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Comments

Psychosis and Psychoticism: A Reply to Bishop

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This brief rejoinder to Bishop's critique of the Eysenck Personality Questionnaire, and in particular the concept of psychoticism (P), emphasizes that when all the evidence now available is taken into account and when the theory is seen in its proper development, the criticisms advanced by Bishop will be seen not to be tenable. Some of her views are taken up in detail and are contradicted by empirical facts while others depend on misunderstandings. It is concluded that there is much evidence for the viability of the concept of psychoticism and for the validity of the questionnaire measurement of P.

Bishop's (1977) criticism of the concept of "psychoticism" (P), embodied in the Eysenck Personality Questionnaire (EPQ; Eysenck & Eysenck, 1976a), deals with many points that have been treated in great detail in our book *Psychoticism as a Dimension of Personality* (Eysenck & Eysenck, 1976b). This book came out after Bishop wrote her critique and was therefore not available to her. It is unfortunate that Bishop's views are based on the manual of the test, as this is not usually the place to discuss fundamental theoretical points. The proper reply to Bishop will be found in the book, rather than this short rejoinder; quite obviously it would not be possible to go into experimental details here, nor would such replication of what has been said elsewhere be useful. Bishop would not have been aware that strong support for the conception of psychoticism comes from certain genetic studies given in detail in our book. This contains a lengthy chapter that reports a large scale twin study, using 544 pairs of twins, and demonstrates that potentially identifiable environmental factors account for 19% of the reliable variation in P, the remaining 81% being our estimate of the heritability of P, after correction for unreliability of measurement. (P shows no evidence of directional dominance or assortative mating.) We also found that a genetic model containing only D_R and E_1 (i.e., additive genetic variance and within-family environmental variance) was adequate to account for our empirical findings; it is interesting that this model is identical to that which was found to be most in accord with empirical findings concerning schizophrenia (Eaves & Eysenck, 1977). This is powerful support for our conception of P as psychoticism, although, of course, it does not *prove* this interpretation to be correct. In the nature of things, no such proof is possible as we are dealing with concepts that can be useful or useless, but they cannot be true in any meaningful sense. Much of Bishop's critique is vitiated by a failure to understand this point.

Bishop might rightly say that this book was not available to her when she wrote her critique; such a reason would not apply to her failure to deal with my original article, which states my genetic hypotheses in relation to psychoticism and psychosis (Eysenck, 1972). This article contains references to back up such claims as are made in the manual of the EPQ, which Bishop uses as her main source; thus she quotes one of our suggestions and complains that "no references are provided to support this claim" (p. 131). It would be very unusual to give numerous citations in support of particular theoretical statements in a manual for a personality test, particularly when the references have already been set out in detail elsewhere!

I will take up in this rejoinder only a few of the points made by Bishop, referring the interested reader to our book for a much more detailed treatment of the points at issue (Eysenck & Eysenck, 1976b). To begin with, Bishop (1977) is wrong in stating that "unlike N [neuroticism] and E [extraversion], P was not formulated to account for empirical data; rather, it was postulated on theoretical grounds" (p.

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127). All three concepts grew out of a consideration of psychiatric writings of the early 1920s, in particular those of Jung and Kretschmer, as I have made clear elsewhere (Eysenck, 1970). Kretschmer (1948), in particular, insisted very strongly on the continuity between psychotic, prepsychotic, and "normal" individuals along his cyclothymia–schizothymia scale; he was followed by a large number of German psychiatrists in this view. That there is true continuity between psychosis and normality (what Bishop calls "Hypothesis A") is of course an arguable point; certain symptoms, such as paranoia, depression, lack of insight, and so on, would on commonsense grounds suggest an affirmative answer, whereas others, such as having hallucinations, would not. No writer known to me has suggested a method of disproving the notion of continuity, and it was for this reason that I proposed the method of *criterion analysis*. Bishop (1977) is critical of this; she states that

> Eysenck (1952) uses the continuity claim to refer to continuity in *indices* of psychosis, which are correlated with, but not identical to clinical symptoms. Hypothesis B is a much weaker claim than Hypothesis A, unless there are grounds to believe that the indices used directly reflect the underlying condition responsible for the behavioral symptoms. (pp. 127, 128, italics added)

I would say that there are such grounds; how else would a well-variegated set of tests discriminate very significantly between normals and psychotics? And how else would we find the correlations actually observed? Bishop's extension of the Costello (1970) analogy is irrelevant; it concerns a battery of homogeneous tests, that is, in fact a single measure of a known factor. I was using a battery of heterogenous tests; this, in my view, completely alters the picture and makes the Costello/Bishop argument untenable.

I do not, of course, deny the distinction between psychosis and psychoticism; if I did, there would be no point in labeling the scale one of psychoticism! The distinction is the same as that between neurosis and neuroticism. I accept the widely held diathesis-stress theory of mental disorder; the diathesis part refers to the -ism concepts (neuroticism and psychoticism), which, when multiplied by suitable stress, produce the neurosis or psychosis states. These are conceived of as exaggerated or blown-up versions of the inherited -isms; the odd behavior of the high P scorer becomes even odder, the fearful behavior of the high N scorer becomes even more fearful. I would think that the change was a quantitative one, not a qualitative one, but, of course, Bishop is entitled to ask for more convincing evidence.

Bishop seems to doubt the usefulness of my dimensional view of psychosis, on the odd grounds that "Eysenck's scheme alone does not distinguish between disorder and health" (p. 130). This is clearly untrue, as we have demonstrated elsewhere (Eysenck, White, & Eysenck, 1976). It is shown in Eysenck et al. (1976) that when a discriminant analysis is carried out on the scores of seven male and seven female groups of 63 subjects each (normal, criminal, schizophrenic, endogenous depressive, personality disorder, anxiety state, reactive depression), the groups are separated in a perfectly meaningful manner, with the first variate separating out the normal from the abnormal groups and the second separating out the psychotic from the neurotic groups. The accuracy with which the test allocates each person to his or her proper group is not impressive, but it is not very much lower than the reliability of psychiatric diagnosis, which obviously sets an upper limit on the accuracy of matching. Certainly this scheme makes no absolute distinction between disorder and health; I know of no evidence that would require us to make such a clear break. It does, however, succeed in allocating the psychiatrically abnormal subjects to the abnormal end of the continuum and the normal subjects to the normal end. It is difficult to see what else could be expected from such an instrument or such a theory.

Bishop seems to rest much of her argument on the fact that some nonpsychotic groups have higher P scores than some psychotic groups. This argument is based on an elementary fallacy, namely, the use of one scale by itself. As we have insisted, psychotics often have high Lie scale (L) scores, and the negative correlation between P and L makes good psychological sense in terms of the conformist behavior indicated by high L scores (Eysenck & Eysenck, 1976b). Eysenk et al. (1976) show how scores must be combined to give maximum discrimination, and the necessary formulae are given in the manual. P scores without regard to dissimulation (L) are not very meaningful in our system and should not be used to support such an argument as Bishop's.

We must next turn to Bishop's discussion of the validity of P as a measure of psychotic predisposition. She states that we have used two methods of validation, the first of which is that psychotics should have elevated P scores, the

second that high P scorers should, on objective and laboratory tests, score in such a manner, as compared with low P scorers, as do psychotics as compared with normals. There is much more evidence on these points than is discussed by Bishop, and our conclusion from a consideration of this evidence was that tentatively at least they support our identification of P with psychoticism (Eysenck & Eysenck, 1976b). There is a third method of validation, namely, that within a psychotic population, those more seriously ill (by psychiatric and objective test criteria) should have higher P scores than those less seriously ill. This also has been established, at least provisionally. As part of this method of validation, we might also consider the fact that psychotics who improve in their clinical status decline in P scores, whereas those who do not improve do not decline in P scores. We consider this third method equally as important as the other two; Bishop might have to reconsider many of her arguments in light of these facts.

We consider the fact that other groups that have been found by genetic studies to form part of the genetic *Erbkreis* of psychosis have high scores on P as support of our hypothesis; this fact was certainly predicted from our theory. Criminality and antisocial behavior generally are predicted to be correlated with high P scores, and this has uniformly been found. Bishop suggests that institutionalization and/or alienation from society as a consequence of criminals' condition might provide alternative similarities. This is unlikely; identical relations have been found in uninstitutionalized children (Eysenck & Eysenck, 1976b), for instance. For any single finding, there will always be alternative possibilities of theoretical explanation; it is when the totality of the facts is looked at that this becomes much more difficult.

I have suggested that the P dimension may find a biological basis in the hormonal field, with high P being related to high androgen content. The evidence has been reviewed in my book *Sex and Personality* (Eysenck, 1976), and although it would be quite premature to come to any definitive conclusions, the empirical data tentatively support such a conclusion. Bishop (1977) is not convinced; she maintains that "if the mean P score for men is higher than that for women, we would expect many more male than female schizophrenics" (p. 132). This leads her to state that "the sex difference in P scores, far from being a source of validation for the P scale, goes directly against it" (p. 132). This argument goes counter to the discussion of this point offered by Gray (1973), who stated:

> In looking at the sex differences in the incidence of psychosis itself, account must be taken of the age of onset of the disorder. In the case of schizophrenia, there is initially a high incidence in males, who show a particularly marked rise in first admissions between the ages of 15 and 25; females begin to catch up with males at about the age of 35 and there is a marked preponderance of females after age 45. . . . A natural implication of this pattern is that the onset of full male sexuality, and of the social interactions which entry into adulthood requires of the male, somehow facilitates the occurrence of a schizophrenic illness, while female sexuality actually affords protection against schizophrenia, this protection being removed at the time of the menopause. With regard to depression, the psychotic form of this symptom is associated with the male sex in Kendell's (1968) factor-analytic study, in distinction to reactive depression, which is predominantly found in females. (p. 449)

Bishop's superficial view of overall figures pays no attention to the support that is given to our hypothesis by Gray's more detailed discussion. I conclude that the figures about sex and psychosis support our theory, if only mildly. (More details can be found in Gray's, 1973, article; I have only quoted a small part of his argument.)

Bishop (1977) concludes that it would be desirable to demonstrate that P had construct validity by prospective studies of high P scorers. Until this is done, "it cannot be concluded that the P scale measures anything other than the consequences of psychiatric disorder" (p. 133). Prospective studies have in fact been carried out (Eysenck & Eysenck, 1976b), demonstrating that high P scorers tend to respond less to psychotherapeutic measures than low P scorers; this is an important finding that may prove useful in clinical psychology. Other such studies are at present being carried out in relation to the future development of high- and low-P children. But even without such studies, I would dispute the meaningfulness of regarding P scores as nothing but "consequences of psychiatric disorder." Most of our studies have in fact been carried out on normal nonpsychotic populations, including large numbers of children; we have tried to test on these groups predictions originating in our general theory, for example, that high-P children are characterized by antisocial and criminal conduct. The successful outcome of such studies clearly indicates the viability of the concept and owes nothing to the concept

of psychiatric disorder patently absent from these populations. Much the same might be said of the prediction, also tested on normal groups, that high P scorers would be more "original" in their responses to typical creativity tests, or that high P scorers would draw more bizarre pictures. There is a wealth of evidence to show that testable predictions can be made from the hypothesis that P is a measure of psychoticism and that these predictions can be verified on normal groups of adults and children. We might include here our predictions regarding the heritability of P and the nature of the genetic model required, fitting exactly the model elaborated for schizophrenia. Indeed, the list of different kinds of evidence showing that the concept of psychoticism is viable is far too long for this short rejoinder; the reader must be referred to our book (Eysenck & Eysenck, 1976b).

One difficulty encountered in the construction of the P scale should be mentioned. Many of the items in the scale give rise to rather extreme breaks. There is no doubt that psychometrically the P scale is inferior to the E and N scales, as Baumann and Dittrich (1976) have pointed out in their German adaptation of the EPQ. This may be due to properties of the P dimension or to our inability to find items that would come nearer to the desired state of 50% responding. It might be thought that the dimension of P might just be an artifact of random responding; that is, consisting of items that are infrequently endorsed in a normal population, the P scale might find psychometric support by random responding, thus producing correlations between such "rare response" items artificially. We have looked into this possibility (Thompson, 1975) but had to discard this hypothesis.

It is also true that the distribution of scores on the P scale is skewed, very much like the distribution of scores on the Raven's Matrices test of intelligence. This may be a function of the actual distribution of P in the population or it may be an artifact of the particular choice of items involved (just as the skewed distribution of the Matrices test scores is due to the particular choice of items, having certain item difficulties); we cannot say at the moment. Only further work with the scale and attempts to construct other similar scales will give us some insight into these matters. They are less worrisome to us than to Bishop because we are more concerned with the question of validity than that of reliability. The value of a scale stands and falls with the provision of data regarding its validity; on this point we believe that the data summarized in our book (Eysenck & Eysenck, 1976b) will be found convincing.

References

Baumann, U., & Dittrich, A. Überprüfung der Fragebogendimension P (Psychotozismus) im Vergleich zu Extraversion und Neurotizismus. *Zeitschrift für Klinische Psychologie,* 1976, *5,* 1–23.

Bishop, D. V. M. The P scale and psychosis. *Journal of Abnormal Psychology,* 1977, *86,* 127–134.

Costello, C. G. Classification and psychopathology. In C. G. Costello (Ed.), *Symptoms of psychopathology*. London: Wiley, 1970.

Eaves, L., & Eysenck, H. J. A genetic model for psychoticism. *Advances in Behaviour Research and Therapy,* 1977, *1,* 1–38.

Eysenck, H. J. *The structure of human personality* (3rd ed.). London: Methuen, 1970.

Eysenck, H. J. An experimental and genetic model of schizophrenia. In A. R. Kaplan (Ed.), *Genetic factors in schizophrenia*. Springfield, Ill.: Charles C Thomas, 1972.

Eysenck, H. J. *Sex and personality*. London: Open Books, 1976.

Eysenck, H. J., & Eysenck, S. B. G. *Manual of the Eysenck Personality Questionnaire*. London: Hodder & Stoughton, 1976. (a)

Eysenck, H. J., & Eysenck, S. B. G. *Psychoticism as a dimension of personality*. London: Hodder & Stoughton, 1976. (b)

Eysenck, S. B. G., White, P. O., & Eysenck, H. J. Personality and mental illness. *Psychological Reports,* 1976, *39,* 1001–1022.

Gray, J. S. Causal theories of personality and how to test them. In J. R. Royce (Ed.), *Multivariate analysis and psychological theory*. London: Academic Press, 1973.

Kretschmer, E. *Körperban und charakter*. Berlin: Springer, 1948.

Thompson, A. H. Random responding and the questionnaire measurement of psychoticism. *Social Behavior and Personality,* 1975, *3,* 111–115.

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