response		
	Yes	*No*	
Reactive	12	4	16
Endogenous	4	12	16
	16	16	32

As discriminators of the classification all items perform well; the χ^2 is 8 on 1 degree of freedom. Yet the number-endorsed distribution is unimodal, even with only two symptoms. Scores of 0, 1, 2 occur with frequencies of 8, 16, 8.

The key to understanding this apparent anomaly lies in the observation that the symptoms are uncorrelated:

		Symptom 1		
		Yes	*No*	
Symptom 2	*Yes*	8	8	16
	No	8	8	16
		16	16	32

Bimodality arises with a preponderance of patterns of two types, almost all zeros and almost all ones. This requires intercorrelations among the symptoms; it is not sufficient that they discriminate well individually. The lack of such intercorrelations indicates that the symptoms are discriminating on unrelated criteria. Constructing an inventory which is heterogeneous in this sense will ensure, even when the symptoms individually discriminate a common classification, unimodality on some dimension which is a pot-pourri of them all. Conversely, an inventory with symptoms tapping the same construct, and with all symptoms centrally located, must yield bimodality whether the construct is dimensional or categorical.

This phenomenon may underlie the apparent conflict between the depression bimodality found by Carney *et al* (1965) and the failure of Kendell & Gourlay (1970*a*) to replicate it. Close reading of the symptom selection procedures of the former shows that a major factor in selection was *a priori* content validity of the symptoms in their instrument, while the latter authors report including symptoms which discriminated their classification but appeared unrelated conceptually to the distinctions between the depressives. They also report excluding symptoms from their final inventory that would usually be included on the basis of their content. Similar comments apply to unimodality found by Kendell & Gourlay (1970*b*) in their search for schizophrenic and affective categories.

Implications

Latent Trait models have been used as a heuristic device to demonstrate that bimodality may arise from a dimensional illness. This counter-intuitive result has not been noticed in the past (Moran, 1966; Maxwell, 1971; Kendell, 1975; Garside & Roth, 1978). Far from being a pathological outcome, the likelihood of such dimensional bimodality is enhanced by symptom and subject selection biases.

Statistical analysis of symptom inventory data

If one accepts the reasoning in this paper then mixture analysis, as recommended by Fleiss (1972)

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and Everitt (1981), and practised by Cloninger *et al* (1985) is, unfortunately, useless for distinguishing between categorical and dimensional illness models. This statement is no less true of discriminant analysis, factor analysis, clustering, numerical taxonomy (Pilowsky *et al*, 1969) and Boolean factor analysis (Weber & Scharfetter, 1984). All of these techniques are applied to the basic symptom inventory data, and insofar as these data are incapable of distinguishing the two illness models, any subsequent analysis of such data is also incapable of making such a distinction.

The technique of Kendell & Brockington (1980) is also suspect. Non-linearity between an outcome score and a symptom inventory scale score would be quite possible whenever bimodality arises in the latter. Since this can occur with dimensional illnesses, so can the non-linearity they would interpret as a hallmark of categorical illness states.

Recommendations

From this perspective, certain conditions must be met before bimodality can be used to infer that an illness is categorical. First, the study population must not be artificially constructed so that it conforms to a categorical structure if in fact the illness is dimensional. In this sense, the community population is an ideal study population.

Secondly, it must be shown that the symptoms chosen are not preselected on *a priori* grounds which relate to an erroneous belief that the illness is categorical. This is tantamount to ensuring that no symptoms can be found which discriminate among the study population at locations other than the putative boundary between the categories.

Thirdly, the symptoms must be homogeneous, or discriminating among those with and without the disease on the same basis, namely that related to the disease in question.

Even then the inference is more an evaluative judgement than a rejection of the dimensional view on qualitative grounds.

Conclusion

In exploring the ramifications of a dimensional view of illness, the nature of a symptom inventory as an instrument has been emphasised. What is seen of the world is an interactive function of three components: reality, the viewing instrument and the viewer. It is quite possible for reality to be continuous, for the instrument to function best at just one point on that continuum, and for the viewer to point the instrument selectively at that region and to conclude that this region is a qualitative boundary. This is not to say that it is not a boundary, but it is not to say that it is. Before one so concludes, there is an onus to demonstrate that the instrument is not selective, but is perceiving reality as it is.

Where the instrument is a psychiatric inventory, Latent Trait techniques seem the most promising way of unravelling this interaction.

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D. A. Grayson, BA(Hons), PhD, *Postdoctoral Research Fellow, NH&MRC Social Psychiatry Research Unit, GPO Box 4, Australian National University, Canberra ACT 2601, Australia*