HANS EYSENCK

Consensus and Controversy

EDITED BY

Sohan Modgil, PhD

Reader in Educational Research and Development
Brighton Polytechnic

AND

Celia Modgil, PhD

Senior Lecturer in Educational Psychology
London University

CONCLUDING CHAPTER

BY

Hans Eysenck
London University

The Falmer Press
A member of the Taylor & Francis Group
(Philadelphia and London)

USA The Falmer Press, Taylor & Francis Inc., 242 Cherry Street, Philadelphia, PA 19106-1906

UK The Falmer Press, Falmer House, Barcombe, Lewes, East Sussex, BN8 5DL

First published in 1986

Library of Congress Cataloging in Publication Data

Main entry under title:

Hans Eysenck: Consensus and Controversy.

(Falmer International Masterminds Challenge)

1. Psychology. 2. Social psychology. 3. Eysenck, H. J. (Hans Jurgen), 1916– . I. Modgil, Sohan. II. Modgil, Celia. III. Series.

BF121.H214 1986 150 86-11473

ISBN 1-85000-021-2

Jacket design by Caroline Archer

Typeset in 10/12 Times
by Imago Publishing Ltd., Thame, Oxon.

Printed in Great Britain by Taylor & Francis (Printers) Ltd, Rankine Road, Basingstoke, Hants.

Contents

therapy in this book. My role as host at the feast is to comment on some of the salient points of difference, and sometimes express my puzzlement at some of the features presented. For instance, what are we to make of the following comment by Lazarus? 'On any bookshelf the volumes that are dog-eared from frequent reference by clinicians in search of pragmatic leads are not likely to bear the Eysenckian imprimatur.' Obviously he has done much research on the state of the books on his colleagues' bookshelves, but perhaps this finding tells us something about the nature of his colleagues. It accords oddly with the findings of Rushton, Endler and others who have done scholarly research over the years as to the frequency with which psychologists' names are cited in the SCI and SSCI. I think that the latter indices are probably a better guide than the Lazarus Dog-Ear Test.

For those who like the fine bold style of writing that characterizes Eysenck's work, let me recommend the strong meat offered by Martin and Jardine. With the coming of these authors Eysenck must look to his stylistic laurels! They triumphantly present the results of their large Australian study involving 3810 pairs of adult twins, writing with enormous self-confidence and having no false modesty in choosing the words to express their satisfaction with their study:

> The single most astonishing finding from this very powerful study is the complete lack of evidence for the effect of shared environmental factors in shaping variation in personality, and their relatively minor contribution to variation in social attitudes.... The conclusion is now so strong that we must suspect those who continue to espouse theories of individual differences in personality which centre on family environment and cultural influences, of motives other than scientific.

I do not think that Eysenck would go as far as this last sentence in his writing, whatever he might think privately. But as Martin and Jardine advance in triumph, one may think of them caparisoned in purple and gold, like the cohorts of the Assyrian, as they descend in ferocity upon the cowering rabble of the hapless environmentalists. They admit that 'outsiders' like myself cannot be expected entirely to appreciate the simple glory of their banners—the thirty-three tables that support their victorious advance (and I must agree):

> It may be difficult for the outsider to the field to appreciate how strikingly good are the fits of our simple models when consideration is given to the power with which they are tested and the many opportunities for them to fail should the assumptions on which they are based be false.

It is a pity that this challenging chapter was not available to John Loehlin when he wrote the companion chapter, for here would have been something most impressive to get his teeth into, in addition to the studies with which he deals. I predict that this chapter of Martin and Jardine will be, above all others in this book, the one that will provoke most comment and controversy.

And where does Eysenck come into all this work on behaviour genetics? It is to be noted that Martin and Jardine entitle their chapter 'Eysenck's Contributions to Behaviour Genetics', which is quite modest of them as the bulk of the chapter concerns their own work, and they might merely have assigned to Eysenck a role similar to that of John the Baptist. In behaviour genetics Eysenck tends to be the second author—as in his partnership with Lindon Eaves and Martin, apart from his earlier studies with Prell. But both of these chapters give due credit to Eysenck for the role he has played in facilitating behaviour genetic research by others. John Loehlin concludes that:

> Perhaps if Eysenck did not believe so firmly in the high heritability of his personality dimensions, all this would not have come to pass. If so, we who are interested in behaviour genetics would indeed

Part III: Behavioural Genetics

3. Eysenck's Contributions to Behaviour Genetics

NICHOLAS MARTIN AND ROSEMARY JARDINE

Hans Eysenck has done more than anyone to promote the necessity for those interested in behaviour to take a serious interest in genetics. He has railed against the concept of the 'typical individual', arguing cogently that the best way to understand mechanisms is to study differences. This has long been recognized by geneticists. Thus, when Beadle and Ephrussi (1937) wished to understand the physiology of eye colour determination in *Drosophila*, they started with mutant individuals having eye colours different from normal (or 'wild type'). By crossing them in various configurations they were able to deduce the biochemical pathways responsible for eye colour. They later applied this paradigm to a much wider array of metabolic processes in the bread mould *Neurospora* (Beadle and Tatum, 1941), and in a short time others applied it to bacteria and their viruses. To this paradigm, which is but an extension of Mendel's experiments in his pea garden, can be attributed the scientific revolution which in only thirty years or so has revealed the structure of DNA, the mechanism of protein synthesis and now even the nucleotide sequences of genes responsible for major clinical disorders. Within two years or so, perhaps even by the time this book is published, we expect to know sequences for the genes responsible for Huntingdon's chorea and Duchenne's muscular dystrophy, an advance unimaginable even ten years ago.

The achievements of psychology and psychiatry in the same period can only be regarded as modest by comparison. Obviously the problems and the nature of the material are far less tractable than those to which geneticists have devoted their energies. But one cannot avoid the suspicion that it is the reluctance of many behavioural scientists either to analyze the causes of individual differences in the field, or to manipulate or control them in the laboratory, which is responsible for their discipline's indifferent performance in the post-war era. Too much sway has been held by those who have more allegiance to ideologies than to the scientific method. It is paradoxical that perhaps the greatest achievement of pre-war psychology, mental

testing theory, should have been subject to virulent and sustained attack in the past twenty years. Environmentalism untainted by biology has been the fashionable *Weltanschauung* during the lifetime of those aged less than forty. Many academics have preferred to engage in sterile semantic debates about 'whether IQ measures intelligence' or to advise governments on 'how to eliminate inequalities in educational achievement' than to undertake the more difficult tasks of measurement and openminded inquiry into the causes of individual differences.

In this bleak intellectual landscape perhaps no-one more than Eysenck has stood as vigorously against the tide of pop psychology and sociological pap. 'I have no faith in anything short of actual Measurement and the Rule of Three,' said Darwin, and neither has Eysenck, except perhaps that biometrical genetics might be added to the list. For, like Darwin, he has consistently been interested in the possibility that many of the observed differences in behaviour might be inherited and that from genetic studies might ultimately come an understanding of their physiological basis and their evolutionary significance.

Eysenck was early in the field with his own small twin studies of neuroticism and extraversion (Eysenck and Prell, 1951, 1956) which indicated that there was genetic variation for these personality traits. We shall not attempt to review his later contributions to the 'heritability of IQ debate' because these have been thoroughly aired elsewhere. It is arguably Eysenck's greatest contribution to behaviour genetics that he managed to interest professional geneticists, with backgrounds in plant and animal breeding, in the causes of variation in human behaviour. Most notably, Jinks and his students Eaves and Fulker started applying the methods of biometrical genetics to many of the measurements which Eysenck himself had developed (Jinks and Fulker, 1970; Eaves and Eysenck, 1974). The achievements of this synthesis have recently been summarized by Eaves and Young (1981) and by Fulker (1981). In the present chapter we report some new work, results of a study of personality and attitudes in 3810 pairs of twins, which owes its origins to Eysenck's earliest forays into the genetics of personality and which powerfully tests and vindicates his hypotheses.

PREVIOUS WORK ON THE CAUSES OF INDIVIDUAL DIFFERENCES IN PERSONALITY AND ATTITUDES

The pioneering twin study of Newman *et al.* (1937) is often cited as indicating the lack of importance of genetic factors in variation in personality. Others have pointed out that this conclusion is neither supported by the data nor in agreement with the results from more recent studies. Certainly there is evidence for a substantial genetic component in variation in extraversion (Eysenck and Prell, 1956; Shields, 1962; Eaves and Eysenck, 1975), psychoticism (Eaves and Eysenck, 1977), neuroticism (Eysenck and Prell, 1951; Shields, 1962; Eaves and Eysenck, 1976a) and lie (Martin and Eysenck, 1976).

In a study of 837 twin pairs by Eaves and Eysenck (1975), it was found that variation in extraversion could be explained by the additive action of genes and individual environmental differences. There was no evidence for the importance of family environment. This simple genetic model has also been found to be appropriate for explaining variation in psychoticism (Eaves and Eysenck, 1977) and lie (Martin and Eysenck, 1976). For neuroticism, a simple genetic model is again adequate (Eaves

and Eysenck, 1976a), although there is evidence that genetic differences in neuroticism become more pronounced with age (Eaves and Eysenck, 1976b).

In general, the results suggest that genetical variation in personality is mainly additive. The extensive data of Floderus-Myrhed *et al.* (1980), however, question the validity of an additive model for extraversion. Eaves and Young (1981) reanalyzed their data from 12,898 same-sex Swedish twin pairs and found that dominant gene action affects the expression of extraversion. Despite the difficulty in detecting dominance in twin studies (Martin *et al.*, 1978), with the number of twins available in this present study we have an opportunity to replicate this important finding.

While individual differences in the personality traits of extraversion, psychoticism, neuroticism and lie undoubtedly have a substantial genetic basis, the data on the genetics of the neurotic symptoms of anxiety and depression are much less clear. The dominant theories of causation have been overwhelmingly in the experiential domain, although Freud (1937) made it clear that to him the aetiology lay in the interaction of constitutional and experiential factors. A recent study of 587 pairs of twins found evidence for a substantial genetic component in both these symptoms (Eaves and Young, 1981). However, Torgersen (1983) in a study of 229 same-sex twins found evidence for a genetic component in neurosis only for male twins and for twins admitted to psychiatric hospitals. He has argued that different findings on the importance of genetic factors in the neuroses may be due to differences in sample selection. We hope to avoid some of the problems of sampling bias by conducting our study in a large sample free of the selection effects found in a treated population.

As with the neuroses, it is often assumed that individual differences in conservatism are due mainly to the socializing influence of the family (e.g., Feather, 1978). Indeed, Cavalli-Sforza *et al.* (1982) found in their analysis of the transmission of various traits that religious and political attitudes were mostly determined within the family. They discounted the suggestion that the transmission of these traits may have a genetic basis, despite the fact that it was not possible with their data to distinguish between cultural and biological inheritance. Certainly there is evidence from three independent twin studies (see Eaves *et al.*, 1978, for a summary) that genetic factors are a major source of variation in conservatism.

It is the aim of this present study to explore the extent to which different genetical and environmental sources of variation are important in determining variation in personality traits, neurotic symptoms and social attitudes. It is an opportunity to replicate and expand previous findings of personality traits and attitudes, as well as to clarify the role of genetic factors in the aetiology of neuroses.

THE TWIN SAMPLE

A questionnaire which included instruments for measuring personality and attitudes was mailed to all twins aged eighteen years and over who were enrolled on the Australian NH&MRC Twin Registry. Between November 1980 and March 1982 questionnaires were mailed to 5967 adult twin pairs throughout Australia, and, after one or two reminders to non-respondents, completed questionnaires were returned by both members of 3810 pairs, a 64 per cent pairwise response rate. With this response rate from an enrolment which is already voluntary and unsystematic, there is ample scope for bias from population frequencies. We shall compare, where possible, the

distribution of scores in this sample with those obtained in random samples in Australia.

Prior to mailing the questionnaire to the entire adult sample, a pilot questionnaire had been mailed to 100 pairs of adult twins in order to assess likely response rate and any problems in construction of the questionnaire. Completed responses were obtained from both members of sixty-five pairs, and thus the pilot predicted the total final response rate very accurately. Only minor changes were made to the final questionnaire as a result of problems observed in the pilot and perhaps because of this, only ninety-six responses from the original pilot sample of 200 were obtained when the final questionnaire was mailed some months later. However, we thus have ninety-six individuals who completed the entire questionnaire twice and whose duplicate responses have been used to assess the short-term repeatability of the various measures.

Diagnosis of the zygosity of same-sex pairs was based on their response to questions concerning their physical similarity and the frequency with which they were mistaken as children. If twins differed in their response to these items, they were asked to send recent photographs of themselves. This method of zygosity diagnosis has been found by other workers (Cederlöf *et al.*, 1961; Nichols and Bilbro, 1966; Martin and Martin, 1975; Kasriel and Eaves, 1976) to be about 95 per cent correct as judged against diagnosis based upon extensive typing, and this is approximately the same reliability as obtained by typing for the most common six or seven blood group polymorphisms. The sex, zygosity and age distribution of the twin sample is shown in Table 1.

Table 1. *Age, Sex and Zygosity Composition of the Sample*

	MZ Females	MZ Males	DZ Females	DZ Males	DZ Opposite-Sex
Number of pairs	1233	567	751	352	907
Mean age (years)	35.66	34.36	35.35	32.26	32.90
Standard deviation	14.27	14.02	14.27	13.88	13.85
Age range	18–88	18–79	18–84	18–83	18–79

TESTS

1 Delusions-Symptoms-States Inventory: Anxiety and Depression Scales (DSSI/sAD)

The DSSI/sAD (Bedford *et al.*, 1976) consists of seven state of anxiety and seven state of depression items. Each item is scored 0, 1, 2 or 3 according to the degree of distress claimed, e.g., none, a little, a lot or unbearably. The possible range of scores is 0–21 for both the anxiety and depression scales. This screening instrument was chosen because its reliability and validity have been established (Bedford and Foulds, 1977) and it is brief. Unlike other screening instruments, it provides separate scores for states of anxiety and depression. It had previously performed well in the course of an epidemiological study of neurosis and the social environment in Australia, proving

itself to be a high-threshold instrument for the detection of states of anxiety and depression in a general population (Henderson *et al.*, 1981): only 3 per cent of men and 3.5 per cent of women had scores of 7 or more for depression, and only 1.0 per cent and 5.6 per cent for anxiety. It has been used here as an appropriate instrument for measuring symptoms by self-report in a large postal survey.

2 Eysenck Personality Questionnaire (EPQ)

The EPQ (Eysenck and Eysenck, 1975) attempts to summarize individual differences in personality by reference to three main constructs: extraversion (E), psychoticism (P) and neuroticism (N), along with a fourth factor, the lie scale (L), which is a measure of social desirability or the tendency to 'fake good'. The scale consists of ninety items of the Yes/No type. The reliability and validity of the EPQ scales, and the relationship between experimental definitions of E, P, N and L and the behavioural ones given by the EPQ are discussed in Eysenck and Eysenck (1975).

3 Conservatism Scale (C-Scale)

The C-Scale (Wilson and Patterson, 1968) was developed to measure the general personality dimension of conservatism with specific reference to 'resistance to change'. The scale, slightly abbreviated for Australian use by Feather (1975), consists of fifty items concerning attitudes to such topics as the death penalty, birth control, church authority and white superiority. The twins were asked to indicate whether or not they agreed with an item by circling 'Yes', '?' or 'No'. Conservative responses score 2, equivocal responses 1 and radical responses 0 so that total conservatism scores could range from 0 to 100 in the direction of increasing conservatism.

METHODS FOR TESTING HYPOTHESES

The classical twin method is based upon the comparison of the degree of similarity of monozygotic (MZ) and dizygotic (DZ) twin pairs, and is the most common procedure for estimating the relative importance of genetic and environmental contributions to human individual differences. Any excess similarity of MZ over DZ twins is usually taken to indicate the presence of genetical factors producing variation in the trait concerned, and there have been numerous formulae suggested for estimating the proportion of variance due to genetical factors, the heritability. The inadequacies of such conventional analyses of twin data have been discussed in detail elsewhere (Jinks and Fulker, 1970). It suffices to say here that in the past ten years the advantages of a hypothesis testing approach to the investigation of the causes of individual differences over traditional formula estimates of heritability based upon untested assumptions have become apparent.

Several hypothesis testing approaches have been espoused, including path analysis of familial correlations (Rao *et al.*,1974), variance components analysis by maximum likelihood or weighted least squares, or pedigree analysis of raw scores from regular or irregular family structures (Eaves *et al.*, 1978). Each method has its

strengths and weaknesses, but one thing they all have in common is a superiority over classical methods which make no attempt to test basic assumptions, obtain maximum likelihood estimates, or compare objectively one model of trait variation against another. Here we use the procedure of variance components analysis.

This procedure has been described extensively in the literature (Eaves and Eysenck, 1975; Martin, 1975; Clark *et al.*, 1980), so only a brief account will be given. The starting point for an analysis of twin data is an analysis of variance which is used to compute the variation, measured as the meansquares, between and within twin pairs. These are calculated for each sex and zygosity group or five in all, including DZ opposite sex pairs.

From standard statistical and genetical theory we can then write expectations for these meansquares in terms of the following parameters or unknowns (Jinks and Fulker, 1970). E_1 is environmental variance within families, specific to the individual and shared with no-one else, not even members of the same family. It also includes measurement error. E_2 is environmental variation shared by cotwins but differing between twin pairs and will include cultural and parental treatment effects. V_A is the genetic variance due to the additive effects of genes in the absence of assortative mating (the tendency of like to marry like). Where there is assortative mating, the additive genetic variance between families is increased by an amount $V_A(A/1 - A)$, where A (Fisher's assortative mating parameter) is the correlation between the additive deviations of spouses and is related to the marital correlation μ (the correlation between husbands and their wives) by $h^2\mu$ (h^2 is the heritability). V_D is the genetic variance due to dominant gene action.

Collectively these expectations form a set of simultaneous equations known as a 'model' of variation and, for the parameters described above, this model is shown in Table 2. A standard procedure known as iterative weighted least squares is now used to estimate the parameters of the model. Providing that the observed meansquares are normally distributed (which they should be given the very large degrees of freedom in our sample), the parameter estimates are approximately maximum likelihood, and the fit of a given model can be tested by calculating the residual chisquare with $k - p$ degrees of freedom, where there are k observed meansquares and p parameter estimates.

Table 2. *Model for Meansquares of Twins Reared Together*

		E_1	E_2	V_A	V_D
MZ	Between	1	2	$2 + 2A/1 - A$	2
	Within	1	0	0	0
DZ	Between	1	2	$3/2 + 2A/1 - A$	5/4
	Within	1	0	1/2	3/4

In choosing the parameters we wish to estimate, we want to provide the most parsimonious description compatible with the data. Therefore a sensible hierarchy of models is as follows. First fit E_1 alone. Failure of this most simple model will indicate that there is significant between-families variation to be explained. A model including both E_1 and E_2 will test whether the between-families variation is entirely environ-

mental in origin, while the E_1V_A model will test whether the between-families variation is entirely genetic. If both two-parameter models fail, then models including all three sources of variation, either $E_1E_2V_A$ or $E_1V_AV_D$ may be tested. As the model matrix (Table 2) is not of full rank, a maximum of three parameters can be estimated, and all such three-parameter models will yield the same chisquare, the fourth degree of freedom simply testing the equality of MZ and DZ total variances.

The restriction to three parameter estimates means that we cannot test directly the relative importance of E_2 and V_D. Also, it should be noted that the coefficients of the extra additive variance due to assortative mating are the same as for E_2 and so they will be completely confounded. It is thus more appropriate to rename E_2 as B (for 'between-families variation') where

$$B = E_2 + V_A(A/(1 - A)).$$

Only if we have an estimate of the phenotypic marital correlation can we estimate A, and make some inference about the relative contributions of E_2 and the genetic variance due to assortative mating, to B.

The twin design is a poor one for the detection of dominance, but with the number of twin pairs available in the present study there was some chance that we would be able to detect its presence. Martin *et al.* (1978) showed that in the case of a trait with 90 per cent heritability, complete dominance and no assortative mating or E_2 (i.e., $B = 0$), 3330 twin pairs would be sufficient to detect dominance at the 5 per cent level with 95 per cent probability, and our sample size is somewhat larger than this. However, the number of twin pairs required rises to over 30,000 when there is only intermediate dominance. Even when significant estimates of V_D are obtained, it should be noted that the expectations for H_R and for additive x additive epistasis (I_R) are identical in MZ and DZ twins (Mather, 1974) and so are completely confounded. Thus when significant estimates of V_D are obtained, it should be remembered that these will include contributions from both sources of non-additive genetic variance.

As there is no necessary reason why the components of variation will be the same in both sexes, models are first fitted to the meansquares for males and females separately and then to all eight statistics combined. We can then calculate a heterogeneity chisquare for k *df* by adding the male and female chisquares, each for $4 - k$ *df*, and subtracting from the chisquare ($8 - k$ *df*) for the corresponding model fitted to all eight statistics. The heterogeneity chisquare for k *df* will indicate whether the same parameters are appropriate for both sexes. If it is not significant, then the DZ opposite-sex data may be added and the same model fitted to all ten statistics.

RESULTS

Scaling

In a genetic analysis it is most appropriate to choose a scale where there is no genotype-environment interaction so that genetic and environmental effects are additive. Jinks and Fulker (1970) have shown that in MZ twins the regression of absolute within-pair differences on pair sums provides a test for any systematic $G \times E_1$ interaction. Table 3 shows these regressions for MZ male and female twins for the raw scores and various transformations.

Table 3. *Proportions of Variance in Absolute Within-Pair Differences Accounted for by Regression on Pair Sums for the Raw Personality and Attitude Scores and Various Transformations*

		MZF		MZM	
		L	Q	L	Q
Anxiety	raw	.32***	.03***	.44***	.04***
	angle	.14***	.02***	.23***	.04***
	$\sqrt{x+1}$	.11***	.05***	.21***	.06***
	$\log_{10}(x+1)$	.00*	.11***	.05***	.15***
Depression	raw	.47***	.05***	.64***	.03***
	angle	.33***	.05***	.49***	.05***
	$\sqrt{x+1}$	.30***	.09***	.46***	.08***
	$\log_{10}(x+1)$	.15***	.21***	.30***	.21***
Extraversion	raw	.02***	.08***	.01**	.10***
	angle	.01*	.01***	.00	.02***
	$\sqrt{x+1}$	.11***	.04***	.09***	.05***
	$\log_{10}(x+1)$	.28***	.01***	.25***	.01*
Psychoticism	raw	.15***	.00*	.14***	.00
	angle	.01**	.01**	.01*	.00
	$\sqrt{x+1}$	.01***	.01**	.01*	.00
	$\log_{10}(x+1)$	.04***	.02***	.06***	.00
Neuroticism	raw	.00	.05***	.01**	.09***
	angle	.00	.00	.00	.02**
	$\sqrt{x+1}$	.04***	.03***	.02***	.06***
	$\log_{10}(x+1)$	.20***	.02***	.17***	.04***
Lie	raw	.00*	.03***	.00	.03***
	angle	.01**	.00	.00	.00
	$\sqrt{x+1}$	.09***	.01***	.05***	.01**
	$\log_{10}(x+1)$	.30***	.00	.26***	.00
Conservatism	raw	.00	.00	.01*	.00
	angle	.00**	.00	.02***	.00
	$\sqrt{x+1}$	.05***	.00	.09***	.00
	$\log_{10}(x+1)$	.15***	.00	.24***	.02***

*.01 < p < .05 **.001 < p < .01 ***p < .001

Notes: Linear (L) and quadratic components after the linear regression has been removed (Q) are shown. These significance conventions apply in all subsequent tables.

The anxiety and depression scales both show significant and substantial linear regressions. These are best reduced by logarithmic transformation and although this results in an increase in the quadratic components, more extreme transformation (e.g., $\log_{10}(\log_{10}(x+1)+1)$) produces no greater improvement so we regard $\log_{10}(x+1)$ as most appropriate for both scales. The quadratic regressions of the extraversion, neuroticism and lie scales, and the linear regression of the psychoticism scale, are best reduced by angular transformation (arcsin $\sqrt{p}$) (Snedecor and Cochran, 1980). For conservatism, only the linear regression in males is significant, and even then it only accounts for a trivial proportion of the variance. Thus it is not necessary to transform conservatism scores, the almost perfect normality of the distribution of C-Scores indicating that the scale has uniform discriminating properties across the range, at least to the level of second-order effects.

Although in most cases transformations to minimize $G \times E$ interaction have a negligible effect on the results of fitting models to variance components, when there are extreme deviations from normality, as for the anxiety, depression and psychoticism scales, the results may differ markedly (Martin and Eysenck, 1976).

Table 4. *Means and Variances of the Twin Sample for Raw and Transformed Personality and Attitude Variables*

		MZF		MZM		DZF		DZM		DZO	
		Mean	Variance	Mean	Variance	Mean	Variance	Mean	Variance	Mean	Variance
Anxiety	raw	2.37	6.90	1.76	5.04	2.37	6.99	1.75	4.05	2.15	6.09
	log(x + 1)	0.42	0.10	0.33	0.09	0.41	0.10	0.34	0.09	0.39	0.10
Depression	raw	1.46	5.90*	1.05	4.33	1.54	6.99	1.03	2.92	1.41	6.23
	log(x + 1)	0.26	0.09	0.19	0.08	0.27	0.10	0.21	0.08	0.25	0.10
Extraversion	raw	12.52	24.45	12.79	23.97	12.22	24.72	13.11	25.55	12.74	24.87
	angle	51.22	240.21	52.19	233.70	50.34	239.10	53.27	258.34	52.02	243.96
Psychoticism	raw	2.73*	3.89*	3.93***	6.45**	2.91	4.43	4.19	6.74	3.61	6.89
	angle	18.19*	53.07	22.26***	68.31	18.83	56.34	23.08	69.33	21.06	74.80
Neuroticism	raw	11.23	27.57	8.81*	26.42	11.38	27.03	9.12	27.42	10.48	26.55
	angle	44.24	218.10	37.39**	215.15	44.69	211.21	38.26	223.50	42.19	208.57
Lie	raw	10.26*	19.50	8.97	19.20	10.05	20.11	8.72	18.58	9.22	18.61
	angle	44.28*	178.44	40.40	181.19	43.70	182.31	39.73	172.67	41.20	169.49
Conservatism	raw	49.53**	148.61	45.32	175.27	49.23	151.45	45.08	192.22	46.17	158.85

Note: Asterisks denote significant differences between MZ and DZ means and/or variances.

Distribution of Scores and Sex Differences

Before fitting models to explain trait variation it is important to test whether the individuals in the MZ and DZ groups have been drawn at random from the same population by testing whether the subgroup means and variances are equal. Table 4 lists the means and variances of the raw and appropriately transformed scores for the twin sample. Two-tailed t-tests and variance ratio tests were performed between MZ and DZ means and total variances, separately for males and females (Table 4). In the raw scores, five of the sixteen t-tests and four of the sixteen F-tests were significant at least at the 5 per cent level. However, there was no consistent pattern in these differences, and they tended to be trivial and significant only because of the very large numbers available. Transformation left differences in means unchanged whilst differences in variances were totally removed.

It is sometimes argued that the twin method is invalid because DZ twins may have less similar environments than MZ pairs. If this inequality were real and influenced the traits under study, then we would expect to find that the total variance of DZ twins was greater than that of MZs. Even granted that the variance ratio test for inequality is not very powerful in detecting such differences, the total variances of the transformed scores for MZ and DZ pairs are so similar that any such differential environmental effects must be of minor importance. Since the groups appear to be comparable, the MZ and DZ classes were combined in the examination of sex differences.

Table 5 presents the means and variances for the sample broken down by sex. Two-tailed t-tests and variance ratio tests were performed between male and female means and variances for the raw and transformed scores. Females have significantly higher anxiety, depression, neuroticism and lie scores and lower extraversion, psychoticism and conservatism scores than males. The distributions of scores in the twin sample are similar to those obtained in previous studies using the C-Scale

Table 5. *Means and Variances for Raw and Transformed Personality and Attitude Variables Separately for Males and Females*

		Females		Males	
		Mean	Variance	Mean	Variance
Anxiety	raw	2.37***	6.92***	1.82	4.88
	log(x + 1)	0.42***	0.10*	0.34	0.09
Depression	raw	1.50***	6.40***	1.12	4.41
	log(x + 1)	0.26***	0.10***	0.21	0.08
Extraversion	raw	12.45***	24.60	12.89	24.70
	angle	51.03***	240.56	52.53	243.67
Psychoticism	raw	2.79***	4.08***	4.15	7.24
	angle	18.43***	54.61***	22.94	72.76
Neuroticism	raw	11.32***	27.04	9.12	26.42
	angle	44.50***	212.58	38.29	213.74
Lie	raw	10.12***	19.45	8.77	18.75
	angle	43.89***	176.89	39.86	175.03
Conservatism	raw	49.00***	151.36***	45.21	174.77

Note: Asterisks denote significant differences between female and male means and/or variances.

(Feather, 1977, 1978), DSSI/sAD (Henderson *et al.*, 1981) and EPQ (Eysenck *et al.*, 1980) in Australian samples. Although Eysenck *et al.* (1980) found in their Australian sample of approximately 600 males and females that females had higher extraversion scores than males, in their larger English standardization sample the pattern of differences was the same as we found. While it could be argued that there is less potential for bias in the sample of Eysenck *et al.* (1980), in view of our much larger sample one could question which is more representative of the Australian population. We also found that females have a greater variance than males in both the anxiety and depression scales, and are less variable in their psychoticism and conservatism scores. These results are identical for both the raw and transformed scores.

From the standardization data that exist, then, there is no evidence that our twin sample is atypical of the population from which it is drawn in the characteristics under study.

Repeatability

Table 6 shows the distribution of age, and the raw and transformed personality and attitude scores for the ninety-six individuals who completed both the pilot and the main questionnaire. They were typical of the total sample in age and distribution of scores except that the males tended to have lower conservatism and neuroticism scores, and higher extraversion scores than those of the total sample.

Estimates of repeatability (Table 6) were obtained by examining consistency of scores from the pilot and main questionnaire. Separate analyses of variance were performed to obtain meansquares between (MS_{bi}) and within (MS_{wi}) individuals and repeatabilities (intraclass correlations) were calculated as $R_i = (MS_{bi} - MS_{wi})/(MS_{bi} + MS_{wi})$. Where there were significant differences between scores on the two occasions, corrected correlations were calculated by removing the between-occasions effects from the within-individuals meansquare. The within-individual variance components (S_w^2) are also shown in Table 6. These are estimates of the portion of the total variance which is unrepeatable, or measurement error.

The repeatabilities for the three EPQ scales are all high, ranging from 0.70 to 0.92, and are similar in males and females. This is consistent with previous results (Eysenck and Eysenck, 1975). As the interval between the completion of the pilot and the main questionnaire ranged from one to ten months (mean three months), it is unlikely that memory would be an important factor in these results.

The reliabilities of conservatism in males and females are similarly high. This is consistent with an earlier finding (Eaves *et al.*, 1978) of a correlation of 0.60 between the conservatism score from Eysenck's Public Opinion Inventory and the conservatism score from a modified version of the C-Scale used here, administered three years apart to nearly 400 pairs of twins.

The reliabilities of the anxiety and depression scales range from 0.55 to 0.67 and are no lower than one would expect of symptoms which fluctuate in their severity. In a longitudinal study of a general population sample (N = 230) Henderson *et al.* (1981) administered the DSSI/sAD on two occasions three months apart. The anxiety scores correlated 0.62 and the depression 0.54. This sensitivity to change has also been reported by Bedford *et al.* (1976)

Table 6. *Distribution of Age, and Raw and Transformed Personality and Attitude Scores for Individuals Who Completed Both the Pilot and the Main Questionnaire.*

		Females (n = 64)				Males (n = 32)			
		Mean	Variance	Repeatability	S_w^2	Mean	Variance	Repeatability	S_w^2
Age		35.98	195.16	—	—	32.59	177.42	—	—
Anxiety	raw	2.55	6.85	0.67	2.29	1.67	5.30	0.61	2.15
	log(x + 1)	0.44	0.10	0.63	0.04	0.30	0.10	0.62	0.04
Depression	raw	1.75	9.17	0.55	4.09	0.94	3.23	0.58	1.35
	log(x + 1)	0.29	0.10	0.66	0.03	0.18	0.07	0.58	0.03
Extraversion	raw	11.98	24.98	0.82	4.56	14.30	25.74	0.90	2.67
	angle	49.43	242.78	0.81	46.09	57.06	277.11	0.89	29.66
Psychoticism	raw	2.93	3.86	0.74	0.99	4.20	7.21	0.75	1.81
	angle	19.04	48.77	0.73	13.23	23.08	73.15	0.70	21.73
Neuroticism	raw	11.53	22.61	0.84	3.73	7.56	26.85	0.83	4.53
	angle	45.21	174.72	0.85	27.04	33.92	231.39	0.83	38.46
Lie	raw	10.53	20.57	0.83	3.52	7.80	12.77	0.78	2.80
	angle	45.24	187.61	0.84	31.78	37.08	116.87	0.79	25.19
Conservatism	raw	49.27	153.87	0.86	21.14	36.61	169.48	0.92	13.52

Correction for Sex Differences and Regression on Age

A sex difference in means will inflate the within-pairs meansquare (WMS) of DZ opposite-sex pairs (DZOS). Since significant sex differences in means were found for all variables (Table 5) the variance terms due to these differences (and the degree of freedom associated with them) were removed from the WMS of DZOS pairs (Clark *et al.*, 1980).

If a variable is strongly age-dependent, this accentuates the differences between twin pairs and inflates the between-pairs meansquare (BMS). Linear correlations of age with the appropriately transformed variables are shown in Table 7. The correlations are significant in every case, but only for the lie and conservatism scales are they substantial. We corrected for age dependence in these two variables by regressing within-pair sums on age and replacing the BMS with one-half of the residual meansquare (with n − 2 d.f.). Meansquares and their degrees of freedom, corrected for sex differences and regression on age where appropriate, are shown in Table 8.

Table 7. *Two-Tailed Linear Correlations of the Personality and Attitude Scores with Age, Transformed Where Necessary*

		Females	Males
Anxiety	$\log(x + 1)$	−.06**	−.09***
Depression	$\log(x + 1)$	−.14***	−.17***
Extraversion	angle	−.16***	−.14***
Psychoticism	angle	−.20***	−.28***
Neuroticism	angle	−.13***	−.14***
Lie	angle	.36***	.38***
Conservatism	raw	.44***	.37***

We may also examine whether twins become more or less similar with age by correlating absolute within-pair differences with age; these are shown in Table 9. The correlations are small and non-significant for anxiety and extraversion, and for psychoticism only the DZ opposite-sex correlation is significant, with opposite-sex pairs becoming more similar with increasing age. For neuroticism and the lie scale the correlations are only significant for DZ females. This indicates that for females genetic differences in neuroticism and lie become more pronounced with age, but no such effect is apparent in males. Eaves and Eysenck (1976b) also found that genetic differences in neuroticism increase with age. Their sample was too small to subdivide by sex, but it was comprised mainly of females and we can therefore consider this a replication of their interesting finding. For conservatism the reverse is true: in males genetic differences become more pronounced with age, but not in females. In the case of depression, both MZ and DZ males become more similar with advancing age, but not females. While this latter finding is open to a number of interpretations, it is clear that if environmental circumstances of cotwins become more different as they get older, these do not appear to produce any greater differences in any of the personality and attitude variables we have measured here.

Table 8. *Observed Meansquares for the Appropriately Transformed Personality and Attitudes Variables, and their Degrees of Freedom*

		Anxiety[a]		Depression[a]		Extraversion[a]		Psychoticism[a]		Neuroticism[a]		Lie[a, b]		Conservatism[a, b]	
		df	Mean-square	df	Mean-square	df	Mean-square	df	Mean-square	df	Mean-square	df	Mean-square	df	Mean-square
MZF	Between	1229	0.134	1229	0.128	1232	368.00	1232	71.92	1232	330.85	1231	238.15	1231	200.39
	Within	1230	0.060	1230	0.061	1233	112.46	1233	34.18	1233	105.38	1233	77.54	1233	43.66
MZM	Between	566	0.122	566	0.103	565	347.84	565	98.61	565	315.18	564	220.57	564	250.49
	Within	567	0.057	567	0.054	566	116.46	566	37.98	566	115.65	566	98.59	566	62.44
DZF	Between	749	0.117	749	0.118	750	283.65	750	69.11	750	265.12	749	195.57	749	175.62
	Within	750	0.081	750	0.081	751	194.62	751	43.59	751	157.38	751	112.61	751	64.25
DZM	Between	351	0.097	351	0.091	350	292.18	350	86.66	350	263.16	349	186.59	350	238.67
	Within	352	0.076	352	0.059	351	224.60	351	52.05	351	183.94	351	105.36	352	85.06
DZO	Between	901	0.106	901	0.108	904	295.30	904	84.61	904	227.82	903	167.93	904	179.92
	Within	901	0.083	901	0.082	904	192.98	904	51.55	904	174.64	904	114.34	905	76.05

Notes: [a]Corrected for sex differences.
[b]Corrected for regression on age.

Table 9. *Two-Tailed Correlations of Absolute Within-Pair Differences in the Transformed Personality and Attitude Scores with Age*

		MZF	MZM	DZF	DZM	DZO
Anxiety	$\log(x+1)$	.02	−.03	−.01	−.06	−.01
Depression	$\log(x+1)$	−.04	−.18***	−.01	−.13*	−.14**
Extraversion	angle	.03	.03	.07	−.04	.01
Psychoticism	angle	−.02	−.04	−.03	−.01	−.07*
Neuroticism	angle	.02	.01	.12**	.02	.01
Lie	angle	−.03	−.01	.09*	.03	.05
Conservatism	raw	.05	.00	.04	.20***	.12***

Genetical Analysis of Trait Variation

We shall discuss the results of the model, fitting separately for each factor. In every case a model (E_1) postulating that all variation was due to individual environmental experiences and error and that there were no greater differences between pairs than between members of the same pair failed badly and is omitted from summary tables. Our first conclusion then is that there are greater differences in personality and attitudes between twin pairs than between cotwins. We shall now see whether this familiacity is due to shared environment, shared genes, or both.

Anxiety

The results of fitting models to log transformed anxiety scores are shown in Table 10. A purely environmental model (E_1E_2) fails adequately to describe the data in either males or females, while a simple genetic model (E_1V_A) gives a good fit in both sexes. No further reductions in chisquare were seen with addition of extra parameters. When the E_1V_A model is fitted to the combined male and female data, the chisquare for the heterogeneity of fit over sexes (obtained by adding the chisquare values for males and females and subtracting from the chisquare of the combined male and female data) is non-significant ($\chi^2_2 = 5.18$, $P > 0.05$). Although we are thus entitled to fit the same model to the joint data, we notice that, while the estimates for E_1 are similar, there is a larger V_A component for females than males.

A full model incorporating different-sized E_1, E_2 and V_A effects for males and females has been developed by Eaves (1977), illustrated in Eaves *et al.* (1978), and is shown in Table 11. V_{Amf} is the covariance between the genetical effects acting in males and those acting in females. If the genes affecting a trait in males are quite different from those affecting the trait in females, then we expect $\hat{V}_{Amf}$ to be zero. If the genes acting in males and females are exactly the same but produce scalar differences in the two sexes, then we expect the correlation between the effects

$$r_{VAmf} = \hat{V}_{Amf} / \sqrt{\hat{V}_{Am} \cdot \hat{V}_{Af}}$$

to be one. A similar argument applies to E_{2mf}, the covariation between E_2 effects acting in males and females.

The results of fitting a model which specifies a common E_1 parameter but different-sized V_A effects in males and females are shown in Table 12.

Table 10. *Summary of Model-Fitting to Log Transformed Anxiety Scores*

	$\hat{E}_1$	$\hat{E}_2$	$\hat{V}_A$	$\hat{V}_D$	*df*	χ^2	h^2
Female							
E_1E_2	.068***	.030***	—	—	2	25.47***	
E_1V_A	.061***	—	.037***	—	2	0.23	.38 ± .02
$E_1E_2V_A$	.060***	−.002	.039***	—	1	0.16	
$E_1V_AV_D$	.060***	—	.033**	.004	1	0.16	
Male							
E_1E_2	.064***	.024***	—	—	2	15.06**	
E_1V_A	.058***	—	.031***	—	2	1.29	.35 ± .03
$E_1E_2V_A$	.056***	−.010	.042***	—	1	0.26	
$E_1V_AV_D$	.056***	—	.012	.020	1	0.26	
Female and Male							
E_1E_2	.067***	.028***	—	—	6	45.37***	
E_1V_A	.060***	—	.035***	—	6	6.70	.37 ± 0.2
$E_1E_2V_A$	.059***	−.004	.040***	—	5	6.23	
$E_1V_AV_D$	.059***	—	.027**	.009	5	6.23	
Female and Male and Opposite-Sex							
E_1E_2	.071***	.024***	—	—	8	67.68***	
E_1V_A	.060 ***	—	.034***	—	8	10.36	.36 ± 0.2
$E_1E_2V_A$	.059***	−.008	.043***	—	7	7.33	
$E_1V_AV_D$	.059***	—	.020*	.016*	7	7.33	

Table 11. *Model for Twin Meansquares Incorporating Different Genetic and Environmental Components of Variation for Males and Females*

		E_{1_M}	E_{1_F}	E_{2_M}	E_{2_F}	$E_{2_{MF}}$	V_{A_M}	V_{A_F}	$V_{A_{MF}}$
MZF	Between	0	1	0	2	0	0	2	0
	Within	0	1	0	0	0	0	0	0
MZM	Between	1	0	2	0	0	2	0	0
	Within	1	0	0	0	0	0	0	0
DZF	Between	0	1	0	2	0	0	3/2	0
	Within	0	1	0	0	0	0	1/2	0
DZM	Between	1	0	2	0	0	3/2	0	0
	Within	1	0	0	0	0	1/2	0	0
DZO	Between	1/2	1/2	1/2	1/2	1	1/2	1/2	1/2
	Within	1/2	1/2	1/2	1/2	−1	1/2	1/2	−1/2

Table 12. *Estimates (± s.e.) Obtained after Fitting a Model Allowing Different Genetic Components of Variation in Males and Females for Log Transformed Anxiety Scores*

	$\hat{E}_1$	$\hat{V}_{A_M}$	$\hat{V}_{A_F}$	$\hat{V}_{A_{MF}}$
	0.060***	0.030***	0.038***	0.023***
±	0.002	0.003	0.002	0.006
	$\chi^2_6 = 2.15 (p = .91)$			
	$h^2_{males} = 0.33 \pm .03$		$h^2_{females} = 0.39 \pm .02$	

Table 13. *Sources of Variance (percentages) for Log Transformed Anxiety Scores*

	Females	Males
E_1 error	38	45
E_1 individual environment	23	22
E_1	61	67
V_A	39	33

Fitting separate V_A parameters for males and females causes a significant reduction in chisquare ($\chi^2_2 = 8.21$, $P < 0.05$). The correlation $r_{V_A mf} = 0.67$ is not significantly different from unity and indicates that the same V_A effects which also act in females act in males, but with a smaller effect on the variance. Thus, in males approximately 33 per cent of the variation in anxiety is genetic in origin while in females this rises to approximately 39 per cent, with the remaining variance due to individual environmental differences and error. We may subtract the values of the residual meansquare (Table 6), obtained from the repeatability data, from the estimates of E_1 and so estimate the proportion of variance due to non-repeatable individual environmental differences (Table 13).

Depression

As in the case of anxiety, in both males and females, the $E_1 V_A$ model best describes the data, although in males there is some evidence that E_2 effects are also important (Table 14). The chisquare for the heterogeneity of fit over sexes is highly significant ($\chi^2_2 = 27.26$, $P < 0.001$), and inspection of the parameter estimates shows that there are larger $\hat{E}_1$ and $\hat{V}_A$ components for males than females.

Fitting separate E_1 and V_A parameters for males and females (Table 15) causes a significant improvement ($\chi^2_3 = 24.97$, $P < 0.001$). The correlation $r_{V_A mf} = 0.73$ is not significantly different from unity which indicates that, as in the case of anxiety, the same V_A effects which act in females also act in males but with smaller effect. Addition of an E_2 parameter in males results in a non-significant reduction of chisquare ($\chi^2_1 = 1.40$, $P > 0.05$), indicating that this effect is not necessary to describe variation. While the heritabilities are similar to those for anxiety, true within-family environment accounts for a greater proportion of the variance in depression than anxiety (Table 16).

Extraversion

The $E_1 V_A$ model is able to account for variation in female extraversion, but addition of the parameter V_D results in an even better fit ($\chi^2_1 = 5.20$, $P > 0.05$). The latter model also provides the best description of the data in males, although the estimate of V_A is negative. There is no heterogeneity of fit of the $E_1 V_A V_D$ model over the sexes ($\chi^2_3 = 1.92$, $P > 0.05$), so we may fit it to the joint male, female and opposite-sex data (Table 17). All three sources of variation are significantly greater than zero; their contributions to the total are shown in Table 18.

Table 14. *Summary of Model-Fitting to Log Transformed Depression Scores*

	$\hat{E}_1$	$\hat{E}_2$	$\hat{V}_A$	$\hat{V}_D$	*df*	χ^2	h^2
Female							
E_1E_2	.069***	.028***	—	—	2	22.04***	
E_1V_A	.062***	—	.035***	—	2	1.20	.36 ± .02
$E_1E_2V_A$	.062***	−.000	.035***	—	1	1.20	
$E_1V_AV_D$	.062***	—	.034**	.001	1	1.20	
Male							
E_1E_2	.056***	.021***	—	—	2	2.46	
E_1V_A	.052***	—	.025***	—	2	1.85	.32 ± .04
$E_1E_2V_A$	.053***	−.010	.013	—	1	0.46	
$E_1V_AV_D$	.053***	—	.044**	−.021	1	0.46	
Female and Male							
E_1E_2	.065***	.026***	—	—	6	54.72***	
E_1V_A	.059***	—	.032***	—	6	30.31***	
$E_1E_2V_A$	.059***	.003	.028***	—	5	30.22***	
$E_1V_AV_D$	.059***	—	.037***	−.006	5	30.22***	
Female and Male and Opposite-Sex							
E_1E_2	.069***	.023***	—	—	8	76.11***	
E_1V_A	.060***	—	.032***	—	8	33.70***	
$E_1E_2V_A$	.059***	−.002	.034***	—	7	33.21***	
$E_1V_AV_D$	.059***	—	.028***	.005	7	33.21***	

Table 15. *Estimates (± s.e.) Obtained after Fitting a Model Allowing Different Genetic and Environmental Components of Variation in Males and Females for Log Transformed Depression Scores*

	$\hat{E}_{1_M}$	$\hat{E}_{1_F}$	$\hat{V}_{A_M}$	$\hat{V}_{A_F}$	$\hat{V}_{A_{MF}}$
	0.053***	0.062***	0.026***	0.036***	0.022***
±	0.003	0.002	0.003	0.003	0.006
			$\chi^2_5 = 8.73$ (p = .12)		
	$h^2_{\text{males}} = 0.33 \pm .03$			$h^2_{\text{females}} = 0.37 \pm .02$	

Table 16. *Sources of Variance (percentages) for Log Transformed Depression Scores*

	Females	Males
E_1 error	33	29
E_1 individual environment	30	38
E_1	63	67
V_A	37	33

According to Fisher's fundamental theorem of natural selection (Fisher, 1931), the pattern of variation demonstrated, where the additive genetic variance is small relative to the non-additive genetic variance, indicates that extraversion is a character which has undergone selection in the course of human evolution. We speculate that selection has been favouring individuals with intermediate extraversion

Table 17. *Summary of Model-Fitting to Angle Transformed Extraversion Scores*

	$\hat{E}_1$	$\hat{E}_2$	$\hat{V}_A$	$\hat{V}_D$	*df*	χ^2	h^2_{narrow}	h^2_{broad}
Female								
E_1E_2	143.5***	96.3***	—	—	2	91.18***		
E_1V_A	115.2***	—	125.3***	—	2	5.26		
$E_1E_2V_A$	112.4***	−37.9	165.3***	—	1	0.01	.52 ± .02	
$E_1V_AV_D$	112.4***	—	51.6	75.8*	1	0.01		
Male								
E_1E_2	157.9***	84.4***	—	—	2	53.99***		
E_1V_A	124.7***	—	119.7***	—	2	10.35***		
$E_1E_2V_A$	118.6***	−64.7	189.6***	—	1	2.26		
$E_1V_AV_D$	118.6***	—	−4.4	129.3**	1	2.26		
Female and Male								
E_1E_2	148.1***	92.5***	—	—	6	150.84***		
E_1V_A	118.1***	—	123.7***	—	6	17.33**		
$E_1E_2V_A$	114.3***	−47.3	173.9***	—	5	4.19		
$E_1V_AV_D$	114.3***	—	32.2	94.5**	5	4.19		
Female and Male and Opposite-Sex								
E_1E_2	158.7***	82.7***	—	—	8	166.92***		
E_1V_A	119.7***	—	122.9***	—	8	19.59*		
$E_1E_2V_A$	114.4***	−38.2	165.6***	—	7	5.42		
$E_1V_AV_D$	114.4***	—	50.9**	76.4***	7	5.42	.21 ± .09	.53 ± .02

Table 18. *Sources of Variance (percentages) for Angle Transformed Extraversion Scores*

E_1 — error	17
E_1 — individual environment	30
E_1	47
V_A	21
V_D	32

scores. However, data other than those on twins are needed to clarify this issue (Martin *et al.*, 1978; Eaves *et al.*, 1977a, 1978).

Psychoticism

Once again the environmental model fails badly, while the $E_1 V_A$ model gives a good fit in both men and women (Table 19). However, there is highly significant heterogeneity of fit over ($\chi^2_2 = 33.26$, $P < 0.001$) and inspection of the parameter estimates shows that there is a larger V_A component in males than females. Allowing for different genetic components in males and females (Table 20) causes a great improvement ($\chi^2_3 = 36.31$, $P < 0.001$), but the correlation $r_{VAmf} = 1.09$ indicates that the same genes act in both sexes but produce twice as much variance in males. Thus in females approximately 35 per cent of the variation in psychoticism is due to additive genetic effects, while in males it accounts for 50 per cent. Eaves and Eysenck (1977) found that 49 per cent of the variation in psychoticism is genetic in origin but did not look for differences in gene expression between the sexes.

Table 19. *Summary of Model-Fitting to Angle Transformed Psychoticism Scores*

	$\hat{E}_1$	$\hat{E}_2$	$\hat{V}_A$	$\hat{V}_D$	*df*	χ^2	h^2
Female							
$E_1 E_2$	37.74***	16.56***	—	—	2	14.87***	
$E_1 V_A$	34.20***	—	20.14***	—	2	2.81	.37 ± .0
$E_1 E_2 V_A$	34.65***	4.15	15.56***	—	1	1.59	
$E_1 V_A V_D$	34.65***	—	28.00***	−8.29	1	1.59	
Male							
$E_1 E_2$	43.36***	25.34***	—	—	2	13.16**	
$E_1 V_A$	37.78***	—	30.91***	—	2	0.28	.45 ± .0
$E_1 E_2 V_A$	38.09***	3.38	27.26***	—	1	0.05	
$E_1 V_A V_D$	38.09***	—	37.41**	−6.77	1	0.05	
Female and Male							
$E_1 E_2$	39.52***	19.33***	—	—	6	63.69***	
$E_1 V_A$	35.32***	—	23.56***	—	6	36.35***	
$E_1 E_2 V_A$	35.71***	3.84	19.35***	—	5	35.12***	
$E_1 V_A V_D$	35.71***	—	30.86***	−7.67	5	35.12***	
Female and Male and Opposite-Sex							
$E_1 E_2$	42.37***	18.67***	—	—	8	88.22***	
$E_1 V_A$	35.80***	—	25.37***	—	8	48.39***	
$E_1 E_2 V_A$	36.39***	3.34	21.46***	—	7	46.94***	
$E_1 V_A V_D$	36.39***	—	31.47***	−6.67	7	46.94***	

Table 20. *Estimates (± s.e.) Obtained after Fitting a Model Allowing Different Genetic Components of Variation in Males and Females for Angle Transformed Psychoticism Scores*

	$\hat{E}_1$	$\hat{V}_{A_M}$	$\hat{V}_{A_F}$	$\hat{V}_{A_{MF}}$
	35.70***	35.40***	19.92***	28.96***
±	1.09	2.35	1.40	4.03
	$\chi^2_6 = 12.08$ (p = .06)			
	$h^2_{males} = 0.50 \pm .02$		$h^2_{females} = 0.36 \pm .02$	

Table 21. *Sources of Variance (percentages) for Angle Transformed Psychoticism Scores*

	Females	Males
E_1: error	24	30
E_1: individual environment	40	20
E_1	64	50
V_A	36	50

In females, true individual environment accounts for a greater proportion of E_1 than error, while in males the reverse is true (Table 21). However, in both males and females, the contribution of true individual environment to variation in psychoticism is greater than has previously been reported (Eaves and Eysenck, 1977).

Neuroticism

In both males and females the simple genetic model provides the best fit to the data. Although the chisquare for the heterogeneity of fit over sexes is non-significant ($\chi^2_2 = 3.17$, $P > 0.05$), we notice that there are smaller $\hat{E}_1$ and larger $\hat{V}_A$ components in females than males (Table 22).

Fitting a model allowing different E_1 and V_A components in males and females (Table 23) results in a significant reduction in chisquare ($\chi^2_3 = 12.64$, $P < 0.01$), the correlation $r_{VAmf} = 0.58$ indicating that there are differences in gene action in males and females. In both sexes approximately one-half the variation in neuroticism is genetic in origin, with individual environment accounting for just over a third of the total variation (Table 24). The correlation of age with absolute within-pair differences in DZ females discussed earlier also indicates that genetic differences become more pronounced as females get older.

Lie

The genetic model describes the lie data adequately, although there is some evidence that E_2 effects are also important in males. There is significant heterogeneity of fit of the $E_1 V_A$ model over sexes ($\chi^2_2 = 12.73$, $P < 0.005$), and we notice that there are larger

Table 22. *Summary of Model-Fitting to Angle Transformed Neuroticism Scores*

	$\hat{E}_1$	$\hat{E}_2$	$\hat{V}_A$	$\hat{V}_D$	*df*	χ^2	h^2
Female							
E_1E_2	125.1***	90.5***	—	—	2	51.12***	
E_1V_A	104.7***	—	110.5***	—	2	0.42	
$E_1E_2V_A$	104.8***	1.2	109.2***	—	1	0.42	.51 ± .02
$E_1V_AV_D$	104.8***	—	112.8***	−2.4	1	0.42	
Male							
E_1E_2	141.8***	76.8***	—	—	2	28.48***	
E_1V_A	118.9***	—	100.3***	—	2	1.72	.46 ± .03
$E_1E_2V_A$	116.5***	−26.4	128.8***	—	1	0.27	
$E_1V_AV_D$	116.5***	—	49.7	52.7	1	0.27	
Female and Male							
E_1E_2	130.3***	86.1***	—	—	6	86.65***	
E_1V_A	109.1***	—	107.4***	—	6	5.85	.50 ± .02
$E_1E_2V_A$	108.4***	−8.2	116.2***	—	5	5.30	
$E_1V_AV_D$	108.4***	—	91.5***	16.5	5	5.30	
Female and Male and Opposite-Sex							
E_1E_2	140.9***	72.0***	—	—	8	136.90***	
E_1V_A	110.9***	—	102.1***	—	8	18.42*	
$E_1E_2V_A$	107.6***	−24.1	128.9***	—	7	12.16	
$E_1V_AV_D$	107.6***	—	56.7**	48.1**	7	12.26	.27 ± .09

Table 23. *Estimates (± s.e.) Obtained after Fitting a Model Allowing Different Genetic and Environmental Components of Variation in Males and Females for Angle Transformed Neuroticism Scores*

	$\hat{E}_{1_M}$	$\hat{E}_{1_F}$	$\hat{V}_{A_M}$	$\hat{V}_{A_F}$	$\hat{V}_{A_{MF}}$
	117.4***	104.2***	95.4***	108.0***	59.4***
±	6.4	3.9	8.0	5.6	13.9
			$\chi^2_5 = 5.78$ (p = .33)		
	$h^2_{males} = 0.45 \pm .03$			$h^2_{females} = 0.51 \pm .02$	

Table 24. *Sources of Variance (percentages) for Angle Transformed Neuroticism Scores*

	Females	Males
E_1 error	13	18
E_1 individual environment	36	37
E_1	49	55
V_A	51	45

$\hat{E}_1$ and smaller $\hat{V}_A$ components for males than females (Table 25). Fitting separate E_1 and V_A parameters for the males and females (Table 26) results in a significant reduction in chisquare ($\chi^2_3 = 13.77$, $P < 0.01$), the correlation $r_{VAmf} = 0.93$ indicating that the same V_A effects which act in females act in males but with smaller effect. Addition of an E_2 parameter in males does not improve the fit ($\chi^2_1 = 3.46$, $P > 0.05$).

The breakdown of total variation (Table 27) is similar to that obtained in previous studies of lie (Martin and Eysenck, 1976; Eaves *et al.*, 1978).

Table 25. *Summary of Model-Fitting to Angle Transformed and Age Corrected Lie Scores*

	$\hat{E}_1$	$\hat{E}_2$	$\hat{V}_A$	$\hat{V}_D$	*df*	χ^2	h^2
Female							
E_1E_2	90.8***	65.8***	—	—	2	42.96***	
E_1V_A	76.8***	—	79.6***	—	2	0.55	.51 ± .02
$E_1E_2V_A$	77.3***	6.7	72.5***	—	1	0.18	
$E_1V_AV_D$	77.3***	—	92.6**	−13.4	1	0.18	
Male							
E_1E_2	101.2***	53.2***	—	—	2	3.37	
E_1V_A	93.8***	—	60.0***	—	2	4.95	.39 ± .03
$E_1E_2V_A$	96.7***	32.7*	24.8	—	1	1.52	
$E_1V_AV_D$	96.7***	—	122.8***	−65.3	1	1.52	
Female and Male							
E_1E_2	94.1***	61.8***	—	—	6	49.39***	
E_1V_A	82.3***	—	73.2***	—	6	18.23**	
$E_1E_2V_A$	83.5***	14.9*	57.3***	—	5	15.34**	
$E_1V_AV_D$	83.5***	—	102.1***	−29.9	5	15.34**	
Female and Male and Opposite-Sex							
E_1E_2	98.9***	53.5***	—	—	8	82.00***	
E_1V_A	81.9 ***	—	70.0***	—	8	24.90**	
$E_1E_2V_A$	82.6***	5.0	64.4***	—	7	24.09**	
$E_1V_AV_D$	82.6***	—	79.4***	−10.0	7	24.09**	

Table 26. *Estimates (± s.e.) Obtained after Fitting a Model Allowing Different Genetic and Environmental Components of Variation in Males and Females for Angle Transformed and Age Corrected Lie Scores*

	$\hat{E}_{1_M}$	$\hat{E}_{1_F}$	$\hat{V}_{A_M}$	$\hat{V}_{A_F}$	$\hat{V}_{A_{MF}}$
	92.04***	76.26***	56.68***	77.44***	61.47***
±	4.98	2.88	5.69	4.03	9.69
			$\chi^2_5 = 11.13$ (p = .05)		
	$h^2_{\text{males}} = 0.38 \pm .03$			$h^2_{\text{females}} = 0.50 \pm .02$	

Table 27. *Sources of Variance (percentages) for Angle Transformed and Age Corrected Lie Scores*

	Females	Males
E_1 error	21	17
E_1 individual environment	29	45
E_1	50	62
V_A	50	38

Conservatism

In contrast to the personality variables, not only the E_1E_2 model but also the E_1V_A model gives a bad fit to the conservatism data in both sexes. However, a model which includes all three sources of variation ($E_1E_2V_A$) gives an excellent fit in both males and females (Table 28). But when this model is applied to the combined male and female data it fails badly, apparently because of heterogeneity of fit over sexes ($\chi^2_3 = 60.66$, $P < 0.001$). Inspection of the parameter estimates reveals that there are larger E_1 and E_2 components for males than females but a similar estimate of V_A in both sexes.

Table 28. *Summary of Model-Fitting to Age Corrected Conservatism Scores*

	$\hat{E}_1$	$\hat{E}_2$	$\hat{V}_A$	$\hat{V}_D$	*df*	χ^2	h^2
Female							
E_1E_2	51.45***	69.79***	—	—	2	41.31***	
E_1V_A	41.82***	—	77.82***	—	2	21.31***	
$E_1E_2V_A$	43.58***	35.67***	41.92***	—	1	0.11	.35 ± .06
$E_1V_AV_D$	43.58***	—	148.92***	−71.33	1	0.11	
Male							
E_1E_2	71.11***	87.43***	—	—	2	11.24**	
E_1V_A	59.59***	—	97.28***	—	2	13.32**	
$E_1E_2V_A$	62.69***	52.71***	43.28**	—	1	0.20	.27 ± .09
$E_1V_AV_D$	62.69***	—	201.41***	−105.42	1	0.20	
Female and Male							
E_1E_2	57.67***	75.35***	—	—	6	108.31***	
E_1V_A	47.42***	—	83.99***	—	6	97.73***	
$E_1E_2V_A$	49.58***	40.95***	42.50**	—	5	60.97***	
$E_1V_AV_D$	49.58***	—	165.35***	−81.90	5	60.97***	
Female and Male and Opposite-Sex							
E_1E_2	62.04***	69.78***	—	—	8	135.90***	
E_1V_A	46.43***	—	83.51***	—	8	110.94***	
$E_1E_2V_A$	49.45***	34.33***	47.97***	—	7	64.41***	
$E_1V_AV_D$	49.45***	—	150.95***	−68.66	7	64.41***	

Table 29. *Estimates (± s.e.) Obtained after Fitting a Model Allowing Different Environmental Components of Variation in Males and Females for Age Corrected Conservatism Scores*

	$\hat{V}_A$	$\hat{E}_{1_M}$	$\hat{E}_{1_F}$	$\hat{E}_{2_M}$	$\hat{E}_{2_F}$	$\hat{E}_{2_{MF}}$
	41.54***	62.05***	43.41***	49.41***	34.57***	37.13***
±	6.34	3.26	1.69	7.49	6.12	4.96
			$\chi^2_4 = 4.40$ (p = .35)			
	$h^2_{\text{males}} = 0.27 \pm 0.4$				$h^2_{\text{females}} = 0.35 \pm 0.5$	

Fitting separate E_1 and E_2 parameters for males and females (Table 29) causes a great improvement in fit ($\chi^2_3 = 60.01$, $P < 0.001$) and excellent agreement with the joint data. The correlation $r_{E2mf} = 0.90$ is not significantly different from unity and indicates that the same E_2 effects which act in males act in females but with a smaller

effect on the variance. The significant correlation of absolute within-pair differences with age in DZ males and opposite-sex pairs (Table 9) indicates that in males genetic differences for conservatism become more pronounced with age.

As discussed above, our estimate of E_2 can be better described as a parameter B which may be attributable to cultural variation (E_2) or additional genetic variation due to assortative mating (AM) or both. In fact $B = E_2 + V_A(A/(1 - A))$ where $A = h^2\mu$, A is the marital correlation between additive deviations of spouses, h^2 the heritability and μ the observed marital correlation (Eaves, 1977). If an estimate of μ is available we can solve the quadratic equation

$$A = h^2\mu$$
$$= \mu(V_A(1 + (A/(1 - A)))/V_T$$

in A, where ($V_T = E_1 + B + V_A$), obtain $AM = V_A(A/(1 - A))$ (the extra additive genetic variation due to assortative mating) and by subtraction of this term from B we can obtain an estimate of 'true E_2'.

We do not have an estimate of the phenotypic marital correlation for conservatism in the parents of twins in this study, but Feather (1978) in his use of the C-Scale in an Australian sample obtained a marital correlation of 0.675 from 103 husband-wife pairs. Using this value as our estimate of μ and the mean of V_T for males and females as V_T, we obtain the breakdown of B into E_2 and AM as shown in Table 30. Thus, approximately 38 per cent of the variation in conservatism in males is genetic in origin and in females this rises to approximately 49 per cent. Cultural influences and parental transmission account for about 21 and 14 per cent of the variation in males and females respectively, the remaining variation being due to individual environmental experiences and error.

Table 30. *Sources of Variance (percentages) for Age Corrected Conservatism Scores*

	Females	Males
E_1	36	41
error	18	9
individual environment	18	32
V_A	35	27
total genetic	49	38
assortative mating	14	11
E_2	29	32
family environment	15	21

Correlations between Personality and Attitude Scores

Partial correlations, controlling for age, between the transformed personality and attitude variables are shown in Table 31. The correlations are similar for both sexes. Individuals who are more anxious and depressed tend to be introverted, more psychotic and neurotic, and have lower lie scores. Although the EPQ scales were designed to measure independent personality attributes, they do depart slightly from orthogonality, a result found previously (Eysenck and Eysenck, 1975). Extraverts tend to be more psychotic, less neurotic and lie less. More psychotic individuals tend

to be more neurotic and, like neurotics, have lower lie scores. Less conservative individuals tend to be more psychotic while more conservative individuals score higher on the lie or social desirability scale. Similar correlations have been found with the Eysenck Radicalism scale elsewhere (Martin and Eysenck, 1976). An interesting sex difference is found with extraversion where introverted females appear to be more conservative but no such relationship is found in men. There is also a slight tendency for more liberal men to be more anxious and depressed. While many of these correlations are statistically significant, with the exception of those between anxiety, depression and neuroticism they are quite low. We are led to speculate whether it is environmental or genetic factors which are responsible for the covariation of the symptom states of anxiety and depression and the personality trait of neuroticism.

Causes of Covariation between Anxiety, Depression and Neuroticism

We know from the univariate analyses that for anxiety, depression and neuroticism, within-family environment (E_1) and additive gene effects (V_A) are important causes of variation, although there are differences in the importance of these effects in males and females. We now investigate the extent to which these two sources of variation are responsible for trait covariation by using the technique of genetical analysis of covariance structures developed by Martin and Eaves (1977). This method tests simultaneously hypotheses about both the sources and the structure of covariation. Just as univariate models were fitted to meansquares, multivariate models are fitted to the between-and within-pairs meanproducts matrices. Detailed explanation and applications of this maximum likelihood technique can be found in Eaves *et al.* (1977b), Fulker (1978), Martin *et al.*(1979), Martin *et al.* (1981) and Clifford *et al.* (1981).

The simplest E_1V_A model includes a single general factor causing covariation between anxiety, depression and neuroticism plus a variance component specific to each variable for both the E_1 and V_A causes of variation. For each source, then, we estimate three factor loadings and three specific variance components, or twelve parameters in all. Each meanproducts matrix contributes three meansquares from the diagonal and three off-diagonal meanproducts, making twenty-four unique statistics from the four between- and within-pairs matrices of MZ and DZ twins of the same sex. We are thus left with twelve degrees of freedom to test the goodness of fit.

Maximum likelihood estimates of factor loadings and specific variance components from each source are then obtained. The proportions of variance in each measure accounted for by these estimates are shown in Table 32. In both sexes this model gives an excellent fit to the data and all parameter estimates are significantly greater than zero ($P < 0.01$).

The results suggest that genetic variation in the symptoms of anxiety and depression is largely dependent on the effects of the same genes which determine variation in the trait of neuroticism. This follows from the finding that the specific genetic components of variation are small, nearly all of their genetic variance being due to the common factor. However, it is interesting that there is still substantial specific genetical variance for neuroticism, and it is possible that this may be manifested relatively independently of the two symptoms we have considered.

A factor of individual environmental effects also appears to influence all three variables, although specific E_1 variation is equally or more important in most cases.

Table 31. *Partial Correlations, Controlling for Age, between the Transformed Personality and Attitude Variables Separately for Females, Upper Triangle, and Males, Lower Triangle*

		Anxiety log(x + 1)	Depression log(x + 1)	Extraversion angle	Psychoticism angle	Neuroticism angle	Lie angle	Conservatism raw
Anxiety	log(x + 1)	—	.66***	−.08***	.15***	.61***	−.12***	−.01
Depression	log(x + 1)	.60***	—	−.10***	.18***	.57***	−.09***	−.03*
Extraversion	angle	−.12***	−.16***	—	.08***	−.17***	−.09***	−.11***
Psychoticism	angle	.12***	.16***	.04*	—	.12***	−.31***	−.21***
Neuroticism	angle	.60***	.55***	−.19***	.10***	—	−.15***	.03*
Lie	angle	−.11***	−.08***	−.08***	−.31***	−.16***	—	.26***
Conservatism	raw	−.05*	−.05**	−.03	−.17***	−.02	.23***	—

Table 32. *Results of Fitting a Multivariate E_1V_A Model to Transformed Anxiety, Depression and Neuroticism Scores*

		E_1		V_A	
		factor	specific	factor	specific
Females					
Neuroticism	angle	.20***	.29***	.35***	.16***
Anxiety	log(x + 1)	.35***	.27***	.35***	.03***
Depression	log(x + 1)	.33***	.31***	.30***	.06***
			χ^2_{12} = 6.90 (p = .86)		
Males					
Neuroticism	angle	.22***	.32***	.34***	.12***
Anxiety	log(x + 1)	.31***	.35***	.30***	.04**
Depression	log(x + 1)	.33***	.35***	.23***	.09***
			χ^2_{12} = 12.52 (p = .40)		

Note: Results are in terms of the proportion of variance accounted for by each source.

The proportion of variance due to error or fluctuating environment in anxiety and depression (Tables 13 and 16) is equal to or slightly greater than the specific environmental variance, which suggests that some of this fluctuating environment may contribute to E_1 factor variance. The specific variance component for neuroticism, on the other hand, is somewhat greater than the unrepeatable variance, so that there may be systematic environmental experiences influencing the trait of neuroticism which do not influence the symptoms we measure.

Genetic and environmental correlations of the variables are shown in Table 33. In both sexes, genetic correlations are much higher (around 0.8) than the corresponding environmental correlations (around 0.4), and are similar for the three variables. While the distinction has been made between personality traits and states (Foulds, 1965, 1974,), for the neurotic symptoms measured here, there is good evidence for a common genetic and within-family environmental basis.

Table 33. *Genetic and Environmental Correlations between Transformed Anxiety, Depression and Neuroticism Scores for Females, Upper Triangle, and Males, Lower Triangle*

ENVIRONMENTAL

		Neuroticism angle	Anxiety log(x + 1)	Depression log(x + 1)
Neuroticism	angle	—	0.47	0.45
Anxiety	log(x + 1)	0.44	—	0.54
Depression	log(x + 1)	0.45	0.48	—

GENETIC

		Neuroticism angle	Anxiety log(x + 1)	Depression log(x + 1)
Neuroticism	angle	—	0.80	0.76
Anxiety	log(x + 1)	0.81	—	0.88
Depression	log(x + 1)	0.73	0.79	—

DISCUSSION

The results of this very large twin study vindicate in the strongest possible way many of the hypotheses proposed and supported by Eysenck during his career. It is possible to measure dimensions of personality and attitudes which are consistent in their pattern from study to study and culture to culture. These are highly repeatable, at least in the medium term. Work by others has shown them to have high validity in their ability to discriminate between important external criterion groups. A considerable proportion of variation in all these dimensions is due to genetic factors.

The single most astonishing finding from this very powerful study is the complete lack of evidence for the effect of shared environmental factors in shaping variation in personality, and their relatively minor contribution to variation in social attitudes. This replicates earlier studies based on smaller numbers in which it was possible that lack of power was responsible for the lack of evidence. The conclusion is now so strong that we must suspect those who continue to espouse theories of individual differences in personality which centre on family environment and cultural influences, of motives other than scientific.

While previous studies on the aetiology of neuroses and minor depression have yielded conflicting results (Young *et al.*,1971; Torgersen, 1983), our large twin study has provided a clear answer to the causes of individual differences in the symptoms of anxiety and depression. The data suggest that population variance in these measures is due only to additive genetic effects and the influence of environmental factors which are unique to the individual. Both symptoms appear to be influenced largely by the same genes in both sexes, but have greater effect in females than males. Environmental variance for depression is also greater in females, a result found previously by Eaves and Young (1981). We found no evidence for the importance of environmental influences shared by members of the same family, effects such as social class and parental treatment. Workers who postulate that early environmental experiences are a major influence on anxiety and depression in adulthood (Parker, 1979, 1981a, 1981b) must recognize that such experiences are not necessarily shared by cotwins; experience from parents is more likely to be a function of the child's genotype than of the family environment (Eaves, 1976; Eaves *et al.*, 1978).

Cultural theories of determination are also strongly rejected as an explanation for the development of the personality traits we have measured. Individual differences in psychoticism, neuroticism and lie can be explained simply by the additive effects of genes and individual environmental experiences. For extraversion there is also evidence that dominance is important. It may be difficult for the outsider to the field to appreciate how strikingly good are the fits of our simple models when consideration is given to the power with which they are tested and the many opportunities for them to fail should the assumptions on which they are based be false.

It is not necessarily true, however, that the same genetic effects are acting in males and females for all traits, or if they are that they will produce deviations on the same scale in both sexes. In psychoticism and lie there are scalar differences between the sexes: genetic differences are more pronounced in males than females for psychoticism, while for lie the reverse is true. Environmental variance for lie is also greater in males than females. A simple genetic model has previously been found to be most appropriate for explaining variation in psychoticism (Eaves and Eysenck, 1977) and lie (Martin and Eysenck, 1976), although no significant differences between the

sexes in environmental and genetic contributions to variance were found in these smaller studies.

Neither is it true that gene effects must stay constant with age. The correlation of age with absolute within-pair differences in DZ females indicates that genetic differences in neuroticism become more pronounced as females get older, confirming a similar result in a smaller sample by Eaves and Eysenck (1976b). This sex difference is reflected in the striking evidence we found for the action of different genes on neuroticism in males rather than females, although their heritabilities are very similar. Our results for neuroticism are similar to those of Eaves and Young (1981), who found that both age and sex affected the expression of additive genetic and environmental differences in the extensive Swedish twin data of Floderus-Myrhed *et al.* (1980).

By contrast with the other variables, the results for extraversion are consistent over sexes and age. The fascinating finding for this variable which sets it apart from the others is the significant and substantial variation due to genetic dominance (Mather, 1966). This would indicate that extraversion is a character which has been subject to an evolutionary history of strong natural selection. Eaves and Young (1981), reanalyzing the data of Floderus-Myrhed *et al.* (1980), found similarly that dominant gene action affects the expression of extraversion, although there was also evidence that both age and sex affected the expression of genetic and environmental differences in extraversion.

The detection of considerable genetical non-additivity for extraversion contrasts well with the lack of evidence for dominance variance affecting neuroticism, and reinforces the view that these two traits are not only statistically independent but also quite independent in fundamental biological aspects. This finding may have important implications for the continuing controversy about the physiological basis of Eysenck's personality dimensions. Gray (1970) has argued that a 45 degree rotation of Eysenck's extraversion and neuroticism dimensions is justified on several biological grounds. Our genetical analysis ascribes quite different origins to the genetic variation for E and N. Since rotation would obscure this distinction, our results may favour Eysenck's position.

It has been asserted that cultural transmission from parents to offspring is the most important cause of familial aggregation in conservatism scores (Feather, 1978) and related attitudes (Cavalli-Sforza *et al.*, 1982). Our analysis shows, however, that a model which includes only individual and family environmental effects is totally inadequate as an explanation of variation in conservatism. In contrast to Eaves and Eysenck (1974), we also found that a model incorporating only individual environmental differences and additive genetic effects is inappropriate, although these authors acknowledge that a larger study, such as ours, might identify common environmental influences that are important to variation.

Our results are similar to those of three independent twin studies (Eaves *et al.*, 1978) which measured conservatism by three different instruments. The three studies showed remarkable consistency in assigning approximately equal proportions of variance to additive genetic effects, within-family environment and a between-families component of variation. When corrected for the effects of assortative mating, the heritabilities were around 50 per cent, while cultural effects accounted for less than 20 per cent of the total variation, and this is similar to our result.

In contrast to these studies, however, we find evidence for environmental (E_1 and

E_2) effects of different size in males and females. It seems that there is greater environmental variation in males than females, and although the cultural effects are qualitatively the same, they have less influence on female variation. While the genetic component is estimated to be the same in samples of both sexes, genetic effects apparently become more pronounced as males get older but not females. Conservatism scores are also apparently more stable over time in males, but genuine individual environmental influences are considerably more important than in females.

The high marital correlation reported for conservatism by Feather (1978) considerably inflates the genetic variance between families and appears to be as important a cause of familial aggregation of attitudes as cultural differences between families. The correlation of 0.675 is amongst the highest marital correlations for any character, physical or behavioural (Spuhler, 1968; Vandenberg, 1972), and the role of attitude concordance in mate selection and marital success needs further investigation. It might be objected that such a high marital correlation arises from convergence of attitudes after marriage rather than being an initial correlation at the time of mate selection. We know of no direct evidence to support or contradict this view. However, in an earlier study Martin (1978) found no correlation between the absolute difference in radicalism scores of husband and wife pairs and the number of years they had been married. The apparent lack of divergence between conservatism scores of MZ cotwins with age (Table 9) is not what one would expect if attitudes tended to converge towards those of spouses, although a high correlation between spouses might vitiate this test.

The final test of the validity of making generalizations from twin data about the sources of variance in the general population must be the ability to make predictions about the sources of covariation between other non-twin relatives. Such a study of conservatism was carried out by Eaves *et al.* (1978) on 445 individuals from pedigrees including parents, natural and adopted children. Fitting models to these irregular pedigrees yielded parameter estimates very similar to those from the present study, except that the most parsimonious model included only E_1, V_A and the assortative mating parameter A. Inclusion of a family environment parameter in the model did not improve the likelihood and the estimate of E_2 was small and non-significant. Competing models which included effects of cultural transmission were less parsimonious, gave no improvement in likelihood and yielded estimates of cultural transmission parameters which were small and not significantly different from zero.

In view of the current interest in cultural transmission (e.g., Cavalli-Sforza and Feldman, 1973; Cavalli-Sforza *et al.*, 1982), it would be interesting to see which items are more culture- or sex-dependent and thus stimulate the development of new scales which could be used to illustrate the mechanisms of non-hereditary transmission between generations. Our results show that conservatism, as it is currently measured, is much more dependent on genetic and within-family environmental differences than between-family cultural differences. Eaves and Eysenck (1974) have suggested that this may be due to society promoting individuality and mobility, which in turn gives greater importance to genetic and individual environmental experiences, irrespective of family environment.

The fact that attitudes are, at least in part, sensitive to cultural differences may make them a useful paradigm for the exploration of models in which gene expression and cultural effects are not independent. This is in contrast to the personality traits and symptoms studied, where the environmental differences which determine dif-

ferences are not organized on a cultural basis. The contrast between the causes of variation for social attitudes and personality supports the distinction previously made between the two and implies that attitudes do not simply result from the projection of personality variables onto the level of social attitudes.

The significant and substantial correlations between anxiety, depression and neuroticism replicate a previous finding that neuroticism is a trait which is closely associated with vulnerability to neurotic symptoms (Henderson *et al.*, 1981). Our analysis of the causes of genetical and environmental covariation of these measures shows that additive genetic effects are equally if not more important in their covariation than individual environmental factors and that genetic correlations are much higher (0.8) than environmental correlations (0.4). While the distinction between personality traits and symptoms may be justified because symptoms are transitory and take different forms (Foulds, 1965, 1974), the fact that correlations between neuroticism and the two symptoms are as high as between the symptoms themselves provides little evidence for this distinction.

Nevertheless, there are also substantial genetic effects on neuroticism (16 per cent of the total in females, 12 per cent in males) which are independent of the two symptoms we have measured. Although specific genetic variance is a small proportion of the total for depression (6 per cent in females, 9 per cent in males), it is possible that this fraction estimates the contribution made in this sample by the major gene polymorphisms which are alleged to predispose to major depression (Comings, 1979; Weitkamp *et al.*, 1981). On the other hand, the genetic factor variance (30 per cent in females, 23 per cent in males) may be regarded as the fraction contributing to neurotic or minor depression.

One hallmark of a good theory is its ability to stimulate new work. By this criterion, Eysenck's theories have certainly been successful over the past thirty years. His hypotheses concerning the nature and origin of individual differences in personality and attitudes have been subjected to increasingly stringent tests, of which the present study is one of the most exacting,and have passed them well. But where do we go from here? Numerous 'wrinkles' in the basic findings have come to light in our powerful study. What is the basis of sex and age differences in gene expression and environmental influences? If it is individual environmental influences rather than shared environment which are important in the differentiation of personality, what is the nature of these influences? Why do we detect no assortative mating for the personality dimensions when we do for most biologically important traits? Are there genes for major depression which are independent of those for minor depression? Is the genetical non-additivity detected for extraversion ambidirectional, indicating an evolutionary history of stabilizing selection towards intermediate values on this dimension? And many more questions could be asked. There is much to be done!

ACKNOWLEDGMENTS

The Australian NH&MRC Twin Registry is supported by the National Health and Medical Research Council of Australia, as was the work described in this chapter. Rosemary Jardine is supported by an Australian Commonwealth Postgraduate Award.

REFERENCES

Beadle, G. W. and Ephrussi, B. (1937) 'Development of eye colours in *Drosophila*: Diffusable substances and their interrelations', *Genetics*, **22**, pp. 76–86.

Beadle, G. W., and Tatum, E. L. (1941) 'Genetic control of biochemical reactions in *Neurospora*', *Proc. Nat. Acad. Sci.*, **48**, pp. 499–506.

Bedford, A., and Foulds, G. A. (1977) 'Validation of the delusions-states inventory', *Brit. J. Med. Psychol.* **50**, pp. 163–71.

Bedford, A., Foulds, G. A. and Sheffield, B. F. (1976) 'A new personal disturbance scale (DSSI/sAD)', *Brit. J. Soc. Clin. Psychol.*, **15**, pp. 387–94.

Cavalli-Sforza, L. L. and Feldman, M. W. (1973) 'Cultural versus biological inheritance: Phenotypic transmission from parents to children (a theory of the effect of parental phenotypes on children's phenotypes)', *Am. J. Hum. Genet.*, **25**, pp. 618–37.

Cavalli-Sforza, L. L., Feldman, M. W., Chen, K. H. and Dornbusch, S. M. (1982) 'Theory and observation in cultural transmission', *Science*, **218**, pp. 19–27.

Cederlöf, R., Friberg, L., Jonsson. E. and Kaij, L. (1961) 'Studies on similarity diagnosis in twins with the aid of mailed questionnaires', *Acta Genet. Stat. Med.*, **11**, pp. 338–62.

Clark, P., Jardine, R., Martin, N. G., Stark, A. E. and Walsh, R. J. (1980) 'Sex differences in the inheritance of some anthropometric characters in twins', *Acta Genet. Med. Gemellol.*, **29**, pp. 171–92.

Clifford, C. A., Fulker, D. W. and Murray, R.M. (1981) 'A genetic and environmental analysis of obsessionality in normal twins', *Twin Research 3: Intelligence, Personality, and Development*, New York, Alan R. Liss, pp. 163–8.

Comings, D. E. (1979) 'Pc I Duarte, a common polymorphism of a human brain protein, and its relationship to depressive disease and multiple sclerosis', *Nature*, **277**, pp. 28–32.

Eaves, L. J. (1976) 'A model for sibling effects in man', *Heredity*, **36**, pp. 205–14.

Eaves, L. J. (1977) 'Inferring the causes of human variation'. *J. Roy. Statist. Soc.*, A, **140**, pp. 324–55.

Eaves, L. J. and Eysenck, H. J. (1974) 'Genetics and the development of social attitudes', *Nature*, **249**, pp. 288–9.

Eaves, L. J. and Eysenck, H. J. (1975) 'The nature of extraversion: A genetical analysis', *J. Person. Soc. Psych.*, **32**, pp. 102–12.

Eaves, L. J. and Eysenck, H. J. (1976a) 'Genetical and environmental components and unrepeatability in twin's responses to a neuroticism questionnaire', *Behav. Genet.*, **6**, pp. 145–60.

Eaves, L. J. and Eysenck, H. J. (1976b) 'Genotype x age interaction for neuroticism', *Behav. Genet.*, **6**, pp. 359–62.

Eaves, L. J. and Eysenck, H. J. (1977) 'A genotype-environmental model for psychoticism', *Adv. Behav. Res. Therapy*, **1**, pp. 5–26.

Eaves, L. J. and Young, P. A. (1981) 'Genetical theory and personality differences', in Lynn, R. (Ed.), *Dimensions of Personality*, Oxford, Pergamon Press, pp. 129–79.

Eaves, L. J. Last, K., Martin, N. G. and Jinks, J. L. (1977a) 'A progressive approach to non-additivity and genotype-environmental covariance in the analysis of human differences', *Br. J. Math. Statist. Psychol.*, **30**, pp. 1–42.

Eaves, L. J. Martin, N.G. and Eysenck, S. B. G. (1977b) 'An application of the analysis of covariance structures to the psychogenetical study of impulsiveness', *Br. J. Math. Statist. Psychol.*, **30**, pp. 185–97.

Eaves, L. J., Last, K., Young, P. A. and Martin, N. G. (1978) 'Model-fitting approaches to the analysis of human behaviour', *Heredity*, **41**, pp. 249–320.

Eysenck, H. J. and Eysenck, S. B. G. (1975) *Personality Questionnaire* (Junior and Adult), Hodder and Stoughton Educational, Buckhurst Hill, Essex, Chigwell Press.

Eysenck, H. J. and Prell, D. B. (1951) 'The inheritance of neuroticism', *J. Ment. Sci.*, **97**, pp. 441–65.

Eysenck, H. J. and Prell, D. B. (1956) 'The inheritance of introversion-extraversion', *Acta Psychologica*, **12**, pp. 95–110.

Eysenck, S. B. G., Humphery, N. and Eysenck, H. J. (1980) 'The structure of personality in Australian as compared with English subjects', *J. Social Psych.*, **112**, pp. 167–73.

Feather, N. T. (1975) 'Factor structure of the conservatism scale', *Australian Psychologist*, **10**, pp. 179-84.

Feather, N. T. (1977) 'Generational and sex differences in conservatism', *Australian Psychologist*, **12**, pp. 76–82.

Feather, N. T. (1978) 'Family resemblances in conservatism: Are daughters more similar to parents than sons are?' *J. Personality*, **46**, pp. 260–78.

Fisher, R. A. (1931) *The Genetical Theory of Natural Selection*, London, Oxford University Press.
Floderus-Myrhed, B., Pederson, N. and Rasmuson, I. (1980) 'Assessment of heritability for personality, based on a short-form of the Eysenck Personality Inventory. A study of 12898 pairs', *Behav. Genet.*, **10**, pp. 153–62.
Foulds, G. A. (1965) *Personality and Personal Illness*, London, Tavistock Publications.
Foulds, G. A. (1974) *The Hierarchical Nature of Personal Illness*, London, Academic Press.
Freud, S. (1937) *Analysis Terminable and Interminable*, London, Collected Papers, No. 5, Hogarth, pp. 316–57.
Fulker, D. W. (1978) 'Multivariate extensions of a biometrical model of twin data', *Twin Research: Psychology and Methodology*, New York, Alan R. Liss, pp. 217–36.
Fulker, D. W. (1981) 'The genetic and environmental architecture of psychoticism, extraversion and neuroticism', in Eysenck, H. J. (Ed.), *A Model for Personality*, Berlin, Springer-Verlag, pp. 89–122.
Gray, J. (1970) 'The psychophysiological basis of introversion-extraversion', *Behav. Res. Therapy*, **8**, pp. 249–66.
Henderson, A. S., Byrne, D. G. and Duncan-Jones, P. (1981) *Neurosis and the Social Environment*, Sydney, Academic Press.
Jinks, J. L. and Fulker, D. W. (1970) 'Comparison of the biometrical genetical, MAVA, and classical approaches to the analysis of human behaviour', *Psychol. Bull.*, **73**, pp. 311–48.
Kasriel, J. and Eaves, L. J. (1976) 'A comparison of the accuracy of written questionnaires with blood-typing for diagnosing zygosity in twins', *J. Biosoc. Sci.*, **8**, pp. 263–6.
Martin, N. G. (1975) 'The inheritance of scholastic abilities in a sample of twins', *Ann. Hum. Genet.*, **39**, pp. 219–29.
Martin, N. G. (1978) 'Genetics of sexual and social attitudes in twins', *Twin Research: Psychology and Methodology*, New York, Alan R. Liss, pp. 13–23.
Martin, N. G. and Eaves, L. J. (1977) 'The genetical analysis of covariance structure', *Heredity*, **38**, pp. 219–29.
Martin, N. G. and Eysenck, H. J. (1976) 'Genetic factors in sexual behaviour', in Eysenck, H. J. *Sex and Personality*, London, Basic Books, pp. 192–219.
Martin, N. G. and Martin, P. G. (1975) 'The inheritance of scholastic abilities in a sample of twins. I. Ascertainment of the sample and diagnosis of zygosity', *Ann. Hum. Genet.*, **39**, pp. 213–18.
Martin, N. G., Eaves, L. J., Kearsey, M. J. and Davies, P. (1978) 'The power of the classical twin study', *Heredity*, **40**, pp. 97–116.
Martin, N. G., Eaves, L. J. and Fulker, D. W. (1979) 'The genetical relationship of impulsiveness and sensation seeking to Eysenck's personality dimensions', *Acta Genet. Med. Gemellol.*, **28**, pp. 197–210.
Martin, N. G., Gibson, J. B., Oakeshott, J. G., Wilks, A. V., Starmer, G. A., Craig, J. and Perl, H. (1981) 'A twin study of psychomotor performance during alcohol intoxication: Early results', *Twin Research 3: Epidemiological and Clinical Studies*, New York, Alan R. Liss, pp. 89–96.
Mather, K. (1966) 'Variability and selection', *Proc. Roy. Soc. Lond. B*, **164**, pp. 328–40.
Mather, K. (1974) 'Non-allelic interactions in continuous variation of randomly breeding populations', *Heredity*, **32**, pp. 414–19.
Newman, H. H., Freeman, F. N. and Holzinger, K. J. (1937) *Twins: A Study of Heredity and Environment*, Chicago, University of Chicago Press.
Nichols, R. C. and Bilbro, W. C. (1966) 'The diagnosis of twin zygosity', *Acta Genet. Stat. Med.*, **16**, pp. 265–75.
Parker, G. (1979) 'Parental characteristics in relation to depressive disorders', *Brit. J. Psychiat.*, **134**, pp. 138–47.
Parker, G. (1981a) 'Parental reports of depressives: An investigation of several explanations', *J. Affect. Dis.*, 3, pp.131–40.
Parker, G. (1981b) 'Parental representations of patients with anxiety neurosis', *Acta Psychiat. Scand.*, **63**, pp. 33–6.
Rao, D. C., Morton, N. E. and Yee, S. (1974) 'Analysis of family resemblance II', *Am. J. Hum. Genet.*, **26**, pp.331–59.
Shields, J. (1962) *Monozygotic Twins*, Oxford, Oxford University Press.
Snedecor, G. W. and Cochran, W. G. (1980) *Statistical Methods*, 7th ed., Iowa State University Press.
Spuhler, J. N. (1968) 'Assortative mating with respect to physical characteristics', *Eugen. Quart.*, **15**, pp. 128–40.
Torgersen, S. (1983) 'Genetics of neurosis: The effects of sampling variation upon the twin concordance ratio', *Brit. J. Psychiat.*, **142**, pp. 126–32.

Vandenberg, S. G. (1972) 'Assortative mating, or who marries whom?' *Behav. Genet.*, **2**, pp. 127–57.
Weitkamp, L. R., Stanger, H. C., Persad, E., Flood, C. and Guttormsen, S. (1981) 'Depressive disorders and HLA: A gene on chromosome 6 that can affect behavior', *New Engl. J. Med.*, **305**, pp. 1301–6.
Wilson, G. D. and Patterson, J. R. (1968) 'A new measure of conservatism,' *Brit. J. Soc. Clin. Psychol.*, **7**, pp. 264–9.
Young, J. P. R., Fenton, G. W. and Lader, M. H. (1971) 'The inheritance of neurotic traits: A twin study of the Middlesex Hospital Questionnaire', *Brit. J. Psychiat.*, **119**, pp. 393–8.