Inferring the Causes of Human Variation

(with Discussion)

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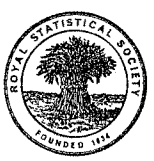

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Inferring the Causes of Human Variation

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SUMMARY

Examples are given of the way in which simple models for variation can be fitted to data relating to human behaviour. Data for different traits are consistent with different explanations. Power calculations are presented in an attempt to quantify possible errors of inference.

Keywords: MODEL FITTING; BEHAVIOUR; GENETICS; TWINS; PEDIGREES; POWER; COVARIANCE; INTELLIGENCE; FACTOR ANALYSIS; PERSONALITY; ATTITUDES: MULTIVARIATE

1. INTRODUCTION

RECENT controversy about the causes of variation in human abilities has caught the attention of scientists who would normally be occupied with other problems and left many wondering whether methods exist which enable even the most superficial analysis of human behavioural differences to be conducted. This paper illustrates one approach to the resolution of the problem by reference to existing data relating to human behavioural traits as they have been analysed by the methods currently employed in biometrical genetics. There are other approaches, many of which do not rely on statistical inference. Some of these have been reviewed by Jinks and Fulker (1970) and compared with the approach of biometrical genetics.

Whenever geneticists have studied continuous variation in other organisms they have been forced to the conclusion that individual differences are the result of the effects of a great many genes, usually of quite small effect, distributed widely throughout the genome. It is the recognition of the cumulative and interactive properties of such polygenic loci which forms the basis of a quantitative and testable theory of human differences. The foundation of such a theory was laid by Fisher in 1918, but in response to new and more effective analysis of differences in a variety of species, biometrical genetics has extended in several further directions. Thus, for example, the properties of genotype–environment interaction, in which the expression of genes depends on the environment, or in which the effectiveness of the environment is under genetical control, have been examined in great detail. The capacity of genetical systems to modify the environment in which development takes place has been documented and theoretical models developed to further its analysis. The combination of a powerful quantitative theory of variation with the basic tools of statistical inference has provided an approach to individual differences which has been vindicated in a large number of technological and scientific applications from improving crop and antibiotic production to the understanding of the intricate relationship between genetical variation and evolution. Many of the recent theoretical developments in quantitative genetics are relevant to any attempt to interpret human variation.

2. PRINCIPLES OF BIOMETRICAL GENETICS

Although we may never know whether a particular explanation of individual differences is "true" we may, given a suitable experiment, decide whether it is false. Central to biometrical genetics is the concept of the "scaling test" (Mather and Jinks, 1971). That is a test which would lead to the rejection of one explanation and favour the provisional adoption of another.

Before any adequate scaling test can be devised, the contribution of various types of effects to different statistics has to be expressed in the form of a model. Some of the simpler models will be illustrated subsequently.

Consider, for example, a large randomly mating population in which the expected frequencies of the three possible genotypes at a single dimorphic locus are given by the Hardy–Weinberg equilibrium. Let the frequency of the alleles A and a be u_a and v_a respectively. The frequencies of the genotypes are expected to be:

Genotype	*AA*	*Aa*	*aa*
Frequency	u_a^2	$2u_a v_a$	v_a^2

The effect of the three genotypes on the phenotype for a given trait may be expressed as a deviation from the mean of the two homozygous forms (*AA* and *aa*) thus:

Genotype	*AA*	*Aa*	*aa*
Effect	d_a	h_a	$-d_a$

The additive genetical deviation is denoted by d_a and the dominance deviation (that of the heterozygote, *Aa*, from the mean of the two homozygotes) is h_a. A positive dominance deviation implies that the allele which increases the expression of the trait is dominant. If h_a is zero there is said to be no dominance.

The effects of any other locus (B, b say) could be represented by corresponding frequencies u_b, v_b and genotypic effects d_b and h_b. In a large randomly mating population, in which there are many such loci segregating and acting independently, we may define parameters to represent the additive and dominance components of the variation due to the loci segregating in the population. Mather and Jinks (1971) define

$$D_R = 4\sum_a u_a v_a\{d_a+(v_a-u_a)h_a\}^2,$$

$$H_R = 16\sum_a u_a^2 v_a^2 h_a^2.$$

In the absence of dominance (i.e. if h_a is zero at every locus) H_R will be zero. In the presence of dominance, D_R will only reflect purely additive-gene effects if the allele frequencies are equal ($u_a = v_a = \frac{1}{2}$) at each locus involved in the expression of the trait. Providing we restrict our consideration to the variances and covariances of relatives in randomly mating populations, we can use these two parameters to represent the contribution of genetical additive and dominance effects to the variances and covariances.

Thus, it can be shown that the genetical contribution to the total phenotypic variance is $\sigma_g^2 = \frac{1}{2}D_R + \frac{1}{4}H_R$ (Mather and Jinks, 1971). Similar expectations can be derived for the genetical contribution to other statistics obtainable from natural populations. The treatment has been restricted to the simplest case. Random mating has been assumed and the higher order effects of the interaction between loci have been ignored. Two of the examples will consider the effects of assortative mating. It takes very large and careful experiments, even in ideal organisms, to detect epistatic effects. Although their biological significance may be substantial because certain types of epistasis together with directional dominance are associated with traits under directional selection, their actual contribution to the total variation for a trait is usually quite small. In human studies the effects of such interactions between loci are unlikely to be separable in practice from the non-additive effects due to dominance. The expectations also include no specification of environmental effects. These will be considered in relation to particular examples. In the initial treatment the effects of genes and environment may be regarded as independent. This does not imply, however, that the presence of genotype–environmental covariance cannot be detected, or that its presence would not have major significance for our understanding of the social and biological significance of particular traits. The

specification and interpretation of such covariation have been the subject of a detailed examination elsewhere (Eaves *et al.*, 1977). Genotype–environmental interaction, like interaction between loci, has not been included in the range of models to be discussed. The specification of $G \times E$ and the detection of some of its forms is possible in man (see, e.g., Jinks and Fulker, 1970; Eaves *et al.*, 1977). The various second-order effects are often ignored in the analysis of human differences. However, the consequences of these various effects can be represented in the form of a mathematical model and the extent to which they might be detected in different studies determined through a variety of power calculations. The biometrical genetical approach is also analytical so that we do not cross the many bridges of speculation until the effects of such factors are shown by a suitable scaling test to account for a significant proportion of the variation.

3. EXAMPLES

Before we can consider the many statistical problems which are encountered when we try to interpret individual differences it is necessary to look in some detail at several examples of the practical application of the model-fitting approach, as it is applied to the analysis of human behaviour. The examples have all been chosen from the many available to illustrate certain patterns of variation and to show how these might be interpreted. Most of the examples are drawn from twin studies because the twin design, though much maligned, is that which has still been the basis of research in this area until now. This is not to say that the twin study is ideal, nor that there is not a need for much more extensive data on other kinds of individuals. It is, however, a sad fact of life that even those who have used twins have rarely exploited their potential to the full.

3.1. *Example* 1: *Psychoticism*: *A Trait Consistent with the Simplest Model*

Eysenck has argued (e.g. 1952) that many of the behavioural disorders familiar to clinical psychologists are extreme manifestations of an underlying continuous distribution of personality differences. The Eysenck Personality Questionnaire (EPQ) attempts to summarize individual differences in personality by reference to three main constructs: Psychoticism (P), Extroversion (E) and Neuroticism (N). The first of these dimensions of personality is assessed in the EPQ by responses to questions relating to irrational fears, lack of sensitivity to others and disregard for social conventions. The P scores derived from the responses have a distribution which is virtually J shaped and display a marked dependence of mean and variance which is removed very effectively by a square root transformation.

In a recent study of twins, replies to the EPQ were obtained from a large number of twin volunteers in the London area. In Table 1 the data summary is presented in the form of

TABLE 1

Analyses of variance for psychoticism scores of twins

Twin type	*Item*	*Degrees of freedom*	*Mean square*
MZ_f	Between pairs	231	0·03269
	Within pairs	233	0·01380
MZ_m	Between pairs	68	0·04575
	Within pairs	70	0·01382
DZ_f	Between pairs	123	0·03881
	Within pairs	125	0·01912
DZ_m	Between pairs	45	0·02911
	Within pairs	47	0·01936
DZ_{mf}	Between pairs	66	0·03456
	Within pairs	67	0·02205

analyses of variance for each of five twin groups: male and female monozygotic twins (MZ_m and MZ_f); male, female and male–female dizygotic pairs (DZ_m, DZ_f and DZ_{mf}). The ages of the twin pairs were variable so the between-pairs sums of squares have all been corrected, with the loss of one degree of freedom (d.f.), for the linear regression of transformed P score on age. There is no suggestion that the regression on age is other than linear for these twins. Further, the effect corresponding to the significant mean difference between male and female twins has been extracted, again with the loss of one degree of freedom, from the sum of squares within DZ_{mf} pairs.

Our data summary consists of ten mean squares which are to be explained in terms of as few causal parameters as possible. No explanation involving only one parameter is admissible since there is clearly significant variation between pairs of twins. Furthermore, it is apparent that the variation within pairs of dizygotic twins exceeds that within monozygotic pairs. One possible explanation is the segregation of genes within *DZ* families. We explore the very simplest of all models for gene action, namely that which assumes all the genes are acting additively. Given such a model for gene action it is possible to determine the contributions of additive gene effects to the covariance of the different types of twins and hence their contribution to the components of variance between *MZ* and *DZ* twins. Similarly, the contribution of additive genetical effects to the variation within *DZ* pairs can be determined. Obviously there is expected to be no genetical variation within *MZ* pairs because members of a pair are genetically identical. The relative frequencies of the possible types of *MZ* and *DZ* pair, at a single locus A/a, are given in Table 2, together with the contribution that the locus

TABLE 2

The contribution of the additive effects of a single locus, A/a, to the phenotypic deviations of MZ and DZ twins

Pair type		*Frequency*		*Effect on phenotype*	
Twin 1	*Twin* 2	*MZ pairs*	*DZ pairs*	*Twin* 1	*Twin* 2
AA	AA	u_a^2	$u_a^4+u_a^3 v_a+\frac{1}{4}u_a^2 v_a^2$	d_a	d_a
AA	Aa	.	$u_a^3 v_a+\frac{1}{2}u_a^2 v_a^2$	d_a	.
AA	aa	.	$\frac{1}{4}u_a^2 v_a^2$	d_a	$-d_a$
Aa	AA	.	$u_a^3 v_a+\frac{1}{2}u_a^2 v_a^2$	.	d_a
Aa	Aa	$2u_a v_a$	$u_a^3 v+3u_a^2 v_a^2+u_a v_a^3$	.	.
Aa	aa	.	$\frac{1}{2}u_a^2 v_a^2+u_a v_a^3$	.	$-d_a$
aa	AA	.	$\frac{1}{4}u_a^2 v_a^2$	$-d_a$	d_a
aa	Aa	.	$\frac{1}{2}u_a^2 v_a^2+u_a v_a^3$	$-d_a$	.
aa	aa	v_a^2	$\frac{1}{4}u_a^2 v_a^2+u_a v_a^3+v_a^4$	$-d_a$	$-d_a$

makes to the phenotypic expression of the trait in the individual twins. The contribution of the locus to the covariance of *MZ* twins is $2u_a v_a d_a^2$. The locus contributes $u_a v_a d_a^2$ to the covariance of *DZ* twins, and the same to the variance within *DZ* pairs. If there are many such loci, all acting independently and additively, we may define their joint contribution to the covariance between and variances of relatives in terms of $D_R = 4\sum_a u_a v_a d_a^2$. This parameter is the expectation of the additive genetical variance component in the absence of dominance. Given random mating, the contributions of D_R to the various mean squares can be obtained from the expectations of the variance and covariance components simply by equating the within pairs mean squares to their corresponding expectations in terms of the variance components. The expected mean square between pairs is that within pairs plus twice the expected covariance between twins.

Since we have not specified any further types of gene effect (for example dominance) and assumed that gene action does not depend on sex or zygosity, the single parameter D_R is all that is necessary to represent the contribution of genetical differences to the mean squares, given our very simple model for gene action. These contributions are specified in the expectations given in Table 3. Some workers are uneasy about such a simple assumption

TABLE 3

Expectations of mean squares for MZ and DZ twin pairs, assuming additive gene action, random mating and no common environments

		Expected mean square	
Twin type	*Mean square*	D_R	E_1
MZ_f	Between pairs	1	1
	Within pairs	.	1
MZ_m	Between pairs	1	1
	Within pairs	.	1
DZ_f	Between pairs	$\frac{3}{4}$	1
	Within pairs	$\frac{1}{4}$	1
DZ_m	Between pairs	$\frac{3}{4}$	1
	Within pairs	$\frac{1}{4}$	1
DZ_{mf}	Between pairs	$\frac{3}{4}$	1
	Within pairs	$\frac{1}{4}$	1

for the genetical system. In fact, the effects of non-additive gene effects are small relative to the additive effects, except in those traits which display a marked linear relationship with reproductive fitness. In such cases other methods are likely to be more fruitful for the detection of the kinds of non-additivity produced by directional selection (see Jinks and Fulker, 1970; Eaves *et al.*, 1977).

No contribution of environmental factors has been proposed. We may distinguish two primary sources of environmental variation; that due to errors of measurement and the specific experiences of individual twins; and that due to the systematic environmental effects which are shared by members of a twin pair. The former will contribute to variation within pairs and the latter to variation between pairs, when the design does not include individuals who have been reared in separate families. We may use the symbol E_1 to denote the variation due to environmental differences within twin pairs and E_2 to denote the contribution of environmental variation between twin pairs. These two sources of environmental variation are of quite a different character, they may arise for quite different reasons, and may affect the organism in quite different ways. Thus the distinction between E_1 and E_2 is not merely formal it is likely also to be causal. Just by noticing that *MZ* twins are not exactly identical we can see that E_1 cannot be zero (though it could conceivably be due solely or substantially to errors of measurement). This does not follow for E_2 however. The presence of family environmental effects has to be inferred over the background of any genetical differences, particularly when our experimental design is restricted to twins reared together. The expectations of Table 3, therefore, include E_1 but not E_2 on the grounds that the effect of E_2 (and other more subtle effects) can only be inferred when the simple model fails to account for the observations. The next example will illustrate the detection of effects which could contribute to environmental differences between families.

The question now arises "Is this simple model adequate for the variation in psychoticism scores, and if so what are the best estimates of the parameters?" One way of attempting to answer the question is by fitting the model to the ten mean squares by the method of weighted

least squares. This is done iteratively allowing the weight matrix to be modified at each cycle to take account of the improved approximation to the amounts of information about the mean squares which can be obtained from each successive cycle.

Thus, writing A for the model matrix (10×2, in this case), x for the vector of ten observed mean squares $\hat{\theta}$ for the two-element vector of estimates of D_R and E_1, we solve

$$\hat{\theta} = (\mathbf{A}'\mathbf{W}\mathbf{A})^{-1}\mathbf{A}'\mathbf{W}\mathbf{x}$$

where W is the matrix of weights. Since the mean squares are independent, W is diagonal. Writing Ex_i for the expected value of the ith mean square we have $w_{ii} = N_i/2Ex_i^2$, N_i being the df for the ith mean square. The Ex_i are only known when the estimates have been obtained so we substitute the observed mean squares for their expected values and seek iterative refinement of the trial weights until satisfactory convergence is obtained for the Ex_i. The weighted sum of squared residuals is approximately χ^2 for 8 df, 2 df being accounted for in estimating D_R and E_1. In this instance the estimates were obtained using the GLIM package developed at Rothamsted (Nelder, 1975). The χ_8^2 was 7·04 indicating a close fit. The resulting estimates were:

$$\hat{D}_R = 0{\cdot}02455 \pm 0{\cdot}00277; \quad \hat{E}_1 = 0{\cdot}01391 \pm 0{\cdot}00104.$$

The fact that the model fits suggests that several sources of variation must be too small to be detectable with samples of this size. The twin data thus give no indication that mating is other than random and there is no suggestion of non-additive gene action. In biological terms this is the simplest system that can be envisaged. It could indicate that psychoticism, whatever may be its clinical significance, displays no obvious relationship to reproductive fitness. The trait could represent the phenotypic effects of loci whose main contribution to fitness lies elsewhere. The absence of any detectable effects due to the environment shared by twins suggests that cultural effects have little part to play in creating and maintaining differences in behaviour represented by the P scale. Obviously, these effects could still be present but remain undetected because the power of the experiment is low. Generally the result of power calculations (see, e.g., Eaves, 1972; Eaves and Jinks, 1972) is rather depressing. Some examples are given at the end of this paper.

The total variance in the population, after correction for the effects of age and sex is given by $\frac{1}{2}\hat{D}_R + \hat{E}_1 = 0{\cdot}026185$. The proportion of the total variance attributable to genetical factors is thus $\frac{1}{2}\hat{D}_R/(\frac{1}{2}\hat{D}_R + \hat{E}_1) = 0{\cdot}469$. This is the so-called and much maligned "heritability" estimate for the trait. If our model had failed, this calculation or any other calculation would not be legitimate. The remaining part of the variation is apparently due to the effects of measurement error and the individual experiences of the twins. The virtue of such models does not lie specifically in the estimation of heritability, but in the information they give about the significance of the traits under study and the areas in which research is likely to be successful. In the absence of demonstrable cultural effects, for example, it makes little sense to look to parents as a source of environmental treatments responsible for behavioural differences. Furthermore, the model can be used for predicting the outcome of further studies. Thus, we do not rest upon a model but seek to test it in other ways by looking at other kinds of family grouping. The results of this twin study suggest, for psychoticism at least, that any future findings are going to be very simple.

3.2. *Example 2: Conservatism–Radicalism: Culture or Mating System?*

The case of P suggested that the mating system and environmental differences between families made little contribution to variation. Twin pairs seemed to differ no more nor less than would be expected if mating were random and gene action were additive. Such a simple model will fit by no means every set of data. Indeed, if this were the case, we would begin to doubt either our capacity to design useful psychological tests or the power of our analytical methods.

Early research on social attitudes confirmed that attitudes were correlated across the population. Eysenck (1954) suggested that the variation in attitudes could be summarized by reference to two principal orthogonal factors which were called "conservatism" and "tendermindedness". The former involved endorsement of more "traditional" attitudes to morality and religion, less liberal approaches to the treatment of criminals, in fact the whole cluster of attitudes which might be called "conservative" with a small "c".

A recent study, one of three studies of conservatism which yield similar results, gave the mean squares in Table 4 for conservatism scores derived by extracting a general factor from

TABLE 4

Mean squares from analysis of conservatism scores of twins after correction for age and sex

Twin type	*Item*	*Degrees of freedom*	*Mean square*
MZ_f	Between pairs	231	1·242
	Within pairs	233	0·242
DZ_f	Between pairs	142	1·164
	Within pairs	144	0·363
MZ_m	Between pairs	81	1·106
	Within pairs	83	0·211
DZ_m	Between pairs	50	1·348
	Within pairs	52	0·359
DZ_{mf}	Between pairs	73	1·196
	Within pairs	74	0·398

68 items of a social attitudes questionnaire similar to that of Wilson (1975). As before, the mean squares have been corrected for the effects of age and sex, since there is a significant increase in conservatism with age. When an attempt is made to explain the observations by reference only to the additive genetical effects and within family environments (see Table 3) the model clearly was inadequate ($\chi^2_8 = 15{\cdot}62$, $0{\cdot}025 < P < 0{\cdot}05$) suggesting some alternative explanation is more appropriate.

At least one further parameter is required to account for the results. On common-sense grounds we might expect cultural effects to play a significant role in the determination of attitudes, so it is likely that the inclusion of a parameter to summarize the contribution of environmental differences between families could lead to a significant improvement in fit. There is, however, a further complication. Several authors (e.g. Wilson *et al.* 1972) have reported significant correlations between spouses for conservatism. Both cultural effects and assortative mating would contribute to variation between families of individuals reared together. In many respects the evolutionary consequences of cultural transmission and assortative mating are likely to be similar in the presence of genetical variation. Both increase the apparent genetical variation. Cultural transmission does this by perpetuating environmentally the consequences of genetical segregation in previous generations. Assortative mating does so by associating alleles of like effect. When our data are restricted to twins we can only ask whether the effects of mating system and culture are playing a jointly significant role in the determination of differences between families.

We may summarize the additional contribution of assortative mating and environmental differences between families in the parameter, B, which may be added to the covariance of twins. The expectations of the twin mean squares are thus modified as in Table 5. Our model assumes that there is still no non-additive genetical variation. Genotype–environment interaction effects will be confounded with our estimates of D_R, E_1 and B. Alternative parameterizations of the model are possible, but these do not lead to any predictions about the findings for other relationships.

TABLE 5

Expectations for twin mean squares allowing for joint effects of common environments and assortative mating

Twin type	*Mean square*	*Expected mean square*		
		D_R	E_1	B
MZ_f	Between pairs	1	1	2
	Within pairs	.	1	.
MZ_m	Between pairs	1	1	2
	Within pairs	.	1	.
DZ_f	Between pairs	$\frac{3}{4}$	1	2
	Within pairs	$\frac{1}{4}$	1	.
DZ_m	Between pairs	$\frac{3}{4}$	1	2
	Within pairs	$\frac{1}{4}$	1	.
DZ_{mf}	Between pairs	$\frac{3}{4}$	1	2
	Within pairs	$\frac{1}{4}$	1	.

The three-parameter model gives an excellent fit to the ten mean squares ($\chi^2_7 = 2{\cdot}51$) and represents a significant improvement over models which assume either no B or no contribution from D_R. The estimates of parameters were:

$$\hat{D}_R = 0{\cdot}5174 \pm 0{\cdot}1420, \quad \hat{E}_1 = 0{\cdot}2361 \pm 0{\cdot}0187, \quad \hat{B} = 0{\cdot}2600 \pm 0{\cdot}0678.$$

All three parameters differ significantly from zero, confirming that the data are consistent with an explanation in terms of genetical and cultural effects. The total variance is $\frac{1}{2}\hat{D}_R + \hat{E}_1 + \hat{B}$. The contribution of each source of variation to the total is thus:

$$\tfrac{1}{2}D_R = 0{\cdot}343, \quad E_1 = 0{\cdot}313, \quad B = 0{\cdot}344.$$

Each source of variation thus contributes about one-third to the total variation. If B is entirely environmental, as could be the case as a result of cultural effects or treatment differences between families, then 34·3 per cent of the total variation in conservatism is due to genetical effects. In passing it may be observed that two other analyses of conservatism, using different measures obtained at different times, give almost identical results to these for the relative contributions of the different sources of variation (Hewitt, 1974; Martin and Eysenck, 1976).

In order to resolve more completely the issue of assortative mating and cultural effects other data need be analysed, in particular data on adopted subjects. These have been collected but are still undergoing analysis. However, we could make some crude predictions on the basis of our data, in conjunction with figures reported elsewhere. Insel (Wilson *et al.*, 1972) reports many familial correlations for the Wilson conservatism scale. These are heterogeneous but an unweighted mean of three estimates of the correlation between spouses is 0·64. Other figures in the literature suggest this result is typical. The precise genetical and cultural consequences of the mating system will depend on the factors responsible for the inter-generational transfer of information. If the only influence of parents on their children is genetic, we may represent the genetical effect of assortative mating by the additional contribution it makes to the additive genetical variance as a result of the correlation produced between genes. Fisher showed this contribution to be $\frac{1}{2}(A/1-A)D_R$, where A represents the correlation between the additive genetical deviations of spouses. When parents only affect their children genetically, A can be estimated from the marital correlation, μ, and the

narrow heritability, h_n^2. The narrow heritability is the proportion of the total variation due to additive effects and is estimated from

$$\hat{h}_n^2 = \tfrac{1}{2}(1/1-A)\,\hat{D}_R/\hat{V}_T,$$

where $\hat{V}_T$ is the total variance. i.e. $\frac{1}{2}\hat{D}_R+\hat{E}_1+\hat{B}$, in terms of our current model for twins. When assortative mating is based on the phenotype for the measured trait (this is the possibility usually considered, though others are conceivable) we may obtain

$$A = h_n^2\mu.$$

Since we have estimates of D_R and V_T from the twins we can use these in conjunction with μ to obtain a quadratic in $\hat{A}$:

$$\hat{A}(1-\hat{A}) = 0{\cdot}64 \times 0{\cdot}343 = 0{\cdot}21952$$

Solutions for $\hat{A}$ are 0·3254, 0·6746. The larger root is greater than μ so our estimate of A is 0·3254. The contribution of assortative mating to the total variance is thus estimated to be $\frac{1}{2}(\hat{A}/1-\hat{A})\,\hat{D}_R$, which amounts to 16·1 per cent of the total. Subtracting this from the contribution of B to the total leaves a contribution of 18·3 per cent to environmental differences between families. All these later calculations depend on two main assumptions, one is that the equation $A = h_n^2\mu$ is an adequate model for the relationship between the correlation between spouses and their genetical correlation, the other is that environmental differences between families do not depend on the parental genotype for conservatism. Adding the estimated contribution of assortative mating to that which would still persist if mating were random we find that approximately 50·4 per cent of the variation in conservatism could be attributable to genetical factors. Quite clearly, other predictions might be made on the basis of this model. For example, if our assumption that the parental genotype is not affecting the offspring's environment is correct we would expect adopted individuals to be no more or less variable than individuals reared by their natural parents. If the model proposed is generally applicable we would be able to predict the similarity between parents and offspring, and between individuals and their more remote ancestors without having to introduce additional parameters into the model.

As far as can be discerned from the data available so far there is a clear indication that the causes of variation in conservatism are more complex than those inferred for psychoticism.

Of particular significance to biologists and psychologists is the finding that the genetical effect of the mating system and environmental differences between families, which could reflect the cultural impact of parents on children, are contributing significantly to individual differences. At first sight many of these findings seem opposed to common sense, but they suggest that the domain of social attitudes is one worthy not merely of sociological interest but also of the concern of biologists, because conservatism could become the first clearly documented case of the capacity of genetical differences in parents to express themselves in the environment they provide for their offspring.

3.3. *Example 3: Illustrating the Effect of Sibling Competition*

The two situations studied so far have given no reason to believe that the environmental differences responsible for the variation within and between pairs depend on the behaviour of the twins themselves. This example illustrates what might be found when the performance of one individual in a family is influenced environmentally by the behaviour of a sibling. The data concern a questionnaire study of attitudes to sex, and particularly with the responses of female twins to questions which relate to twins' assessment of their degree of sexual satisfaction. The twins were volunteers who completed the questionnaire anonymously. The response rate was low which could lead us to suspect sampling bias but the twins gave quite typical patterns of responses to other questionnaire data included in the study. Martin

and Eysenck (1976) calculated the mean squares for female twin pairs. These are given in Table 6. It was found (see Martin and Eysenck, 1976) that a simple model involving D_R and E_1 gave a relatively poor account of the variation even with these relatively small samples, ($\chi^2_2 = 4{\cdot}91, 0{\cdot}05 < P < 0{\cdot}10$). Simple attempts to explain the failure of the model by reference

TABLE 6

Mean squares obtained from analysis of sexual satisfaction scores in female twins

Twin type	*Item*	*Degrees of freedom*	*Mean square*
MZ	Between pairs	93	268·9809
	Within pairs	95	140·9404
DZ	Between pairs	52	240·9316
	Within pairs	54	274·6038

to the additional parameter introduced for conservatism produced an estimate of B which was almost significant and negative. Such a nonsense result suggests that the model was still inadequate. Attempts to reparameterize the model to take account of the effects of dominance still yielded nonsensical results so Martin and Eysenck resorted to explanation in terms of a model for sibling effects proposed by Eaves (1976). If there is genetical variation for a trait and the phenotype of one sibling can be influenced by the phenotype of another in the same family, a particular pattern of variation results which can be detected in the pattern of variances and covariances from twin studies and studies of other degrees of relatives. Eaves distinguished two situations on the basis of whether a high performance of one twin improved that of his co-twin ("co-operation") or led to a reduction in the performance of his co-twin ("competition"). In the latter case, the results would appear as a negative correlation between genes and environment which would lead to greater total variance in *DZ* twins, compared with *MZ* twins, and a reduction in the covariance of *DZ* twins even to the point where the *DZ* covariance might be negative.

The simplest form of the model is outlined in Table 7. Once more the possible types of twin pair are tabulated. The frequencies with which the pairs are expected to occur in a randomly mating population are those of Table 2. In this case, however, we not only specify

TABLE 7

The contribution of the additive effects of a single locus A/a in the presence of sibling effects

Type of pair		*Effect on phenotype*	
Twin 1	*Twin* 2	*Twin* 1	*Twin* 2
AA	*AA*	$d_a + d'_a$	$d_a + d'_a$
AA	*Aa*	d_a .	. d'_a
AA	*aa*	$d_a - d'_a$	$-d_a + d'_a$
Aa	*AA*	. d'_a	d_a .
Aa	*Aa*	. .	. .
Aa	*aa*	. $-d'_a$	$-d_a$.
aa	*AA*	$-d_a + d'_a$	$d_a - d'_a$
aa	*Aa*	$-d_a$.	. $-d'_a$
aa	*aa*	$-d_a - d'_a$	$-d_a - d'_a$

the effect of the alleles on the phenotypes of the individuals in whom they are expressed directly but, in addition, allow for each allele to have a corresponding environmental effect on the other twin in the pair. Thus, in a twin pair with individuals of genotypes AA and aa, the AA individual will receive a contribution d_a from his own genotype, but an additional, environmental contribution $-d'_a$ from his co-twin. The aa individual, reared with an AA sibling, however, will receive a total contribution of $-d_a+d'_a$ from the same locus. The particular example illustrates only the additive effects of genes. The model may be modified quite simply to include dominance deviations of the direct and environmental expressions of the genes (Eaves, 1976). Each individual's genotype now expresses itself in the performance of the individual and of his siblings. To the extent to which the same genes exert both direct and indirect environmental effects on behaviour there will be covariation between genotype and environment reflected in the cross-products of the direct and environmental effects of genetical differences. When the model is restricted to the purely additive effects of genes we may define:

$$D_R = 4\sum_a u_a v_a d_a^2 \quad \text{(as before)}, \quad D'_R = 4\sum_a u_a v_a d_a d'_a, \quad D''_R = 4\sum_a u_a v_a d'^2_a,$$

where D''_R represents the environmental effects of the genes on sibling phenotypes and D'_R represents the genotype-environmental covariance. If individuals are all reared at the same density (e.g. in twin or sibling pairs) then the effects of D_R and D''_R will be confounded in any expectations.

The contribution of D'_R, however, will depend on the genetical similarity between members of the pair and hence on whether the pairs are MZ or DZ twins, or indeed quite unrelated biologically. In Table 8 the contributions of D''_R and D'_R are added to those of D_R and E_1

TABLE 8

Expectations of mean squares on simplified sibling effects model

		Expected mean square		
Twin type	*Mean square*	$D_R+D''_R$	D'_R	E_1
MZ	Between pairs	1	2	1
	Within pairs	.	.	1
DZ	Between pairs	$\frac{3}{4}$	$1\frac{1}{2}$	1
	Within pairs	$\frac{1}{4}$	$-\frac{1}{2}$	1

to give the expected mean squares of identical and fraternal twins on the sibling effects model. Martin and Eysenck (1976) fitted this model to the data on sexual satisfaction and obtained the following estimates:

$$(\hat{D}_R+\hat{D}''_R) = 331{\cdot}8 \pm 118{\cdot}9, \quad \hat{E}_1 = 141{\cdot}1 \pm 20{\cdot}4, \quad \hat{D}'_R = -101{\cdot}2 \pm 55{\cdot}5.$$

The residual χ^2_1 falls to 0·005 suggesting a significant improvement in fit over that of the two-parameter model. The fit is perhaps too good to be true, which may reflect the inspection of the data which preceded the fitting of this model. Replication of this result is desirable but the estimates clearly indicate the principle of competition since the effect of genotype–environment covariance (D'_R) is clearly negative and approaches significance.

Although the sample and the data suggest that we should regard this as no more than an illustration, it shows how the biometrical–genetical approach detects objectively what would be difficult to detect in any other way, namely the effect of one individual on the performance of another. In this case it seems that twins are competing with one another for sources of

sexual satisfaction. A similar pattern of covariation between twins has been found for the time two-year-old male twins spend playing with their father (Martin, 1977).

The potential biological significance of our ability to detect sibling effects in practice relates to the emerging discipline of sociobiology. Several authors (e.g. Hamilton,1964) have argued that the mechanism of kin selection could be an important component in the evolution of social behaviour. That is, the fitness of a particular genotype depends both on the contribution the allele makes directly to the phenotype of the individual but also on the contribution that the allele makes to the survival of relatives. One prerequisite for kin selection may be the existence of genotype–environmental covariance for traits related to fitness. The absence of genotype–environment covariance could indicate that individual selection is usual.

It is important to stress that analyses of twin data which rest only on comparisons of correlations, as is still the case for the bulk of published twin studies, will not only miss many of the indications of competitive effects but be misleading their authors into a false, and unduly simple interpretation of the pattern of variation for the traits they study.

3.4. *Example 4: Sex Differences in the Determination of Tendermindedness*

An earlier example dealt with the determination of conservatism, the first of Eysenck's two principal dimensions of social attitudes. It is also recognized that a second dimension, which Eysenck terms "tendermindedness", is required to account for the covariation among social attitudes. The social attitudes questionnaire reported in Eysenck (1954) was administered in a postal study to a large sample of *MZ* and *DZ* twins. The expected factor structure was replicated in the twin sample (Hewitt *et al.*, 1977). Scores on the "tendermindedness" dimension were obtained and summarized by Hewitt (1974). The mean squares (which have not been corrected for age) are given in Table 9. An attempt to represent the ten statistics in terms of

TABLE 9

Mean squares for twin "tendermindedness" data

Twin type	*Item*	*Degrees of freedom*	*Mean square*
MZ_f	Between pairs	323	10·3697
	Within pairs	324	1·8643
MZ_m	Between pairs	141	7·8197
	Within pairs	142	3·1980
DZ_f	Between pairs	193	7·5213
	Within pairs	194	2·9982
DZ_m	Between pairs	36	6·5694
	Within pairs	37	3·3720
DZ_{mf}	Between pairs	126	8·1251
	Within pairs	126	4·3155

either of the first two simple models considered above failed, so Hewitt chose to exclude the opposite sex pairs from the analysis and found that male and female twin pairs were consistent with the D_R, E_1 model provided that different values for the parameters were allowed in each sex.

The data were thus consistent with a model in which the expression of genes and the effects of environment were dependent on sex. That is, there is underlying variation in tendermindedness, some mechanism of sex limitation mediated genetically or culturally. This finding is now explored further in relation to the opposite sex pairs which were omitted from the early analysis. In Table 10 a simple model is given for the additive effects of a single

TABLE 10

The additive effects of a single locus A/a on the phenotypes of twin pairs when gene effect depends on sex

Type of pair		*Male pairs*		*Female pairs*		*Male–female pairs*	
Twin 1	*Twin 2*	*Twin 1*	*Twin 2*	*Twin 1*	*Twin 2*	*Male twin*	*Female twin*
AA	*AA*	d_{ma}	d_{ma}	d_{fa}	d_{fa}	d_{ma}	d_{fa}
AA	*Aa*	d_{ma}	.	d_{fa}	.	d_{ma}	.
AA	*aa*	d_{ma}	$-d_{ma}$	d_{fa}	$-d_{fa}$	d_{ma}	$-d_{fa}$
Aa	*AA*	.	d_{ma}	.	d_{fa}	.	d_{fa}
Aa	*Aa*	.	.	.	.	.	.
Aa	*aa*	.	$-d_{ma}$	.	$-d_{fa}$	.	$-d_{fa}$
aa	*AA*	$-d_{ma}$	d_{ma}	$-d_{fa}$	d_{fa}	$-d_{ma}$	d_{fa}
aa	*Aa*	$-d_{ma}$	.	$-d_{fa}$	.	$-d_{ma}$	.
aa	*aa*	$-d_{ma}$	$-d_{ma}$	$-d_{fa}$	$-d_{fa}$	$-d_{ma}$	$-d_{fa}$

locus on like and unlike-sex twin pairs. The effect of the increasing homozygote AA is d_{ma} in males and d_{fa} in females. In like-sex pairs all the gene effects which are detectable in one sex will co-vary over pairs. We can thus define:

$$D_{Rm} = 4\sum_a u_a v_a d_{ma}^2 \quad \text{and} \quad D_{Rf} = 4\sum_a u_a v_a d_{fa}^2.$$

The model can easily be extended to include the effects of dominance. The expectations of mean squares of like-sex pairs will thus follow exactly those of the usual additive genetical model except that D_{Rm} will appear in the expectations for males and D_{Rf} in the expectations for females. The opposite sex pairs are slightly different since the covariance of opposite sex pairs will depend only on those loci which are expressed in both sexes. The covariance is thus $\frac{1}{4}D_{Rmf}$ where $D_{Rmf} = 4\sum_a u_a v_a d_{ma} d_{fa}$. The variance within male–female DZ pairs, on the other hand, will be $\frac{1}{4}D_{Rm} + \frac{1}{4}D_{Rf} - \frac{1}{4}D_{Rmf}$ after correction for the overall difference between sexes. In the case where the variation in males depends on the expression of quite different genes from those expressed in females we would expect D_{Rmf} to be zero, and for there to be no genetical covariation between members of unlike-sex twin pairs. In the other extreme, when the same genes have identical effects in males and females $D_{Rm} = D_{Rf} = D_{Rmf}$ and the genetical expectations reduce to those in Table 3. In Table 11 the expectations for the tendermindedness mean squares are given on the assumption that the genes constitute the only source of twin covariation and making allowance for the possible sex differences in the gene effects. Allowance is also made for the possible effects of sex on the environmental differences within pairs by including E_{1m} to denote the contribution of such effects to variation in males, and E_{1f} to denote the corresponding component in females. Using the ordinary approach of weighted least squares, as before, the estimates are:

$$\hat{D}_{Rm} = 4{\cdot}83 \pm 0{\cdot}92, \quad \hat{D}_{Rf} = 7{\cdot}93 \pm 0{\cdot}60, \quad \hat{D}_{Rmf} = 6{\cdot}46 \pm 1{\cdot}85,$$

$$\hat{E}_{1m} = 3{\cdot}16 \pm 0{\cdot}36, \quad \hat{E}_{1f} = 1{\cdot}79 \pm 0{\cdot}14.$$

At first glance these look very pleasing in that they suggest a considerable dependence of gene action on sex but imply that virtually the same genes are affecting both sexes since $D_{Rmf}/\sqrt{(D_{Rm} D_{Rmf})} = 1{\cdot}04$. This value slightly violates the constraint $D_{Rmf} \leqslant \sqrt{(D_{Rm} D_{Rf})}$ but this is not sufficient of itself to suggest there is much wrong with the model. The fit of the model is relatively good ($\chi_5^2 = 7{\cdot}29, P \simeq 0{\cdot}20$), but it still seems appropriate to ask whether there is further room for improvement.

TABLE 11

Expectations of mean squares for twin pairs when gene expression and within family environmental effect depend on sex

		Expected mean square				
Twin type	*Mean square*	D_{Rm}	D_{Rf}	D_{Rmf}	E_{1m}	E_{1f}
MZ_f	Between pairs	.	1	.	.	1
	Within pairs	.	.	.	.	1
MZ_m	Between pairs	1	.	.	1	.
	Within pairs	.	.	.	1	.
DZ_f	Between pairs	.	$\frac{3}{4}$	.	.	1
	Within pairs	.	$\frac{1}{4}$	.	.	1
DZ_m	Between pairs	$\frac{3}{4}$	.	.	1	.
	Within pairs	$\frac{1}{4}$	.	.	1	.
DZ_{mf}	Between pairs	$\frac{1}{4}$	$\frac{1}{4}$	$\frac{1}{4}$	$\frac{1}{2}$	$\frac{1}{2}$
	Within pairs	$\frac{1}{4}$	$\frac{1}{4}$	$-\frac{1}{4}$	$\frac{1}{2}$	$\frac{1}{2}$

Particularly, in view of the results obtained for the other dimension of social attitudes (see above) it is worth investigating the possibility of cultural influences (or assortative mating) on the trait. The analysis was repeated, including a parameter B, as in Example 2, to specify any additional effects which could be due to such factors. The contribution of B was simply added to all the expectations for the mean squares between pairs (c.f. Table 5). The extra parameter produced a marked improvement in fit (the new residual was $\chi^2_4 = 2{\cdot}58$) and led to a marked alteration in the interpretation of the data, as can be seen from the following estimates:

$$\hat{D}_{Rm} = 1{\cdot}94 \pm 1{\cdot}48, \quad \hat{D}_{Rf} = 5{\cdot}11 \pm 1{\cdot}22, \quad \hat{D}_{Rmf} = 0{\cdot}88 \pm 2{\cdot}92,$$

$$\hat{E}_{1m} = 3{\cdot}19 \pm 0{\cdot}36, \quad \hat{E}_{1f} = 1{\cdot}84 \pm 0{\cdot}14, \quad \hat{B} = 1{\cdot}36 \pm 0{\cdot}56.$$

The estimate of B is now significant, but the genetical parameters look very different. The genetical effects in males do not appear to be significant at all and, as we might expect, the estimate of D_{Rmf} has fallen sharply until it too no longer differs from zero. The principal characteristics of tendermindedness therefore seem to be those of a trait whose pattern of determination shows a marked interaction with sex. Both sexes seem to be subject to the effects of the family environment (as indicated by the significance of B) but when allowance is made for this, there is only indication of genetical variation in females. The genes appear not to be contributing at all to individual differences in males. This finding has been replicated by Martin (1977) in a second study of tendermindedness.

3.5. *Example 5: General and Specific Inherited Abilities—A Multivariate Approach*

Outside behavioural genetics most geneticists restrict themselves the analysis of one variable at a time. Whenever they have succumbed to the temptation of multivariate methods sound genetics has usually been obscured by inappropriate statistics. The standard methods of multivariate analysis—the various forms of factor analysis, canonical analysis and cluster analysis—do not help very much in the understanding of genetical problems because they usually let the statistical tail wag the genetical dog. This may reflect a widespread ignorance of multivariate techniques among geneticists but there have been few so-called genetical applications of multivariate methods which have won universal acclaim.

There are, however, circumstances especially in the analysis of human behaviour, when the analysis of multiple variables in the context of some joint psychological and genetical model

could improve our understanding of the traits that are measured. One instance is the need to test hypotheses about the causes of variation and covariation among multiple measures of ability.

Loehlin and Vandenberg (1968) published the covariances within and between *MZ* and *DZ* twin pairs for five of the Primary Mental Ability scales devised by Thurstone. These purport to assess different facets of cognitive function. In Table 12 we give the matrices of mean products within and between pairs for both types of twin as they can be derived from the results tabulated by the original authors. Martin and Eaves (1977) showed how hypotheses such as those we have already considered may be combined with hypotheses about the covariance structure of multiple variables to give greater insight about the causes of trait covariation. There is considerable evidence now accumulating that individual differences in ability are partly under genetical control. Various authors (e.g. Jinks and Fulker, 1970; Eaves, 1973, 1975; Rao *et al.*, 1975) have shown how the various available bodies of data are consistent with a model in which genetical factors play an important part in determining differences in cognitive performance. It has been shown that there is a substantial effect of assortative mating.

On the psychological side it is also well known that the different measures of ability are quite highly correlated with one another. Indeed, this finding is the basis of Spearman's classical model for general and specific abilities (Spearman, 1904), which implies that each separate ability stems from the superimposition of factors specific to that ability on a substrate of variation in a factor common to all abilities. The question now arises, "Do the genes and the environment affect all the traits in the same way or is the covariance of different abilities largely dependent on one source of variation?" Secondly, to what extent do the components of variation specific to the individual measurements depend on the segregation of genetical effects which have influences specific to the different traits? The twin data of Loehlin and Vandenberg provide an illustration of how this might be attempted.

Our basic model is now a model for mean products rather than simply for mean squares. Just as we wrote a genotype–environmental model for the variables considered singly above, so may we write an analogous model which specifies the genetical and environmental components of trait covariation. Bearing in mind the findings of more extensive studies of general intelligence, we propose to approximate the variation for these abilities by a model allowing for the additive effects of genes, within family environmental effects, and the joint effects of assortative mating and culture. The effect of genetical non-additivity, which several analyses have shown to be significant, is ignored in this study. Its contribution will lead to over-estimation of the additive genetical contribution and to the under-estimation of the effects of culture and assortative mating.

The model proposed therefore, is a multivariate extension of that found appropriate for the radicalism data above. We write expectations for the four observed matrices of mean products as follows:

$$\boldsymbol{\Sigma}_{BMZ} = \mathbf{D}+\mathbf{E}+2\mathbf{B}, \quad \boldsymbol{\Sigma}_{WMZ} = \mathbf{E}, \quad \boldsymbol{\Sigma}_{BDZ} = \tfrac{3}{4}\mathbf{D}+\mathbf{E}+2\mathbf{B}, \quad \boldsymbol{\Sigma}_{WDZ} = \tfrac{1}{4}\mathbf{D}+\mathbf{E};$$

the subscripts *B* and *W* noted between-pairs and within-pairs matrices respectively.

The matrix **D** represents the components of variance and covariance due to the additive effects of the genes. **E** represents the within family environmental covariances, and **B** denotes the effects on trait covariance of assortative mating and family environment. We could attempt to estimate all the elements of **D**, **E** and **B**, subject only to the constraint that the matrices of components should be positive definite. However, this would not make any use of our previous knowledge of the structure of abilities. It is appropriate in this case to express **D**, **E** and **B** in factorial form thus:

$$\mathbf{D} = \boldsymbol{\Delta}\boldsymbol{\Delta}' + \boldsymbol{\delta}^2, \quad \mathbf{E} = \mathbf{H}\mathbf{H}' + \boldsymbol{\eta}^2, \quad \mathbf{B} = \boldsymbol{\Gamma}\boldsymbol{\Gamma}' + \boldsymbol{\gamma}^2.$$

TABLE 12

Mean products within and between pairs for five primary mental abilities (data of Loehlin and Vandenberg, 1968)

	Monozygotic twins									
	Between pairs (122 d.f.)					*Within pairs* (123 d.f.)				
	Numerical	*Verbal*	*Spatial*	*Word fluency*	*Reasoning*	*Numerical*	*Verbal*	*Spatial*	*Word fluency*	*Reasoning*
Numerical	3603·41	1521·73	1449·47	889·60	1034·20	372·74	−1·48	1·36	−2·57	30·04
Verbal		2047·49	712·91	837·76	959·43		161·25	18·13	58·41	69·88
Spatial			4059·80	304·36	453·67			449·28	28·26	13·66
Word fluency				1161·32	497·75				196·90	43·60
Reasoning					937·22					126·89
	Dizygotic twins									
	Between pairs (74 d.f.)					*Within pairs* (75 d.f.)				
	Numerical	*Verbal*	*Spatial*	*Word fluency*	*Reasoning*	*Numerical*	*Verbal*	*Spatial*	*Word fluency*	*Reasoning*
Numerical	3943·48	2160·52	2248·69	1282·17	1425·28	1183·74	242·25	464·66	307·51	226·77
Verbal		2161·06	1683·94	1062·12	1199·44		325·00	111·14	198·69	132·65
Spatial			4595·79	877·62	1084·55			1110·50	478·80	114·34
Word fluency				1064·25	682·36				313·88	183·25
Reasoning					1064·74					177·84

TABLE 13

The multivariate analysis of five cognitive abilities in twins: parameter estimates and standard errors

	Genetical				*Within family environment*				*Assortative mating and cultural effects*			
	Loading		*Specific*		*Loading*		*Specific*		*Loading*		*Specific*	
Factor	*Estimate*	*Standard error*	*Estimate*	*Standard error*	*Estimate*	*Standard error*	*Estimate*	*Standard error*	*Estimate*	*Standard error*	*Estimate*	*Standard error*
Numerical	50·897	7·710	20·972	18·219	1·159	1·989	19·216	1·218	19·463	5·237	1·761	70·668
Verbal	16·830	4·461	17·114	4·167	10·201	1·525	7·821	1·746	26·662	3·132	2×10^{-6}	2×10^{7}
Spatial	31·027	6·352	37·647	8·339	2·123	2·132	21·094	1·346	11·918	4·887	27·504	5·664
Word fluency	14·686	3·433	22·926	4·552	6·580	1·376	12·706	0·918	12·453	2·434	1×10^{-5}	3×10^{6}
Reasoning	14·451	3·140	1·628	26·125	6·629	1·150	8·853	0·845	14·720	2·333	11·414	2·012

Following the model of Spearman we propose to test the hypothesis that **D**, **B** and **E** can each be explained by a (different) single common factor and corresponding sets of specifics. We do not, in the first case, propose any relationship between the elements of the different factors though this can be done (Martin and Eaves, 1977). With five traits, the initial model involves a total of thirty parameters. Five loadings and five specifics for each of the three causal components represented by **D**, **E** and **B**. The estimates may be obtained by maximum likelihood in the manner outlined by Jöreskog (1973). Writing $\mathbf{S}_i$ for the ith observed matrix of mean products, based on $N_i df$ and $\mathbf{\Sigma}_i$ for its corresponding expected value we obtain the maximum likelihood estimates of the parameters by minimizing

$$-\ln L = \tfrac{1}{2}\sum_i N_i[\ln|\mathbf{\Sigma}_i| + \mathrm{tr}(\mathbf{S}_i\mathbf{\Sigma}_i^{-1})].$$

The numerical analysis was implemented on the University of Manchester CDC7600 using the NAG library routine E04HAF to perform the minimization of $-\log L$ (Numerical Algorithms Group, 1976). Standard errors of the estimates were obtained by inversion of the information matrix evaluated for the maximum likelihood estimates (see Martin and Eaves, 1977). The estimates are given in Table 13. The adequacy of the model H_1 may be assessed, against the alternative hypothesis that each observed statistic must be represented by a separate parameter (H_0) by calculating twice the difference between the log-likelihood values obtained for the two hypotheses. This yielded $\chi^2_{30} = 33{\cdot}0$, $0{\cdot}25 < P < 0{\cdot}50$, indicating a good fit. Other explanations, including the attempt to reduce the model by attempting to represent the loadings of $\mathbf{\Delta}$ and $\mathbf{\Gamma}$ as simple scalar functions of one another, and by removing **B** altogether from the model led to a significant reduction in the quality of fit. This suggests that such simplifications are not justified by the data. Examination of the estimates and their standard errors in Table 13 suggests that some of the parameters are small compared with their standard errors. Unlike the factor loadings, which can take negative values, the specifics are constrained to be positive so it is difficult to know exactly what their expected values might be. It would appear, however, that the specific contributions of cultural effects and the mating system to numerical and verbal ability and to work fluency are small enough to be discounted on virtually any test. Similarly the first and last genetical specifics are small in relation to their standard errors so there would be some justification for allowing these to take zero values. All the specific components of **E**, however, are large in comparison with their standard errors. Similarly there is little justification for ignoring the contribution of either the genetical or the cultural factor to variation in any of the five tests since all the loadings are many times greater than their standard errors. Only the contribution of the within-family environmental loadings approaches zero in the case of the numerical and spatial aspects of cognitive performance.

The parameters of the model can be estimated once more, allowing the non-significant parameters to take zero values. The new estimates (Table 14) differ little from those of Table 13. Using these new estimates it is possible to estimate the proportional contribution of the different common and specific factors to the variation in the five tests of ability. The figures are given in Table 15. The most striking feature is the finding that the contribution of environmental differences within families is largely trait-specific. This is what might be expected if most of the environmental variation within families is due to errors of measurement, as has been shown in the past for cognitive measures. Any environmental differences within families seem also to have quite specific consequences for behavioural development. They appear to determine the particular profile of abilities manifest by individuals rather than an overall level of competence on the five tests. The chief point in introducing this example was not so much for the substantive findings, since any twin analysis of intelligence cannot do full justice to the known complexity of the causes of individual differences in intelligence, as to illustrate the ease with which the model fitting approach of biometrical genetics can be extended to the multivariate case. The analysis does, however, carry several implications for

TABLE 14

Multivariate analysis of cognitive abilities in twins: parameters of reduced model

	Genetical		*Within-family environment*		*Assortative mating and cultural effect*	
Factor	*Loading*	*Specific*	*Loading*	*Specific*	*Loading*	*Specific*
Numerical	56·142	—	—	19·343	17·768	—
Verbal	17·211	17·063	10·582	7·281	26·560	—
Spatial	28·914	39·003	—	21·202	12·990	27·316
Word fluency	14·279	23·016	6·481	12·795	12·534	—
Reasoning	14·263	—	6·410	9·052	14·761	11·527

TABLE 15

Summary of contributions to each of five ability measures of specific and common sources of variation

	Proportional contribution of factors					
	Genetical		*Within family environmental*		*Assortative mating and cultural effects*	
	Common	*Specific*	*Common*	*Specific*	*Common*	*Specific*
Numerical	0·696	—	—	0·165	0·139	—
Verbal	0·127	0·125	0·096	0·046	0·606	—
Spatial	0·164	0·299	—	0·177	0·066	0·294
Word fluency	0·140	0·363	0·058	0·224	0·215	—
Reasoning	0·177	—	0·071	0·142	0·369	0·231

our understanding of general intelligence. It suggests strongly that the basis of general intelligence, as opposed to any specific ability, is dependent on the diversity of effects of the same genes. Such a conclusion follows from the finding that it is the genetical factor of **D** which loads most consistently on all the variables. The joint effects of culture and the mating system share some of the generality of the genetical factors, although there is evidence against the view that culture and assortative mating contribute in the same way as other causes of variation to the profile of abilities assessed by these five traits. The fact that there is substantial variation expressed in the common genetical factor and not just in **B** rules out the possibility that the trait covariation was simply a secondary consequence of the mating system rather than a primary consequence of gene action.

On the whole geneticists have been sceptical about the methods of multivariate analysis because the standard approaches did not seem to yield any answers to the questions implied in the design of genetical experiments. The approach outlined above in the analysis of cognitive behaviour in twins is one which can be extended to the analysis of any situation in which the experimenter is able to formulate a model for both the causes and structure of variation in multiple traits. For this reason it should be more attractive to those engaged in genetical research.

3.6. *Example 6: Beyond Twins: Assortative Mating and Intelligence*

It would be a mistake to suppose that the analysis of individual differences can, or should, depend on twin data alone. Nor can the interpretation depend on any other single set of

relationships, for example parents and offspring. The results from any one study of a particular constellation of relatives have to be viewed against the background of all the other kinds of relationship which have been studied. Any model which is advanced has to be sufficiently general to encompass the bulk of the data. Any critique which is developed must be developed against the whole of the data and must propose an equally general alternative if it is to be a serious competitor for professional recognition. Finally, therefore, it is necessary to illustrate the analysis of data other than twins to show how these can be used to test general hypotheses about the causes of variation. It would be incautious not to indicate the reservations which may be felt about the quality of data in this area. There are many competent studies of intelligence which have concentrated on the collection of data on only one group of relatives, for example twins or parents and offspring. These give results which are remarkably consistent in the picture they give of variability in intelligence but there have been few systematic studies of cognitive behaviour which report data on a whole range of relatives for the same trait. Any joint analysis of these studies is open to the criticism that heterogeneous data have been pooled.

Reed and Reed (1965) attempted to study mental retardation by the examination of a large number of extensive pedigrees of mentally retarded individuals in Minnesota. Eaves (1973) gave the mean squares derived from an analysis of variance of 3,556 individuals from 53 such pedigrees. The data consisted of the scores on various I.Q. tests of individuals in the final generation of pedigrees whose ancestry could be traced backwards for five generations. The analysis of variance was thus able to identify the nested contributions of parents, grandparents, great-grandparents and great-great-grandparents to the variation observed in the last generation. The mean squares reported by Eaves are given in Table 16 together with their

TABLE 16

Analysis of variance of I.Q. data from pedigree study

Item	*Degrees of freedom*	*Mean square*	*Expected mean square*
Between great-great grandparents (*GGGP*)	52	971·8298	$\sigma_w^2+2{\cdot}9496\sigma_p^2+9{\cdot}0202\sigma_{gp}^2+25{\cdot}7406\sigma_{ggp}^2+66{\cdot}6369\sigma_{gggp}^2$
Within great-great grandparents : between great grandparents (*GGP*)	113	554·5149	$\sigma_w^2+2{\cdot}9548\sigma_p^2+7{\cdot}8901\sigma_{gp}^2+19{\cdot}3830\sigma_{ggp}^2$
Within great grandparents : between grandparents (*GP*)	401	308·5967	$\sigma_w^2+2{\cdot}3754\sigma_p^2+5{\cdot}4514\sigma_{gp}^2$
Within grandparents: between parents (*P*)	902	234·3704	$\sigma_w^2+2{\cdot}3436\sigma_p^2$
Within families (*W*)	2089	121·6629	σ_w^2

expectations in terms of the basic components of variance model. The coefficients reflect the considerable imbalance in the data due to the great inequality of numbers in different parts of the pedigree.

The task is to provide an explanation of these data in terms of a causal model. It had long been recognized that much of the available I.Q. data from other sources were consistent with an explanation partly in genetical terms so the first attempt was to explain the observations by reference to a model which assumed additive gene action and the absence of environmental differences between families. Because of the established finding that the intelligence scores of spouses were correlated it was decided to allow for the genetical consequences of assortative mating using the equilibrium expectations of Fisher (1918). Table 17 gives the expectations

TABLE 17

Expectations of variance components of pedigree study in terms of genotype–environmental model in presence of assortative mating

Component	*Expectation*
σ^2_{bggpp}	$\frac{1}{2}\left(\frac{1}{1-A}\right)\left(\frac{1+A}{2}\right)^7_{DR}$
σ^2_{ggp}	$\frac{1}{2}\left(\frac{1}{1-A}\right)\left(\frac{1+A}{2}\right)^5\left\{1-\left(\frac{1+A}{2}\right)^2\right\}_{DR}$
σ^2_{gp}	$\frac{1}{2}\left(\frac{1}{1-A}\right)\left(\frac{1+A}{2}\right)^3\left\{1-\left(\frac{1+A}{2}\right)^2\right\}_{DR}$
σ^2_{p}	$\frac{1}{2}\left(\frac{1}{1-A}\right)\left(\frac{1+A}{2}\right)\left\{1-\left(\frac{1+A}{2}\right)^2\right\}_{DR}$
σ^2_{w}	$\frac{1}{4}D_R+E_1$

for the five variance *components* in terms of this model. The parameters have been described already. It is important to notice that the expectations do not involve the correlation of spouses *phenotypes*, only the additive genetical correlation between spouses, A. We can ask later whether the observed value of A is consistent with that to be expected from the known marital correlation in the population. The model thus involves three parameters. Since the model is non-linear weighted least squares estimates of the parameters were obtained using an iterative approach to solve the non-linear minimization problem for a given set of weights and then to repeat the minimization for a new set of weights based on the estimated mean squares until a satisfactory minimum χ^2 was obtained. For the genotype–environmental model the minimum was $\chi^2_2 = 1{\cdot}19$, suggesting that the model fits quite well. The parameter estimates were $\hat{D}_R = 173{\cdot}06 \pm 25{\cdot}99$, $\hat{E}_1 = 78{\cdot}72 \pm 8{\cdot}39$, $\hat{A} = 0{\cdot}27 \pm 0{\cdot}07$.

The standard errors are approximate, obtained from the inverse of the approximate matrix of second derivatives of χ^2 with respect to the estimates. All the parameters are significantly different from zero. Of particular interest is the significance of A which implies that the genetical variation in intelligence cannot be described adequately without reference to the mating system. Indeed, Eaves (1973) showed how a model which assumed random mating failed to account for these data.

If the genetical consequences of assortative mating are a secondary result of the phenotypic correlation between spouses the value of A can be used to predict the phenotypic correlation, μ, from $A = h^2_n \mu$, where $h^2_n = \frac{1}{2}D_R(1-A)^{-1}/V_T = 0{\cdot}60$. The predicted marital correlation is thus 0·449. Reed and Reed report a marital correlation of 0·464 for their data, after correction for unreliability of measurement, which is very close to that predicted on the basis of the causal model. This ability of the model to predict another aspect of the data is a further basis for confidence in its usefulness and validity. The biological implication is that the mating system is leading to a significant correlation between the loci affecting I.Q. This increases the variability in the trait, relative to that expected for a randomly mating population. Apart from the effect that this has in maintaining a greater supply of individuals in the upper tail of the ability distribution, the mating system also increases the susceptibility of individual differences to the effects of natural selection, if the trait displays any consistent relationship with reproductive fitness.

Clearly, the fit of the simple genotype–environmental model is very satisfactory but it has been suggested that certain other (unspecified) environmental explanations might be no less suitable. An attempt may be made to verify this by fitting alternative models which do not

depend in any way on genetical differences but allow for the non-hereditary transmission of information between generations. Once the mathematical constraints of biological inheritance are removed there is a large number of possible environmental explanations which could be induced in an attempt to explain the data. One simple example, having much similarity of form to the model of Fisher already exploited for these data, is a path model which allows for the dependence of the similarity between siblings for I.Q. entirely on the phenotypes of parents for the same trait. We denote the path from parental I.Q. to offspring I.Q. by p. The correlation between siblings in terms of the model is thus $r_s = 2p^2(1+\mu)$, where μ denotes the phenotypic correlations of parents. The component of variance between sibships is thus $r_s V$, where V denotes the total phenotypic variance. The expectations of the other components are given in Table 18. The estimates obtained when this model is fitted are:

$$\hat{V} = 197{\cdot}05 \pm 5{\cdot}65, \quad \hat{p}^2 = 0{\cdot}09 \pm 0{\cdot}01, \quad \hat{\mu} = 1{\cdot}09 \pm 0{\cdot}25.$$

The χ^2 for testing the goodness of fit suggests formally that the fit is excellent ($\chi^2_2 = 1{\cdot}24$, $P \simeq 0{\cdot}50$) but the outstanding difficulty of this model is the improbably large value for the predicted marital correlation compared with the observed value. Therefore we are forced to conclude that the model fails to provide an adequate account of variation in intelligence, for these data. Taken by itself, this finding does not lead us to reject all purely environmental models for differences in IQ. It might, for example, be argued that the model for transmission is wrong. In particular it may be mistaken to suppose that cultural effects depend entirely on the phenotypes of the parents. A better fit to the empirical data might be obtained by postulating latent cultural factors. The correlation between the spouses of siblings may depend on factors other than the sibling and marital correlations. Further ingenuity might lead to an environmental model which fits these data *and* gives sensible parameter values, but the results reported here show that at least one plausible model for the cultural transmission of intelligence gives a nonsensical answer.

TABLE 18

Expectations of variance components on simple environmental model

Component	*Expectation*
σ^2_{bgggp}	$2p^8(1+\mu)^7\ V$
σ^2_{ggp}	$2p^6(1+\mu)^5\ \{1-p^2(1+\mu)^2\}\ V$
σ^2_{gp}	$2p^4(1+\mu)^3\ \{1-p^2(1+\mu)^2\}\ V$
σ^2_p	$2p^2(1+\mu)\ \{1-p^2(1+\mu)^2\}\ V$
σ^2_w	$\{1-2p^2(1+\mu)\}\ V$

4. DISCUSSION

The examples illustrate the practical possibility of using the model fitting approach to preclude certain plausible explanations of variation. The possibility of error cannot be ignored, however, and before concluding it is important to consider briefly the extent to which the results of a given study might lead to mistaken inference about the causes of variation. Several attempts have been made to consider the power of various experimental designs for the analysis of human variation (e.g. Eaves, 1972; Eaves and Jinks, 1972). More recently, Martin *et al.* (1977) have attempted a more general consideration of the statistical problems of inference in the twin study. Their approach is outlined here. The method is not completely general, in the sense that it is an approximation which would not work well in every conceivable situation, but it seems to give satisfactory results in the areas which are of practical relevance.

The approach is best considered by a simple example. Consider a study of variation in a particular trait in which equal numbers of monozygotic and dizygotic twins are sampled from a randomly mating population in which half the variation is due to the additive effects of many gene loci and the remainder due to the effects of environmental differences within families. For unit total variance we can thus write the population parameter values $\frac{1}{2}D_R = E_1 = 0{\cdot}5$. Using these values in conjunction with the expectations in Table 3 the expected mean squares for *MZ* and *DZ* twins can be obtained as:

Mean square	*Expected value*
Between *MZ* pairs	1·5
Within *MZ* pairs	0·5
Between *DZ* pairs	1·25
Within *DZ* pairs	0·75

Let **x** denote the vector of the four population values of the twin mean squares.

Suppose now that an investigator attempts to fit a model to mean squares derived by sampling *MZ* and *DZ* twins from this population. Not knowing the actual causes of variation, he mistakenly tries to fit a model which ignores the contribution of additive genetical effects (D_R), but specified in addition to E_1, a parameter (E_2) to represent the hypothesized contribution of environmental differences between families. The model representing the false hypothesis (H_F) is thus:

Mean square	*Expected mean square*	
	E_1	E_2
Between *MZ* pairs	1	2
Within *MZ* pairs	1	.
Between *DZ* pairs	1	2
Within *DZ* pairs	1	.

Let **A** denote the model matrix. The expected values of the false parameters of H_F are obtained iteratively by solution of:

$$\boldsymbol{\theta} = (\mathbf{A}'\mathbf{W}\mathbf{A})^{-1}\mathbf{A}'\mathbf{W}\mathbf{x}$$

The weight matrix, **W**, is calculated at each cycle using the parameters of H_F to provide the expected predicted values of the mean squares. Since **W** is a linear function of the total sample size an arbitrary number of pairs can be assumed to obtain $\boldsymbol{\theta}$, although the values in $\boldsymbol{\theta}$ will depend on the relative weight given to the two types of twin. With equal numbers of *MZ* and *DZ* twins we obtain $E_1' = 0{\cdot}625$ and $E_2' = 0{\cdot}375$. The primes denote the parameter estimates to be expected under the false hypothesis. These values may be substituted in $\mathbf{y} = \mathbf{A}\boldsymbol{\theta}$ to give the values of the predicted mean squares to be expected when fitting H_F to data derived from this population. In this case $\mathbf{y}' = (1{\cdot}375, 0{\cdot}625, 1{\cdot}375, 0{\cdot}625)$. Providing the ratios x_i/y_i do not depart too greatly from unity the scaler

$$\lambda' = (x-y)'W(x-y)$$

is the non-centrality parameter of a non-central χ^2 for 2 df. With 100 pairs divided equally between *MZ* and *DZ* the non-centrality parameter is 2.41.

The value of λ' can be used in conjunction with tables of non-central χ^2 to determine the sample sizes necessary to achieve a particular discrimination with a given degree of confidence, or to determine, for a given sample size, the probability of mistaken inference. Given the population values we have assumed for D_R and E_1, the total sample size required to be 95 per cent certain of rejecting H_F at the 5 per cent level is that necessary to ensure $\lambda' = 15{\cdot}443$, being the value of $\lambda_{(0{\cdot}05, 0{\cdot}95, 2)}$ tabulated by Pearson and Hartley (1972). The required sample

size is thus $(15{\cdot}443 \times 100)/2{\cdot}41$, or 604 pairs. In order to be even 50 per cent certain of rejecting H_F at the 5 per cent level a total of $(4{\cdot}957 \times 100)/2{\cdot}41 = 206$ pairs are required. These results are very similar to those obtained in other work on the power of studies of individual differences.

Because the reasoning of the above procedure was based, perhaps ashamedly, on intuition rather than on any sound analytical proof, we sought to verify our power calculations empirically by computer simulation. Using a package developed by Kearsey for the simulation of biometrical genetical experiments, Martin *et al.* similated 500 samples of 103*MZ* and 103*DZ* twin pairs from a randomly mating population with $\frac{1}{2}D_R = E_1 = 0{\cdot}5$. Under these circumstances it was expected that attempts to explain the data with reference to H_F would fail in 50 per cent of samples.

The 500 sets of data were summarized by analysis of variance to give the mean squares within and between pairs for the two types of twin. Then the E_1, E_2 model (H_F) was fitted by weighted least squares. The χ^2 test of goodness of fit exceeded the 5 per cent level of 5·991 in 243 out of 500 simulated experiments, which does not differ significantly from the 50 per cent expected ($\chi^2_1 = 0{\cdot}4$, $P > 0{\cdot}75$). Furthermore, the observed distribution of the simulated χ^2's can be compared with that expected for a non-central χ^2 for 2 d.f. with $\lambda = 4{\cdot}957$. These statistics (Table 19) show good agreement for mean, variance and skewness, although the

TABLE 19

Comparison of observed distribution of simulated non-central χ^2's with their expected distribution

Statistic	*Expectation*	*Expected value*	*Observed value*
Mean	$\nu+\lambda$	6·957	6·657 ± 0·205
Variance	$2(\nu+2\lambda)$	23·828	21·112
Skewness	$8\frac{(\nu+3\lambda)^2}{(\nu+2\lambda)^3}$	1·346	1·351
Kurtosis	$3+\frac{12(\nu+4\lambda)}{(\nu+2\lambda)^2}$	4·845	3·561

Note: $\nu = 2$, $\lambda = 4{\cdot}957$.

kurtosis suggests that slightly more extreme values occur than might be expected. These results give reasonable grounds for confidence that our approach to the problem of mistaken inference, if approximate, is not seriously misleading in practice.

This simulation study of what is a very realistic situation should make us cautious about the explanations we adopt on the basis of studies of modest dimensions. The example we have considered is of an attempt to falsify quite a crude set of assumptions when the degree of falsehood is quite substantial. Even in such circumstances the power of the test can be quite low. These considerations are ignored in most published analyses but they do not disappear. Indeed, we may suppose that the adoption of even less efficient statistics by many workers in this field can only make matters worse still.

Weighed against the consideration of simulation studies, however, there must be the practical observation that meaningful discrimination is possible even in twin studies. Quite clearly, from the examples discussed earlier, it can be seen that useful analysis can be conducted when the sample sizes are large enough.

5. CONCLUSION

There can be little advance in the statistical apparatus with which human variation is analysed until we have a basic grasp of the theoretical and practical framework within which

models for individual differences are developed. This paper is not an encylopaedia. It serves merely to introduce the kinds of consideration which occur daily in the analysis of differences in man.

Many aspects remain untouched. No consideration has been offered of the specification and detection of genetical non-additivity and genotype environmental interaction. Apart from the treatment of sibling effects, little mention was made of genotype–environmental covariance. The contribution of first and third degree statistics to the analysis of the genetical system and its interaction with the environment has not been discussed. This is not because the approach of biometrical genetics has nothing to say on these areas. Rather the reverse is true. Many of the earliest contributions to the analysis of such effects come from within the overall scope of biometrical genetics. These various factors have all been considered elsewhere in some detail (e.g. Jinks and Fulker, 1970; Eaves *et al.*, 1977).

The apparent weight given to twin data in the choice of examples does not arise out of any conviction that twins are definitive for this type of research. The simplicity of the twin design, however, makes it well suited to both for the task of illustration and for the initial stages of a research programme. All the principles and procedures which are here illustrated with reference to twins do not change with the collection of data on other kinds of relationship. The strength of the biometrical genetical approach is its provision of a systematic and positive framework within which a general understanding of human variation can be realized.

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DISCUSSION OF DR EAVES'S PAPER

Professor CEDRIC A. B. SMITH (University College London: This is an interesting and informative application of the methods of biometrical genetics to various problems of social, psychological interest. Biometrical genetics, as developed by Fisher, Mather and Jinks, extends Mendelian genetic theory to characters where the individual genes are not recognizable, and must be dealt with on a statistical rather than an individual basis. Social and psychological traits are presumably partly genetically determined, so their analysis is important from many points of view, but also presents formidable problems of theory and methodology. Dr Eaves shows courage in attempting an analysis, and disarming humility in recognizing the limitations. He has taken the point of view that, although the data are not all one could wish, they are about the best at present available. It would clearly be much better to obtain data on complete families. Also, in the case of intelligence and similar tests, there may be problems in sampling and in ensuring comparability between the scores for subjects of different ages or social strata, although twin data, as used here, may at least reduce these problems. Dr Eaves has clearly succeeded in showing that, as far as the available data go, some simple models combining genetic and environmental effects do adequately fit the data, whereas other apparently plausible ones do not.

However, one may speculate to what extent these very simple models would still prove adequate if further data were obtained. In the first place, the classical theory of biometrical genetics deals with crosses between inbred lines of experimental organisms, whose behaviour is closely controlled. Even so, several crosses (between different lines, between the hybrids themselves and with the parental lines) are usually taken into account in estimating the parameters (such as variance components). In contrast, human populations are heterogeneous and mate as they will. Hence it seems daring of Dr Eaves to attempt to reach substantial conclusions from nothing more than sets of human twins. Of course, twins have the valuable property that when the variance within pairs of (genetically alike) monozygotic twins is smaller than that within (genetically diverse) dizygotic twins, this almost certainly indicates genetic influence. (Inbreeding depression and linkage are other good indicators of genetic influence.) But precise quantitative deductions about the environmental components of variation need great care, particularly because twins share specially similar biological environments before birth, and social ones afterwards.

Recent studies of qualitative characters (such as inherited diseases) show that alternative explanations (simple Mendelian or polygenic inheritance or infection) can be distinguished only with great difficulty, even when much reliable information on large families is available. One might suspect by analogy that the same would hold in biometric genetics; indeed Dr Eaves's Example 4 (Section 3.4) seems to partially confirm this. Furthermore, any model of the kind used by Dr Eaves is almost certainly oversimplified. Certain effects (such as non-additivity or epistasis) are neglected because they are not statistically significant, though it is hardly plausible that they are strictly zero. This procedure of neglecting non-significant effects completely is often adopted

in a wide spectrum of statistical applications. It is difficult to feel very happy about it, though it is difficult to think of an alternative procedure which would be generally acceptable. (And, of course, almost all statistical and mathematical models are oversimplified.)

As an outsider, with no responsibility for collecting or analysing the data, I would also wonder about the correctness of some of Dr Eaves's interpretations. For example, some children copy their parents, other rebel. Could correlations in "conservatism" be due to reactions to parental attitudes as much as direct genetical influence? It is also not obvious why the matrices **D**, **E**, **B** should be expressible in the factorial form suggested ($\mathbf{D} = \Delta\Delta' + \delta^2$, etc.)

Finally, one might regard the title of the paper as too ambitious. Mendelian genetics (including biometrical genetics) shows the pattern of inheritance of characters. But other approaches (such as biochemical studies) give deeper understanding. Thus we now know in considerable detail how a biochemical blockage causes phenylketonuria (a kind of mental retardation and physical weakness), and hence how it can be cured or alleviated by suitable diet. The formal Mendelian fact that it is a simple recessive is considerably less useful. However, complete understanding requires an enormous amount of work and patience, and I would therefore like to propose the vote of thanks to Dr Eaves in congratulating him on how much information he has succeeded in extracting from limited data.

Professor R. N. CURNOW (University of Reading): I regret that, owing to illness, I was unable to attend the meeting and personally second the vote of thanks to Dr Eaves for his interesting paper. There were three main points that I wished to make.

First, there is the question of the representativeness of the samples of twins available for work of this kind. Availability is unlikely to be random in relation to the various expressions of human behaviour analysed in this paper. The assumption generally made that the twins are a random sample from an infinite population in Hardy–Weinberg equilibrium could be crucial to the interpretation of the statistical analyses. The problem of representativeness also applies with at least equal strength to the pedigreed population in Example 6 on assortative mating and intelligence. In addition to the problem of non-random samples, there is reason to question the assumption, implicit in ascribing Hardy–Weinberg values to the genotypic frequencies, that there is no selection, in terms of differential fertility and viability, operating on loci directly or indirectly affecting personality.

My second point concerns the fitting of models and, in particular, the interpretation of a good fit. Dr Eaves shows that very simple models often fit the data satisfactorily. The fact that more complex models do not improve the fit should not be used on its own to argue that the simple model is correct or even sufficiently accurate for some purpose or other. Dr Eaves appears to argue along these lines in, for example, his analysis of the data on psychoticism (Example 1) with its failure to include a term, E_2, for environmental differences between twin pairs. This approach relies far too much on the data and not enough on our knowledge (from evolutionary arguments, from studies of other traits and from a consideration of these particular traits) that there is almost certain to be considerable complexity in the form of genotype × environment interactions, genotype environment correlations, epistasis, sex effects and the non-genetic effects of zygosity. Similarly, I can find no explicit mention in the paper of the consequences of linkage or of the importance of the assumption of linkage equilibrium. The possibility of non-additive genetic effects cannot be dismissed, as attempted by Dr Eaves in his Example 1 on psychoticism, by saying that they only occur when there is a marked linear relationship between the trait and reproductive fitness. Our knowledge of the evolutionary and more recent history of behavioural traits is insufficient to argue that these effects are second order. A simple model may fit because, as Dr Eaves discusses, of the lack of power in the test and the data but also because important effects may cancel each other. This last comment also points to the dangers of interpreting established components of variance as due to one cause when it may be a mimicry of that cause by some other. The difficulties of seperating in humans the effects of cultural or social inheritance and of genetic inheritance are well known. Unfortunately, effects that are confounded with each other in a particular set of data do not necessarily have the same general consequences.

I would favour fitting models as complex as the data will logically allow and only omitting small terms if by so doing the estimates of the other parameters are not appreciably affected. Dr Eaves gives an example of the importance of the order of fitting when discussing the inclusion of a term for "cultural influences (or assortative mating)" in Example 4, the Tendermindedness

study. I would also want to attempt detailed studies of environmental or genetic correlates with differences between twin pairs and also with differences within twin pairs.

My third point concerns the purpose of studies of human inheritance and particularly the inheritance of human behaviour. To say that a simple model is good enough must imply that it is good enough for some purpose. What is the purpose of Dr Eaves's analyses? If it is to increase understanding then there is really no level at which we can justifiably ignore a complexity of causation which we know might well be present to a substantial degree. If the purpose is to lead to the formulation of social policy then we must be sure that complexities and parameter values that would make that policy detrimental are very unlikely. I do not believe that Dr Eaves's limited range of models nor the power of the tests he uses justify any firm conclusions about policy.

My final point is a minor one—but heartfelt! Please could we have some means as well as variances and covariances. Differences between means are important and the size of second-order statistics are difficult to interpret without some measure of average values.

I have been critical but this is because of the interest and importance of work of this kind. I hope that the Society will encourage more people, like Dr Eaves, to submit their analysis and interpretation of data to the scrutiny of this Society and hope that the possibility of critical comments will not discourage potential contributors of these papers.

The vote of thanks was passed by acclamation.

Dr A. VETTA (Oxford Polytechnic): Dr Eaves says that "whenever geneticists have studied continuous variation in other organisms they have been forced to the conclusion" that the polygenic hypothesis is necessary. This does not appear to be the case. Thompson (1975) is the latest in a long line of research workers who question the necessity of such a hypothesis. The argument is that a trait exhibiting continuous variation can be studied by assuming a few genes only. In this situation the theory given by Fisher (1918), particularly his theory of assortative mating which is used by Dr Eaves, will not be correct. Vetta (1976a) discusses a situation where the polygenic hypothesis, if it did not exist, will have to be invented. This, however, does not justify the claim made by Dr Eaves. It is also far from certain that the success, or rather a lack of it (Sheldon, 1963), in predicting the improvement in animal breeding experiments will not be equalled if only a few genes are assumed to determine the trait.

A minor question of terminology, d_a is *usually* regarded as additive deviation when $h_a=0$. Otherwise, the additive deviation for allele A, for example, is $u_a\, d_a+v_a\, h_a$.

Dr Eaves uses Fisher's (1918) model of assortative mating and refers to Wright's model rather briefly. The two models differ a great deal. In Table 5 he uses coefficients 0 and 2 for within and between pairs, respectively, for his column *B*. These coefficients are obviously appropriate for *MZ* twins. For *DZ* twins their use is a little difficult to justify. He has probably based himself on Fisher (1918) who adds all the increase in additive variance to the sib covariance and no part of it to sib variance. This is not correct (Vetta, 1976b). Assortative mating introduces association between phases of factors but cannot affect Mendelian segregation. Part of the increased variance should be reflected in sib covariance and a part in the mean sibship variance.

The main thrust of the paper is on fitting genetical models to mean squares. The basis for this exercise is the variance analysis invented by Fisher (1918). This is a type of local perturbation analysis and its use to study human population is open to serious criticisms (Feldman and Lewontin, 1975). One of the objections is that it ignores the norm of reactions of genotypes. A study of causes of human variation could be more useful if it led to isolation of factors, environmental or genetical, which affect a trait.

Anyone who wishes to fit a genetic model to a behavioural trait should, in my view, be required to say why an environmental model should not be fitted. Unless this type of self discipline is accepted we shall be in danger of being swamped with "genetical" explanations for traits where other explanations are more reasonable. For data on Psychoticism, for example, a simple environmental model would give coefficients similar to those in Table 4 but with different meanings. Such an "environmental" model will give an equally good fit. The moral is obvious, namely, model fitting does not assist us very much in understanding the *causes* of human variation.

It is probably common ground between Dr Eaves and myself that an understanding of genetics is essential if we wish to understand human behaviour. Perhaps we differ as to how this knowledge is to be used. My own preference is for isolating environmental and genetical factors which affect a

trait. Perhaps behaviour geneticists need to be reminded of a statement by Hirsch (1970). He says, 'I believe that in order to study behaviour we must understand genetics quite thoroughly. Then, and only then, can we . . . forget about it intelligently.'

Professor K. MATHER (University of Birmingham): First, arising from the contribution to the discussion by Dr Vetta; there is a widely held misapprehension that the type of analysis which Professor Jinks, Dr Eaves, myself and others endeavour to undertake on continuous variation carries implications about the number of genes in the system—indeed that it carries the implication that continuous variation requires a large number of genes to produce it. I can show Dr Vetta examples of continuous variation where there are no genetic differences at all, let alone many. This is a complete misapprehension. I have become rather tired of hearing it.

Secondly, with regard to the causation of continuous variation, it is possible with great labour in favourable material such as *Drosophila* to arrive at a minimum count of units which can be located moderately accurately in the chromosome, and which must therefore be genic in nature. With the two characters I know best this minimum is over 15, and I have no doubt that it could be increased considerably if one were prepared to devote the necessary labour to it. I do not know whether that would be regarded as polygenic—how poly is "poly"? I have no doubt either that a reasonable interpretation of quite a lot of data can be obtained by postulating six or eight genes. But why postulate any specific number of genes when the analysis does not require one to do so?

Furthermore, the units that are being inferred from the properties of polygenic systems are very seldom single genes. Almost inevitably they are linked complexes of genes—complexes that have been called effective factors. The confusion between genes and the effective factors into which they are so commonly associated in the experiments is about as widespread as the misinterpretation of continuous variation.

I hope that it will not be necessary to labour these points further because they are not basic to Dr Eaves's paper.

Professor Smith said that he had never known a selection experiment that led to a verifiable prediction. He cannot have read the literature on selection experiments. There are many in which theory tells us that something is to be expected—something which time and time again appears. For example, if we select for almost any character in almost any organism (and there is evidence from a number) it is known, initially from empirical observation but justified by subsequent theory, that we will get what are called correlated responses to selection. These arise sometimes from pleiotropic action of the genes, but much more often because, as I said earlier, the genes operate in linked combinations, which means that if we select for one set of genes they will drag along other genes linked to them. One of the commonest of these correlated responses is the reduction of fertility. It is in fact possible to obtain near sterility in this way.

Now quite ordinary basic genetical theory tells us that if there are linked combinations of genes, it will be possible given time and opportunity to resolve the linkages and with them the correlated responses. Whenever this has been tried it has happened, as theory predicts. If that is not verification I do not know what is.

Or at another level; the response reflects the nature of the selection being applied. Commonly the selection is straightforwardly directional, but other types have been used, notably the disruptive selection applied by Thoday and his colleagues. As was predicted they found that this results in the rise of switching genes leading to polymorphisms, with some evidence of the further expected build up of a genetic background enhancing the difference in the character being switched. Again a prediction both verifiable and verified.

I could go on, but this is enough. I have not been talking about Dr Eaves's paper, and for that reason it is not relevant to record my comments. But I cannot listen to such misrepresentations of the genetical aspects of the study of continuous variation with making some protest.

Professor J. L. JINKS (University of Birmingham): I can only reiterate what Professor Mather has said, namely that the remarks of Dr Vetta on the number of genes involved are irrelevant to the discussion of this paper. Our biometrical analyses do not presume any number of genes.

I also cannot accept that the evidence Dr Vetta quotes, suggesting that the numbers of genes are small, has any general relevance or indeed any relevance at all to continuous variation in a natural population of complex characters of the kind we are discussing. Over the years my colleagues

and I have estimated the number of genes involved in this kind of variation in a variety of organisms, mainly plants, and I can assure Dr Vetta that any estimate of the number is determined solely by the persistence of the experimenter and the precision of the experiment. Zero will be found if the experiment is conducted in a sloppy way, or 100 if the experiment is progressively refined so that increasingly small effects can be detected. I could quote many examples to show that the number of genes found is proportional to the patience and effort which the experimenter is willing to put into their detection.

Dr J. ROSTRON (North East London Polytechnic): I would like to take up Dr Eaves's comments on the multivariate approach. I have been working on this for the past few years and have recently completed a thesis on it. Dr Eaves's comment that "the standard methods of multivariate analysis . . . do not help much" is indeed true. I have sought linear combinations of phenotypic variables (human ridge counts) with specific genetic behaviours, such as having maximum parent–offspring correlation, or intra-family correlation. Since there are ten variates, with high inter-correlations and fairly high heritabilities, the major problems encountered are ill-conditioning, with non-positive definite matrices. The only useful method I found was to use factor analysis to reduce the dimensionality of the data from ten to two, and then to rotate the factors so that they had maximum (genetic) correlations between parent and offspring or intra-family correlation. This method is perhaps less rigorous than that of Dr Eaves, but is rather easier to perform and uses less computer time. It is not, of course, as is Dr Eaves's method, parameter-orientated.

Dr C. SMITH (Animal Breeding Research Organisation): The value of statistical analysis in inferring the causes of human variation has been recently queried (e.g. Feldman and Lewontin, 1975). It is thus encouraging to see quantitative human geneticists improving their tools and their data to tackle some of the criticisms and deficiencies of earlier analysis. The use of model or hypothesis testing and assessing the power of the tests used are two important tools, as shown by Dr Eaves and by workers in other schools (e.g. MacLean *et al.*, 1975).

The procedure adopted by Dr Eaves is first to test simple models, with non-rejection implying "provisional acceptance". However, choice of models may depend on the biases or ingenuity of the scientist. A model may be rejected because of lack of fit or because of unlikely (nonsense) parameter estimates, as judged by the experimenter? Unfortunately different models may lead to similar data sets (Kidd and Cavalli-Sforza, 1973) and discrimination may be difficult (Smith, 1971). Confirmation of a model and parameters across data sets may be reassuring but does not resolve the dilemma. Rejection of simple models leads to more complex models, these often being suggested by the form of the data, and so statistical tests may be spurious. Even though we know the situation is likely to be complex, scepticism (and the number of alternative models possible) grows as the complexity of the models increases.

In farm animals, with possible controlled experimentation, it is usually not possible to resolve the causes of variation but only describe and utilize its statistical properties. In human data, with possible biases in ascertainment, in measurement and in interactions with social and cultural factors (Cavalli-Sforza and Feldman, 1973), the resolution of causes of variation is more difficult and uncertain. Dr Eaves is perhaps not sufficiently critical of his data, of the assumption in his models or of his results when they fit his experience. For example, the biases due to unequal ascertainment of male and female twins in Tables 4 and 9 might be queried. Or the assumption of a constant level of assortative mating (A) over 5 generations in Table 17 seems dubious. A genetic model (Table 7) for competition between female co-twins for sexual satisfaction (with different spouses or partners!) might well be ruled out on commonsense grounds.

Scientists who are unsympathetic to Dr Eaves's aims and methods will find much to criticize and may remain unconvinced by these results. But in the end it will only be through the accumulation of sound data and through varied analyses such as these, that the field may move into an area of "beyond reasonable doubt" on the role of genetics in human variation.

The author replied later, in writing, as follows:

Many of the comments lack the specificity necessary to permit a detailed reply in limited space. My views, and those of other biometrical geneticists, have been expressed at length elsewhere (e.g. Jinks and Fulker, 1970; Mather and Jinks, 1971; Eaves *et al.*, 1977). I would have no one doubt, however, that I see the statement by Professor Curnow that "This approach relies too much on

the data" as a compliment rather than a criticism. In a discipline where prejudice and speculation thrive upon ignorance of the facts I make no apology for preferring data.

Would-be critics often appeal to the "almost certain complexity" of human variation. On what measurements is such certainty based? Biometrical genetics provides examples both of great complexity and relative simplicity. On the whole the models I have fitted to human data are somewhat more complex and the procedures more rigorous than those applied in other analyses of human differences. Even so, the level of complexity which need be assumed for a particular trait, human or otherwise, is decided neither by appeals to evolution nor by invocation of "common sense" but by the analysis of data. Anyone who points to the poverty of data or the uncertainty of statistical inference merely repeats what I have said in my paper and elsewhere (Eaves and Jinks, 1972; Martin *et al.*, 1977). Likewise, it is impossible to speak of the complexities of genotype–environment interaction and correlation without acknowledging the fundamental contribution of biometrical geneticists to their specification and practical analysis (see Eaves *et al.*, 1977, for a recent review). There is little to be gained from a pious rehearsal of doubt. There is everything to gain from the collection of more extensive and reliable data.

On a more specific matter, Professor Smith queries my formulation of the multivariate model for abilities. There are alternatives which I rejected because the factor model embodied the theoretical position first proposed by Spearman. I hoped that its application in this analysis would shed additional light on the cause of the communality between abilities.

I think, from one of Professor Curnow's comments, that my text was unclear since he has gained the impression that I used an *a priori* evolutionary argument to justify my simple model for psychoticism. Quite the contrary is true. Within the limitations of the design and power of the study the data yield little evidence of non-additivity in gene action. In the normal way, the evolutionary conclusion would follow from the data, not vice versa, that the effects of genes on psychoticism were not linearly related to their effects on fitness. He is, in my view, mistaken in his statement that I did not consider linkage disequilibrium. I expended much energy in the detection of the consequences of assortative mating, among which linkage disequilibrium is the foremost.

I can add little to the replies of Professors Mather and Jinks to Dr Vetta's misreading of the evidence about the polygenic basis of continuous variation. Recent work on fungi provides the most compelling evidence of what can be done when more refined analysis is possible.

As far as I know, Dr Vetta has never published any analyses which illustrate the comparatively trivial difference his postulated correction to Fisher's model produces in real data. Furthermore, I cannot resolve his apparent self-contradiction when he asserts, on the one hand, that Fisher was wrong in assigning to the covariance between siblings all of the variance due to assortative mating whilst maintaining, on the other, that assortative mating does not affect Mendelian segregation.

Dr Vetta is impressed by Feldman and Lewontin's reference to "local perturbation analysis". I am not. It is no more than mathematical obfuscation for the platitude that anything might happen if we change the *status quo*.

As far as environmental models are concerned, I have done as much as most geneticists to further the specification and testing of environmental models (Eaves, 1976, 1976a). Indeed, my paper gives one explicit example of an environmental model for familial similarity which can hardly have escaped Dr Vetta's notice. I do not know how he, or anyone else, can have any idea of what is "reasonable" except by some version of the exercise I have outlined.

Dr Rostron highlights some of the problems I encountered in trying to apply multivariate methods in genetics. He is correct in observing the enormous computer time required for the approach. This partly reflects my inefficiency as a programmer. The problems of non-positive definiteness can be overcome with careful parameterization of the model and by an appropriate choice of algorithm. One solution is the invocation of a penalty function to ensure that any search for a minimum never departs from a region in which all the eigenvalues of particular matrices are positive. This is messy but seems to work in practice. I certainly did not embark upon this approach for convenience or speed but because the methods which seek to maximize a particular correlation or variance do not help the geneticist who designs his study with a particular model in mind. It is possible that dermal ridge counts could be a candidate for specifying a structural model prior to the analysis. One of our applications involves ten variables and our difficulties are not numerical. Although our measurements are not as reliable as Dr Rostron's his problem may not be insuperable.

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