THE DEFINITION AND MEASUREMENT OF PSYCHOTICISM

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Summary—In this paper an attempt is made to answer certain questions and criticisms concerning the concept of psychoticism (P) as a dimension of personality. The points addressed are: (1) Is it reasonable to talk about psychosis as a unitary concept, rather than about separate, unrelated disorders (schizo-phrenia, manic-depressive disorder)? (2) Is such a concept generalizable to form a continuum of 'psychoticism' with normality? (3) Is psychoticism related to psychopathy rather than to psychosis? (4) What methodology can be used to answer questions like those raised above to make answers more compelling than the suggestive naming of psychometric factors? It is suggested that an *experimental* approach must be combined with a *psychometric* one to obtain answers which go beyond the sterility often associated with a purely correlational approach, as suggested by Cronbach (1957; *American Psychologist*, 12, 671–684).

INTRODUCTION

In 1952, I suggested that in addition to neuroticism (N) and introversion-extraversion (E) there existed a third major dimension of personality, called psychoticism (P) which was orthogonal to N and E (Eysenck, 1952). Work testing various deductions from this hypothesis has been published periodically (Eysenck, Granger & Brengelmann, 1957; Eysenck, 1970a; Eysenck & Eysenck, 1976; Eaves, Eysenck & Martin, 1989), with largely positive results; reviews of the many studies generated by the original theory have been published by Claridge (1981, 1983) and Zuckerman (1989; Zuckerman, Kuhlman & Camac, 1988). A recent paper (Eysenck, 1991b) has attempted to consider the relation between the three dimensions postulated in the PEN model, and other typologies, such as Cattell's 16 PF (Cattell, Eber & Tatsuoka, 1970) and the 'Big Five' (John, 1990). It is suggested that the PEN model constitutes a paradigm (in the Kuhnian sense) in personality research (Eysenck, 1983a), and fulfils the stringent criteria suggested for acceptance of a paradigm (Eysenck, 1991b).

In this paper I shall be concerned with certain substantial criticisms of the P dimension, i.e. criticisms which are concerned not with purely psychometric issues, but with issues of great theoretical importance. To understand these issues, it may be useful to introduce the model I am putting forward. Figure 1 illustrates its major features. The abscissa constitutes a dispositional personality trait, psychoticism, which extends from the left (low P-high empathy, socialization, co-operativeness) to the psychotic characteristics and syndromes shown on the right (traits characteristic of high P are shown in Fig. 2). The distribution of P is more or less normal; in actual fact it has usually been skewed to the right (Eysenck & Eysenck, 1975), and although recent improvements in the scale have ameliorated this tendency, it has not been abolished (Eysenck, Eysenck & Barrett, 1985). Whether this feature is the product of psychometric faults in questionnaire construction, or inherent in the 'true' distribution of P, is not known; for our present purpose the answer is irrelevant. It may be noted in passing, however, that J-shaped distributions are quite common in psychology (Walberg, Strykowski, Rowai & Hung, 1984), and that Allport (1934) many years ago demonstrated the applicability of his J-curve hypothesis to conforming behaviour, which in many ways is the obverse of psychoticism.

Psychosis (schizophrenia, manic-depressive illness) is postulated to occur under environmental stress with a probability P_A , which is a monotonic function of psychoticism, as shown in the figure. Psychosis is not regarded as a category qualitatively different from normality, a point I have tried to establish by reference to criterion analysis (Eysenck, 1950, 1952a), and similarly different psychoses are not regarded as categorically different; in both cases we are dealing with *continua*



Fig. 1. Diagrammatic representation of the continuity theory of 'psychoticism'.

of one kind or another. Close to psychosis at the right of the diagram are behaviours variably diagnosed as schizoid, 'spectrum', or psychopathic, with 'personality disorder' a more recent synonym. Figure 2 shows some of the traits the intercorrelations between which is the ultimate justification for the postulation of P as a dimension of personality (Eysenck & Eysenck, 1976).

This model clearly violates a number of psychiatric assumptions, and it is important to answer these objections if the model is to prove acceptable. In addition, there have been some psychological criticisms which also demand an answer. In the following sections I shall try to answer these criticisms in some detail.

'PSYCHOSIS' OR DIFFERENT PSYCHOSES?

During the last century it used to be assumed that there was a common feature to all functional psychoses (the theory of the Einheitspsychose); Griesinger (1861), Guislain (1833), Neumann (1859) and Zeller (1837) may be quoted in support. Kraepelin (1897) separated manic-depressive insanity from dementia praecox (schizophrenia), conceiving of them as unrelated diseases; this conception is prevalent in most if not all textbooks of psychiatry and clinical psychology. (See Berrios, 1987, for an historical introduction.) Kraepelin (1920) himself pointed out some of the difficulties raised by this bifurcation; as he explained: "No experienced psychiatrist will deny that there is an alarmingly large number of cases in which it seems impossible, in spite of the most careful observation to make a firm diagnosis... it is becoming increasingly clear that we cannot distinguish



Fig. 2. Traits intercorrelating to form a higher-order factor of 'psychoticism'.

satisfactorily between these two illnesses and this raises the suspicion that our formulation of the problem may be incorrect." The well-known difficulty of obtaining acceptable reliabilities in the diagnosis of psychotic disorders bears ample testimony to this problem.

Few studies, oddly enough, have been devoted explicitly to the solution of this very fundamental problem. Kendell and Gourlay (1970) constitute an important exception to this criticism. Selecting 146 patients with a group diagnosis of schizophrenia and 146 with a group diagnosis of affective disorder, they devised a discriminant function analysis that achieved maximum separation between the groups. Diagnostic items favouring affective illness (early morning waking, delusion of guilt) were counted to one side, items favouring a diagnosis of schizophrenia (affective flattening, Schneiderian first-rank factors) to the other. The expectation on the basis of Kraepelin's hypothesis was that the distribution would be bimodal; in actual fact it is trimodal, with the major peak in the middle. In other words, schizo-affective cases are more common than 'pure' cases of either hypothetical disorder. When the experiment was repeated on a second sample a unimodel (normal) distribution was obtained. These results are quite incompatible with the Kraepelin theory, and suggest rather a general factor of psychosis, with a rather weak second bipolar factor. The position is similar to that in intelligence testing, where we also find a prominent general factor (g), and a much weaker bipolar factor dividing verbal from non-verbal tests (Eysenck, 1979).

Cloninger, Martin, Guze and Clayton (1985) have presented some contrary evidence to suggest that there does exist a 'point of rarity' between the symptom complex of schizophrenia and that of other psychiatric disorders. They used the self-report ratings of a series of 500 psychiatric outpatients and 1249 of their first-degree relatives, and derived a discriminant function distinguishing between the symptoms of schizophrenia and those of other conditions from the ratings of half their subjects; they then obtained a bimodal distribution of scores on this function when it was cross-validated using the ratings generated by their remaining subjects.

It is interesting that within schizophrenia, there are two major types which explain much of the variance: schizophrenia of acute onset and schizophrenia of gradual (invidious) onset. This constitutes the strongest predictor of the subsequent pattern of development, i.e. of the likelihood that a patient would develop a remitting or a continuous type of illness. As Jablensky (1988) points out: "Moreover, the follow-up demonstrated that patterns of course, significantly predicted by the mode of onset, tended to cluster at two extremes. On the one hand, there were cases of acute onset, in which the cumulative duration of psychotic episodes amounted to less than 15% of the length of the follow-up period. On the other hand, the majority of the cases of an insidious onset tended to be severely psychiatric for more than 75% of the follow-up period. Relatively few cases fell between these two extremes, and the statistical distribution was strongly suggestive of bimodality" (p. 27). Thus there may be no bimodal distribution when we look at phenomenologically different psychotics (schizophrenics, manic-depressives), but there is a bimodal distribution within a phenomenologically undifferentiated group of schizophrenics As Jablensky emphasizes: "At the height of the initial psychotic episode the two groups could not be distinguished from one another in terms of symptomatology, and in CATEGO class membership was not predictive of one or other patterns of course" (p. 287). If there are different kinds of psychosis, type of onset of schizophrenic illness creates a much clearer differentiation than does symptomatology of schizophrenia vs affective disorder!

It seems certainly true, as Farmer, McGuffin and Bebbington (1988) point out, that "in general, criteria that incorporate longitudinal variables such as duration of illness in their definition . . . fare better (with respect to the prediction of short and long-term outcome) than those relying purely on cross-sectional psychopathology" (p. 43) but this, combined with the varying nature of such criteria, leaves us in the curious position where, as Brockington, Kendell and Leff (1978) have put it, the previous state of inarticulate confusion in the diagnosis of schizophrenia has been replaced by a babble of precise but differing formulations of the same concept. This uncertainty must add considerably to the problem of deciding between a dimensional and a categorical basis of classification.

Kendell and Brockington (1980) tested another deduction from the Kraepelinian model, namely that discontinuity between diagnostic groups could be discovered by analysing the relation between diagnostic score and some outcome variable, such as time in hospital, occupational record, social outcome, etc. Non-linear regression would be indicative of a genuine discontinuity, but in testing

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eight outcome criteria against the schizophrenic-affective continuum, Kendell and Brockington failed to discover any such lack of linearity. As they say, "the results of this further analysis do not lend support to the view that schizophrenic and affective psychoses are distinct entities" (p. 266).

Quite generally, it appears that the likelihood of schizo-affective patients getting better is intermediate between that of schizophrenics (worst prognosis) and affective (best prognosis) (Crougham, Welner & Robins, 1974; Brockington, Kendell & Wainwright, 1980). Bimodality of outcome is not usually found, but continuity seems to be the rule (Crow, 1986). It should be noted, however, that there are statistical difficulties in using the shape of the distribution (unimodal, bimodal, etc.) as evidence for a categorical or dimensional model.

Grayson (1987) has given a thorough discussion of the considerations involved, and has shown 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 had previously been assumed. He is thus opposing the more common view expressed by Everitt (1981) that "for a population frequency curve, bimodality is (except in pathological cases) a sufficient (although not 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". In principle Grayson is right, but even so the evidence cited in this section strongly suggests, although it does not prove, that we are dealing with a continuum rather than with separate illnesses (which of course might still have differentiating features); for more conclusive proof we have to rely on other types of evidence.

When Kasanin (1933) introduced the concept of 'schizo-affective' psychoses, these were supposed to be relatively rare. Clearly they are not, and neither do they constitute a third type of psychosis; most psychotics seem to fall into this category, and there are no clear boundaries between them, schizophrenics and manic-depressives. Kendell and Brockington (1980) developed a method for establishing a non-linear relationship between symptomatology and outcome, but failed to find any such relationship in a sample of 127 unselected psychotic and 105 schizo-affective patients; as they say, "it has to be noted that yet another attempt to demonstrate discontinuity has failed". Dimensional rather than categorical taxonomic thinking is clearly indicated.

It may be useful to look at one further type of evidence, namely that furnished by factor analysis and cluster analysis. The data were collected and originally analysed by Everitt, Gourlay and Kendell (1971), who rated 146 schizophrenics and an equal number of affective psychotics on 44 variables which were section scores on the Mental State Schedule used in the U.S.–U.K. Diagnostic Project (Cooper, Kendell, Gurland, Sharpe, Copeland & Simon, 1972). Means and standard deviations on each variable are given by Maxwell (1972), who factor analysed the correlations between the items separately for each group. If each group was suffering from a completely different illness, and if some items were relevant to one type of illness, others to the other, then one would not expect similar co-variance matrices, or similar factor patterns. In actual fact there is considerable similarity. Equally, on the hypothesis of different diseases one would expect a quite different distribution and frequency of symptoms for the two samples. Maxwell's Table 1 shows that this is not so; the two distributions are very highly correlated, as our Fig. 3 shows very clearly. There are 44 symptoms listed on the abscissa, with the percentage incidence indicated on the ordinate.

There are a few symptoms indicated in Fig. 3 where slight differences do appear, and they are pretty much where one would expect them. Affectives have higher scores on worry, muscular tension, secret thoughts, depressed mood, signs of depression, somatic symptoms, fading interests and lack of concentration; schizophrenics on frequency of voices, subjective thought disorder, delusions of persecution, blunting and incomprehensibility, but the differences are nowhere absolute, but only relative. It should be added that the items used were but a small selection of those employed in the original study, concentrating on those which best discriminated the two groups; had another sample of items been employed, the similarities would have been much greater. These results are not compatible with a rigid form of the Kraepelinian dichotomy, but fit very well into a dimensional view, considering a continuum of psychoticism, together with a small bipolar factor contrasting schizophrenic and affective disorders. Such a view of course requires other types of confirmation, and these are discussed below.



Fig. 3. Trait frequency of schizophrenia and affective patients. Data from Maxwell (1972).

It might be thought that the prophylactic value of medication might show sufficient specificity to mark a clear discontinuity, lithium preventing relapse in affective illness, and neuroleptic medication in schizophrenia. However, some schizophrenic illnesses respond to lithium (Biederman, Lerner & Belmaker, 1979; Delva & Letemendia, 1982, 1986), and in addition to their effectiveness in mania, neuroleptics may be of value in depression (Hollister, Overall, Shelton, Pennington, Kimbell & Johnson, 1967). There has never been any doubt that mania responds to phenothiazines, as do some depressions (Klerman & Cole, 1965). It is also well-documented that many schizophrenics respond well to ECT (Brandon, Cowley, McDonald, Neville, Palmer & Wellstood-Garson, 1985; Taylor, 1980). As Crow (1986) sums up, "no unequivocal demarcation of the functional psychoses can be made on the basis of symptoms, outcome or response to treatment" (p. 421). Response to treatment is perhaps the weakest of these three sets of arguments, partly because of the poor reliability of psychiatric diagnoses, partly because it is not unlikely that such diagnoses are often influenced by the responses of the patients to treatment. The major problem, of course, is an ethical one, as long as we believe that certain drugs are better for treating certain types of psychosis, whether such beliefs are true or false, so long will it be ethically impossible to mount decisive experiments incorporating random allocation of patients to drug treatments.

Kendell (1987) makes another important point. As he says, "it is disconcerting how frequently the biological abnormalities reported in schizophrenia and assumed to be of aetiological significance are subsequently found in affective disorders also" (p. 501). He mentions the enlargement of the lateral ventricles (Dolan, Calloway & Mann, 1985), abnormal smooth pursuit eye movement (Iacono, Peloquin, Lumry, Valentine & Tuason, 1982), the role of 'high expressed emotion' in precipitating relapse (Horley, Orley & Teasdale, 1986), and even the season of birth (Hare, 1987). These similarities certainly speak against a rigid adoption of the Kraepelinian system, although they do not necessarily support Crow's apparent denial of any distinction between the different functional psychoses.

Another source of evidence is epidemiology. As Hare (1987) has pointed out, "comparison of the findings in schizophrenia and affective psychosis shows the two groups to be similar in sex ratio, age-incidence, risk of suicide, and seasonal variations in onset and birth: and to be different in personality type, premorbid impairment, and age of onset by sex. Differences in prognosis and fertility are less marked now than formerly" (p. 514). Thus here also there are factors favouring a dimensional, rather than a categorical approach. 68. F.F 1. 1.5.18

The data so far considered are certainly not in accord with what one might have expected to find if Kraepelin's hypothesis had been correct; as far as they go the data are rather in accord with a dimensional theory which would suggest that differential diagnosis of psychophrenic patients grade them along a continuum of severity, rather than classifying them in terms of non-overlapping categorical disease entities, with schizophrenia the most severe, followed by schizo-affective disorder, bipolar affective illness, and finally unipolar affective illness. What does genetic research have to say on this topic (Crow, 1987; McGuffin, Murray & Reveley, 1987; McGuffin & Murray, 1991).

First, let us look at the genetic approach which has also been used in studies of genetically identical individuals. McGuffin, Reveley and Holland (1982) reported on a set of monozygotic triplets, two of whom had received a hospital diagnosis of schizophrenia, while the third was considered to be a manic-depressive. Re-evaluation and the use of 'blind' raters suggested that the discordance was not simply due to misdiagnosis or differing diagnostic bias. Nor is this finding an isolated instance; Dalby, Morgan and Lee (1986) and Farmer, McGuffin and Gottesman (1987) have also reported similar discrepancies for twins. These data illustrate "some of the shortcomings of a strictly applied Kraepelinian dichotomy" (McGuffin, Murray & Reveley, 1987), and support the existence of a general psychoticism factor.

Following a rather different line of argument, Decina, Luscas and Linder (1989) concluded a survey of parent-child pairs who both required hospital admission for a psychotic illness, by saying that "while no patient with affective disorder was found amongst the children of schizophrenic parents, 50% of children of parents with affective disorder presented with schizophrenia". These results are difficult to understand genetically. If we agree that on the psychoticism continuum schizophrenia is further removed from normality than affective disorder, the law of regression to the mean would lead us to expect that the parent-child trend would be from schizophrenia to affective disorder; instead we get an increase in severity of pathology from one generation to another. Crow (1990) has suggested a genetic theory, namely that psychosis is a disorder of the cerebral dominance gene, and also that the psychosis locus is in the pseudoautosomal region, and he has brought forward evidence that the cerebral dominance gene is located within the pseudoautosomal region (Crow, 1989). It is not appropriate here to take issue with the differences between his concepts, and the now classical views of Gottesman and Shields (1972).

These studies are very relevant to the question of whether schizophrenic and affective psychosis are genetically related. Gershon and Rieder (1980) answer the question in the negative: "evidence from twins and family studies suggests that bipolar manic-depressive illness and chronic schizophrenia are distinct entities". Similarly, Shields, Heston and Gottesman *et al.* (1973) declare that "the genetic diathesis for affective disorders is independent of that for other psychiatric disorders". Crow (1986) disagrees: "affective illness in one generation may predispose to schizophrenia in the next" (p. 421), and again (Crow, 1990): "attempts to draw a line of genetic demarcation between schizophrenic and affective illnesses have failed. It must be assumed that these diseases are genetically related" (p. 788). In view of such diametrically opposed opinions, a detailed look at the evidence may be in order.

Rosenthal (1970) already noted that in 5 studies, there was an excess of schizophrenia in children of parents with affective disorder; there was a mean incidence of 2.3%, as compared with the 0.8% lifetime prevalence in the general population. This would not be expected on the 'separate diseases' hypothesis. Two studies of psychotic parents with psychotic children amplify these early findings (Penrose, 1968; Powell, Thomson, Hall & Wilson, 1973). In 621 such pairs, Penrose found that among children of parents with affective disorder, schizophrenia was almost as common as affective disorder (205 vs 232). Similarly, in the Powell *et al.* study the number of schizophrenic children of schizophrenic parents, the majority was schizophrenic (150 vs 34 for Penrose; 9 vs 0 for Powell *et al.*). In a methodologically rather less satisfactory study, Cammer (1970) found that 26.6% of 533 children of 273 manic-depressive parents were schizophrenic. In a smaller way, Elsasser (1952) found that of 169 children of two parents with non-schizophrenic psychoses, 18 suffered from definite affective illness and 6 from definite schizophrenia. Schultz (1940), in a small-scale study, found that of 25 children of two manic-depressive parents, 7 suffered from the same disorder, but 3 suffered from schizophrenia.

Equally interesting is the evidence from Pollock and Malzberg (1940), who collected family histories of psychosis over three generations, and found an excess of affective illness (15 cases) over schizophrenia (11 cases) in relatives of preceding generations who had been diagnosed as suffering from schizophrenia. Slater (1953) found a ratio of 4:3 (affective disorder to schizophrenia) in the parents of schizophrenic patients. The corresponding ratio for siblings was 3:5, also showing an unexpectedly large number of affective psychoses. He did not find a similar excess of schizophrenia in the parents or siblings of patients with affective disorders. These findings support an earlier one by Slater (1936), to the effect that in a study of manic-depressive illness there occurred a surprisingly large number of schizophrenics among the children; in 10 of 15 such cases he was unable to find schizophrenia in other members of the patient's family or that of the husband or wife.

An interesting study by Kant (1942) suggested that the proportion of schizophrenic to manic-depressive cases of relatives of deteriorated schizophrenics was 5:1, whereas, in relatives of recovered schizophrenics, the ratio was 1:5. Thus, in milder cases of schizophrenia, i.e. cases closer to affective disorder on our continuum; relatives actually are much more likely to suffer from affective than schizophrenic disorders, a finding difficult to explain on Kraepelinian lines. Similarly, Weinberg and Lobstein (1943) found in a study of 199 schizophrenic personalities that there was a higher percentage of affective disturbances in the ancestry of remitting schizophrenics than in that of steadily deteriorating ones. Again, Vaillant (1962) compared the heredity of 30 recovered schizophrenics with a non-recovered group, and found that at least 50% of the recovered group had heredity positive for an affective, mostly depressed psychosis, as contrasted with 7% in the control group.

Particularly impressive is a large-scale study by Tsuang, Winokur and Crowe (1980) of 1587 first-degree relatives of schizophrenics, manics, depressives, and controls who were personally interviewed without knowledge of the proband's diagnosis. Schizophrenia in first-degree relatives carried a morbidity risk of 0.6 in the controls; for schizophrenia it was over 5 times higher, for mania over 3 times higher, for depression over twice as high, but not significantly different. For affective disorder in first degree relatives, morbidity risks of depression was twice as high as in controls; mania twice as high, and schizophrenia just a little higher. Thus depression is twice as high in first-degree relatives of schizophrenics as in controls, and also twice as high in first-degree relatives of affective disorder patients as in controls. (I have combined the findings from two forms of data collection (personal interview and records) used by the author because they give very similar results.) Bipolar affective disorder was if anything more common in relatives of schizophrenics than of depressives, while unipolar disorder was much less so. The authors emphasize that "schizophrenia and affective disorder were different and support heterogeneity of major functional psychoses" (p. 500); they rather underplay the equally clear indications of overlap, linking bipolar affective disorder rather more closely with schizophrenic than unipolar disorders. These two disorders may then be conceived as respectively mild and severe forms of the same disorder (Tsuang, Faraone & Fleming, 1985), with the more severe disorder more closely related to schizophrenia.

Equally impressive is a more recent paper by Angst and Scharfetter (1990), looking at over 250 probands of variously diagnosed psychotics, and examining the ratio of schizophrenic to affective illnesses in the first degree relatives. This ratio rose from 0.30 in unipolar to 0.47 in bipolar affective disorders, to 0.92 in predominantly affective and 2.99 in predominantly schizophrenic schizo-affective disorder, and finally 5.05 in schizophrenia. "As the formal illness in the proband changes from affective to schizophrenic, the ratio of schizophrenia to affective illness in the first-degree relatives increases. There is no discontinuity such as would allow one to assert that there are two quite separate genetic components." (Crow, 1990, p. 791).

On such a continuum model, where would we expect schizo-affective disorder to go? Clearly it should appear between schizophrenia and affective disorders, giving us a continuum ranging from normal through unipolar and bipolar affective disorders to schizo-affective disorder and finally schizophrenia. The evidence supports such a view of schizo-affective disorder. Angst, Felder and Lohmeyer (1979) found that the risk of schizophrenia and affective disorder was approximately equal in first-degree relatives of schizo-affective probands, while that of schizo-affective illness was appreciably less. Tsuang, Dempsey, Dvoredsky and Strauss (1977), and Baron, Gruen, Asnis and

Kane (1982) found schizo-affective illness to be more closely related to affective illness than to schizophrenia; they agreed with Tsuang (1979) that schizo-affective illness was not a genetic entity.

Gershon *et al.* (1982), in a study of 1254 relatives of patients with major affective disorder concluded that "these data were compatible with the different affective disorders representing thresholds on a continuum of underlying multifactorial vulnerability. In this model schizo-affective illness represents greatest vulnerability, followed by bipolar . . . then unipolar (affective) illness." They also found that there was a highly significant (P < 0.001) excess of schizophrenics among the relatives of patients with schizo-affective illness in comparison with those having other types of affective illness. Curiously, Gershon *et al.* do not extend their continuum concept to include schizophrenia!

It is true that the morbid risks of psychosis in the relatives of probands with schizo-affective disorders is particularly high, but the suggestion that these disorders 'breed true' (Perris, 1974) is unwarranted. As McGuffin, Murray and Reveley (1987) point out, "a number of studies have now failed to support the concept of schizo-affective psychosis as a distinct genetic entity. Although schizo-affective disorder does occur in the families of probands with schizo-affective disorder, these relatives are also at increased risk of both schizophrenia and affective disorder" (p. 550). They also suggest that schizo-affective disorder could be heterogeneous with some cases part of a phenomenological spectrum attributable to mainly 'schizophrenic' genes and other cases a severe variant of affective psychosis, but this seems far-fetched and much less likely than the dimensional hypothesis outlined above.

Even when schizophrenia is narrowly defined, the risk of schizo-affective disorder is significantly increased in relatives of schizophrenics so diagnosed (Kendler, Gruenberg & Tsuang, 1985). Crow (1986) concludes his survey of the evidence relating to schizo-affective disorder by saying that "parsimony requires the conclusion that schizo-affective disorder is but the bridge between affective disorders and schizophrenia. The psychoses constituted a genetic continuum rather than two unrelated diatheses" (p. 424). This conclusion has been elaborated by Crow and Cooper (1986) along genetic lines. Crow (1986) enumerates several additional studies which tend to support the thesis of a continuum (see also Hupis, 1954; Meninger, Ellenberger, Prayser & Mayerson, 1958; Karlsson, 1974; Rennert, 1982; Flor-Henry, 1983 in support). Such a conclusion does not oblige one to agree to the specific genetic theories developed by Crow (1990), and it does not rule out the existence of specific genes responsible for different manifestations of the underlying vulner-ability concept (Eysenck, 1970c).

Kendell (1987) has expressed the position admirably. "Four main groups of functional psychoses have been recognised since the early years of the century: schizophrenia and affective psychoses, acute psychoses of good prognosis; and chronic paranoid psychoses. The air of permanence and stability is misleading. None of these four groupings, or of the individual psychoses included within them, has been clearly demonstrated to be a disease entity. All are still defined by their clinical syndromes and these syndromes appear to merge inevitably into one another and into other syndromes in the domain of neurotic illness and personality disorder. As a result it is not clear where the boundaries should be drawn" (p. 499). This is a clear statement of the facts which demand a form of *dimensional* rather than categorical description, even though some categorical specificity cannot be ruled out as an additional descriptive variable.

All the data so far surveyed have been based on psychiatric classification, and it is well known that diagnoses in this field are far from reliable or accurate (Beck, 1962; Hunt, Wittson & Hunt, 1953; Kreitman, 1961; Kreitman, Sainsbury, Morissey, Towers & Scrivener, 1961; Ley, 1970; Norris, 1959; Sandifer, Hordern, Timburg & Green, 1968; Schmidt & Fonda, 1956). These studies have been chosen because of the number of psychiatrists involved, and the number of categories used (Ley, 1972). Other references are to Ash (1949) and Shepherd, Brooke, Cooper and Lin (1968). (It might be thought that more recent data involving DSM-3 would be more appropriate, but of course the sources cited are more relevant because they reflect practices during the time when most of the studies here mentioned were planned and executed.)

The amount of agreement is not high. The actual figures for percentage agreement are 55% for Schmidt and Fonda, 58% for Norris, 63% for Kreitman *et al.*, i.e. averaging around 59% for two observers. For the Sandifer *et al.* study the figure is 34% (4 raters) and 10% (6–10 raters). These

figures are clearly unsatisfactory, but not far removed from those obtained for medical diagnoses of physical diseases generally (Eysenck, 1991a).

The relativity of psychiatric diagnosis is most clearly shown by the huge national differences which have been found. Thus Kramer (1961) showed that in some age/sex categories 10- or even 20-fold differences are involved when comparing British and American diagnoses. Cooper *et al.* (1972) have shown that these differences arise from a much more inclusive conception of schizophrenia, leading to a much higher frequency of this diagnosis in the U.S.A. than in the U.K., with an opposite tendency as regards the diagnosis of manic-depressive disorder. The recent adaption of DSM-3 criteria has of course considerably altered the picture, but the new standards are more relevant to future studies.

Granted that reliability of psychiatric diagnoses is poor, and often owes more to the nationality of the psychiatrist making the diagnoses than to the actual mental state and behaviour of the patients (Cooper *et al.*, 1972), it is worth noting that diagnoses of major psychotic illnesses show little overlap with neurotic disorders of various kinds, and have somewhat higher rates of agreement. It does not seem likely that random errors (Ley, 1972) can explain the observed relationships. This is the 'observer agreement' model of reliability; more interesting from our point of view is the consistency or stability model, i.e. comparing diagnoses assigned to patients on successive admission, or at other widely separated points in time.

An early study by Masserman and Carmichael (1938) looked at a series of 100 patients admitted to a university psychiatric clinic in Chicago and found that a 'major revision' of diagnosis was necessary in over 40% only 12 months later. In a much larger study, Babigian, Gardner, Miles and Romano (1965) compared the diagnoses assigned to 1215 patients on more than one occasion and found that diagnoses of schizophrenia were more reliable than of affective disorder. However, the actual time interval between diagnoses was only a few days or weeks, so there was little room for change of symptomatology over time. Odegaard (1966), in a comparison of first and last diagnoses of patients first admitted in 1950–1954, and then re-admitted at the end of 1963, found that many patients originally diagnosed as having a reactive psychosis were subsequently diagnosed as having either schizophrenia or a manic-depressive illness-but of course these are patients re-admitted, and thus probably atypical of all first cases so diagnosed. Cooper (1967) studied diagnoses of 200 patients on four different occasions, finding that only 54% were allocated to the same broad category on all four occasions. However, many changes were due to changes in the doctor rather than to changes in the patient's symptoms. Such changes were found in 16% (32 cases), the commonest being for schizophrenic symptoms to develop in patients who had previously had purely depressive symptoms, but with some paranoid features. Here, as in the transgenerational shift from affective (parent) to schizophrenic (child), the direction is toward a more severe disorder.

Of particular interest for our purpose is a study by Kendell (1974) who followed up 1913 patients originally diagnosed in 1964 and re-admitted at least once before the end of 1969. It was found that 58% of patients did not undergo any significant change in diagnosis. Unchanged at final diagnosis were 69% of all depressive illness, 75% of all schizophrenics, and 35% of all personality disorders. A change from schizophrenia to depressive illness was found in 10.9%, from depressive illness to schizophrenia in 7.4%. From mania to schizophrenia there was a change in 17.0%, but only 2.3% in the reverse direction. (Depressive illness here included reactive and endogenous; there appears to have been little stability to the more refined diagnosis.) It is interesting that there was a significant interchange of diagnosis for personality disorder and depressive illness, as usually personality disorder is considered to belong with the schizophrenic *Erbkreis*.

The data so far considered are incompatible with a purist interpretation of Kraepelin's theory, but they only partly support Crow's view. It is true that the major two psychotic disorders do not 'breed true', but equally there is no blending of the two into schizo-affective disorders when the parents are one of each (Gottesman & Bertelsen, 1991). Clearly, it is as important to avoid exaggeration in the denial of genetic differentiation between psychoses as it is to avoid denial of common features (Crow, 1987).

What would happen if we relied exclusively on the statistical study (factor analysis, multiple discriminant analysis) of reliably rated symptoms in a very variegated psychotic population, including depressives and schizophrenics of all kinds? Lorr, Klett and McNair (1963) have carried out such a study, as well as including discussions of previous works employing similar method-

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ologies. They isolated 10 syndromes: excitement; hostile belligerence; paranoid projections; grandiose expansiveness; perceptual distortions; anxious introspectiveness; retardation and apathy; disorientation; motor disturbance; and conceptual disorganization. Symptoms were carefully rated by specially-trained interviewers, and these 10 syndromes emerged after oblique solution of the factors emerging. The intercorrelations between the factors gave rise to 3 second-order factors: Excitement vs retardation, schizophrenic disorganization, and paranoid process. They again correlated positively together, to form a higher-order factor which the author labelled 'schizophrenia', although 'psychosis' would have been a more appropriate name in view of the explicit inclusion of depressive patients (see statement on p. 32). This analysis then eventuates in a hierarchical model, very much as hypothesized here.

It is only fair to mention studies which suggest a lack of generality between different types of psychosis. Gottesman and Bertelsen (1991) report on in-patient psychotics who had children by other in-patients, thus making possible a variation of the diallel cross-method used in plant and animal research. In homotypic manic-depressive couples, the kind of risk (68%) in offspring for manic-depressive diagnosis approached the value expected for a dominant gene with complete dominance (78%); as the writers emphasize, "the results do not support the view of a continuum of psychopathology between affective and schizophrenic psychoses because the risk of schizophrenia from such dual-mating manic-depressives is close to the base rate for the general population" (p. 95). The numbers involved are too small as yet to constitute a definite contradiction to the view here taken that we have *both* generality *and* specificity, but the results should act as a warning not to disregard genetic specificity.

It is often believed that the so-called 'new genetics' might, in principle at least, settle issues of dimensionality vs categorical discrimination between hypothetical disease entities (Pato, Lander & Schulz, 1989). Roberts and Claridge (1991) have taken up this challenge and argue convincingly that the 'new genetics' is quite compatible with a dimensional view of schizophrenia, and need not favour the single gene hypothesis.

It is also important to realize that the inclusiveness of criteria used to define schizophrenia may determine in part the outcome of genetic studies. An interesting comparison of inclusiveness criteria is the comparison by Farmer, McGuffin, Harvey and Williams (1991) of the heritability (MZ/DZ concordance ratios) of different DSM-III categories. Heritability goes up from a simple 'schizophrenia' diagnosis to one adding schizotypal personality, with another increase by adding affective disorder with (mood-incongruent) psychosis, reaching its highest value with the addition of a typical psychosis, with a slight decline when schizophreniform disorder is included. The inclusion of any form of affective disorder produces a distinct lowering of the concordance ratio, which is again lowered by the inclusion of paranoid disorder, with a final lowering by the inclusion of any axis-I DSM-III category (Farmer, McGuffin & Gottesman, 1987; Farmer *et al.*, 1991). The results suggest both a general psychoticism factor, and also a fair degree of specificity.

We have looked at eight different types of studies which might give evidence concerning the continuity vs categorical conception of the major functional psychoses. It is possible in each case to evaluate the contribution made by each type of study in numerical form; of course these evaluations are inherently subjective, but being based on factual material rational discussion is possible. If we denote the continuity hypothesis G (for generality), and the Kraepelinian theory of complete distinction S (for specificity), we can assess the support given by each of our 8 types of study to G or S in a 5-point scale, on which 5:0 would mean complete support for G; 4:1 strong support for G; 3:2 weak support for G; 2:3 weak support for S; 1:4 strong support for S, and 0:5 complete support for S. Table 1 shows the outcome.

Clearly the outcome does not favour either side at the expense of the other. There is strong evidence for the existence of a continuum covering all the functional psychoses, and ordering them in relation to severity. But there is also strong evidence for the reality of differentiation, following Kraepelinian lines. It would not be reasonable to stress either line to the exclusion of the other; psychoticism is a reality, but so is the distinction between schizophrenia and indeed different types of schizophrenia (Kendell, 1987) and manic-depression and indeed unipolar or bipolar types of affective disorder (Kendell, 1987). For special purposes we may wish to emphasize one line of argument or the other, but clearly there is no victory in all these studies for either rigid Kraepelinian distinctions or for the ancient 'Einheitspsychose'.

The definition and measurement of psychoticism

Table 1. Empirical evidence favouring continuum (G for general) or categorical (S for specificity) theories of psychosis

	G	S	
1. Distribution of symptoms	4	1	
2. Symptom similarity	4	1	
3. Outcome	3	2	
4. Medication	1	4	
5. Biological abnormality	2	3	
6. Genetic research: markers	2	3	
7. Genetic research: familial incidence	2	3	
8. Diagnostic stability	2	3	
Total	20	20	

SCHIZOTYPY AND PERSONALITY DISORDER

The theory of 'psychoticism' has two major components. The first of these, the existence of some degree of generality among the functional psychoses, has been dealt with in the preceding section. The second postulates an extension of this continuum to less serious disorders ('spectrum' disorders), and indeed to normal behaviour. Some material regarding 'spectrum' disorders has already been cited, but this section will deal specifically with the problem of extending the continuum from psychosis to normality.

Before turning to the psychiatric literature, I shall mention a study specially designed to answer the question of continuity from psychosis to normality, and introducing a method of analysis specially created to make such an answer possible. I have called the method 'criterion analysis' (Eysenck, 1950), because it uses a criterion (psychosis-normality) to furnish us with a model which can either show continuity or discontinuity.

The study was designed to test Kretschmer's (1946, 1948) theory of a schizothymia-cyclothymia continuum, as well as my own theory of a normality-psychosis continuum. Kretschmer was one of the earliest proponents of a continuum theory linking psychotic and normal behaviour. There is, he argued, a continuum from schizophrenia through schizoid behaviour to normal dystonic (introverted) behaviour; on the other side of the continuum we have syntonic (extraverted) behaviour, cycloid and finally manic-depressive disorder. He is eloquent in discussing how psychotic abnormality shades over into odd and eccentric behaviour and finally into quite normal typology. Yet, as I have pointed out (Eysenck, 1970a,b), the scheme is clearly incomplete. We cannot have a single dimension with 'psychosis' at both ends; we require at least a two dimensional scheme, with psychosis-normal as one axis, and schizophrenia-affective disorder as the other.

In order to test this hypothesis, I designed the method of 'criterion analysis' (Eysenck, 1950, 1952a,b), which explicitly tests the validity of continuum vs categorical theories. Put briefly, we take two groups (e.g. normal vs psychotic), and apply to both objective tests which significantly discriminate between the groups. We then intercorrelate the tests *within each group*, and factor analyse the resulting matrices. If and only if the continuum hypothesis is correct will it be found that the factor loadings in both matrices will be similar or identical, and that these loadings will be proportional to the degree to which the various tests discriminate between the two criterion groups.

An experiment has been reported, using this method. Using 100 normal controls, 50 schizophrenics and 50 manic-depressives, 20 objective tests which had been found previously to correlate with psychotics were applied to all the subjects (Eysenck, 1952a). The results clearly bore out the continuum hypothesis. The two sets of factor loadings correlated 0.87, and both were proportional to the differentiating power of the tests (r = 0.90 and 0.95, respectively). These figures would seem to establish the continuum hypotheses quite firmly; the results of the experiment are not compatible with a categorical type of theory.

Another study investigated 153 psychotic patients prior to the application of therapy (Verma & Eysenck, 1973). These patients were interviewed and rated on the In-patient Multidimensional Psychiatric Scale (IMPS), published by Lorr, Klett, McNair and Lasky (1963), were administered the PEN Inventory (an early form of the Eysenck Personality Questionnaire; Eysenck & Eysenck, 1975), as well as several other tests and questionnaires. A factor analysis was carried out on altogether 34 test or interview scores, and 2 major factors emerged. The first was clearly a general psychosis factor, with its highest loading on the P scale, and high loadings also on ratings, objective

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tests and other questionnaires. Factor 2 discriminated the outgoing, extraverted, extrapunitive type of psychotic from the inward-looking, introverted, intropunitive type, this factor loaded on the extraversion-introversion side. The P factor did not discriminate between the types of psychosis, but the E factor did. These results suggest that within the psychotic field extraverted and introverted behaviour patterns may be distinguished with a considerable degree of clarity, and thus reinforce the findings of Armstrong, Hottusson, Ries and Holmes (1967) and Venables and Wing (1962). The former concluded, from an examination of the MPI scores of schizophrenic patients, that "these results raise the possibility that a significant degree of what is included within the process-reactive frame of reference may be considered a function of extraversion-introversion" (p. 69).

Our results (Verma & Eysenck) suggest that some even broader grouping is possible, embracing not only schizophrenics, but also other types of psychotics. Thus depressive disorders had relatively high E scores, paranoids and schizophrenics low ones. (It is interesting to note that in the Kendell and Gourlay study the item most highly correlated with the affective end of the continuum was "More outgoing and gregarious recently", a typical extraversion item.) The possibility certainly exists, and should be investigated, that the major difference between functional psychoses are related to the other major dimensions of personality, i.e. E and N.

When we turn from psychotic states to types of personality lying close to them on the psychoticism continuum, we encounter specifically the so-called 'schizoid personalities', 'spectrum' and personality disorders. Manfred Bleuler (1911, translation 1978) first described the schizoid personality:

"He is taciturn or has little regard for the effect on others of what he says. Sometimes he appears tense and becomes irritated by senseless provocation. He appears as insincere and indirect in communication. His behaviour is aloof and devoid of human warmth; yet he does have a rich inner life. In this sense he is introverted ... Ambivalent moods are more pronounced in the schizoid than in others, just as he distorts the meanings of, and introduces excessive doubts into his own concepts. But on the other hand, the schizoid is also capable of pursuing his own thoughts and of following his own interests and drives, without giving enough consideration to other people and to the actual realities of life. He is autistic. The better side of this autism reveals a sturdiness of character, and inflexibility of purpose, an independence, and a predisposition to creativity. The worse side of it becomes manifest in a lack of consideration for others, unsociability, a world-alien attitude, stubbornness, egocentricity, and occasionally even cruelty."

Bleuler concluded that at least half of his patients had shown some degree of schizoid behaviour before their psychotic breakdown, and he noted that similar characteristics were also very noticeable in their siblings and their offspring. This seems a clear indication of an extension of the psychotic *Erbkreis* to non-psychotic individuals.

Early work has been well summed-up by Reich (1976) who concentrated on the 'schizophrenia spectrum'; thus the studies reviewed are very relevant to the continuity vs categorical discrimination (Gottesman, 1987). Reich defines the spectrum concept as a theory which maintains that there exists a cluster or spectrum of psychopathological states, some characterized by psychosis and others not, which share a genetic etiology with schizophrenics, and which, therefore, constitute, together with schizophrenia itself, a 'spectrum of schizophrenic disorders' (Kay, Rosenthal, Wender & Schulsinger, 1968). The theory implies a diathesis-stress conception (Gottesman & Shields, 1972), with the genetic diathesis being a requisite for the development of a spectrum-related illness. "Any particular schizophrenic spectrum disorder is, therefore, seen as representing not a discreet state which is unrelated to the other disorders, but a point on a genetic continuum, with differences among the points reflecting differences in intensity, or in some other clinically evident quality, which may be environmentally and/or genetically determined'' (Reich, 1976, p. 4). (My own theory would extend this 'spectrum' beyond schizophrenic disorders to *all* functional psychoses (Eysenck, 1952b; Eysenck & Eysenck, 1976).)

Tests of the 'schizophrenic spectrum' theory may be carried out along several lines. The first consists of the use of 'markers', i.e. characteristics of schizophrenics which are also found in a

significant amount in relatives of schizophrenics not themselves psychotic. Examples are deviant eye-tracking which is frequently found in schizophrenics (Holzman, Proctor & Hughes, 1973), as well as in their relatives (Holzman, Proctor, Levy, Yasillo, Metzer & Hunt, 1974). Another example would be a reduced level of platelet monoamine oxydase, which is found not only in schizophrenics but also in their non-schizophrenic monozygotic twins (Wyatt *et al.*, 1974). The study of 'markers' will be found to be a favourite method of analysis to show that psychoticism scores do indeed map into the psychotic continuum, as discussed in a later section.

The second method uses the concept of familial incidence of spectrum cases, based on the assumption that they represent 'thresholds' along a genetic continuum. This notion is shown in Fig. 1, where different spectrum disorders have differential 'thresholds' on the P continuum. In other words, the threshold for schizophrenics is higher than that for psychopathy, or schizoid disorders, the latter requiring less genetic predisposition and/or environmental stress to appear. Although the model has only recently been specified in testable form, the notion itself goes back a long time (Planansky, 1972). Thus Ruedin (1916) had already looked for psychiatric pathology among the relatives of (strictly defined) schizophrenics; so did Kallman (1938), who searched the families of schizophrenics for many kinds of pathology, and found two frequently occurring types of relatives whom he called 'eccentric personalities' and sufferers from 'schizoid psychopathy'. Slater (1953) used spectrum notions in studying behavioural traits in discordant twins of schizophrenic probands, as did Gottesman and Shields (1972) in looking for 'schizophrenic equivalents'.

Reich (1976) also cites a number of adoption studies in which the adopted children of schizophrenic parents are found to have a variety of spectrum disorders. In particular, the Extended Family Study (Kety *et al.*, 1968) and the Adoptees Study (Rosenthal *et al.*, 1971) gave strong evidence for the existence of such spectrum disorders. The adoptees were personally examined and compared with adoptees with non-schizophrenic parents; significantly more diagnoses of schizophrenic spectrum disorders were made in index adoptees than control cases. (For later analyses, and a more detailed collection of studies, see McGuffin & Murray, 1991.)

Let us now turn to a consideration of the psychological literature. Much research has gone into the measurement of schizotypy and schizotypal personality disorders in recent years (Claridge, 1985), and we know much more about it as a result.

There are many schizotypy scales which have proliferated in recent years, and which owe much to Bleuler's description, although they tend to go beyond schizophrenia into 'psychosis-proneness', and usually concentrate on a single concept, like perceptual aberration (Chapman, Chapman & Raulin, 1978), magical ideation (Eckblad & Chapman, 1983), impulsive non-conformity (Chapman, Chapman, Numbers, Edell, Carpenter & Beckfield, 1984), intense ambivalence (Raulin, 1984), social fear (Raulin & Wee, 1984), cognitive slippage (Miers & Raulin, 1985), etc. More general are scales like the STA and STB scale of Claridge and Broks (1984). All these scales seem to measure much the same construct, as Kelley and Coursey (1992) have shown by intercorrelating and factor analysing 11 such scales. Only anhedonia (Chapman, Edell & Chapman, 1980) failed to correlate with the other scales; the Claridge and Broks scale (STA), being the most general, had the highest loading (0.86), followed by cognitive slippage (0.79), intense ambivalence (0.78) and magical ideation (0.71). Non-conformity (0.65) also had a sizeable loading and the MMPI Mini-Mult (1968), i.e. the sum of the Depression, Psychasthenia and Schizophrenia scales, had a loading of 0.74. Other factorial studies (e.g. Muntaner, Garcia-Sevilla, Fernandes & Torrubia, 1988; Bentall, Claridge & Slade, 1989) gave similar results.

What is noticeable in the studies mentioned is that while the psychoticism scale is uncorrelated with neuroticism, schizotypy scales usually show high correlations with N; indeed, these are sometimes so high as to suggest that what is being measured is N, rather than P. Thus in the Muntaner *et al.* (1988) study, the first factor (schizotypy) had a high loading on N, but not on P. In the Bentall *et al.* (1989) study, their second factor had high loadings on N, the Claridge and Broks STA and STB, the schizoidia and the schizophrenism scales. In view of the genetic distinctiveness of neurotic and psychotic disorders this suggests that many schizotypy scales measure two dimensions of personality simultaneously, which is an undesirable state of affairs. Montag and Levin (1992) have correlated the Claridge and Broks STA with the Coursey Personality Scales, and found much the highest correlation with emotional instability. The Bentall *et al.* (1989) study also shows that introversion, as well as N and P, is involved in typical schizotypy scales. They factor analysed 10 such scales, as well as P, E and N, and found 3 factors, each of which had high loadings for some but not all of the schizotypy scales. Factor 1 was labelled 'perceptual cognitive', with a high loading (0.72) on psychoticism. Factor 2 was labelled 'social anxiety', with its highest loading on neuroticism (0.85). Finally, factor 3 was labelled 'introverted anhedonia', with a high loading on extraversion (-0.61). It is difficult to agree to factor 3 having much to do with schizophrenia or schizotypy. Similarly, factor 2 is simply a factor of neuroticism, with N having much the highest loading. This leaves factor 1 as roughly representing psychoticism, although 'hypomanic personality' has the highest loading—hardly suggestive of 'schizotypy'.

If these data have anything to tell us, it may be that differences between different *types* of psychosis may be mediated by differences in N and E, at least partially (Eysenck, 1970c). This is not the place to argue the case, but just as there are different kinds of psychopathy defined by differences in N and E (Eysenck, 1987), so there may be similar associations between different types of psychosis and the major dimensions of personality.

There are clear connections between the concept of schizotypy and that of personality disorder, as is clear from Bleuler's description of the schizoid personality. Kallman (1938) was one of the first to recognize a relationship between psychopathy and schizophrenia in his description of what he calls 'schizoid psychopaths', and since then retrospective and longitudinal high risk studies of schizophrenics, together with studies of delinquents and criminals, have confirmed the association between psychopathy and schizophrenia.

Of particular importance in this connection has been the work of Heston (1966, 1970) who studied the children of schizophrenic mothers who were adopted away within 3 days of birth; there were 58 subjects who comprised the first experimental group, with another 58 matched controls whose mothers were not schizophrenic. Blind evaluation of subjects followed after they had reached maturity. Many differences were found. Controls had much lower scores on the Menninger Mental Health-Sickness Rating Scale, had no member diagnosed as schizophrenic, compared to 5 for the experimentals, had 2 as opposed to 9 members diagnosed as sociopathic personality (P < 0.017), had 2 as opposed to 11 members spending more than 1 year in an institution (jail or psychiatric), and had 2 as opposed to 7 labelled 'felon' (P < 0.054). Heston describes in detail personalities more frequently found in the experimental group who, he states, fit the older diagnostic category of 'schizoid psychopath' (Kallman, 1938). Rosenthal *et al.* (1968) and Kety *et al.* (1968) have also found evidence for such 'schizophrenic spectrum' disorders in adopted children of schizophrenic mothers. These data would seem decisive in extending the 'spectrum' from psychotic to non-psychotic disorders of a schizoid, psychopathic or criminal nature.

Heston (1966) found that in addition to psychopathy and criminal subjects in his experimental group, he also had an excess of neurotic personality disorders (13 as compared to 7), but this was just on the borderline of significance. (It should be remembered that Heston, like many others, gives estimates of significance levels which are appropriate to *single* comparisons while listing 20 such comparisons; this means that 1 apparently significant comparison could have arisen by chance.)

Do psychopaths have elevated levels of psychoticism? Hare (1985) showed that criminals with some features of psychopathy showed some signs of paranoid schizophrenia, schizotypal personality, and a relatively high incidence of neuropsychological and neurophysiological abnormalities. Raine (1992) studied 36 prisoners rated on the Hare Psychopathy Check List (Hare, 1980) and divided them into high, medium and low scorers. They were also given 4 schizotypal personality scales, which were summed to give an overall index. Prisoners were also assessed according to DSM-III criteria for borderline and schizotypical personality by interviewers blind to the questionnaire scores. The results showed significant relations between psychopathy and borderline disorder, schizotypal disorder, and schizotypal personality. There was also support for Heston's (1966) finding that an unstable, impulsive lifestyle lacking in commitments and long-term plans, represents that element in psychopathy most related to schizotypal personality, and also to psychoticism (Eysenck & Gudjonsson, 1990).

The evidence supporting the view that psychoticism is strongly related to psychopathic, antisocial and criminal behaviour is reviewed in detail by Eysenck and Gudjonsson (1990); it appears at all ages (childhood, youth, maturity) and results in sizeable correlations. The concept of 'personality

disorder' or psychopathy is of course rather fuzzy; Eysenck and Eysenck (1978) have suggested that it is no more than a rather arbitrary combination of high P, N and E, a suggestion along lines of dimensional description which agrees quite well with DSM-III description, which isolates 3 separate clusters to characterize personality disorders, corresponding closely to P, E and N personality description (Eysenck, 1987).

We may conclude that there is good evidence to suggest an extension of the psychotic continuum into non-psychotic types of behaviour variously described as psychopathic, schizoid, criminal, alcoholic, etc., but always genetically linked with psychosis through close relatives of one kind or another. It is curious that advocates of the concept of schizotypy, from Meehl (1962, 1989) onwards have linked this extension to non-psychotic diseases with schizophrenia alone, not with the whole psychotic continuum. Thus the subject matter of this and the preceding section, although obviously closely related, has been looked at in isolation; it is only the concept of psychoticism (Eysenck, 1952a,b; Eysenck & Eysenck, 1976) which has brought them together. This, plus the failure to separate psychoticism from neuroticism, suggests that much of the schizotypy literature is only partially relevant to the classification of the concept of psychoticism.

To end this section, it may be useful to consider a criticism of the P scale that has been made quite frequently from its earliest days of inception (e.g. Davis, 1974; McPherson, Presby, Armstrong & Curtis, 1974). The criticism is based on the hypothesis of a linear relation between P-score and the position of various groups on the psychoticism continuum; it is suggested that schizophrenics should have the highest P scores being furthest to the right on the continuum (Fig. 1), and the fact that other groups (e.g. criminals, psychopaths) tend to have higher scores on P (Eysenck & Eysenck, 1976; Claridge, 1981, 1983) is taken to disprove the identification of P with psychoticism. Perhaps, it is sometimes argued, P should be renamed psychopathy, because of the high scores of psychopaths on this scale.

The next section will be devoted to a discussion of the reason why P is indeed a psychoticism factor; here I only wish to state some reasons why P scores of psychotics are often lower than those of other groups. The first reason, of course, is simply that we attempted to construct a scale of psychoticism, not of psychosis, hence we deliberately left out all the typical symptoms which go to make up such diagnostic scales as the MMPI schizophrenia scale. Our aim was to construct a scale which would measure psychoticism in normal groups (i.e. in non-psychotic groups); the scale was not intended as a diagnostic clinical device. The second reason is that psychosis may easily reduce the patient's insight (a well-known schizophrenic symptom), thus making it more difficult for him to fill in the questionnaire truthfully. The third reason is that patients often have a high Lie score (e.g. McPherson et al., 1974, found an L score of 13 for psychotic patients, 6 for normal controls); this would automatically rule out any meaningful comparison. The fourth reason is the simple fact that psychotics are nearly always under drug treatment, thus altering their mental state in ways that are likely to interfere with accurate answers to P-related questions. The fifth reason is the fact that most of the psychotics tested were held in mental institutions, and institutionalization is likely to affect the responses of inmates to such questions in unpredictable ways. These and many other reasons caused me originally (Eysenck, 1952b) to abandon all thoughts of producing a questionnaire or inventory of psychoticism, and rely rather on experimental tests not open to such objection-or at least not to the same extent. I would thus argue that on a priori grounds we would have expected a linear increase in P score with change of group from left to right on the continuum in Fig. 1, but would not expect a continuation of this increase into the psychotic range. A reasonably high P score, correlated with severity of illness, might be found (Verma & Eysenck, 1973), but not a score higher than anything found in other groups, such as psychopaths, criminals, etc.

This leaves me with the most crucial question of all—how can one *prove* the identification of a statistical factor (P) with a concept like psychoticism? The next section contains a description of the methodology used, and a brief account of the results achieved.

The data surveyed so far do not support the Kraepelinian theory, but surveys such as Crow's (1986, 1990) phrase their conclusion rather too firmly, and disregard contrary evidence. Thus Tsuang, Bucher and Fleming (1983) analysed family data from schizophrenic and control probands, using a multiple threshold model to determine whether a given group of diagnoses were aetiologically related to schizophrenia. The data did not fit the multiple threshold model, and while

mania showed a (non-significant) relationship to schizophrenia, depression failed to do so, and personality disorder showed an increased one They concluded that the hypothesis that schizophrenia and a spectrum of disorders have a common familial aetiology was not accepted. The authors comment on their failure to agree with others (e.g. Reich, 1976) who summarized in a review paper the frequent finding of 'inadequate personality' in relatives of schizophrenics, but although their study is in a minority, it was well done and cannot be disregarded.

THE P-SCALE AS A VALID MEASURE OF PSYCHOTICISM

The most crucial property of a scale designed to measure psychological traits is of course *validity*, but this is difficult to establish. Construct validity is difficult to prove, in the absence of agreement on theoretical constructs. Concurrent validity assumes the validity of some already existing measure, which is usually hard to find. Predictive validity assumes the existence of external criteria, but this poses difficult problems, as we have seen. I wish here to introduce a rather novel, and certainly unusual measure of validity which is based on the nature of the nomological network surrounding the concept (psychoticism) under investigation. This method (proportionality analysis) is related to criterion analysis (Eysenck, 1950, 1952a), and has the added advantage that it allows the objective determination of the psychological nature of the factors that emerge from factor analysis.

It is well known that the psychological identification of factors is a very difficult task, with few agreements and many doubts. The Wechsler Test subscales on analysis tend to divide, after extraction of g, into verbal and non-verbal tests. But is this the psychological principle involved? Verbal scales may measure crystallized ability, non-verbal scales fluid ability. Or the distinction may be between timed and untimed tests. Clearly interpretation is not obvious, and may require elaborate experimental follow-up. Examination of the *content* of the scale is certainly not sufficient. Analysing the Guilford Scale of Social Shyness, which had been declared by him to be a measure of a single entity, I found 2 uncorrelated factors correlating respectively with introversion and neuroticism, suggesting two quite separate types of social shyness. I have discussed in detail the inadequacy of purely psychometric analyses in discerning and identifying personality traits and dimensions (Eysenck, 1991b); what are the alternatives?

Scientific research should begin with a theory; the one here to be tested is incorporated in our Fig. 1, postulating a continuum from empathic, altruistic, socialized behaviour through average, schizoid and psychopathic behaviour to psychotic illness. We can deduce certain consequences which follow from the theory and are testable in relation to the claim that P is a valid measure of this continuum. Such deductions take a number of steps. (1) Select a theoretical concept which postulates a marked difference between schizophrenics and normals. (2) Construct a proper test of the concept in question. (3) Demonstrate that the test is valid, i.e. discriminates well between schizophrenics and normals. Let us call this test T. If the hypothesis of a continuum is correct, and if T and P are good measures of this continuum, then (4) P and T should correlate significantly within the normal group, and possibly also within the psychotic group (the latter prediction is subject to the problem outlined in the preceding section, i.e. the possible effects of the actual psychotic illness). Ideally, therefore, the proportionality of schizophrenia vs normal T scores can be translated to within-group comparisons of high P scoring normals vs low P scoring normals, and high P-scoring psychotics vs low P scoring psychotics. If P does not measure the continuum in question, then none of these consequences follow. The prediction is that on test T schizophrenics: normals =P+:P- in both psychotic and normal groups. We would thus have a powerful method of testing the theory in question, and providing evidence for the validity of the P concept. Alternative theories, e.g. that P is a measure of antisocial personality disorder, and "that the label psychoticism isn't appropriate for the P dimension" (Zuckerman, 1991, p. 375), can thus also be subjected to factual scrutiny.

As an example, consider HLA B27, a subsystem of the human leukocyte antigen system, which is found more frequently in schizophrenics than in normal, non-psychotic subjects (McGuffin, 1979; Gattaz, Ewald & Beckman, 1980). Gattaz (1981) has shown that in a comparison of schizophrenic patients with and without HLA B27, those with the antigen had significantly higher P scores (P < 0.02, n = 11:29). In another study Gattaz, Seitz and Beckman (1985), 17 B27 positive and 16 B27 negative non-psychotic subjects showed a difference on P scores in the expected direction (P < 0.01). This example shows the expected effects of an association between P and T in both a normal and a psychotic group, and may serve to illustrate the method.

Another study concentrated on the prevalence of hallucinations. Slade (1976) contrasted 3 groups—normals, psychotics without and psychotics with auditory hallucinations; on the P-test normals had the lowest scores (2.80), hallucinated psychotics the highest (7.25), with non-hallucinatory psychotics inbetween (4.80). In a later study, Launay and Slade (1987) correlated scores on a 12-item questionnaire testing hallucinatory predispositions with the P scale. The correlations were 0.21 for 100 male prisoners and 0.46 for 100 female prisoners, making a combined r = 0.35. Thus here again there is an association between T (hallucinations) and P in both psychotic and non-psychotic groups.

Eye-tracking is another variable that has been related to P and the general psychoticism continuum. Lipton, Levy, Holzman and Levin (1983) have shown that not only schizophrenics, but often also their relatives, show faulty lack of smoothness in the pursuit or tracking eye movements required when, say, following a swinging pendulum. Similarly, twins discordant on schizophrenia may nevertheless be concordant for this test. Iacono, Peloquin, Lumry, Valentine and Tuason (1982) found this symptom in patients with unipolar and bipolar affective disorder in remission. Bosch (1984) and Iacono and Lykken (1979) have reported positive correlations with psychoticism questionnaires in schizophrenic and normal subjects. There are some contradictions in the data, and a large-scale replication would seem suggested (see also Simons & Natkin, 1989, and Silver *et al.*, 1982).

A rather different approach was used by Jutai (1988), who examined specifically the lateralized cerebral dysfunction in schizophrenia and affective disorder postulated by Flor-Henry and Gruzelier (1983), and based on a model suggested by Venables (1980). Jutai concluded that the results of his study supported Venables' notion that in the development of schizophrenic disorders, there may be an initial disturbance of right-hemisphere mechanisms of attentional control. Psychosis-prone young adults diagnosed on the Chapman tests tended to use visual search strategies similar to those of right-brain damaged patients. He adds the usual disclaimer that at present it is not certain that they do so for similar reasons.

A different technique for investigating hemisphere differences is the dichotic shadowing technique (Rawlings & Borge, 1987). The theory that schizophrenics are characterized by left-hemisphere overactivation (Flor-Henry & Gruzelier, 1983) has been tested by Rawlings and Claridge (1984) and shows that subjects scoring high on a measure of schizotypal personality responded more quickly to verbal material presented tachistoscopically to their left visual field than to their right, while subjects with low scores, showed the usual superiority for material presented to the right visual field. Broks (1984) found a similar difference. Rawlings and Borge (1987) have reported 2 experiments using the dichotic shadowing technique. Both gave positive results, showing differential responding in the two ears, with high P scorers failing to show the right ear superiority shown by the low P scorers; the second experiment gave similar results for the male subjects but gave little evidence for the females. Overall the studies give mild support for the theory; but leave many questions unanswered. It is worth mentioning that Hare and McPherson (1984) found that a group of criminal psychopaths showed a significantly smaller right-ear advantage on a dichotic listening task than did groups of criminals who were not clearly psychopathic; this adds to the evidence that psychopaths belong to the schizophrenic *Erbkreis*.

Related to these studies of attention deficit in schizophrenics is an experiment carried out by Hinton and Craske (1976), who argued that 'attentional effort' is positively correlated with the magnitude of action potentials in those muscles which are not involved in the tasks being undertaken (Easan & White, 1961), and that 'degree of effort' in attentional tasks would lead schizophrenics to show higher action potentials in a simple attention task (Goldstein, 1965; Malmo, Shagass & Smith, 1951). He predicted and found positive correlations between his EMG index and the P score for both males (r = 0.56) and females (r = 0.44), concluding that P score related directly to increase in generalized muscle action potentials on attending to simple perceptual discrimination tasks.

Attentional processes may also be involved in an interesting experiment reported by Badcock, Smith and Rawlings (1988). The topic selected was the effect of a masking stimulus (backward masking) on exposure of a target stimulus, with a specified inter-stimulus interval intervening. Masking deficits, i.e. increased susceptibility to a mask, had been found prior to (Braff, 1981), during (Braff & Saccuzzo, 1981) and following (Miller, Saccuzzo & Braff, 1979) a schizophrenic episode. Succuzzo and Schubert (1981) had used the presence of a masking deficit within various subgroups of schizophrenics and schizotypals to verify the existence of spectrum disorder. Badcock *et al.* successfully extended this research to include high P scorers, who showed significantly more deficit than low P scorers. They also argued that the results of such experiments might be simply the effects of (1) differential no-masking thresholds and (2) differential susceptibility to increasing task difficulty. Both possible determinants were shown to be active, with high P scorers requiring longer target durations, at a particular level of accuracy, than low P scorers, and with high P scorers showing greater effects for more difficult stimuli. These results make interpretation of the masking data more difficult, but agree with reduced original sensitivity levels in schizophrenics (Mannuzza, Spring, Gottlieb & Kietzman, 1980; Braff & Saccuzzo, 1981; Nuechterlein & Dawson, 1984) and in schizotypics (Merritt & Balogh, 1984). Whatever the correct interpretation, the results support a proportionality approach.

Word association tests show a similar result. It is well known that schizophrenics show unusual and rare responses to standard lists of words (Kent & Rosanoff, 1910; Tendler, 1945; Pavy, 1968). A similar effect has been observed in the biological relatives of schizophrenics (Ciarlo, Lidz & Ricci, 1967; Zahn, 1968; Mednick & Schulsinger, 1968; Griffiths, Mednick, Schulsinger & Diderichsen, 1980). Two studies have extended this relationship to normal groups of students, finding P positively and significantly correlated with unusual and rare word associations (Upmanyu & Kaur, 1986; Ward, McConaghy & Catts, 1991). Here again we find agreement with the proportionality criterion.

Low platelet monoamine oxydase (MAO) has been found in psychotic patients, and also in their relatives and inpatients who have recovered, suggesting that low MAO activity may be a marker for 'vulnerability' (Buchsbaum, Coursey & Murphy, 1976; Schalling, Edman & Aesberg, 1987). In a recent study of 61 healthy high school volunteers, Klinteberg, Schalling, Edman, Oreland and Aesberg (1987) found correlations of -0.30 in female and -0.27 in males between MAO and psychoticism. It may also be noted that low MAO activity was found related to extraversion, impulsiveness, and sensation-seeking, as well as monotony avoidance, and that Lidberg, Modin, Oreland, Tucker and Gillner (1985) found it related to psychopathy, again suggesting a relationship between psychopathy and schizophrenia. (See also Checkley, 1980, for a review of MAO in relation to depressive illness.)

These results may be related to serotonin levels which seem to have similar behavioural correlates as MAO, and hence may be predicted to correlate inversely with P (Zuckerman, 1991). Schalling, Aesberg and Edman (1984) have in fact found that CSF 5-HIAA levels were inversely related to P scores; similarly, CSF levels of 5-HIAA were found to be positively related to a measure of inhibition of aggression, suggesting that in humans, P is related inversely to the functioning of the serotonergic system, as is much psychopathology.

The next measure is based on the Venables (1963, 1964) and Claridge (1972) theory that psychotic patients differ from normals not so much in their absolute levels on the range they cover on given psychophysical measures, but rather in the way in which different measures co-vary. Thus in psychosis, whether natural or LSD induced, there occurs a peculiar inversion of the co-variation between autonomic and perceptual function. The most widely used measures were the two-flash threshold and the electrodermal response. Claridge and Chappa (1973) have extended this model to normal subjects, and have shown that high P scorers do indeed behave, when compared with low P scorers, as schizophrenics do when compared with normals. They conclude: "The results provide evidence for psychoticism as a normal personality dimension having, as its biological basis, a particular kind of nervous typological organization seen, in its extreme form, in the psychotic disorders" (p. 175). Later studies have extended this peculiar inversion of perceptual and autonomic functioning to relatives of psychotic patients, i.e. to members of the psychotic Erbkreis (Claridge, Robinson & Birchall, 1985). A more detailed discussion of the whole theory and its relevance to the concept of psychoticism is given in Claridge's (1985) book on the Origins of Mental Illness. (It should be noted that a replication study of the Claridge and Chappa study was only partially successful, for reasons which are not immediately obvious.)

Another interesting variable frequently used in this context is the alleged inefficacy of 'filtering' mechanisms in schizophrenia (Hemsley, 1975, 1976).

In this connection it may be useful finally to list two psychological systems which have received much theoretical attention, and which may have a causal influence on schizophrenia, as well as being related to P. The first of these is *negative priming* (Beech & Claridge, 1987), a concept widely used to explain the schizophrenic's failure to use inhibitory material early in the information processing system, thus allowing material in the pre-conscious to gain conscious representation (Frith, 1979). The general nature of the effect is as follows. First, a distractor is used in a priming display; when next used as a target stimulus, response latency to the latter is increased compared with trials where no such relation is present (Tipper, 1985; Tipper & Cranston, 1985). This concept may be used as a measure of individual differences (Tipper & Baylis, 1987), and if it is true that schizophrenia is associated with a weakening of the inhibitory mechanisms, then we would predict a negative correlation between measures of negative priming and interference, and a positive correlation between psychoticism or schizotypy and interference. In other words, negative priming is a precondition of effective inhibition of interfering stimuli, and inefficient negative priming, as in schizophrenics and high P scorers, would lead to interference and hence poor performance.

Another concept to be discussed here is that of 'latent inhibition', a close relation of negative priming (Weiner, 1990). Passive pre-exposure of a stimulus reduces its ability to enter into new associations when that opportunity is offered in the same context as the initial pre-exposure (Macintosh, 1975; Pearce & Hall, 1980). This phenomenon, originally studied in animals, has now also been widely investigated in human subjects, both adults and children (Lubow, 1989). Lack of latent inhibition would promote attentional deficits, such as occur in schizophrenics, and it has been shown that schizophrenics not under medication, or at an early stage of medication, do indeed show less latent inhibition than controls (Baruch, Hemsley & Gray, 1988). It was found that medication, as expected, reversed this trend.

When the same procedure was tried on normal subjects, using the Claridge schizotypy scale and the Eysenck psychoticism measure as psychosis-prone scales, these were negatively correlated with latent inhibition, supporting the hypothesis. Lubow, Ingberg-Sacks, Zalstein and Gewirtz (1992) have replicated the Baruch, Hemsley and Gray (1988) study, showing that latent inhibition was weaker in high P than in low P subjects. Here also predictions of proportionality are successfully verified.

One final example relates to a characteristic of the schizoid individual already noted by Bleuler (1978) in 1911, in his description quoted above, namely that such persons show "a predisposition to creativity". This speculation, unlikely as it might seem at first, has found some empirical support (Eysenck & Eysenck, 1976), and fits well into our proportionality scheme (Eysenck, 1983). What has been shown is that offspring of schizophrenic mothers raised by foster parents were notably more successful adults, possessing artistic talents and demonstrating imaginative adaptation to life to a degree not found in the control group (Heston, 1966). Similarly, Karlsson (1968, 1970) found among relatives of schizophrenics a high incidence of individuals with great creative achievements. McNeil (1971) found that the mental illness rates in highly creative adoptees and in their biological parents were positively and significantly related to the creativity level in the adoptees. These and many other studies (Hasenfus & Magaro, 1976) suggest strongly that Bleuler was correct in his assertion.

Does P correlate with creativity? The answer would appear to be in the affirmative. Woody and Claridge (1977) have found surprisingly high correlations between P and creativity as measured by the Wallach-Kogan Creativity Tests, with r values in the 60s and 70s. Gotz and Gotz (1979a,b) studied outstanding German artists, both male and female, and found that they had unusually high P scores. These are only some of the studies demonstrating the predicted relationship between P and creativity, again obeying the proportionality extension (Eysenck, 1983).

Efforts to investigate the proportionality criterion have not always been successful. Thus the Kamin blocking effect [impaired learning of an association between a conditional stimulus (CS2) and an unconditional stimulus (UCS) if CS2 is presented simultaneously with a different CS (CS1) already associated with the UCS] is absent in acute schizophrenia (Jones, 1989), but there was no systematic relation between the blocking effect and any of 4 measures of psychoticism (Jones, Gray

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& Hemsley, 1990). The reasons for this failure are not known, but the theoretical model is a promising one that deserves to be studied more intensely.

Robinson and Zahn (1985) carried out a study attempting to link psychoticism with arousal. Postural conditions were used to induce high and low activation levels in normal subjects high or low in P. Electrodermal and heart rate measures were recorded in different activation conditions, with high P scorers showing significantly lower autonomic arousability and poorer two-flash performance while undergoing the low activation condition. Various other differences were found between high and low P scorers, and the authors argue that the results tend to show a similarity of high scorers' performance to that of psychopaths, rather than of schizophrenics, as reported in the literature.

Szelenberger (1979) studied the results of the visual evoked response recovery cycle in 41 schizophrenic inpatients and 41 healthy subjects. Patients showed a lack of P_2 response amplitude facilitation, as previously found by Shagass (1968) and others. A significant correlation of -0.33 was found with P in the normal group, which suggests conformity with the hypothesis linking P and schizophrenics, but Szelenberger points out that the relationship between facilitation is much stronger with neuroticism, so that perhaps the study should be counted among the failures.

Much more could of course be said about the theories involved in these studies, the experimental difficulties of taking into account drug administration in chronic schizophrenics, or indeed the theoretical prediction of changes in experimental behaviour to be expected when acute psychosis becomes chronic (Gray, Feldon, Rawlins, Hemsley & Smith, 1991). Many of the questions find at least a tentative answer in the Gray *et al.* paper, which attempts the construction of a neuropsychological model of schizophrenia (or perhaps psychosis?). This model includes animal studies, amphetamine effects on psychotic-like behaviour, and several other topics indirectly relevant to our purpose, but not sufficiently so to deserve detailed comment here.

We may summarize the findings of this section by stating that the methodology of proportional effect has been surprisingly successful in showing that schizophrenic-normal differences are reproduced when comparing high and low P scorers, both in normal and (less frequently) in psychotic groups. While not universally successful, the great majority of comparisons have shown the expected effects, and it would seem difficult to account for these findings on grounds other than the admission of a *continuum* ranging from the normal to the psychotic, with gradings both within the normal and the psychotic portion. Many details remain to be sorted out, and many other hypotheses remain to be tested, but the outline is becoming clear.

Some of the 'failures' listed above may be due to differences within overall diagnostic groupings like 'schizophrenia'. An example is furnished by a well-known discrimination between different sub-types of schizophrenics. Thus the distinction between paranoid and other types of schizophrenia is clearly made by Venables and O'Connor (1959), who use the term 'withdrawn' to characterize the non-paranoid groups. The former have been shown to be 'hyperscanners', non-paranoid to be 'hyposcanners' of the environment (Silverman, 1964). Goldberg, Schooler and Mattson (1968) argued that hyperscanners would do better than hyposcanners on reaction time tasks, and demonstrated that this was indeed so. One might argue that paranoia is merely an extreme form of psychoticism; withdrawal adds a specific psychotic element.

DISCUSSION AND CONCLUSIONS

In this article I have tried to determine the degree to which the empirical evidence supports three major portions of the dimensional or continuity hypothesis which I originally advanced some 40 years ago (Eysenck, 1952a,b). The three components state:

(1) Psychotic symptoms and illnesses do not form completely separate diagnostic entities, unrelated to each other, but are genetically related and form a general cluster with severity of illness the major distinguishing marker. It is not part of the theory to deny specificity of genetic origin also existing and contributing to the total variance; it is merely asserted that in addition to specificity there also exists a certain amount of generality, suggesting that the term 'psychosis' contains a meaningful generalization.

- (2) Psychosis is not a separate diagnostic entity which is categorically separated from normality; it is merely an extreme along a continuum of abnormality shading into schizoid personality, 'spectrum' disorders, psychopathy and personality disorder, criminality and alcoholism, and average types of behaviour right to the other extreme of empathy, altruism and selflessness.
- (3) This continuum is co-linear with the concept of psychoticism, embodied (however imperfectly) in the P scale of the EPQ, and also in a number of 'schizotypy' constructs and scales. Proof for this proposition makes use of criterion analysis and its derivative, the proportionality criterion. All the elements of this theory are empirically testable, and have been so tested on numerous occasions.

As regards the generality of 'psychosis', it seems clear that there are definite genetic links between different diagnostic categories (schizophrenia, manic-depressive disorder, schizo-affective disorder, unipolar disorder) which make it impossible to regard them as entirely separate disease entities. Some specificity there undoubtedly is, but there is also a generality of disorder which links all these disorders and their subclassifications and diagnoses together to form one end of the psychoticism continuum, with a severity gradient placing schizophrenia at the extreme end, followed by schizo-affective disorder, manic-depressive disorder and finally unipolar illness.

It equally appears clear that there is no absolute barrier between this concept of 'psychosis' and borderline disorders linking these psychoses with more normal behaviours. Many different names have been given to these transitional states (schizoid personality, 'spectrum' disorders, personality disorders, psychopathy) which in turn connect intimately with alcoholism, criminality, eccentricity and anti-social behaviour generally. Again there is probably some degree of specificity connected with all these types of behaviour but there is also the continuum which links them together, and with psychotic states (Rieder, 1979).

Is this continuum adequately measured by the EPQ-P scale? Because of the novelty of the concept, and because of the short time during which it has been investigated, the scale clearly has many faults, but nevertheless when tested it has proved surprisingly successful in marking the continuity from schizophrenia to normal behaviour. Undoubtedly, it is likely to benefit from continuous improvement (as in the EPQ-R scale; Eysenck, Eysenck & Barrett, 1985) but it seems already to have many of the attributes required by an instrument designed to investigate the properties of the psychotic continuum. Various schizotypy scales appear to fulfil a similar function, but they suffer from correlating so highly with neuroticism that we must seriously doubt their adequacy as measures of psychoticism; as Kendler, Gruenberg and Strauss (1981) have shown in their analysis of the Copenhagen adoption study, there were no genetic or familial environmental links between anxiety and schizophrenia. Schizotypy scales seem to measure aspects of both psychoticism and neuroticism, with an emphasis on the latter; this does not suggest that they would be well equipped to measure the former. However, only continued work with all kinds of 'psychosis-prone' measures will ultimately determine which is closest to co-linearity with (true) 'psychoticism'.

In the list of variables used to illustrate the proportionality criterion, I have on purpose included several different types of measures. One class deals with biological variables (HLA B27, MAO; serotonin) of different kinds. A second deals with laboratory behaviours (eye-tracking; dichotic shadowing; sensitivity levels). A third is concerned with learning-conditioning variables (latent inhibition; negative priming). Yet a fourth is concerned with psychological variables (creativity, hallucinatory activity; word association). Physiological variables (EMG, autonomic-perceptual inversion) constitute yet a fifth set of variables. It is the variety of variables which makes the results impressive, together with the theoretical congruence; to obtain successful results over such a wide array of variables suggests that the underlying hypothesis may be along the right lines.

What are the advantages of the perspective suggested here over the traditional categorical viewpoint of psychiatric diagnosis? In the first place, it is more in line with reality, as the experiments and investigations listed in the text suggest. In the second place, it suggests experimental investigation which the orthodox model would fail to generate, or regard as important. In the third place, it obviates certain difficulties in experimentation, such as institution-alization and drug-treatment of patients, which have made proper experimental study of psychotic

patients very difficult—it is always problematic whether observed differences between patients and controls are due to some disease process, or to drug and/or institutionalization (iatrogenic) effects. If the theory here offered is anywhere near correct, we can test our hypotheses by investigating high- vs low-P normals, or even animals (Gray *et al.*, 1991). This greatly expands the horizon of our theory-testing paradigms, and may hopefully lead to a better understanding of psychotic disorders, and their treatment.

The advantages of joining the psychiatric and psychological research efforts devoted to schizophrenia and psychosis generally, and normal behaviour and the rest of the psychoticism continuum, are likely to go both ways. Thus the question of the biological basis of psychoticism may find a solution based on biological model building in the schizophrenic compartment (Gray et al., 1991; Schmajuk, 1987; Swerdlow, Koob, Geyer, Mansbach & Braff, 1988; Weinberger, 1987; Frith & Dove, 1988; McKenna, 1987; Joseph, Frith & Waddington, 1979). Work already referred to concerning MAO and serotonin fits in well with at least some of these models, and it may not be too long before an agreed theory of biological causation for P arises to take its place with the biological theories giving a causal basis for N and E (Eysenck, 1967, 1981). Zuckerman (1991) has attempted to give a local habitation and a name to the entities involved in his book on *The Psychobiology of Personality*, written from the same point of view underlying the planning of this article, and the reader looking for further enlightenment is referred to his summary of the evidence. By accepting the continuity hypothesis, and by working towards a proper theory from both ends (psychosis and normality), we are more likely to arrive at the desired end.

One final comment may be in order. Factor analysis has often been criticized because of its lack of objectivity; the number of factors extracted, the mode of rotation adopted, and the naming of the resulting factors is to some extent at least subjective. The methods and theories described in this paper attempt to avoid such subjectivity; the factor isolated and named 'psychoticism' is firmly based on empirical and experimental studies which have tested a large number of deductions from the original theory. The resulting factor has not been *named* psychoticism *post hoc* and by simple inspection of its contents; it was conceived on theoretical grounds, and on the basis of a large body of empirical evidence, and constantly revised to accord with new evidence. The method of criterion analysis, in its varied forms, tests the fundamental correctness of the assumptions underlying the factor. It is suggested that the objections often made to factorial investigations do not affect in any way the concept of psychoticism, because of the efforts made to avoid precisely those criticisms. It is also suggested that the method used may be of much wider applicability in the personality sphere.

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