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Correspondence

PREDICTIVE ACCURACY AND CLINICAL UTILITY OF THE GRID TEST OF SCHIZOPHRENIC THOUGHT DISORDER

DEAR SIR,

Hill (1976) has argued that the Grid Test of Schizophrenic Thought Disorder (GTSTD) shows appreciable statistical validity in terms of agreement with clinical judgements of thought disorder but that clinical utility is suspect because, in part, prediction on the basis of baserates often exceeds test prediction. I wish to show that Hill has misapplied the logic of discriminative efficiency and clinical assessment.

Wiggins (1973) has stated that a valid test always possesses greater predictive accuracy than that possible for baserate prediction alone, and has called attention to a misleading example in Meehl and Rosen (1955) that apparently has given rise to the belief that baserate prediction can exceed in accuracy the use of a valid indicator. The correct relationship is shown in the expression (Wiggins, 1973, p. 252):

$$P(VP) = (BR)(SR) + phi\sqrt{BR(1-BR)SR(1-SR)};$$

where P(VP) = probability of valid positives; BR = the baserate; SR = the selection ratio; and phi = the validity coefficient as the phi coefficient.

That is, given a validity coefficient greater than zero, the P(VP) always is greater than that predicted on the basis of baserates (when phi = 0 in the random case).

Using Hill's (1976) example, data pooled from Frith and Lillie (1972) and Bannister, Fransella and Agnew (1971) give a phi value of .286. Let this be the validity of the GTSTD. Imagine a new sample of patients. Suppose the baserate for thought disorder is 10 per cent, as Hill estimates, then retaining the selection ratio implicit in his example, baserate prediction gives P(VP) = 2 per cent. However, with phi = .286, P(VP) is now 5.5 per cent, an increase in predictive accuracy of 3.5 per cent. The importance of this gain in accuracy is a matter of incremental validity (Sechrest, 1963). This example illustrates that a test of validity greater than zero will always yield greater predictive accuracy than that possible from baserate prediction alone.

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HYSTERIA AND URBANIZATION

DEAR SIR,

We should like to report a recent study that we carried out on the incidence of hysteria in one part of Japan. We defined hysteria as the development of physical symptoms in the absence of physical illness but in the presence of some significant psychological change in life circumstances. Most of our patients complained of breathing disturbances, fits or pain, and all displayed importunate and suggestible behaviour in their interactions with medical staff.

We reviewed the notes over two decades (1952–1973) of all women attending the psychiatric outpatient departments of two general hospitals which between them provided the main hospital services for a mixed rural and urban population of one million.

There were two main findings. First the incidence of hysteria in women fell over this period. In the early 1950s it was diagnosed in about 6 per cent of new outpatients; by the early 1970s less than 2 per cent were so diagnosed, despite a comparable attendance rate. Secondly, the incidence bore some relationship to the type of area in which the patient lived. It was highest in the suburbs but low in both rural and inner city areas.

Our finding of a decline in hysteria has been reported from the United States (Stefansson *et al*, 1976) and elsewhere in Japan. Our finding that the incidence is affected by population density has also

been reported previously (Imura *et al*, 1954; Higashimura and Kamiya, 1974). We were, however, intrigued by the relatively high incidence in the suburbs. These suburbs contain a population who have recently undergone radical changes in life-style, representing for the most part rural families who have moved into the satellite towns of the city, to take up factory jobs. We speculate that it may be a loss of traditional sociocultural ties which increases the risk of an hysterical disorder.

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RAISED MONOAMINE OXIDASE IN ACUTE PSYCHOSIS?

DEAR SIR,

We read Drs Brockington and Owen's report on platelet monoamine oxidase activity in acute psychosis (*Journal*, March 1980, **136**, 312) with much interest. They found significantly increased levels of MAO in acutely psychotic patients, as well as a significant correlation between affective flattening and activity of the enzyme. Since the most obvious differences between acute and chronic psychosis are in levels of anxiety, agitation and restlessness, increased anxiety would seem the most likely explanation for increased MAO activity in acutely psychotic individuals.

Stress has been shown to elevate MAO in animals (Pryor *et al*, 1972); in humans, subcutaneous injections of epinephrine have activated the enzyme (Gentil *et al*, 1975; Gentil *et al*, 1976; Owen *et al*, 1977).

We assayed MAO activity via phenylethylamine and tryptamine substrates in 20 drug-free subjects. Patients were physically healthy and had a diagnosis of generalized anxiety disorder; an equal number of normal control subjects were also included. Assays were done before and after four weeks of relaxation training. Blood samples were drawn after the subjects relaxed in a supine position for 30 minutes in a semi-dark room; all samples were taken at the same time of the day. Subjects were instructed to avoid coffee, tea and tobacco prior to the assays. Anxious subjects showed significantly higher pre-treatment levels of MAO as compared to the controls (Table I). The post-treatment evaluation indicated a reduction in levels of anxiety and MAO activity. At the post-treatment level there were no significant differences in MAO activity between the index and control groups. When the pre-treatment and post-treatment levels of enzyme activity were compared, anxious subjects showed significant reductions following treatment (index group post-treatment mean, PEA substrate 32.10, SD = 10.42, $t = 2.42$, $P < .03$ and tryptamine substrate 7.66, SD = 2.25, $t = 3.34$, $P < .003$); controls showed no significant differences. Plasma levels of epinephrine and platelet MAO were assayed in 15 anxious subjects before and after relaxation training. Significant correlations were found between epinephrine and MAO both at the pre-treatment (tryptamine substrate $r = .46$, $P < .05$, PEA substrate $r = .46$, $P < .05$) and post-treatment (tryptamine substrate $r = .54$, $P < .05$; PEA substrate $r = .51$, $P < .05$) levels. Our data indicate that anxiety influences MAO activity. This finding may explain the increased activity of monoamine oxidase in acutely psychotic patients.

TABLE I

Comparison of pre-treatment levels of MAO activity between anxious and normal subjects

	Anxious subjects		Normal subjects			
	Mean	SD	Mean	SD	t	P
PEA	37.26	14.33	29.29	10.79	1.99	<.05
TRYPT	9.42	3.57	6.94	2.42	2.56	<.01

MAO expressed as nmoles/4 × 10^8/hr.

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