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A GENOTYPE–ENVIRONMENTAL MODEL FOR PSYCHOTICISM*

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Abstract – This study reports the results of a biometrical genetical analysis of scores on a personality inventory (The Eysenck Personality Questionnaire, or EPQ), which purports to measure psychoticism, neuroticism, extraversion and dissimulation (Lie Scale). The subjects were 544 pairs of twins, from the Maudsley Twin Register. The purpose of the study was to test the applicability of various genotype–environmental models concerning the causation of *P* scores.

Transformation of the raw scores is required to secure a scale on which the effects of genes and environment are additive. On such a scale 51% of the variation in *P* is due to environmental differences within families, but the greater part (77%) of this environmental variation is due to random effects which are unlikely to be controllable.

The genetical consequences of assortative mating were too slight to be detectable in this study, and the genetical variation is consistent with the hypothesis that gene effects are additive. This is a general finding for traits which have been subjected to stabilizing selection. Our model for *P* is consistent with these advanced elsewhere to explain the origin of certain kinds of psychopathology.

The data provide little support for the view that the "family environment" (including the environmental influence of parents) plays a major part in the determination of individual differences in *P*, though we cite evidence suggesting that sibling competition effects are producing genotype–environmental covariation for the determinants of *P* in males.

The genetical and environmental determinants of the covariation of *P* with other personality dimensions are considered. Assumptions are discussed and tested where possible.

INTRODUCTION

There have been many developments in the genetic analysis of personality since Newman *et al.* (1937) suggested that heredity made relatively little contribution in this field, as compared with that of intelligence. Bracken (1969), Roubertoux and Carlier (1973) and Eysenck (1976) have reviewed the literature and have concluded that there is considerable evidence in favour of the opposite view; heredity plays a powerful part in the genesis of personality differences. As Eysenck (1976) makes clear, this change in view is due to two major developments: (1) an improvement in the personality models available to present-day research workers, and (2) an improvement in the methods of analysis contributed by biometrical geneticists. As much of this paper will be concerned with the application of these new methods to a particular problem, no more will be said here about our second point.

As regards the first point, Royce (1973) has recently reviewed the literature, and has concluded that there is evidence from many replicated studies for the existence of three "superfactors" which have received various divergent names, but which we may perhaps call *N* (neuroticism, anxiety, or emotionality vs. stability) *E* (extraversion vs. introversion) and *P* (psychoticism vs. super-ego strength). These factors have much better standing scientifically than the traits (and measures) used by Newman *et al.* in their pioneering study. The general

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dimensional personality model underlying this general system of measurement has been discussed elsewhere (Eysenck, 1970a), as has the heritability of N and E (Eysenck, 1976). Here we shall be concerned with the construction of a genetic model for the P dimension.

The hypothesis of a major personality dimension underlying psychotic behaviour, and running from normal to abnormal conduct, was first suggested by Eysenck (1952a) who also worked out a statistical method for testing the theory of continuity (criterion analysis – Eysenck, 1950). Application of this method to the analysis of experimental test results from normal and psychotic groups demonstrated the tenability of the continuity (dimensional) hypothesis (Eysenck, 1952b). The theory has recently been stated in a more formal manner, emphasizing its genetic derivation and implications (Eysenck, 1972). The conception of psychoticism has been given formal empirical expression in the form of the P scale of a new questionnaire, the EPQ (Eysenck Personality Questionnaire – Eysenck and Eysenck, 1976a) which also measures E,N and L (lie or dissimulation scale). This questionnaire is the end-point of a long history of development, through various less satisfactory scales; this development has been traced for adults (Eysenck and Eysenck, 1968, 1971; Sybil Eysenck and Hans Eysenck, 1968, 1969a, 1972) and for children (Sybil Eysenck and Hans Eysenck, 1969a, 1973; Eysenck *et al.*, 1971). It is an essential feature of the theory that psychotics should have high P scores, and that within the psychotic group, size of P score should be commensurate with severity of psychosis; this is so (Verma and Eysenck, 1973). A number of predictions have been made, based on the theoretical conception underlying P; a summary of tests carried out to verify these predictions is given elsewhere (Eysenck, 1973). It is part of the theory that psychotic behaviour and psychopathic behaviour should show a large measure of communality, and studies have been carried out to verify the hypothesis that criminals have exceptionally high P scores (Eysenck, 1970b; Sybil Eysenck and Hans Eysenck 1970, 1971a, b, 1974). In the present study we have used the final version of the P scale; previous heritability studies, (Eaves, 1970, 1973; Eysenck, 1972, Eaves and Eysenck, 1974, Insel, 1974) have used earlier and less satisfactory inventories (named PEN and PI). The theories underlying the concept, and the empirical works supporting it, are now available in book form (Eysenck and Eysenck, 1976b).

A brief discussion of the general theory, and of some of the empirical material supporting it, may be of interest before turning to the discussion of the genetic and environmental factors responsible for variations in P. Originally, genetic studies of the first- and second-degree relatives of probands with schizophrenic or manic-depressive psychosis had shown that these relatives manifested psychosis, not always of the same type as the proband, much more frequently than relatives of non-psychotics; this was true even when the relatives of psychotics were brought up by adoptive non-psychotic parents from birth. This suggests the existence of some underlying general predisposition (*diathesis*) for psychosis, genetic in nature, but requiring some environmental *stress* to produce actual psychotic breakdown (diathesis–stress model). Edwards' model of polygenic inheritance (Edwards, 1969) puts the matter very clearly, and is reproduced here from Shields (1971) as Fig. 1; the legend makes clear the meaning of the

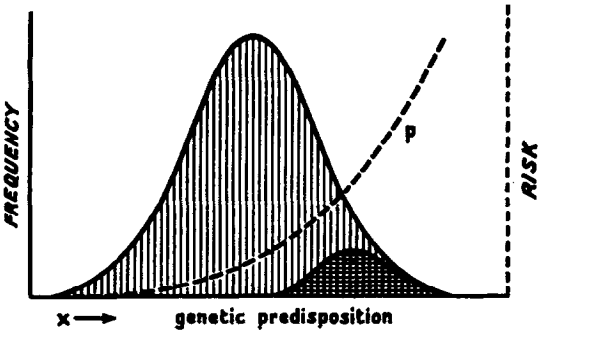

FIG. 1. Edwards' model of polygenic inheritance. For X (genetic predisposition) read P (psychoticism) in the case considered in this paper. Taken with permission from Shields (1971).

various parts of the figure. The cross-hatched portion of the diagram denotes diagnosed psychotics; the normal distribution covering the major part of the figure denotes the distribution of our hypothetical psychoticism variable (*P*). The abscissa is labelled "genetic predisposition" to denote the hypothesis that *P* is in fact largely controlled by genetic mechanisms; it is of course the main purpose of this paper to support or disprove this hypothesis.

Of particular interest in connection with this theory are those persons lying close to the small group of affected individuals; while not themselves affected, they should show elevated scores on a measure of *P*, and thus a high genetic predisposition towards abnormal behaviour. Such groups can be found with undue frequency among the first- and second-degree relatives of psychotics, particularly schizophrenics (Eysenck, 1972, 1976; they show evidence of psychopathy, criminality, schizoid behaviour, alcoholism, and other forms of abnormality. It is important for our theory that such groups also show elevated *P* scores; this is an obvious requirement of our theory. We thus regard *P* as theoretically a phenotypic measure of the hypothetical genetic predisposition towards psychotic behaviour diagrammed along the abscissa of Fig. 1, and we consider that *p* in the diagram (i.e. the likelihood of being affected by the disorder in question) is a monotonic function of *P* (the score of the EPQ).

This monograph explains our attempts to analyse genetically the variation we observed in subjects' scores on the *P* scale of the EPQ (Eysenck Personality Questionnaire) both as a trait in its own right and in relation to other traits for which scores are usually derived from this questionnaire, namely neuroticism (*N*), extraversion (*E*) and the lie scale (*L*). We shall discuss our methods in some detail, hopefully without undue technicality because the methods we have adopted, though long familiar to quantitative geneticists, have so far been applied but rarely to the analysis of human behaviour. Our approach is that of biometrical genetics (see Mather and Jinks, 1971). The application of these methods to human behaviour was considered in greater technical detail by Jinks and Fulker (1970) but since their publication, further theoretical and analytical advances have been made to which we shall allude in this paper. We have adopted the biometrical–genetical approach because it is completely general and provides a framework in which assumptions can be specified in precise quantitative terms so that various models for the causes of variation can be tested statistically and falsifiable predictions can be made about the outcome of further studies.

Our account of the analysis of *P* will involve some symbols which we shall usually define verbally, though often a precise mathematical definition can be given for those terms which involve the frequencies and effects of alleles at a number of genetic loci. We shall give the numerical estimates of relevant parameters but will avoid precise details of the estimation procedures which are given in more technical accounts. It is sufficient to acknowledge that we have tried to obtain the best possible estimates, but we are aware that the properties of our data are not ideal for such purposes.

The data we present have been collected from twin volunteers who comprise the twin register of the Department of Psychology of the Institute of Psychiatry, London. As is common among volunteer samples we find that females are more frequent than males, and there is a marked excess of monozygotic over dizygotic twins. Zygosity of the twins was usually established by questionnaire, although some were diagnosed by blood-typing. Twins were diagnosed as monozygotic if they reported being frequently mistaken for one another in childhood and stated that they did not differ markedly in physical appearance or colouring. Pairs which gave inconsistent responses were diagnosed from the knowledge of the pair accumulated by the collator of the data and reference to documents such as letters sent by the twins. Pairs which claimed marked dissimilarity in colouring or appearance and which reported no confusion in childhood were classified as dizygotic.

Kasriel and Eaves (1976) have examined the responses of 178 pairs of twins whose zygosity had been established with a high degree of confidence by competent blood-typing. They found 2% of monozygotic pairs were misclassified as dizygotic, and a greater (6%) tendency erroneously to classify dizygotic pairs as monozygotic. The precise consequences of such misclassification remain to be analysed and will depend on the relationship between the classification criterion and the traits under study.

We proceed in this paper as if we had exact knowledge of zygosity. We believe that the

analysis is still worthwhile even if the more extensive data currently being collected show our conclusions to be unjustified.

We deal mainly with the *P* scale of the EPQ although we shall compare our results with those we obtained in an earlier (and larger) study of a similar scale from the PEN. The questionnaires were posted to twins on the register and the completed questionnaires were returned to the Institute of Psychiatry for coding. About half of the mailed questionnaires produced complete responses for the 554 pairs of twins whose scores are analysed here.

TRANSFORMATION OF THE *P* SCALE

The bizarre quality of extreme psychotic behaviour is to some extent reflected in the distribution of responses to *P* items in personality questionnaires. Generally items of a *P* scale are endorsed only rarely in a "psychotic" manner, thus few people admit to hating their mother or confess to feeling no discomfort when faced with another's suffering. The rarity with which "psychotic" responses are made contributes to a *P* scale which, in its raw form at least, has many undesirable properties. Although there are twenty-five items contributing to the *P* scale of the EPQ the modal score differs greatly from the mean. A recent survey gave the summary statistics in Table 1. The distribution of raw scores is thus virtually *L* shaped and makes reliable analysis of the present raw *P* scale difficult. In particular the errors of measurement associated with a given *P* score increase as *P* increases. Thus one source of environmental variation in *P*, namely unreliability of measurement, becomes more important when we consider individuals who are more "psychotic", as measured by their raw *P* score. To the extent that individuals' predispositions on the *P* scale are genetically determined we will find that genetical and environmental factors do not operate independently but that they interact. In effect we shall find that sensitivity to the environment is itself apparently under genetical control. We can show that this is so by using the absolute intrapair differences for *P* scores of MZ twins measures of environmental differences within pairs and relating these to the corresponding pair means. We thus take the scores, P_1 and P_2 say, of a pair of MZ twins and calculate, for every pair, the absolute difference $|P_1 - P_2|$ and the sum $P_1 + P_2$. We may study the relationship between sums and differences by plotting a graph or summarize the mathematical relationship by calculating a best-fitting curve relating sensitivity to the environment to predisposition to psychoticism. This is the procedure first applied by Jinks and Fulker (1970) to the analysis of genotype–environment interaction in humans. For the raw *P* scores we find a sum–difference correlation of 0.47 for females and 0.50 for males. Of course, since our MZ twins are reared together there may be environmental influences shared by the twins which may also interact with those environmental factors for which the twins differ and so contribute further to the relationship between sums and differences, but the large sum–difference correlations we find for the raw scores indicate a considerable non-additive component of variation.

Table 1. Summary statistics for raw *P* scores from the P.Q.

	Males	Females
N	318	770
Mean	3.365	2.131
Minimum	0	0
Maximum	14	11
Median	2.657	1.675
Mode	2	0
Variance	8.485	4.184
Skewness	1.174	1.246

Any analysis of the raw scale into component causes is likely to lead to a very complex picture because of the large genotype–environment interactions created by the choice of scale. Similarly, any attempt to use the raw scale for predictive purposes may be complicated unnecessarily by the need to consider the many consequences of genotype–environmental interaction. If there are good biological or psychological reasons for retaining the present scale

of measurement for P then we must accept the complications and be prepared to grapple with the consequences. If, however, the adoption of the present scale is largely a matter of convenience then we should not be afraid to change our scale if such a change can be justified on psychological or analytical grounds.

In the case of P (as indeed in the case of many other scales used in personality assessment) statistical, psychological and genetical considerations all coincide happily to suggest a scale of measurement which has far simpler properties and can be derived by a simple transformation of individuals' raw scores on P.

Let us first consider how an individual of a given predisposition responds to the items of the P scale. We may suppose that a particular individual has a given probability, p, of responding "psychotically" to any one of the n items of the questionnaire. Providing we may assume that p is the same for every item in the scale (i.e. that there are no major differences between the items with respect to their endorsement frequencies) and providing the subject distributes his responses randomly over the n items (i.e. for a population of subjects of the same p, the items are independent) then we expect his P score to be np. If we could envisage many independent repetitions of the study, such that the same subject, with the same p, responded without the benefit of prior experience to the same questionnaire, we would expect the different estimates of his P score to differ. The extent of the differences could be assessed by the variance between repeated tests which is expected to be $np\ (1-p)$ from the properties of the binomial distribution.

Since the frequency, p, with which items are endorsed in a psychotic direction is small, we recognise that the term $(1-p)$ is close to 1 in this case, so the variance of the P score of a given individual is expected to be equal to the mean, i.e. to np, where n denotes the number of items in the scale. This is, of course, the well known property of the Poisson distribution which is the expected distribution of independent rare events. For small p, such as we find for "psychotic" responses, we thus expect the error variance of subjects' responses to increase virtually linearly with their predisposition to make "psychotic" responses, generating precisely the trend we observe for the relationship between pair sums and absolute differences for the raw P scores which has such undesirable properties both for interpreting the causes of variation and for making predictions.

Since, given our assumptions about the way subjects respond to the items of the questionnaire, we expect and find a linear relationship between predisposition and environmental variation for our twin data, it is a fairly simple matter to find a transformation of the raw P scores which is expected to remove this relationship by making the sampling variance constant throughout. If such a transformation is successful it will remove at least that portion of the genotype–environmental interaction which is due to systematic variation in errors of measurement.

It may be shown that a square root transformation of our raw scores should remove such a linear relationship between mean and variance, and thus remove much of the genotype–environmental interaction. In fact, we find that converting our raw P scores to $P^* = \sqrt{P/n}$ removes nearly every trace of sum–difference covariation for the MZ twins. The correlations between pair sums and absolute intrapair differences are now –0.11 for females and –0.02 for males. We find also that transformation of P to P^* removes for the EPQ nearly all the significant heterogeneity of the total variances between the different groups of twins. When we consider the P scale of the earlier PEN questionnaire, we find that a simple square-root transformation is less effective than a transformation of P to $P^* = \sqrt{(P+1)}$ which is suggested as one alternative to the simple square-root transformation when there are many zero scores, (Snedecor and Cochran, 1967). Bartlett (1947) considered the properties of many transformations which might be applied profitably to questionnaire data before attempting any analysis of the causes of variation if unnecessary complexity is to be avoided where possible.

AN ENVIRONMENTAL MODEL

Having satisfied the basic requirements of scale we may turn to the crucial matter of attempting to identify and interpret the causes of variation in psychoticism as we have tried to measure it. We have already demonstrated by our successful transformation of the raw P scores

that a scale can be found on which genotypic and shared environmental experiences apparently do not interact with the unique experiences of individuals within the family, at least not in any systematic way which is likely to be amenable to analysis and aid prediction. If there were such interactions we would expect the transformed scale to show evidence of a systematic relationship between the sums and absolute differences of our MZ twin pairs.

So we may expect a fairly simple explanation of variation in P^* to be adequate since there is nothing to suggest genotype–environment interaction when adjustment is made for the obvious properties of the scale. If there are genetical and environmental factors affecting P^* they are acting additively. That is, the effect of the environment does not depend on the genotype of the individual concerned.

Any theory of individual differences, whether it involves genetical factors, environmental factors or some combination of both, must ultimately be cast in quantitative terms if its predictive utility is to be assessed. We begin, therefore, by seeking a statistical summary of individual differences in P^* and by attempting to predict the relationships among different parts of our data in terms of a simple mathematical model which embodies the principles of our theoretical specification. Suppose, for example, all the individual differences in P were due to environmental influences which were quite specific to individuals, and shared with no one else, not even with members of the same family. We could equate the phenotypic variation observed for P^* in a given population simply to a parameter, E_1 say, and write:

$$\sigma_P^2 = E_1 .$$

A simple test of this model is, of course, provided by observations of individuals reared in the same family. If all our variation were due to quite unique individual experiences we would expect individuals in the same family, to be no more and no less alike than families of individuals produced artificially by grouping our test scores at random. If we were able to determine the means of our natural families exactly, and compute the variance of such means, σ_b^2, we would expect it to be zero because there is expected to be no variation between families as all the individual environmental effects are expected to be distributed randomly among individuals in the population, and in no way related to the classification into families.

One simple way of assessing the importance of σ_b^2 for family groupings is the intraclass correlation coefficient. We have adopted the analysis of variance, however, because such a data summary imposes no artificial scaling on the variation of different groups of individuals.

In Table 2 we give the form of an analysis of variance of paired individuals. The individuals may be paired according to any criterion. Each pair, for example, might be a twin or sibling pair.

Table 2. General form of analysis of variance of paired observations

Item	d.f.	Expected mean square
Between pairs	$n-1$	$\sigma_w^2 + 2\sigma_b^2$
Within pairs	n	σ_w^2

In Table 3 are given the actual analyses of variance for five groups of relatives, male and female MZ twins, male and female DZ twins, and opposite sex DZ twins. (In the latter case we have removed a further component due to the sex difference.) A variance ratio test (F) shows that in most cases σ_b^2 was significantly greater than zero. Clearly this is not what we expect if all the differences are due to individual environmental experiences with the family ("E_1") so we would be forced at this stage to reject any model which attempts to explain the variation in P only in terms of environmental differences within families.

Before we move on in an attempt to explain the similarity between relatives (or the differences between families, which amount to the same thing) we shall use our simple example to introduce a further procedure to which we must refer in a subsequent discussion.

Table 3. Analyses of variance of twin pairs for transformed *P* scores

Twin type	Item	d.f.	mean square	*F*	*P*
Female monozygotic	Between pairs	240	0.033891	2.41	10^{-11}
	Within pairs	241	0.014087		
Male monozygotic	Between pairs	78	0.051267	3.71	10^{-8}
	Within pairs	79	0.013817		
Female dizygotic	Between pairs	132	0.042274	1.99	4×10^{-5}
	Within pairs	133	0.021206		
Unlike-sex dizygotic†	Between pairs	72	0.036167	1.66	0.016
	Residual	72	0.021789		
Male dizygotic	Between pairs	50	0.030715	1.53	0.067
	Within pairs	51	0.020092		

†The residual term is corrected for the mean difference between sexes. Results are pooled from two sets of data distinguished by the order in which the sexes were born.

Suppose we had satisfied ourselves that σ_b^2 was zero for all groups of twins, so that we felt justified in making our original equation relating σ_p^2 to E_1. How would we attempt to estimate E_1? Reference to the d.f. in Table 3 shows that some statistics are known more precisely than others. We have 241 pairs of female MZ twins, but only 57 male DZ pairs. Clearly we know the mean squares for the first group more precisely than those of the second so we need to give additional weight to statistics based on more observations if we wish to obtain our best estimate of E_1. We expect chance variation to affect the smaller groups more, so we do not wish these to affect our final estimate more than is absolutely necessary to ensure the most efficient use of the data.

Similar considerations apply if a model fails. We would wish to give more weight to deviations from our expectations when they relate to statistics based on relatively larger numbers of observations.

Clearly, in our simple case, we have ten mean squares all of which should equal E_1 if our simple theory of the causes of individual differences were right. If we were to obtain our best estimate of E_1 using all ten statistics, appropriately weighted, we could see how much the observed statistics varied around the values we would predict from a single estimate of E_1. If these variations were large we would be tempted to reject our model. If they were small we would conclude that we had no evidence to justify the rejection and so we might adopt it provisionally as a working model for individual differences in *P*.

The formal mathematical procedure we have outlined above is that of "weighted least squares". The application of the technique to problems of this kind, together with some of the assumptions underlying its use have been considered in more detail elsewhere (Mather and Jinks, 1971; Jinks and Fulker 1970; Eaves 1969).

If we attempt to account for all the mean squares purely on the basis of specific environmental experiences (E_1) we find that our best estimate of E_1 is 0.02765 but that a χ^2 test of goodness of fit of the model gives $\chi_9^2 = 108.9$ ($P < 10^{-6}$) indicating that such a model is far from adequate and that alternative, more complex, explanations must be sought.

INCLUDING THE SHARED ENVIRONMENT

Possible explanations of individual differences fall into two general categories:

(1) purely environmental;
(2) genotype–environmental.

Under the first heading we must include the simple E_1 model which we have just rejected, and any serious attempts to account for similarities and differences in terms of environmental causes. The difficulty with environmental models is that any but the simplest have so far escaped quantitative formulation, though some serious attempts have now been made to provide a more rigorous quantitative basis for analysing the mode of operation of environmental factors; (e.g. Cavalli-Sforza and Feldman, 1973; Eaves, 1976a,b). One simple environmental model seeks to explain the variation between individuals in terms of two kinds of environmental components. The first of these, E_1, summarizes the variation due to the

specific environmental experiences of individuals which they do not share even with individuals reared in the same family. The second, E_2, summarizes the variation due to differences in the quality of environment resulting from variation provided by families, natural or foster, in which individuals are raised and which are shared with all members of the same family. The effects which comprise E_2 may be maternal or paternal, social or physical, physiological or behavioural. In fact, on further analysis, they may be shown to be themselves comprised partly of genetical effects. For this reason it may be premature to dismiss as causally environmental those factors of the family background which are called "environmental" by default.

Our E_1, E_2 model which is perhaps the simplest testable environmental model is expressed in

$$\sigma_p^2 = E_1 + E_2,$$

where σ_p^2 denotes the phenotypic variance of the population we cannot, of course, estimate E_1 and E_2 separately if we have σ_p^2 alone; we need to consider the mean squares of Table 3. In particular we must convert our statistical expectations of mean squares into expectations in terms of E_1 and E_2.

Since our model assumes no genetical effects and no differential treatment on the basis of genotype, we expect the variation between pairs of MZ twins to be no different from that between DZ twin pairs, within the limits of sampling variation. So we can write $\sigma_{b\,mz}^2 = \sigma_{b\,dz}^2 = E_2$. Similarly, we can write our expectations for the within-family components, $\sigma_{w\,mz}^2 = \sigma_{w\,dz}^2 = E_1$. Thus, the model assumes that variation between pairs of twins is a measure of variation between the environments provided by families as a whole, and that all variation within families is due to the specific environmental experiences (including errors of measurement) of individuals irrespective of their families. We are, of course, providing a model for *variation*; no model of this kind, genetic or environmental, has any bearing on the effects common to all individuals in a population, be they genetical factors which are not polymorphic, or uniform environmental factors which stem from the prevailing cultural climate.

Our expectations for the ten mean squares of our twin study are given by substituting for the σ^2's of Table 2 their expectations in terms of E_1 and E_2. Thus, all between pair mean squares have the expectation $E_1 + 2E_2$, all the mean squares within families are expected to be estimates of E_1 only. Is our model justified? Weighted least squares estimates are:

$$E_1 = 0.01720,$$
$$E_2 = 0.01050,$$

suggesting that a large common environmental component may prevail in the determination of P^*.

But we cannot take these estimates too seriously because we find that the deviations of observed from predicted values for the mean squares are too substantial to be dismissed purely as sampling variation. Indeed, the χ^2 for assessing the quality of fit of the model is 19.58 for 8df. ($P = 0.012$). Once again, our model has failed to account adequately for the differences in P^* scores among the individuals in our study.

INTRODUCING GENETICAL PARAMETERS

Now we are faced with a dilemma. We can attempt to improve our environmental model by invoking still further parameters. For example, we might attempt to explain the deviations by arguing that we need one or two additional environmental parameters to represent hypothesized differences in the uniformity of treatment of MZ and DZ twins. Alternatively we may relax the restriction that our model be purely environmental and seek an explanation as simple as those we have sought so far by recognizing the regular and predictable properties of inherited differences between individuals.

This is not the appropriate place to recapitulate the history of quantitative genetics. It is sufficient to refer to the repeated demonstration that the pattern of continuous variation for a variety of traits in a variety of organisms is consistent with the view that at least part of such continuous variation is due to the cumulative properties of many genes whose individual effects

are small. Genetical analysis of continuous variation reveals that the genes contributing to such variation differ only in the magnitudes of their measured effects from genes contributing to discontinuous phenotypic differences. They display the properties of segregation, linkage and interaction within and between loci which characterize classical Mendelian inheritance.

Quantitative genetics has continually refined methods of analysis and the theoretical framework for the evolutionary interpretation of the kinds of genetical and environmental factors which give rise to differences between individuals.

The most basic of all models for continuous variation is that which assumes, until evidence is produced to the contrary, that the genetical part of variation for a particular trait is due to the independent and additive effects of many polymorphic loci. Such a model assumes as a first approximation that loci behave as if they were unlinked, that the effect of one locus on the expression of a trait is not altered by changes at other loci, and that heterozygotes at a locus have an affect on the phenotype which is midway between the effects of the two corresponding homozygotes.

In such a case we say that the gene action is "completely additive". Dominance and epistasis are termed "non-additive" effects. If the frequencies of increasing and decreasing alleles are unequal at the loci contributing to the measured variation some (though not all) of the contribution of non-additive effects will be inextricably associated with the additive effects. For this reason we introduce into our model for the genetical variation a parameter "D_R" which contains all the variation due to additive gene action, but in addition it contains *some* of the variation due to non-additive effects when the allele frequencies are unequal. Other parameters are required to specify the remaining contribution of dominance and epistasis if these are present. Mather and Jinks (1971) and Mather (1974) define D_R precisely in terms of gene effects and frequencies. D_R represents a recurring expression in any attempt to simulate algebraically the contributions of genes to variation in a metrical trait for a natural population.

Other authors specify different notations for the same genetical effects. Falconer (1960), for example, uses V_A to denote the additive genetical variance. This is equivalent to $\frac{1}{2}D_R$ in our notation. We find that we can write new expectations for the components of variance of our earlier statistical model for our mean squares. If we assume no non-additive genetical effects, random mating, and independent loci we may write the following expectations for the *genetical* part of the components of variance:

$$\begin{aligned}\sigma^2_{b\,\mathrm{mz}} &= \tfrac{1}{2}D_R,\\ \sigma^2_{w\,\mathrm{mz}} &= 0,\\ \sigma^2_{b\,\mathrm{dz}} &= \tfrac{1}{4}D_R,\\ \sigma^2_{w\,\mathrm{dz}} &= \tfrac{1}{4}D_R.\end{aligned}$$

Such a model makes the assumption that there is no environmental variation. We know such an assumption to be unfounded because identical twins differ. We must therefore, add at least one parameter to specify the additional effect of the environment. We have every reason to include E_1, that is specific environmental variation; we have little reason apart from intuition and opinion to include E_2, so we choose to omit E_2 until we have evidence that it is required.

We thus write our expectations for the components of variance:

$$\begin{aligned}\sigma^2_{b\,\mathrm{mz}} &= \tfrac{1}{2}D_R,\\ \sigma^2_{w\,\mathrm{mz}} &= E_1,\\ \sigma^2_{b\,\mathrm{dz}} &= \tfrac{1}{4}D_R,\\ \sigma^2_{w\,\mathrm{dz}} &= \tfrac{1}{4}D_R + E_1.\end{aligned}$$

Notice that $\sigma^2_{b\,\mathrm{mz}}$ and $\sigma^2_{b\,\mathrm{dz}}$ are the expected variances of *true* family means, that is, they represent what the variances of family means would be if we were to obtain very large random samples of members of each family. Individual within the family influences therefore, *do not* as one might at first imagine, contribute to the true variation between families (σ^2) although they do contribute to the mean square between pairs (since we only sample two out of the possible range of unique genotypes and environmental influences within the family). Our expectations of mean squares between families take account of the finite sample on which family means are

based. By ensuring that the within pairs components (σ_w^2) contribute to the mean square an amount which is, relative to the contribution of σ_b^2, inversely proportional to the family size.

A SIMPLE GENOTYPE–ENVIRONMENTAL MODEL: ASSUMPTIONS

In Table 4 we have the expected contributions of D_R and E_1 to the *mean squares* we have obtained in our study. To get these we simply multiply the expectations of the σ^2's in terms of D_R and E_1 by the coefficients of the σ^2's in the expectation of mean squares and accumulate the contributions of D_R and E_1 over all components contributing to a particular mean square. So, for example, to obtain the expectation for the mean square between MZ pairs we take E_1 (the expectation of σ_w^2) and add D_R (twice the expectation of σ_b^2).

Table 4. Expected mean squares of twin analyses on D_R, E_1 Model

(See text for implied assumptions and Table 2 for the statistical model for the mean squares.)

Twin type	Item	Expected mean square†
Monozygotic	Between pairs	$E_1 + D_R$
	Within pairs	E_1
Dizygotic	Between pairs	$E_1 + \frac{3}{4} D_R$
	Within pairs	$E_1 + \frac{1}{4} D_R$

†The σ^2's of Table 2 are expected to be

$\sigma^2_{w\mathrm{mz}} = E_1$,

$\sigma^2_{b\mathrm{mz}} = \frac{1}{2} D_R$,

$\sigma^2_{w\mathrm{dz}} = \frac{1}{4} D_R + E_1$,

$\sigma^2_{b\mathrm{dz}} = \frac{1}{4} D_R$.

Mather and Jinks (1971, ch.8) explain the derivation of the coefficients.

Table 4 implicitly specifies all the assumptions we are making when we attempt to account for variation in P^* by a simple genotype–environmental model. We may enumerate these:

1. *There are no common environmental effects.* ($E_2 = 0$). If we wished to include them in our model we could do so by adding E_2 to our expectations for the between family components of variance ($\sigma^2_{b\mathrm{mz}}$ and $\sigma^2_{b\mathrm{dz}}$).

2. *There is no dominance.* If we wished to specify the contribution of dominance we could do so by including an additional parameter in a model (e.g. Mather and Jinks, 1971).

3. *There is no epistasis.* If there were, it may well be inseparable analytically from the effects of dominance and E_2 in our study. The specification of epistasis, though tedious, is not impossible for polygenic variation in randomly mating populations (Mather, 1974).

4. *There is no genotype–environment interaction.* We have already suggested that our scale of measurement provides little indication of systematic genotype–environmental interaction. If these were present they could be specified formally (Jinks and Fulker, 1970) but unsystematic GE interactions may not be separable analytically as long as our study was restricted to twins reared together (Eaves and Eysenck, 1975).

5. *Mating is random.* This assumption is implicit in the fact that D_R is given the particular numerical coefficients it receives in the expectations of $\sigma^2_{b\mathrm{mz}}$ and $\sigma^2_{b\mathrm{dz}}$. Any genetical similarity between spouses resulting from their phenotypic similarity would tend to increase the coefficient of D_R in the expectations of $\sigma^2_{b\mathrm{dz}}$ and $\sigma^2_{b\mathrm{mz}}$, although the effect is only large when there is substantial additive variation and the observed correlation between spouses is quite large. Fisher (1918) provides one model for assortative mating, which has recently been explored further by Vetta and Smith (1974). Eaves (1973) showed for IQ how the genetical effects of assortative mating could be detected analytically by the failure of the assumption of random mating to account for the observed pattern of variation for a metrical trait.

6. *There is no sex-linkage or sex limitation.* Although these two assumptions are not identical, their failure in practice would lead to differences in D_R between male, female and opposite-sex pairs. By attempting to fit the same D_R to all groups of twins, irrespective of sex, we are providing an opportunity for failure of these assumptions to lead to significant differences between observed mean squares and their expected values.

7. E_1 *is the same for males and females.* There is no reason why this should be so in principle. By specifying only one E_1 component we are submitting this hypothesis to examination.

8. *Genetical and environmental effect do not covary.* Many of the supposed alternative environmental explanations of genotype–environmental covariation (COVGE). COVGE might arise because one genotype provides the environment for another member of his family. Thus for example, genetical differences between parents may produce environmental differences between offspring. Such COVGE may be expressed precisely in genetical terms so that studies may be designed to aid its detection (Eaves, 1976a). COVGE which arises because of the environmental effect of parental genotypes on the twins in our study will not be separable from the effects of common family environments (E_2). In our case, therefore, they will contribute to failure of a simple model, but we shall not be able to identify the precise reason for failure of our assumptions. COVGE which arises because one twin's genotype is influencing the phenotype of the co-twin may be detected fairly unambiguously in our study, because we may expect the total variances of MZ and DZ twins to differ if twins are reared together (Eaves, 1976b). Furthermore, if the effect is competitive, such that a positive genotypic deviation of one twin produces a negative response in the co-twin, we may find the similarity of DZ twins much reduced, even to the extent of finding negative covariance between DZ twins.

We have thus considered at some length the specific assumptions we have made in representing our expectations of mean squares in terms of two parameters, D_R and E_1.

All the additional effects which we have assumed to be unimportant in the first instance can be represented formally by additional parameters in a more general model. The fact that we can write down parameters corresponding to hypothesized effects, however, does not mean that these can necessarily be estimated if we only have data on twins reared together. Effects of sex linkage and sex-limitation might be detected by the inability of a simple model to explain the pattern of variation in both sexes simultaneously, but it would be difficult without additional data to decide which of the two kinds of gene action were actually causing failure of the D_R,E_1 model.

In our data, the effects of assortative mating and common environments cannot be separated from one another, though a large component of either could conceivably lead us to reject our simple model. Similarity between twins arising because the maternal or paternal genotype for P forms part of the developmentally significant environment of the offspring cannot be separated from similarity due to assortative mating and E_2.

Non-additive genetical variation, especially that due to dominance is likely to be difficult to detect with studies of the size we have used so far in the analysis of P (see Eaves, 1972) but there is an added difficulty in twin studies of this kind which arises because the effects of E_2 (and any effects which in our design are confounded with E_2, such as assortative mating and certain kinds of COVGE) all tend to make DZ twins and MZ twins more comparable in their degree of similarity, whereas dominance tends to make DZ twins less alike relative to MZ twins than we might expect on the basis of our simple model. Thus the various effects might tend to cancel out with two related consequences: firstly we shall find our capacity to detect various sources of failure in our assumptions is sharply reduced; and consequently we shall find, secondly, that we are more likely to adopt our basic model provisionally even though the *real* situation is substantially more complicated. Of course, with data on foster relatives we may be able to sort out the effects of common environments and the related kinds of COVGE but our present study does not have such data. The various studies of similarity between spouses suggest that there is little assortative mating for P (Eysenck, 1975). The largest and most reliable investigation (Nias, 1975) finds a correlation of 0.19 for 586 pairs of spouses. Eysenck (1975) found a correlation of 0.14 with a sample of 241 pairs. Any slight assortative mating suggested by these figures is too small to have detectable genetical consequences in quite large samples for a trait such as P for which the degree of genetic determination is fairly low. In respect of the mating system, therefore, we have some additional evidence that our assumptions are justified.

TESTING THE GENOTYPE–ENVIRONMENTAL MODEL

When we actually fit our D_R, E_1 model to the twin data for the transformed P scores of the EPQ we find that observed and expected mean squares agree quite well ($\chi^2_8 = 9.81, P = 0.28$) so the twin data by themselves give us little reason to doubt the validity of our simple model for individual differences in response to the P items of this scale. Our estimates were:

$$\hat{D}_R = 0.02723 \pm 0.00315,$$
$$\hat{E}_1 = 0.01419 \pm 0.00115.$$

Thus we conclude that we can adopt a working explanation for variation in psychoticism which assumes that there is both genetical and environmental variation in P. Our model suggests that to a first approximation we may discount shared family environmental effects (since a large E_2 might cause the model to fail) and we might consider mating to be random as far as any genetical analysis is concerned. In addition we have found nothing to suggest that gene action is anything other than additive. We have deliberately erred on the side of caution because we are aware of how easy it is to be misled by relatively small bodies of data. The resolution of our study is, however, sufficient for us to reject the simplest environmental explanations but insufficient for us to reject the simplest equivalent explanation in terms of the joint action of genetical and environmental influences. Leaving caution aside for the time being however, let us ask what our model implies if we take it seriously.

Firstly, it implies that about half the variation in P as it is measured on our transformed scale is due to the genetical effects we have attempted to represent by D_R. More precisely we can estimate the proportion of variation due to genetical causes from:

$$\tfrac{1}{2}\hat{D}_R/(\tfrac{1}{2}\hat{D}_R + \hat{E}_1) = 0.46.$$

We can only do this because our model is judged adequate. It would be wrong to give such an estimate if we found our expected mean squares showed significant departures from the corresponding observed values. Most attempts to estimate "heritability" make no such effort to test the underlying assumptions, and use data only in the most inefficient manner.

A "heritability" by itself does not help very much in our understanding of the trait. True, it might help us to predict a response to selection, at least in the short term, but this is more appropriate to the manipulation of cattle than to the understanding of people. If we wish to understand the biology of psychoticism we have to ask whether our genetical model for variation in P has any implications for understanding the polymorphism we observe for psychotic behaviour. Here again, we have to admit to speculation because of uncertainty about our conclusions. Experience of genetical analysis in other organisms has shown that the absence of strong non-additive components of genetic variation is characteristically associated with traits which are not subject to strong uniform directional selection. That is, the kind of genetical variation we observe for P^* may be what we have learnt to associate with stabilizing selection, i.e. natural selection tends to favour individuals of intermediate predisposition to P^* rather than those of extremely high or low "psychotic" predisposition. The precise circumstances under which polymorphism can be maintained under stabilizing selection are fairly restricted (Gale and Kearsey, 1968; Linney, 1972) requiring that most of the genetical variation be due to relatively few loci of fairly large individual effect.

There may be here the making of reconciliation between those who have approached the genetics of psychotic behaviour from the standpoint of major gene theory and those who have attempted to explain the variation in terms of continuous distribution of liability either by attempting to measure the underlying continuum as we have done, or by applying regression theory to tables of incidence in relatives (Gottesman and Shields, 1967) using a method developed by Falconer (1965).

One of the stumbling-blocks to explanations of schizophrenia in terms of a single polymorphic locus has been the need to account for the relatively high incidence of a disease which is apparently so debilitating in terms of reproductive fitness (see e.g. Slater and Cowie, 1971). Attempts have been made to explain such polymorphism in terms of heterozygous

advantage, which assumes that individuals who are intermediate with respect to certain expressions of the schizophrenic genotype are superior in fitness. We have suggested that our additive polygenic model may indicate that the trait is either selectively neutral, or subject to stabilizing selection in which case we are predicting superiority of intermediates as far as fitness is concerned. In this respect our model provides a similar heuristic to that of the single gene models since both yield similar predictions about the relationship between certain forms of psychopathology and fitness. Single gene models, however, have often been unsatisfactory since their proponents have often had to introduce either modifying genes or environmental factors to explain anomalies so that any dissimilarity between such models and the one we propose is greatly reduced. A reanalysis by Fulker (1973) using a form of the threshold method used by Gottesman and Shields of carefully chosen data relating to the incidence of schizophrenia in the relatives of schizophrenics suggested that the incidences in relatives of various degrees could be explained on the assumption that underlying continuous variation in liability to schizophrenia was due to the additive effects of polymorphic loci (D_R) and the specific within-family environmental effects such as those we have considered in our analysis (E_1).

Of course, there is a limited number of possible first approximations to the causes of variation for any trait, so we should not be unduly surprised that the different approaches to aspects of psychotic behaviour give fairly similar answers. Any marked inconsistency, however, would have suggested that the different traits and analyses were not comparable.

There is a sense in which our study of the genetics of P has failed to detect any striking features of the genetical system over and above the "genetical noise" that we find associated with any trait, be it human or otherwise. The model we have failed to reject is the most fundamental of all, and may merely represent the baseline against which other factors of greater biological, social or clinical interest ought to reveal themselves if they are present.

Our analysis of the EPQ for example, provides no evidence that the genotypic predisposition of one twin is in any way influencing environmentally the degree of "psychotic" behaviour demonstrated by the other. Thus, there is no evidence that an individual raised in the presence of a "psychotic" sibling is going to be any more or less "psychotic" than would be predicted purely from a knowledge of his genotype and the specific impersonal environmental experiences to which he is exposed. If there were a marked influence of this kind of one twin upon another we would expect the total variances of MZ and DZ twins to differ significantly and for our simple model to fail (Eaves, 1976a). In particular, if the predisposition of one twin towards "psychotic" behaviour tended to evoke extreme stability from the other and vice versa we would find that DZ twins were much less alike than might be expected on the basis of our D_R, E_1 model. Clearly this is not the case for the EPQ.

ANALYSIS OF A "COMPETITION" EFFECT

An analysis of other data, however, provides some evidence of such a competition effect for an earlier psychoticism scale, that of the PEN. As part of an early unpublished study of P, data were analysed on the responses of 708 pairs of like-sexed twins to the 80 item PEN questionnaire. It was found that this P scale had similar undesirable properties to those seen in the raw P score obtained from the EPQ in that the difference within MZ pairs increased markedly with the pair means. We discovered that a transformation of the raw scores to $\sqrt{P/n}$ ($n = 24$ in this case) removed most of this apparent genotype–environmental interaction so we analysed the transformed scores, much as we have done for those derived from replies to the EPQ. The mean squares of the analyses of variance of the transformed scores are given in Table 5. We found, firstly, that neither the E_1, E_2 model nor the D_R, E_1 model explained the mean squares for all the statistics simultaneously, neither did a model in which we fitted D_R, E_1 and E_2 to the mean squares for males and females jointly ($\chi^2_5 = 15.68, P \cong 0.008$).

It was easy to see why this was so. The variances of males and females differed substantially, and this meant we had to attempt separate explanations of the observations for each sex. This difficulty was paralleled by the psychometric observations that the sexes differed markedly in the characteristics of their responses to the P items of this early scale; this was one reason why the scale was reconstructed. From the view point of genetical "novelty value" this may not have been a fruitful decision. Because the interaction of P with sex appeared to reflect

Table 5. Analyses of variance of transformed P scores from PEN

Twin Type	Item	d.f.	mean square	F	P
Female monozygotic	Between pairs	330	0.0287	2.28	8×10^{-14}
	Within pairs	331	0.0126		
Male monozygotic	Between pairs	119	0.0206	2.31	3×10^{-6}
	Within pairs	120	0.0089		
Female dizygotic	Between pairs	197	0.0269	1.55	0.0012
	Within pairs	198	0.0174		
Male dizygotic	Between pairs	58	0.0176	0.90	0.65
	Within pairs	59	0.0195		

qualitative as well as scalar differences we chose to drop the unlike-sex pairs from the analysis and consider only like-sexed pairs.

We found that a D_R, E_1 model fitted the mean squares for female twins, ($\chi^2_2 = 0.59$, $P \cong 0.74$) but an E_1, E_2 model failed to do so ($\chi^2_2 = 7.41, P \cong 0.025$). For females we found

$$\hat{D}_R = 0.01692 \pm 0.00120,$$
$$\hat{E}_1 = 0.01279 \pm 0.00050,$$

which means that 40% of the total variation in females is apparently inherited. The E_1, E_2 model also failed to account for the variation in males ($\chi^2_2 = 8.45, P \cong 0.015$). Although the D_R, E_1 model just failed ($\chi^2_2 = 6.81$, $P \cong 0.033$), the deviations were small except for the variation within DZ pairs for which the observed value was much greater than expected. This is the kind of departure we would expect if there were a competition effect tending to enhance the phenotypic dissimilarity of unlike genotypes reared in the same family.

Retaining our simple additive model for gene action and our E_1 model for residual environmental effects, we can attempt to represent our competition effect by additional parameters which allow for the fact that the genotype of one individual is part of the developmentally significant environment of his sibling. We give such a model in Table 6. D_R is exactly as before, D_{RS} reflects the environmental effects of the genotype of one sib on the phenotype of another, and D'_{RS}, represents the covariation between the usual effects of alleles affecting psychoticism in the individual directly and those effects which contribute to the environment of an individual's sibling. If D'_{RS} is positive we have a situation in which psychoticism in one sib engenders psychoticism environmentally in another. We may call this a "cooperation" effect. If D'_{RS} is negative we have a situation in which psychoticism in one sib stimulates relative normality in another and vice versa. This may be called "competition".

Table 6. Modified model for twin data allowing for the contribution of competition due to the additive genetical deviations of co-twins†

Twin type	Item	Expected mean square
Monozygotic	Between pairs	$E_1 + D_R + D_{RS} + 2D'_{RS}$
	Within pairs	E_1
	Total variance	$E_1 + \frac{1}{2}D_R + \frac{1}{2}D_{RS} + D'_{RS}$
Dizygotic	Between pairs	$E_1 + \frac{3}{4}D_R + \frac{3}{4}D_{RS} + 1\frac{1}{2}D'_{RS}$
	Within pairs	$E_1 + \frac{1}{4}D_R + \frac{1}{4}D_{RS} - \frac{1}{2}D'_{RS}$
	Total variance	$E_1 + \frac{1}{2}D_R + \frac{1}{2}D_{RS} + \frac{1}{2}D'_{RS}$

†These expectations only apply to twins reared together.

In fact we find such a model fits very well the variation for P in males. The fit has indeed improved significantly over that of the two parameter models since the residual chisquare is now only $\chi^2_1 = 0.004$, ($P \cong 0.98$).

We are unable to separate D_R and D_{RS} analytically, so we have to represent them by a single component. Our estimates are:

$$\begin{aligned}(\hat{D}_R + \hat{D}_{RS}) &= 0.02712 \pm 0.00785,\\ \hat{D}'_{RS} &= -0.00773 \pm 0.00373,\\ \hat{E}_1 &= 0.00889 \pm 0.00114.\end{aligned}$$

All these parameters differ significantly from zero. The fact that $\hat{D}'_{RS}$ is negative and significant implies that there is a significant competition effect within male twinships which is affecting the pattern of variation in psychotic behaviour. If such an effect is real it is clearly of some interest, and even if not, our approach to its detection may have some value in the analysis of other traits and in the interpretation of other twin data. We have no way of identifying the mechanism of such competition. Its effect could be direct and relate to aspects of interpersonal relationships in the pair, or it may be indirect and reflect the relative inability of the "psychotic" to exploit the resources of the family, be they material or psychological, when he is in competition with a normal twin. The opposite aspect of the effect might also be expected. That is, a normal sib may be able to benefit more from the family resources because his relatively abnormal sib is unable to command a fair share by virtue of his abnormal behaviour.

Clearly, we have to view this explanation in context. It relates to one sex in one study, so we should not regard the matter as any more than suggestive of further dimensions in the quantitative analysis of behaviour which illustrate the generality and flexibility of our approach.

THE HERITABILITY OF PSYCHOTICISM

Since we have suggested that, for males at least, variation in the transformed scores from the PEN is affected by the competition arising by virtue of the twin situation we are unable to provide any estimate of heritability for the *P** scale of the PEN which is applicable to the population at large, although we *can* do for the *P** scale of the EPQ. As far as the PEN is concerned we may estimate the proportion of the total variation of male twins which has a genetical basis. For this purpose we include genetical variation due to all sources, whether due to the direct expression of an individual's genes or due to the environmental effects of those alleles possessed by his co-twin. We thus summarize the proportion of variation which is genetically determined in male MZ twins by

$$\tfrac{1}{2}(\hat{D}_R + 2\hat{D}'_{RS} + \hat{D}_{RS})/\tfrac{1}{2}(\hat{D}_R + \hat{D}'_{RS} + \hat{D}_{RS}) + \hat{E}_1 = 0.397.$$

This figure, which includes no correction for errors of measurement, only applies to individuals raised with a partner of identical genotype, i.e. to MZ twins. If we wish to consider the proportion of DZ twin variance which is due to genetic causes we have to recognize that the coefficient of the covariance term (D'_{RS}) is reduced which means that the apparent "heritability" will be increased as $\hat{D}'_{RS}$ is negative in our case.

For DZ twin pairs reared together we have:

$$\tfrac{1}{2}(\hat{D}_R + \hat{D}'_{RS} + \hat{D}_{RS})/\tfrac{1}{2}(\hat{D}_R + \hat{D}'_{RS} + \hat{D}_{RS}) + \hat{E}_1 = 0.522.$$

We may go further than this, and predict how much of the variation would have a genetical basis if we were to consider male pairs of unrelated individuals reared together. In this case there would be no contribution of D'_{RS} so the proportion of variation due to genetical factors is expected to be:

$$\tfrac{1}{2}(\hat{D}_R + \hat{D}_{RS})/\tfrac{1}{2}(\hat{D}_R + \hat{D}_{RS}) + \hat{E}_1 = 0.604.$$

In the absence of genotype–environment interaction we would expect the variance of singletons to be $\tfrac{1}{2}\hat{D}_R + \hat{E}_1$. However, our data do not permit us to separate the variation

ascribed to D_R from that included in D_{RS} so we are unable to produce a heritability appropriate for singletons, although it is expected to be less than that for unrelated individuals reared together.

As far as the EPQ is concerned, we have shown that our twin data are represented more economically by models involving the joint action of genes and environment than by models of similar complexity which are purely environmental. We have suggested that the trait is typical of many in that it displays no clear evidence of non-additive genetic variation or between family environmental effects, but we have exercized justifiable caution in interpreting our results. The advantage of our approach is that it leads to predictions about other kinds of relationships which can be used to test our first approximations derived from twin data.

We have also suggested that a scale of measurement can be found which is apparently free of any significant systematic genotype–environmental interaction, although we showed that this was certainly not the case for psychoticism as it is assessed by the raw test scores.

Since our model for variation in P^* as measured by the EPQ fits the data adequately we can adopt the view, at least provisionally, that any influences of the family environment such as are shared by individuals reared by the same parents are likely to be small relative to other causes of variation. Statistical considerations suggest that we would have roughly a 55% chance of detecting an E_2 effect which accounted for 25% of the total variation if the trait were, in fact, 25% heritable, with the sample sizes we had taken. If E_2 actually accounted for 40% of the variation we would be about 99% certain of detecting its presence. This means that we can assume, fairly confidently, that family environmental effects contribute little more than 25% of the variation in P, given that 25% of the variation in P was in fact genetically determined. Such considerations, of course, are inappropriate if we choose some more subtle explanation of the data, but in the end our model must be judged by its predictive power.

If we are right, the causes of psychoticism, and hence possibly of the variability underlying certain forms of psychopathology, are not going to be amenable to analysis in terms of "family history" when this is regarded in purely environmental terms. Our consideration of family history in this context extends to any features of the family environment which may be correlated with the genotype of the individuals studied.

If we are justified in disregarding E_2 we must also discount any environmental influences of, for example, the parental phenotype, since these are confounded with E_2 in twins reared together. Thus, the contribution of psychotic parents to psychoticism in their offspring may not be in the quality of the environment they provide by virtue of their psychoticism, but in the genes they transmit.

So much for environmental variation between families. What about that within families? According to our estimate of E_1 a little more than half of the variation in P reflects environmental differences between individuals within the same family. These arise, formally, from two sources; individuals' specific experiences and unreliability of measurement. The specific experiences which are relevant, of course, must remain the object of future research, but the fact that they relate to differences between individuals even in the same family suggests that they are likely to be of an accidental rather than of a social or cultural character. Again, the precise nature of the "accidents" remains to be established. They may be physical or behavioural. Clearly, there is scope within E_1 for the discovery of "traumatic" experiences, but any variation due to such experiences is expressed in addition to that already created by genetical polymorphism. Such may be an explanation for the view that certain traumata only induce psychotic behaviour in certain individuals. If we regard overt psychotic behaviour as a threshold character, with an underlying continuously variable trait it is easy to see how environmental accidents occurring to those predisposed genetically to be near the threshold of psychosis may be much more damaging than similar accidents occurring to those much further away from the theshold. Thus, although we choose to eliminate $G \times E$ interaction on our present scale of measurement, we should not be surprised if our trait translated into terms of clinical diagnoses and aetiology shows substantial genotype–environmental interaction, with psychotics being relatively more sensitive to environmental experiences than normals. In this respect, our original P scale, for all its faults, simulates what we might expect on the basis of some kind of threshold model of variation in psychoticism, since genotypes who on average produce high P scores, seem to show greater sensitivity to environmental differences.

Our estimate of E_1 however, does not only reflect the long term developmental consequences of specific experiences, it also reflects the short term fluctuations due to sampling which are unlikely to be explained in simple deterministic terms. We have thus an area of fundamental uncertainty which we can call unreliability of measurement.

Having measured psychoticism on the scale we have chosen, we are able to obtain from theoretical considerations alone a value for the sampling error to be expected on the basis of the model we have assumed for the raw responses to the items. In our case, having assumed that the model of independent rare events applies to responses to the P items, we expect the variance of $\sqrt{P/n}$ to be approximately $0.25/n$. Actually it is found (Bartlett, 1947) that this theoretical expectation is only good for values of P greater than about 9 on the original scale of measurement. The expected error variance for values of P between 1 and 9 are somewhat greater than this, although of course, the variance is zero when P is zero. By using a table of frequencies of the different P scores in conjunction with Bartlett's table of corresponding error variances and remembering to divide by the number of items, we have attempted to estimate as precisely as we are able the average error variance of the P scores in our sample. This value turns out to be 0.0108 for females and 0.0112 for males. Our weighted least squares estimate of E_1 was 0.0142, which includes the component due to errors of measurement. The mean squares within MZ pairs were 0.0141 and 0.0138 respectively, and we may use these in conjunction with our theoretical errors to give some idea of the likely contribution of potentially identifiable environmental factors to the variation in P within families. We may first obtain χ^2 test of the significance of such factors by dividing each of the within-pair *sums of squares* by the corresponding expected error variance. Such a χ^2 has the same d.f. as the sum of squares used in the calculation. We obtain, for females $\chi^2_{241} = 314.63$ ($P \cong 0.0005$) and for males $\chi^2_{79} = 97.34$ ($P \cong 0.079$). This suggests that an experiment which is sensitive enough can detect real environmental effects for P over and above the variation attributable to error, given that our model for the item responses is valid.

Bearing in mind the magnitude of our expected errors of measurement we may now summarize our view of the causes of variation in $\sqrt{P/n}$.

Our best estimates of D_R and E_1 are:

$$\hat{D}_R = 0.02723$$

and

$$\hat{E}_1 = 0.01419.$$

Taking the mean of our two estimates of sampling error to be that appropriate for our data we find the expected error variance is:

$$\sigma^2 = 0.011.$$

We may subtract σ^2 from E_1 to give our estimate of the variation due to "real" environmental differences within the family (E_1^*). This calculation yields:

$$\hat{E}_1^* = 0.00319.$$

Our estimate of the total variation is:

$$\tfrac{1}{2}\hat{D}_R + \hat{E}_1^* + \sigma^2 = 0.027805.$$

This means that $(0.00319 \times 100)/0.027805 \cong 11\%$ of the total variation of P^* can be clearly assigned to environmental causes over and above errors of measurement. Sampling variation, on the other hand, accounts for nearly 40% of the total variance. Our data give us little reason to assign the remaining 49% of the variation to anything other than the effects of several, perhaps many, genes of independent and additive effects.

If we are prepared to consider only those causes of variation which cannot be ascribed to sampling effects, then the relative contribution of genetical variation appears much greater since we must remove the contribution of σ^2 from our estimate of the total variation. Potentially identifiable environmental factors now account for 19% of the *reliable* variation in P^*, the

remaining 81% being due, apparently, to genetical variation. The last figure, 81% is our estimate of the heritability of P^*, after correction for unreliability of measurement. If our response model is appropriate we may wish to regard such unreliability as inherent in the trait we are measuring and thus prefer for most predictive purposes to work with the uncorrected figure of 49%.

The finding that by far the greater part of the environmental variation within families (in our case 77%) can be attributed to errors of measurement, is by no means new in quantitative genetics (see e.g. Falconer, 1960) although it may lack intuitive appeal for many clinicians. Students of man may not be persuaded by references to other organisms, but when the components of environmental variation have been analysed in detail it is often discovered that environmental variation between replicated genotypes reared together is very little greater than that between replicated structures on the same individual. Thus Caligari (personal communication) has demonstrated that variation for sternopleural chaetae number in *Drosophila melanogaster* is barely greater between individuals of the same genotype reared in the same culture than between different sides of the same fly. As far as variation in P is concerned it seems that the culture conditions within a human family are no less uniform than those in the typical Drosophila culture.

RELATIONSHIPS OF P TO OTHER PERSONALITY TRAITS

We have virtually exhausted all that we can say about the genetics of P by itself. It remains to consider the relationship of P to other behavioural traits. Two studies have now been undertaken, only one of which has so far been reported (Eaves and Eysenck, 1974).

In the light of our close examination of the P scale there are many ways in which we feel the earlier study leaves much to be desired because it was based on raw scores without any attempt to remove the obvious difficulties that these created. However, it is still instructive and does indicate something of the nature of the relationship between psychoticism and other behavioural traits.

Using the PEN and a social attitudes inventory (Eysenck 1951) we obtained scores on P, extraversion (E), neuroticism (N), radicalism (R), toughmindedness (T) and emphasis (Em) scales. The P scale used, it will be recalled, is that for which we found some evidence of competition when the males were subjected to closer scrutiny. We obtained the scores of our twins on the six traits and estimates of the mean squares and mean products between and within pairs of male and female MZ and DZ twins. We were unable to find a model which fitted males and females simultaneously; so we left out the opposite sex pairs and fitted D_R's and E_1's to all the variances and covariances at once, allowing the parameters to take different values in each sex. The results of this study, and a statement of our reservations, are given in our earlier paper (Eaves and Eysenck, 1974). As far as P is concerned, we found that there was significant covariation between the effects of genes which contributed to differences in P and those genetic effects which contributed to variation in T, Em and N. In no case were the estimated genetic correlations large, but they were large enough to be statistically significant. This early P scale also showed small but significant environmental correlation with T and E, confirming that all the environmental variation in this scale cannot be attributed to errors of measurement, otherwise we would expect no environmental covariation with other traits.

Of particular interest for our understanding of P is the genetic covariation with Em. At the superficial level the implication is itself interesting that more "psychotic" individuals are genetically disposed to be more emphatic in their attitudes. A finding which is significant about Em is the fact that extreme attitudes on most of the items of the inventory are endorsed relatively infrequently. In this respect the properties of the Em scale are likely to resemble those of P and may reflect tendencies to adopt rare, unpopular and possibly antisocial responses.

We have completed a similar analysis of the EPQ scores for P, E, N and for the Lie scale (L). We conducted a preliminary transformation of the raw scores in order to produce a scale on which the effects of genes and environment were additive. This necessitated an angular transformation of E, N and L. The square-root scores were taken for P as before. It was found that neither a D_R, E_1 model fitted the observed mean squares and mean products ($\chi^2_{80} = 112.9$,

$P \cong 0.009$) nor an E_2, E_1 model ($\chi^2_{80} = 184.4, P < 10^{-6}$). There might be two reasons for this. We might argue that the sexes differ somewhat in the causes of variation and covariation for the traits concerned. There seems to be some justification for this view at least for the lie scale. There may also be different patterns of determination for the different traits so that the same kinds of causes cannot be expected to explain all the variation. We found that a model which allowed D_R, E_1 and E_2 gave a greatly improved fit ($\chi^2_{70} = 88.3, P \cong 0.07$) though still the fit was not excellent.

We give the estimated parameters of this model in Table 7. The significance levels are approximate but are a useful guide to the importance of the various effects. As far as trait *variation* is concerned we find highly significant E_1 effects for all the traits, though we will recall that E_1 contains any variation due purely to errors of measurement. We also find significant additive genetical effects for every trait. Only L, however, shows any indication of a common environmental component above the contribution of genetical variation. A more detailed analysis of L will be published later. We are able to detect significant covariation between the within family environmental differences for P^* and E, and P^* and L, though not for E and L. E and N also show highly significant environmental covariation of this kind. The fact that we can detect significant environmental covariation between P^* and other traits confirms that not all the E_1 for P^* can be due to measurement error. We notice, however, that the environmental correlations (r_{E_1}) are still comparatively small ($r_{E_1} = 0.1404$ for P^* and E; $r_{E_1} = -0.1686$ for P^* and L) so we may conclude that only $100 \times (0.1404^2 + 0.1686^2) = 4.8\%$ of the environmental variation in P^* could be predicted from knowledge of the environmental deviations affecting E and L. This is quite consistent with the small excess of our estimate of E_1 for P^* over the value expected if the environmental differences within families were simply due to sampling variation.

Table 7. Estimates of genetical and environmental variation and covariation for transformed *P,E,N* and *L* scores from the EPQ

	$\hat{D}_R$				$\hat{E}_1$				$\hat{E}_2$			
	P	*E*	*N*	*L*	*P*	*E*	*N*	*L*	*P*	*E*	*N*	*L*
P	0.0228[b]	−0.0155	0.0038	−0.0082	0.0144[c]	0.0033[a]	0.0021	−0.0033[b]	0.0020	0.0071	0.0038	−0.005?
E		0.1169[c]	−0.0148	0.0217		0.0376[c]	−0.0104[c]	−0.0028		−0.0163	0.0012	−0.0116[a]
N			0.0999[c]	−0.0221			0.0416[c]	−0.0023			−0.0015	0.0014
L				0.0389[b]				0.0260[c]				0.0126[a]

[a]significant at the 0.05 level.
[b]significant at the 0.01 level.
[c]significant at the 0.001 level.

All the traits showed significant additive genetical components of variation but no pair of traits showed clear indication of genetical covariation. Only L showed any sign of E_2 variation, which subsequent detailed analyses showed to relate mainly to females. Covariation assigned to E_2 was also detected between E and L, even though we were unable to detect any significant E_2 variation for extraversion.

The absence of signficant D_R and E_2 covariance terms should not mislead us into thinking that there is no significant covariation between the traits apart from that arising on account of environmental difference within families. This is not the case. There is highly significant covariation between traits apart from E_1, but we are simply unable to decide whether, in general, it is due to genetical effects or common environments. An example will help in seeing this. Consider the traits P and L. Our models show significant within family environmental covariation for these traits, but neither the estimate of the genetical covariation parameter ($\hat{D}_{RPL} = -0.008161$) nor the common environmental covariation ($E_{2PL} = -0.005279$) is individually significant. We may, however, estimate the net covariation due either to D_R or E_2 effects from:

$$\hat{COV}_{PL} = \tfrac{1}{2}\hat{D}_{RPL} + \hat{E}_{2PL} = -0.009359.$$

The variance of $\hat{COV}_{PL}$ is thus:

$$V(\hat{COV}_{PL}) = \tfrac{1}{4} V_{\hat{D}_{RPL}} + V_{\hat{E}_{2PL}} + COV_{\hat{D}_{RPL}\hat{E}_{2PL}}.$$

Since the covariance of the estimates of D_{RPL} and E_{2PL} is quite large in relation to their variances we find that the variance of COV_{PL} is quite small and the estimate of the net covariance, due to the joint effects of genes and common environments is highly significant. No doubt similar calculations could be performed in relation to other pairs of traits.

Thus, our analysis of trait covariation, has left us in a slightly unsatisfactory position because we have been unable to distinguish between major genetical and environmental components of covariation. The fact that our genetical model gives a somewhat better fit to the data overall may lead us to prefer a genetical rather than an environmental explanation. It would, however, be undesirable merely to inspect the data and attempt to improve the fit of the model by inserting additional parameters to explain particular anomalies, so until a future study aids our attempts to explain the covariation between *P* and other personality traits, this must remain tentative.

CONCLUSION

As far as our data are concerned *P* represents a paradigm of certain basic principles of quantitative genetical analysis which are frequently forgotten in the genetical interpretation of behavioural differences. Firstly, we have seen a powerful extreme example of the relationship between analytical results and scale of measurement in the way in which genotype–environmental interaction was removed (along with sex interactions) by a choice of scale which took into account the most appropriate model for subjects' responses. Such interactions are virtually inbuilt in psychometric tests of this kind and are likely to make the process of prediction from such scales much more complicated than need be the case. There is nothing mysterious or dishonest in a choice of scale, it is merely that some are better than others and in the absence of any other objective criterion we can justify selecting that which yields the simplest analysis and predictions. If, at a later date, sound psychological or physiological justification can be found for the analysis of raw *P* scores then we must accept the attendant difficulties of genotype–environmental interaction and any other kind of complication generated by such a scale. Our approach to the analysis of *P* has shown how simple genetical criteria can help us to decide on our choice of scale. In this respect, our test of the simple D_R,E_1 model which is the baseline for all causal analyses of variation, is analogous to the scaling tests of classical biometrical genetics (Mather and Jinks, 1971).

Secondly, we have shown how the model fitting procedure is a powerful analytical device in psychogenetics, just as it has proved to be in other branches of quantitative genetics. For all the inadequacies of our data, we have been able to preclude some otherwise plausible interpretations of individual differences and suggest a simple theoretical framework for the analysis of *P* which can yield testable predictions.

Thirdly, our analysis of certain restricted subsets of the data, especially that of the Lie Scale and the *P* scale of the PEN have shown that we are not at a loss about how to proceed even when the simple models of classical quantitative genetics fail. Although we would not wish our *ad hoc* explanation of model failure to be taken too seriously, it stems from the general approach of biometrical genetics to continuous variation and, as such, can be cast in a form which can yield testable predictions beyond the confines of our restricted set of relationships (Eaves, 1976b). An attempt to model the structure of the environment itself in genetical terms might be a serious alternative to the purely empirical approach which characterizes present attempts to comprehend environmental variation.

Fourthly, our analysis provides a heuristic for studies of environmental influences. This is unlikely to be popular, because, as far as *P* is concerned, there are, apparently few which we can detect above the noise of sampling error and the segregation and recombination of genes having purely additive effects. Of course, some of the steps between genotype and final expression in the phenotype may be in the ability or inability of an individual to utilize or modify the environment in which he develops. Such steps, like any others in the developmental process

may be inherently modifiable though formally inseparable from genetical effects in our analysis.

Studies of the simple "survey" type are unlikely to yield fruitful results if steps are not taken to control for the genotypes of the individuals in the study. This implies a need for studies of environmental treatment variables which are firstly independent of genotype and secondly of sufficient extremity to be detectable above the variation due to sampling which is usually assigned to environmental factors. The studies of Record *et al.* (1969) illustrate the possibilities and the difficulties of careful research in this general area, with particular reference to aspects of cognitive ability.

Our demonstration that we do not need to invoke common environmental (E_2) effects, at least in our data, effectively precludes the view that there is any genetical polymorphism determining variation in the quality of environment provided by parents since such effects in our study would be confounded with E_2. We thus conclude that psychoticism in parents apparently makes little contribution to psychoticism in offspring over and above that to be expected on the basis of direct hereditary transmission.

Fifthly, the approach of quantitative genetics goes beyond a formal analysis of causes into the domain of population genetical theory in that it provides some substance for attempts to relate variability to selection. We have shown that the genetical variation we observe for P^* gives no suggestion of non-additivity, though we must admit the power of our test is unlikely to be great. This implies that there is unlikely to be any marked linear relation between pscyhoticism as it is assessed on our transformed scale, and fitness. Our preliminary analysis of the genetical system leads us to the provisional view that the relationship between P^* and fitness is either non-linear, such as might be associated with stabilizing selection, nor non-existent as we might expect if variation in P^* merely reflects the relatively trivial effects of genes whose primary contributions to genetic variation in fitness lie elsewhere. If we adopt the view that selection for P^* is stabilizing then we might regard the maintenance of extremely psychotic and extremely "super-ego" determined behaviour as a genetical consequence of maintaining some intermediate and presumably adaptive level of psychoticism in the population. If, on the other hand, we adopt the view that variation in P^* is adaptively neutral (a view which would not find favour with many geneticists) then we must regard the psychotic continuum as the chance byproduct of genetical variation whose primary effect on the phenotype (from the standpoint of fitness) is expressed elsewhere.

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