

GENERAL

Genetics and the development of social attitudes

WHATEVER the current norms might be with regard to public opinion about various issues, and whatever factors contribute to their change with time, attitudes are far from uniform. Eysenck¹ has shown how responses to a public opinion questionnaire could be resolved into two main factors: 'radicalism' (*R*) against 'conservatism' and 'toughmindedness' (*T*) against 'tendermindedness'. Individual differences in opinion are, to some degree, the social manifestation of individual differences in personality. Significant covariation has been detected between various measures of extraversion (*E*) and *T*. Wilson² has suggested personality correlates of *R*. Such relationships, together with available evidence for the genetic determination of personality differences (see, for example, ref. 3), justify a more penetrating analysis of the extent to which variation in opinion and personality share a common genetic basis.

We consider the causes of trait covariation within and between pairs of monozygotic (MZ) and dizygotic (DZ) twins. From the responses of 708 volunteer pairs of like-sexed twins to an 80-item Personality Inventory and to a 60-item Public Opinion Inventory, scores were derived on scales of psychoticism (*P*), extraversion (*E*), neuroticism (*N*), *R* and *T*, together with an emphasis (*Em*) score which indicated any tendency of subjects to adopt more extreme opinions. The questionnaires and the scoring keys used have been published elsewhere^{1,4}.

On the basis of replies to questions concerning childhood similarity, the twins were diagnosed as MZ or DZ (ref. 5). The pairs differed in their ages and in the time for which they had lived apart. Our analysis is based on the raw scores and the absence of an age correction will not severely affect the conclusions from a study which can only detect gross effects.

The MZ intrapair differences in personality were not related to the duration of separation, so we have pooled separated and unseparated twins. The composition of the sample by zygosity and sex is shown in Table 1, and it can be seen that it is not representative of zygosity or sex, although the twins are representative of the population with respect to variation for the traits in question. The data were summarised by the mean squares and mean products within and between pairs for every trait and group of twins.

Twin data alone are inadequate for a detailed genetic analysis, particularly when the twins were not properly separated⁶, so the structure of our data limited us to tests of comparatively simple hypotheses. We had to accept estimates of components which may be biased and tests which are not very powerful.

We wished to test, and hoped to choose between, two hypotheses of equal rank about the causes of variation and covariation for our six scales of personality and attitude. The hypotheses are: (1) that all the observed trait variation and covariation is attributable either to differences between the environmental influences shared by members of the same pair (E_2), or to differences in the environmental influences specific to individuals within the family (E_1); and (2) that all the variation and covariation is attributable either to the cumulative and additive effects of many loci for which the population is polymorphic (D_R), or to specific (E_1) environmental influences. We refer to the first hypothesis as the 'simple environmental hypothesis' and the second as the 'simple genetic hypothesis'.

For a particular pair of traits, *i* and *j*, we can write expectations for the mean products, in terms of the genetic and environmental covariance components for that pair of traits.

TABLE 1 Composition of the sample of twins (number of pairs)

Zygosity	Sex		Total
	Male	Female	
MZ	120	331	451
DZ	59	198	257
Total	179	529	708

These components are D_{Rij} , E_{1ij} and E_{2ij} , and they have been defined in detail and the restrictions on their interpretation discussed elsewhere¹. The expectations derived for both simple hypotheses are given in Table 2.

The coefficients of D_R are applicable to randomly mating populations. The slight assortative mating observed for personality variables is insufficient to cause a serious departure from these expectations, since the heritability of the traits is quite low. Insel⁸ reported correlations between spouses for the Wilson Conservatism Scale, finding values between 0.59 and 0.66. If this correlation had a large genetic component, its effect in our case, would be inseparable from E_2 and would contribute to failure of our simple genetic model. It is possible to obtain weighted least squares estimates of the parameters of either model⁷. A detailed analysis suggested that the genetic and environmental parameters might be inconsistent over sexes. At the risk of introducing some redundancy into our model, therefore, we allowed different values for our estimates in the two sexes and subsequently compared them. There are 168 raw statistics altogether, and we fit 84 parameters, leaving 84 d.f. for testing either model.

When the simple environmental hypothesis was tested we found that the residual $\chi_{84}^2 = 167.31$ ($P < 10^{-6}$) so we concluded that a model invoking only environmental factors fails to provide an adequate description of the variation and covariation of the six traits. For the simple genetic hypothesis, we found that the residual chi square is smaller ($\chi_{84}^2 = 111.60$, $P \approx 0.025$) and just significant. A model in which D_R , E_1 and E_2 were fitted jointly gave a very satisfactory fit ($\chi_{84}^2 = 47.98$, $P \approx 0.24$), suggesting that some E_2 parameters might be added to our simple genetic model. By this stage, however, there were many redundant parameters in the model and in the absence of any theory about which parameters could be deleted from the more complex model, we present the results of the simple genetic model, bearing in mind that some modification might still be appropriate (Table 3). The results were obtained by pooling estimates over sexes.

The estimates for the components of variance can be used to calculate heritabilities for the six traits from

$$\hat{h}^2 = \frac{1}{2} \hat{D}_R / (\frac{1}{2} \hat{D}_R + \hat{E}_1)$$

and provided we are justified in accepting our model, these estimates refer to the proportion of the population variance which is genetic in origin (Table 4).

The simple environmental model is insufficient to explain the observed variation and covariation of the traits in this study. We are, however, restricted by the structure of the data available. A larger or better designed study might identify common environmental influences in the departures from expectation which, in our study, appear as error. We

TABLE 2 Expectations of mean squares (mean products) on two simple models

Model Parameter	Expectation			
	Environmental E_{1ij}	Environmental E_{2ij}	Genetic D_{Rij}	Genetic E_{1ij}
Mean square/product				
Between MZ pairs	1	2	1	1
Within MZ pairs	1			1
Between DZ pairs	1	2	$\frac{3}{4}$	1
Within DZ pairs	1		$\frac{1}{4}$	1

TABLE 3 Estimates of parameters of a simple genetic model*

Scale	R	T	D_R (genetic)			E	N	R	T	E_1 (environmental)		
			Em	P						Em	P	E
R	12.39¶	-1.43	4.38§	0.81	-0.29	2.08	3.37¶	-1.19¶	1.37‡	0.02	0.03	0.19
T		†21.53¶	-4.19	2.11§	6.62¶	2.61		†9.18¶	-2.67§	0.71§	-0.32	0.06
Em			40.67¶	2.47‡	-0.82	2.85			34.76¶	0.44	1.65	1.73
P				3.19¶	0.48	1.80‡				2.92¶	0.67‡	0.05
E					17.61¶	-1.21					9.41¶	-0.13
N						22.04¶						11.25¶

* Values given after pooling over sexes. Where the estimates were heterogeneous over sexes they are marked †.

‡ Significant at the 0.05 level.

§ Significant at the 0.01 level.

¶ Significant at the 0.001 level.

can, however, be reasonably confident that some kind of genetic mechanism has to be invoked to account for the observations so far. That such a mechanism might involve the correlation of developmentally significant environmental factors with genetic variation should not be discounted. About half the variation between individuals for the six traits has a genetic basis (Table 4).

There are also some predictable significant covariance terms. Our study confirms the correlation between E and T, showing that it has a genetic, rather than an environmental basis, since only the D_R component is significant. There is also a significant genetic correlation between P and Em. This suggests that individuals who achieve high P scores share the strong conviction of being right, which is characteristic of some psychotic behaviour. The more significant tendency, for high P scores to be associated with 'toughmindedness' scores, is perhaps indicative of psychopathic tendencies which are known to be related genetically to psychosis and hence to P (ref. 9).

Although we claim to show some common basis for differences in personality and attitude, we stress that, for the scales in question, the greater part of the genetic and environmental variation is specific to particular traits. 'Radicalism' scores, for example, are totally independent of the measured personality variables, although they have significant associations with the other attitude measures.

Genotype-environmental interaction could inflate our estimates of both D_R and E_1 . Some kinds of correlation between G and E may be discounted because the fit of the D_R , E_1 , E_2 model implies that the total dispersions are homogeneous for the different kinds of twins. In many cases, however, genotype-environment correlation, if present, remains as a source of bias in our estimated genetic components.

Our model assumed that gene action is purely additive. Even without the complication of E_2 it would have been virtually impossible to identify any non-additive variation in our study. The bias in parameters estimated from twin studies in the presence of dominance is discussed elsewhere⁷.

Extrapolation from these results to other populations and cultures is likely to be misleading because they are almost certain to differ with respect to the relative importance of different determinants of variation.

If we restrict ourselves to the British population and contemporary culture, we expect, on the basis of our simple

TABLE 4 Heritability estimates for personality and social attitude scales

Scale	h ²
R	0.65
T	0.54*
Em	0.37
P	0.35
E	0.48
N	0.49

* Based on pooling heterogeneous estimates for two sexes.

genetic model, that attitudes in parents would be relatively poor predictors of attitudes in offspring. Parent-offspring correlations would be about $\frac{1}{2}ht$, given random mating, and thus to be roughly between 0.2 and 0.3. Many of the available familial correlations for attitudes (see for example, ref. 10) come from pre-war studies and show parent-child and sibling correlations which are much higher. The difference, if real, could reflect the consequences of cultural change during the last 30-40 yr. If individuality and mobility were not encouraged, common environmental (E_2) influences might play a significant part in maintaining the pattern of variation in attitude. In such a situation, parent-offspring correlations would resemble those for siblings and MZ twins. A culture which promotes individuality and mobility, however, may reveal a pattern of similarity between relatives which depends more obviously on genetic components and give greater significance to the environmental experiences which are unique to the individual, irrespective of his family environment. More recently, Insel⁸ has obtained correlations between parents and offspring for the Wilson Conservatism Scale which are still too large to be consistent with a simple genetic model, so we cannot regard the issue as settled. The sensitivity of attitudes to cultural change may, however, make them a useful indication of the interdependence of gene expression and culture and encourage further research in this subject.

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