Person & Ind. Diff. Vol. 1, pp. 35 to 55
Pergamon Press Ltd., 1980. Printed in Great Britain

INTERGENERATIONAL STABILITY AND CHANGE IN THE CAUSES OF VARIATION IN PERSONALITY

P. A. YOUNG,* L. J. EAVES† and H. J. EYSENCK‡

(*Received* 15 *July* 1979)

Summary—A combination of twin and parent–offspring data on the EPQ and JEPQ is subjected to genotype–environmental analysis by the balanced pedigree method. The method, based upon Jöreskog's approach to the analysis of covariance structures, employs the principle of maximum-likelihood and permits tests of the model and the extraction of standard errors for parameter estimates. The data for extraversion and neuroticism are on the whole consistent with a simple model which assumes additive gene action, random mating and environmental effects within families. For the psychoticism dimension and the lie scale mating is found to be non-random. The stability of personality expression across generations is investigated. With the exception of the neuroticism dimension there is a marked inconsistency of gene action between juveniles and adults. For the lie scale social interaction between juvenile cotwins is detected and the juvenile scale is suggested as a paradigm of a trait for which the environmental interactions between relatives have a major role in the causes of individual variability.

1. INTRODUCTION

Although animal studies of behaviour have clearly demonstrated that gene-expression can change markedly with age (Broadhurst and Jinks, 1966), the analysis of such developmental changes in man is more difficult, because of the expense of longitudinal studies and the associated problems of maintaining the long-term cooperation of human subjects. The modification of the effects of genes and environment during development, however, remains critical both for our understanding of behavioural changes in general and for the practical application of psychometric tests to the long term prediction of behavioural patterns. Using designs based on the classical twin study, Wilson (1972) has shown how the profiles of twins' cognitive development are apparently under genetical control. The last two decades have seen much effort expended upon formulating and discriminating between hypotheses about the causes of variation in the normal personality. The majority of such studies have considered adolescents or adults in isolation and almost all have relied upon the classical twin study experimental design. The assumption seems to have been that results obtained at one age will be comparable with those from the study of another age group. However, little or no evidence has been provided to support this often implicit assumption. It is now almost ten years since it was demonstrated (Rachman, 1969; Eysenck, 1969) that the personality dimensions of neuroticism and extraversion could be measured in children as young as 7 or 8 and that the factor structures so defined showed a high correlation with those of much older children. Nevertheless, few behaviour genetic studies have addressed the question of the age dependence of personality. Although some studies have attempted to compare results obtained from the administration of the same questionnaire to separate samples at different ages (Reznikoff and Honeyman, 1967; Horn *et al.*, 1976), and others (e.g. Partanen *et al.*, 1966; Claridge *et al.*, 1973) have compared older and younger twins by splitting their samples at the mean, or some other arbitrary age, few substantive conclusions have emerged or could be expected to emerge from such crude designs. Dworkin *et al.* (1976, 1977) employing a retrospective longitudinal approach, present evidence for significant adolescence to adulthood change. They reascertained part of the sample,

* Department of Genetics, University of Birmingham.
† Department of Experimental Psychology, University of Oxford.
‡ Institute of Psychiatry, University of London.

as adults, originally studied 12 yr earlier, in adolescence, by Gottesman (1965, 1966). Dworkin *et al.* argue for the involvement of genetic factors not only in the expression of traits at both ages, but also in the process of change in expression over time. However, the small size of the sub-sample participating at both ages, 42 twin pairs, reflecting typical sample wastage in longitudinal studies, obviously casts doubt upon the importance of the finding that for many scales there was an absence of significant genetic variance. Eaves and Eysenck (1976 a) similarly suggest that long-term changes in neurotic behaviour may be under genetic control. This assertion is based upon the presence of a significant correlation between DZ twin intrapair differences and age, matched by the lack of such a correlation for MZ twins.

Prospective longitudinal studies (e.g. Vandenberg *et al.*, 1968; Wilson, 1977) must in the long run be expected to provide the best description of personality change, and the best source of data to elucidate the cause of change over extended periods of time, so long as the problems of sample wastage can be overcome. However, very few such studies concerned with behavioural variables are in progress, let alone nearing fruition. Therefore, the cross-sectional family study is proposed as a viable and attractive alternative. In its simplest form the family study would consist of a nucleus of twin families comprising twins and both parents. This design may be extended by including other types of family grouping or persons having different degrees of relationship to the members of the nuclear family.

Implicit in the naming and construction of the scales of the adult and junior forms of the Eysenck Personality Questionnaire (EPQ) (Eysenck and Eysenck, 1975) is the belief that the primary dimensions of variation in adult personality have their antecedents in juvenile personality structure. Despite this belief, no previous attempt has been made to relate the causes of variation in the respective scales of the two forms of the questionnaire. This aim would seem to be increasingly desirable, considering the recent proliferation of studies attempting to infer adult behaviour from the personality and behaviour of juveniles.

A family study in the sense outlined above, extended by the inclusion of singleton, that is only-child, families, was conducted. The results of this study were then coupled with data previously collected on a large sample of adult twins, in order to allow the investigation of the causes of variation and covariation of the respective scales of the adult and junior forms of the EPQ.

2. THE DATA

The questionnaire results analysed in this paper were collected from two independent sources. The juvenile families of twins and singletons together with their parents were contacted via appeals in the popular press and on radio programmes. The families were mailed copies of the adult and junior versions of the EPQ and asked to complete them separately. The distribution of the twin and singleton families by sex, and the twin families by zygosity, is summarised in Table 1(i). The ages of the juvenile respondents lay between 7 and 17 yr with a mean of 11.06, and the mean differences in age, between the groups represented in 1(i), were not significant. There was some over representation of middle class families, as is common in volunteer studies.

The second source of data was the EPQ responses of a large sample of twins who are members of the Institute of Psychiatry twin register. The adult twins [Table 1(ii)] were a heterogeneous group with respect to age, having a mean of 30.50 yr and ranging from 16 to 86 yr. There was a significant difference in mean age between the adult twin group and the parents of the juvenile families, the mean age of the latter being 41.36 yr. The twins' zygosities were diagnosed primarily by the responses to the two questions, "In childhood, were you frequently mistaken by people who knew you?" and "Do you differ markedly in physical appearance and colouring?" (Kasriel and Eaves, 1976), although in some cases further information on physical resemblance and the results of blood-typing were available (Young, 1977).

Table 1. Composition of (i) juvenile family and (ii) adult twin samples

	Male	Female	Unlike sex
(i) Juvenile families			
Monozygotic (pairs)	59	50	
Dizygotic (pairs)	40	37	76
Singleton	85	97	
(ii) Adult twins			
Monozygotic (pairs)	70	233	
Dizygotic (pairs)	47	125	68

3. DATA SUMMARY

Previous investigations of the three primary scales of the EPQ (Eaves and Eysenck, 1977; Eaves *et al.*, 1977 p. 26) indicated the possible presence of detectable genotype–environmental interaction in the present data, resulting from heteroscedasticity caused by the choice of scale. The data are consistent with a binomial choice model in the case of the extraversion, neuroticism and lie scales. On the other hand a Poisson model, which assumed 'psychotic' responses are distributed as independent rare events, was more appropriate for the P scale. These results, being in agreement with previous studies, were taken as indications of appropriate transformations to remove the undesirable effects of the present scales of measurement. A modified square-root transformation was applied to the adult and juvenile P scales, whilst an angular transformation (Mosteller and Youtz, 1961) was used on the remaining scales to secure scales with uniform error variance.

The scores were corrected for their linear dependence upon age by removing the linear component of the regression on age, while at the same time the mean was standardised to zero. This procedure was applied separately to the two sexes and to all four adult and juvenile scales.

The covariance matrices were calculated for each of the seven types of family group and for the five classes of adult twin. These matrices are presented separately in Tables 2–5 for each of the four scales of the EPQ. Recognising the symmetry of the individual matrices, the actual covariances are given above the leading diagonal of each matrix, while below it are included the corresponding correlations which may give a better intuitive grasp of the properties of the data. For each trait considered there are 77 independent statistics: 5×10 from the juvenile twin families, 5×3 from the adult twins and 2×6 from the singleton families. The intention is to summarise these in the most economical manner.

Before commencing with the testing of genotype–environmental models, an inspection of the data matrices with the aid of hindsight will throw light upon some of those aspects of the data that must be taken into account in the models. Considering the marital correlations first, only for the lie scale is there an obvious and significant covariance between the parents of the families. In fact, by fitting preliminary empirical models to the data, it was found that the covariance between parents for psychoticism was also significant and positive. However, for extraversion and neuroticism there was no detectable assortative mating. Secondly there is in general significant covariance between members of a twin pair. Thus there appear to be some between family effects to explain. In the case of the adults the covariance of MZ twins is greater than that of DZ twins for all four traits, suggesting that gene effects are probably implicated in all four domains of adult personality; for the juvenile scales the same can be said for E and N but the situation is much less clear for the lie scale and psychoticism.

The correlations between parents and offspring for all four scales are obviously variable in magnitude and suggest the possibility of sex linkage. If a significant proportion of the loci contributing to the expression of a polygenic trait lie on the X chromosome, then we may expect to find differences in the magnitude of the parent–offspring correlation,

Table 2. Covariances (upper triangles), variances (diagonals) and correlations (lower triangles) between relatives for extraversion

Group	Juvenile twin/singleton families					Adult twins	
	df	Mother	Father	Child 1	Child 2	*df*	Twin 1	Twin 2
MZ_m	58	0.070	0.010	0.010	0.016	69	0.075	0.046
		0.143	0.071	0.011	0.003		0.639	0.070
		0.212	0.228	0.033	0.020			
		0.311	0.063	0.562	0.038			
MZ_f	49	0.071	0.014	0.010	0.012	232	0.070	0.032
		0.228	0.056	0.013	0.006		0.472	0.063
		0.213	0.291	0.033	0.024			
		0.217	0.112	0.624	0.043			
DZ_m	39	0.078	−0.001	0.003	0.019	46	0.063	0.010
		−0.011	0.085	0.004	0.010		0.162	0.058
		0.051	0.076	0.034	0.000			
		0.364	0.177	0.009	0.036			
DZ_f	36	0.088	−0.014	0.003	0.005	124	0.080	0.013
		−0.209	0.048	0.005	−0.010		0.169	0.077
		0.053	0.141	0.030	0.006			
		0.091	−0.251	0.177	0.033			
DZ_{mf}	75	0.062	0.001	0.009	0.008	67	0.062	0.009
		0.009	0.079	0.002	−0.003		0.159	0.052
		0.243	0.044	0.024	0.003			
		0.154	−0.057	0.091	0.043			
Singleton m	84	0.086	0.002	0.000				
		0.022	0.068	0.007				
		0.001	0.133	0.046				
Singleton f	96	0.072	0.014	0.008				
		0.170	0.092	0.007				
		0.134	0.104	0.045				

dependent upon the sex of the family members considered. Thus restricting the argument to man, where the female is the homogametic sex and the male heterogametic, we expect in the presence of significant sex linkage (Mather and Jinks, 1971), that

$$r_{\text{Mother/Son}} = r_{\text{Father/Daughter}} \geqslant r_{\text{Mother/Daughter}} > r_{\text{Father/Son}}.$$

Although variable, the parent–offspring covariances do not differ significantly for any one of the four EPQ traits and those small differences that exist do not follow the expected pattern for sex-linked inheritance. Lastly a comparison of total variances can be made. There are no obvious differences between the variance of the MZ and DZ juvenile or adult twins for either of the four traits. Also the variances of the parental groups are not in any systematic way different from the variances of the adult twins, which provides some support for the contention that the twins are not atypical of the general population. The one notable feature to emerge from an inspection of the variances, however, is the difference in variance between singletons and twins for the two traits extraversion and lie. This difference suggests, that possibly for these traits, a simple model involving only additive environmental and gene effects will not be suitable.

4. FITTING THE MODEL

In the past, several estimation procedures have been adopted, ranging from obtaining the first solution which springs readily to the eye without regard for efficiency, to the implementation of the principle of maximum-likelihood for fitting multifactorial models to individual unreduced data (e.g. Lange *et al.*, 1976). The former approach has much to commend it as a thoughtful basis for obtaining trial parameter values, but cannot be defended as a definitive solution, because it yields neither tests of the model nor standard errors for the parameter estimates. The latter approach is necessary, but time consuming, when there is no clearly defined and consistent pedigree structure to the data. Between these two extremes there are a variety of efficient techniques which rely upon prior data summaries. When the data permit summary in terms of the independent mean squares of the analysis of variance, as in the case of the classical twin study, the approach of

Table 3. Covariances (upper triangles), variances (diagonals) and correlations (lower triangles) between relatives for neuroticism

Group	Juvenile twin/singleton families *df*	Mother	Father	Child 1	Child 2	Adult twins *df*	Twin 1	Twin 2
MZ_m	58	0.078	0.009	0.010	0.011	69	0.070	0.037
		0.144	0.054	0.017	0.013		0.511	0.076
		0.153	0.291	0.059	0.030			
		0.149	0.211	0.456	0.072			
MZ_f	49	0.070	−0.011	0.032	0.023	232	0.065	0.028
		−0.155	0.070	0.018	0.012		0.425	0.066
		0.460	0.257	0.069	0.029			
		0.342	0.178	0.436	0.065			
DZ_m	39	0.079	−0.009	0.028	0.004	46	0.054	0.001
		−0.153	0.042	−0.005	−0.003		0.021	0.059
		0.381	−0.085	0.069	0.000			
		0.059	−0.057	0.007	0.058			
DZ_f	36	0.059	0.000	−0.004	0.022	124	0.063	0.004
		−0.005	0.068	−0.007	−0.005		0.066	0.065
		−0.082	−0.138	0.037	0.016			
		0.427	−0.081	0.403	0.045			
DZ_{mf}	75	0.041	0.001	0.009	0.008	67	0.069	0.011
		0.027	0.057	0.022	0.001		0.167	0.060
		0.156	0.329	0.077	0.023			
		0.146	0.010	0.293	0.077			
Singleton m	84	0.086	0.001	0.019				
		0.015	0.071	0.006				
		0.263	0.089	0.058				
Singleton f	96	0.058	−0.005	0.022				
		−0.089	0.060	0.002				
		0.359	0.040	0.063				

Table 4. Covariances (upper triangles), variances (diagonals) and correlations (lower triangles) between relatives for psychoticism

Group	Juvenile twin/singleton families *df*	Mother	Father	Child 1	Child 2	Adult twins *df*	Twin 1	Twin 2
MZ_m	58	0.402	−0.062	−0.010	0.010	69	0.557	0.303
		−0.137	0.503	−0.032	−0.036		0.543	0.558
		−0.027	−0.074	0.362	0.157			
		0.023	−0.074	0.376	0.482			
MZ_f	49	0.447	0.072	0.057	0.093	232	0.398	0.170
		0.164	0.431	0.060	−0.031		0.422	0.408
		0.132	0.140	0.420	0.182			
		0.200	−0.068	0.401	0.488			
DZ_m	39	0.529	0.047	0.034	−0.059	46	0.397	0.047
		0.100	0.412	−0.037	0.012		0.115	0.429
		0.068	−0.085	0.473	0.115			
		−0.138	0.032	0.286	0.343			
DZ_f	36	0.358	0.161	0.102	0.026	124	0.486	0.183
		0.377	0.509	0.111	0.051		0.355	0.548
		0.245	0.224	0.488	0.176			
		0.064	0.109	0.380	0.440			
DZ_{mf}	75	0.584	0.136	−0.019	0.128	67	0.523	0.100
		0.227	0.613	0.074	0.139		0.192	0.515
		−0.036	0.138	0.469	0.117			
		0.238	0.252	0.242	0.499			
Singleton m	84	0.432	0.071	0.065				
		0.161	0.458	−0.013				
		0.159	−0.031	0.388				
Singleton f	96	0.440	0.162	0.055				
		0.328	0.554	0.076				
		0.130	0.160	0.405				

Table 5. Covariances (upper triangles), variance (diagonals) and correlations (lower triangles) between relatives for the lie scales of the EPQ and JEPQ

Group	Juvenile twin/singleton families					Adult twins		
	df	Mother	Father	Child 1	Child 2	*df*	Twin 1	Twin 2
MZ_m	58	0.035	0.016	0.012	0.011	69	0.055	0.027
		0.399	0.044	0.012	0.013		0.513	0.051
		0.347	0.311	0.034	0.023			
		0.285	0.294	0.588	0.045			
MZ_f	49	0.037	0.009	0.010	0.003	232	0.044	0.022
		0.271	0.032	0.010	0.009		0.516	0.042
		0.297	0.300	0.033	0.017			
		0.090	0.239	0.460	0.040			
DZ_m	39	0.025	0.014	0.007	0.003	46	0.025	−0.001
		0.404	0.046	0.005	−0.003		−0.041	0.035
		0.209	0.123	0.038	0.021			
		0.123	−0.081	0.647	0.029			
DZ_f	36	0.047	0.011	0.029	0.005	124	0.047	0.020
		0.299	0.028	0.016	0.006		0.429	0.045
		0.525	0.366	0.067	0.031			
		0.104	0.164	0.549	0.048			
DZ_{mf}	75	0.035	0.012	0.009	0.013	67	0.051	0.008
		0.355	0.034	0.009	0.009		0.170	0.039
		0.229	0.233	0.048	0.022			
		0.304	0.209	0.442	0.050			
Singleton m	84	0.032	0.012	0.002				
		0.328	0.045	0.002				
		0.083	0.048	0.028				
Singleton f	96	0.036	0.008	0.008				
		0.215	0.039	0.004				
		0.251	0.112	0.028				

weighted least squares is simple and yields estimates which approximate closely to the maximum-likelihood estimates. When, as in the present case, the data are more effectively summarised by covariance matrices, the method of WLS becomes more tedious because of the need to specify the covariances between all the different statistics in the sample. The availability of new and powerful computer software for non-linear optimisation places the application of the methods of maximum-likelihood well within the scope of practical analysis.

Following, for example, Jöreskog (1973) we write

$$\text{Log } L = -\tfrac{1}{2}N\,[\log|\boldsymbol{\Sigma}| + \text{tr}\,(\mathbf{S}\,\boldsymbol{\Sigma}^{-1})] + \text{constant}$$

for the log-likelihood of obtaining the observed mean products matrix **S**, where N are the degrees of freedom of the observed matrix and $\boldsymbol{\Sigma}$ is the matrix of expectations. If we assume that the observations are generated by a distribution which is multivariate normal, then the log-likelihood of obtaining a given set of m independent observed covariance matrices, may be obtained from

$$\log L = -\tfrac{1}{2}\sum_{i=1}^{m} N_i[\log|\boldsymbol{\Sigma}_i| + \text{tr}\,(\mathbf{S}_i\,\boldsymbol{\Sigma}_i^{-1})]$$

(omitting the constant term).

Given that the expectations have been formulated in terms of p parameters, we then require the vector of parameter estimates **p** that maximises the Log L. Maximising Log L is equivalent to minimising $-\text{Log } L = F$.

In practice there are many ways of solving numerically a minimisation problem of the type proposed here. We chose, as did Martin and Eaves (1977), to implement a program of our own, based upon the Numerical Algorithms Group (1974) Fortran subroutine E04HAF for non-linear constrained minimisation. There was found to be no need, in practice, for the use of constraints to obtain solutions as the routine moved fairly easily and quickly towards the minimum. The routine E04HAF allows a degree of flexibility in the choice of method used for minimisation. In all analyses reported below,

a method employing first and second derivatives, as well as the actual function values, was used in a modification of Newton's method. The first derivatives were evaluated numerically by the central-difference method, whilst the numerical approximations to the second derivatives were calculated using a forward-difference formula.

5. TESTING THE MODEL

Again following Jöreskog, given our proposed model H_1, we may test this against an alternative model, a less restricting hypothesis, H_0 say, since under H_0, twice the difference between the likelihoods of the alternative models, $2(L_0 - L_1)$, is distributed in large samples as χ^2 with degrees of freedom equal to the difference in the number of parameters estimated under H_1 and H_0. The H_0 used was such as to allow every expected statistic to take its observed value. Thus the test statistic will summarise how much of the observed pattern of variances and covariances is left unexplained by the proposed model.

6. RESULTS

6.1. *The simple genotype–environmental model*

The model fitting procedure will be illustrated by applying it to the simplest of all hypotheses that includes both genetical and environmental causes of variation. Eaves and Eysenck (1975, 1976, 1977) found that variation within and between pairs of adult twins for E, N and P was adequately described by reference to only two parameters, E_1, the result of environmental effects specific to individuals, and, D_R, the combined additive effects of the loci contributing to variation in that trait. This model was, therefore, chosen as the first attempt to explain the present data.

The data consist of the responses of juveniles to the junior version of the EPQ and of adults to the standard EPQ. How do we treat the relationship between each trait as measured on each version of the scale? We could hypothesise that exactly the same range of environments and gene effects are responsible for determining the traits as measured at both stages of development and would therefore be justified in using the same parameters to describe variation in the juveniles and adults. The test of the model would then in part be a test of this hypothesis. However, if the model failed we would have no information concerning the degree to which effects expressed during youth may be expected to continue their expression into adulthood. For the latter reason, the effects were specified separately in the two age groups, and the parent–offspring covariance was used as a means of assessing this continuity of expression.

At least four parameters are required to describe the mean squares and mean products, two representing specific environmental effects in juveniles (E_{1J}) and adults (E_{1A}), and also parameters representing the additive effects of genes at both stages of development, D_{RJ} and D_{RA}. Eaves and Eysenck (1975, 1977) consider in some detail the specification of simple gene–environment models applicable to twin data. Mather and Jinks (1971) explain the specification of additive and dominance, to be encountered later, genic variation in populations having unequal frequencies of increasing and decreasing alleles, in terms of D_R and H_R respectively. A fifth parameter, D_{RAJ}, is also required to represent the covariance of the additive genetical effects expressed in the scores of adults on the standard EPQ, with those additive genetical effects causing variation in the scores of juveniles, on the junior version of the questionnaire.

The assumptions implied by the five parameter model are:

(i) All environmental effects contributing to variation are specific to individuals.
(ii) All gene effects are additive and the loci act independently, i.e. there is no dominance, epistasis or linkage disequilibrium.
(iii) The effects of interactions between genotype and environment are either absent or confounded with the environmental effects specific to individuals.
(iv) There is no covariance of genotypic and environmental deviations.
(v) Mating is random for the traits considered.
(vi) Sex linkage and sex limitation are absent.

These assumptions may be expressed in terms of precise algebraic expectations for the observed variances and covariances (Table 6).

Next, the five element vector of parameter estimates is obtained, representing the maximum likelihood solution for this model (Table 6), given the observed matrices. A test of the model may be applied,

$$\chi^2{}_{n-p} = 2(L_0 - L_1)$$

with $df = 77 - 5 = 72$, in order to judge the adequacy of the model.

In Table 7 are given the maximum-likelihood parameter estimates and their standard errors for the simple genotype–environmental model fitted to the summary statistics of all four traits, together with the chi-square values for testing the model. Only for the lie scale is the five parameter model not an adequate description of the observed mean squares and mean products matrices.

Table 6. Expected variances and covariances of the simple five parameter genotype–environmental model

Statistic	Expectation
Variances	
Juveniles	$\frac{1}{2} D_{RJ} + E_{1J}$
Adults	$\frac{1}{2} D_R + E_{1A}$
Covariances	
Juvenile MZ twins	$\frac{1}{2} D_{RJ}$
Juvenile DZ twins	$\frac{1}{4} D_{RJ}$
Adult MZ twins	$\frac{1}{2} D_{RA}$
Adult DZ twins	$\frac{1}{4} D_{RA}$
Parent-offspring	$\frac{1}{4} D_{RAJ}$
Spouses	0

The covariances of the parameter estimates are obtained from the inverse of the matrix of second partial derivatives of the log-likelihood function with respect to each parameter in the model. For the purpose of constructing tests of significance, the ideal method of differentation would be algebraic. However, the production of algebraic solutions for the second derivatives of different, many non-linear, models, would be extremely time consuming and require repetition for each new model considered. Instead, the feasibility of numerical differentiation was explored. The method finally employed is that represented by formulae 25.3.24 and 25.3.27 in Davis and Polonsky (1965, p. 884), and required $(p^2 + 3p + 1)$ function evaluations given a model containing p parameters. The square roots of the elements of the leading diagonal of the inverse of the matrix of second derivatives may be taken as approximate values for the standard errors of the estimates and used to construct normal deviate tests of the significance of the parameters.

6.2. *Extraversion*

All five parameters are highly significant and this model provides a good explanation of the observed covariance matrices ($\chi^2_{72} = 7\ 3.80$, $p = 0.42$). The suggestion earlier that there was no evidence for assortative marriage for extraversion has been confirmed by the fit of the simple model which specified a null parameter for the parental covariance. Allowing the parental covariance to deviate from zero resulted in only a small and non-significant change ($\chi^2_1 = 2.22$, $0.2 > p > 0.1$) in the χ^2 testing the goodness of fit of the model.

However, before accepting this model, the possibility must be explored that the covariance of twins, and that of parents and their offspring, is due to environmental factors. In other words, is the family environment, as opposed to genetic influences, significant in the development of extraversion? To this end we introduce two parameters, E_{2A} and E_{2J}, to represent the variation and covariation of contemporaries, due to common family

Table 7. Estimate of parameters and chi-square tests of the model for the simple genotype–environmental model fitted to the four scales of the EPQ and JEPQ

Parameter	Scale			
	Extraversion	Neuroticism	Psychoticism	Lie scale
E_{1A}	0.035 ± 0.003	0.038 ± 0.003	0.24 ± 0.02	0.021 ± 0.001
D_{RA}	0.073 ± 0.007	0.053 ± 0.006	0.46 ± 0.04	0.040 ± 0.004
E_{1J}	0.017 ± 0.002	0.036 ± 0.004	0.25 ± 0.03	0.016 ± 0.002
D_{RJ}	0.041 ± 0.006	0.056 ± 0.010	0.36 ± 0.06	0.044 ± 0.005
D_{RAJ}	0.024 ± 0.006	0.046 ± 0.007	0.13 ± 0.05	0.025 ± 0.004
χ^2_{12}	73.80	87.20	70.11	145.47*

* $p < 0.001$.

environmental influences, in adults and juveniles respectively. We also specify E_{AJ} to represent the covariance of parents and offspring resulting from purely environmental influences. Under this model the expectation for the total variance within either generation is $E_1 + E_2$, the covariance of MZ and DZ twins is simply E_2, and the parent–offspring covariance is E_{AJ}. In later model fitting to psychoticism, this covariance, E_{AJ}, will be made more explicit in terms of the regression of the juvenile onto the parental phenotype. However, the fit of this five parameter 'environmental' model to the extraversion data was not good ($\chi^2_{12} = 111.36$, $p = 0.002$) and this model was rejected as an alternative explanation of this data.

The five parameter genotype–environmental model was therefore accepted as an adequate and parsimonious explanation of the data on extraversion. Nevertheless, this does not exclude the possibility that more complicated explanations may explain slightly more of the variation than does the present model. For instance, the possibility that siblings may form part of each other's developmentally significant environment (Eaves, 1976) was considered, since preliminary investigations into the juvenile data alone had suggested this possibility. The model was developed in terms of regression theory. We may follow Eaves (1976) and consider the direct and indirect effects of a single locus with alleles A, a. However, instead of defining separate indirect effects of the locus, we represent the indirect effects by their regression onto the direct effects,

Genotype	AA	Aa	aa
Direct effect on phenotype	$+d_a$	h_a	$-d_a$
Indirect effect on sibling	$+bd_a$	bh_a	$-bd_a$.

Assuming b to be the same at each and every locus, it can be shown that we may replace Eaves' parameters D'_R, D''_R by bD_R and b^2D_R respectively. If the estimate of b is positive cooperation will be implicated, that is the direct and indirect effects will both be in the same direction. The reverse will imply the presence of competition and that the direct and indirect effects have opposite sign. The expectations on this model, of the observed statistics involving juveniles, are given in Table 8. Fitting this model produced a significant improvement ($\chi^2_1 = 6.56$, $0.05 > p > 0.01$) over the simple five parameter genotype-environmental model. The estimates of the parameters E_{1A} and D_{RA} did not alter from

Table 8. Expected variances and covariances of the genetically based sibling effects model fitted to the juvenile statistics for extraversion

Statistic	Expectation
Variances	
MZ twins	$\frac{1}{2}D_{RJ} + bD_{RJ} + \frac{1}{2}b^2D_{RJ} + E_{1J}$
DZ twins	$\frac{1}{2}D_{RJ} + \frac{1}{2}bD_{RJ} + \frac{1}{2}b^2D_{RJ} + E_{1J}$
Singletons	$\frac{1}{2}D_{RJ} + E_{1J}$
Covariances	
MZ twins	$\frac{1}{2}D_{RJ} + bD_{RJ} + \frac{1}{2}b^2D_{RJ}$
DZ twins	$\frac{1}{4}D_{RJ} + bD_{RJ} + \frac{1}{4}b^2D_{RJ}$
Twin with parent	$\frac{1}{4}D_{RAJ} + \frac{1}{4}bD_{RAJ}$
Singleton with parent	$\frac{1}{4}D_{RAJ}$

the estimates (Table 7) obtained under simple genotype–environmental hypothesis. The estimates of parameters in the juvenile model were,

$$\hat{E}_{1J} = 0.015 \pm 0.002$$
$$\hat{D}_{RJ} = 0.053 \pm 0.007$$
$$\hat{D}_{RAJ} = 0.026 \pm 0.006$$
$$\hat{b} = -0.17 \pm 0.06$$

The estimate of b was significant and negative, indicating that the hypothesised sibling effects are competitive in nature. Alleles exerting an increasing effect upon the phenotype of the individual who carries them have an indirect decreasing effect upon the phenotype of that individual's cotwin. However, this apparent effect of the sibling environment is small, and is, as far as we can tell, transitory. Its effect upon estimates of the proportion of genetic variance and the genetic correlation between adults and juveniles is minimal and not significant.

If the simple model is adequate we may calculate summary statistics. As there is no substantial non-additivity or indication of the influence of the family environment, we estimate the heritability of extraversion defined in adults and juveniles as,

$$\hat{h}_A^2 = \tfrac{1}{2} D_{RA}/(\tfrac{1}{2} D_{RA} + E_{1A}) = 0.51 \pm 0.04,$$
$$\hat{h}_J^2 = \tfrac{1}{2} D_{RJ}/(\tfrac{1}{2} D_{RJ} + E_{1J}) = 0.54 \pm 0.07.$$

Thus approximately half of the variation in both adults and juveniles may be attributed to genetic influences. However, are the same genes operative in both adults and juveniles? An attempt to answer this question may be made by looking at the estimate of the covariance of gene effects in parents and offspring. If we expect the only difference between the gene effects in adults and juveniles to be a scalar one, then the correlation $D_{RAJ}/(D_{RA} \cdot D_{RJ})^{1/2}$ should be unity. On the other hand, if this coefficient approaches zero, the conclusion would have to be that few of the genes producing variation in juvenile extraversion are still operative in adulthood. In practice

$$\hat{r}_G = D_{RAJ}/(D_{RA} \cdot D_{RJ})^{1/2} = 0.44 \pm 0.11,$$

implying that slightly less than half of the genetic effects on extraversion are common to both adults and juveniles.

In deriving the standard errors for the above summary statistics we followed Kendal and Stuart (1963, p. 231). Given that a function $g(\mathbf{x})$ of variates $(x_1, x_2, \ldots x_k)$ each has mean θ_i, then g has mean $g(\theta_1, \theta_2, \ldots \ldots \theta_k)$, and to the first order we have,

$$\text{var}(g) = \sum_{i,j=1}^{k} \left[\frac{\partial g}{\partial \theta_i} \cdot \frac{\partial g}{\partial \theta_j} \cdot \text{cov}(x_i, x_j) \right].$$

Numerical differentiation was again chosen in preference to algebraic, in order that extensions of the method to more complicated functions would be simpler. In the case of the simple functions given above, algebraic derivatives were also employed and the result vindicated our choice of the numerical method, since differences were only apparent in the second, and often only in the third, significant figure.

6.3. *Neuroticism*

We turn now to neuroticism, the second major dimension of personality considered here. The fit of the five parameter gene–environment model to the variances and covariances for neuroticism was adequate ($\chi^2_{12} = 87.20$, $p = 0.11$), although there was perhaps some room for improvement. The estimates and their standard errors are given in Table 7. As was the case for extraversion no improvement in the fit of the model was gained by allowing the parental covariance to differ from zero. Therefore, there is no

evidence for assortative marriage for neuroticism. When the purely environmental hypothesis, involving only specific environmental influences and the effects of the common family environment, was put to the test it again failed to account for the data ($\chi^2_{72} = 105.74$, $p = 0.006$).

The simple five parameter gene–environment model has been shown to be adequate and summary statistics may therefore be calculated. The estimates of the heritability of neuroticism in adults and juveniles, as defined above, are,

$$\hat{h}^2_A = 0.41 \pm 0.04$$

$$\hat{h}^2_J = 0.44 \pm 0.07.$$

That is, approximately 40% of variation in neuroticism is due to additive genetic influences, while the estimate of the genetic correlation for neuroticism is,

$$\hat{r}_G = 0.84 \pm 0.14.$$

Allowing for the size of the standard error associated with this estimate, we may conclude that there is no evidence that the correlation differs significantly from unity. This fact in itself is very interesting in that it implies that those genes which predispose children to be neurotic or stable will continue their expression into adulthood, and will therefore lend support to the clinician who attempts prediction of adult behaviour from that of the child. Although we have accepted the above five parameter model as *adequate*, Eaves *et al.* (1978) had noted some non-additivity in the adult neuroticism data and proposed two possible explanations, although were unable to adequately differentiate between them. The present data summary does not allow the consequences of age differences within groups to be examined, by the estimation of $g \times$ age interactions, because model fitting to the raw data is required. However, it is possible to test their second suggestion, which was that the non-additivity might be due to genetic dominance. Since dominance does not contribute to parent–offspring covariance, and we have excluded the involvement of the family environment, and further the genetic correlation, r_G, does not differ significantly from unity, we may replace D_{RAJ} in the expectations by $\sqrt{(D_{RA} \cdot D_{RJ})}$. The estimates of additive and dominance variation derived from twin studies are usually highly negatively correlated and the effects difficult to separate, by virtue of the fact that they share much of the same information. However, by including an estimate of the parent–offspring covariance, which depends only on D_R, it is possible to achieve a better resolution of these two hypothetical sources of variation. The model actually fitted contained six parameters, E_{1A} and E_{1J} for specific environmental variation; H_{RA} and H_{RJ} for dominance variation; and finally $\sqrt{(D_{RA})}$ and $\sqrt{(D_{RJ})}$, thus forcing D_{RA} and D_{RJ} to be non-negative. The exact specification of H_R when allele frequencies are unequal may be found in Mather and Jinks (1971). Fitting the model (Table 9) produced a marginally significant ($\chi^2_1 = 3.86$, $p \approx 0.05$) increase in the log-likelihood, although the improvement was wholly confined to the adults. The estimates of $\sqrt{(D_{RA})}$ and $\sqrt{(D_{RJ})}$ yield values for D_{RA} and D_{RJ} of 0.0016 and 0.08 respectively. Thus the estimate of $\sqrt{(D_{RA})}$ is unacceptably small and that of H_{RJ}, being negative, also points to failure of the model. The model is therefore inappropriate for the present data, although indicating that in more extensive data, a significant contribution due to non-additive gene effects may be found.

6.4. *Psychoticism*

The third dimension in the Eysenckian theory of personality structure is Psychoticism. Referring again to Table 7, the simple gene–environment model apparently provides quite an adequate explanation of the data ($\chi^2_{72} = 70.11$, $p = 0.54$). However, the fixing of the parental covariance parameter to zero is quite unacceptable as we have already noted that there is a significant marital correlation for psychoticism in the present data. Phenotypic assortative mating for a genetically controlled metrical character implies genetical covariance between spouses. Sustained assortation will result in an increase in genetical

variation *between* families as a result of the linkage disequilibrium generated. It is possible to consider more than one mechanism for assortation. Fisher (1918) considered three, distinguished by which of the three spousal covariances, phenotypic, genetic or additive genetic, was assumed to be primary. The first mechanism is usually considered to be the most probable, at least for man, and will be employed here as a first approximation. Following Fisher, if we consider the marital correlation, μ, to be primary, then the genetical correlation between spouses, A, given additivity, is $h^2\mu$, where h^2 is the heritability. The genetic variance of individuals, $\frac{1}{2} D_R$, is increased by an amount $\frac{1}{2}[A/(1 - A)] D_R$, all of which contributes to the covariance of twins reared together.

Table 9. Expected variances, expected covariances and parameter estimates of the genetical dominance model for the neuroticism scale

Statistic	Expectation
Variances	
Adults	$\frac{1}{2}[\sqrt{(D_{RA})}]^2 + \frac{1}{4} H_{RA} + E_{1A}$
Juveniles	$\frac{1}{2}[\sqrt{(D_{RJ})}]^2 + \frac{1}{4} H_{RJ} + E_{1J}$
Covariances	
Adult MZ twins	$\frac{1}{2}[\sqrt{(D_{RA})}]^2 + \frac{1}{4} H_{RA}$
Adult DZ twins	$\frac{1}{4}[\sqrt{(D_{RA})}]^2 + \frac{1}{16} H_{RA}$
Juvenile MZ twins	$\frac{1}{2}[\sqrt{(D_{RJ})}]^2 + \frac{1}{4} H_{RJ}$
Juvenile DZ twins	$\frac{1}{4}[\sqrt{(D_{RJ})}]^2 + \frac{1}{16} H_{RJ}$
Parent–offspring	$\frac{1}{4}\sqrt{(D_{RA})} \cdot \sqrt{(D_{RJ})}$
Parameter	**Estimate**
E_{1A}	0.037 ± 0.003
$\sqrt{(D_{RA})}$	0.041 ± 0.010
H_{RA}	0.108 ± 0.012
E_{1J}	0.037 ± 0.005
$\sqrt{(D_{RJ})}$	0.283 ± 0.067
H_{RJ}	−0.052 ± 0.081

There is one further complication that must be considered before the model is complete. That is, the extent to which genes passed from parents to offspring, and contributing to the expression of the trait in the latter, may be regarded as also in a state of linkage disequilibrium. The situation was resolved by specifying two 'parameters' to describe the additive genetic variation in juveniles. The first represented the effects of genes specifically expressed in juveniles, D_{RS}, the other 'D_{RJC}' was used to describe the effects of genes also expressed in adulthood and which would therefore be in linkage disequilibrium due to the assortative mating of the parents. It was assumed that the only difference in magnitude, of these common gene effects in adults and juveniles, would be a scalar one. Therefore, the relation $D_{RC} = (D_{RA} \cdot D_{RJC})^{1/2}$ was assumed to hold, where D_{RC} was the covariance of these gene effects in parents and offspring. This assumption was put to the test by replacing 'D_{RJC}' in the expectations by $(D_{RC})^2/D_{RA}$. The six parameter genetical model embodied in the first half of Table 10 was fitted to the mean squares and mean products matrices for psychoticism.

The decision to allow a non-zero parental covariance was justified by the highly significant change in the log-likelihood ($\chi^2_1 = 15.44$, $p < 0.001$) on fitting the six parameter model. The fit of the latter genetical model, as to be expected, was exceptionally good ($\chi^2_{71} = 54.67$, $p = 0.92$)—perhaps too good—reflecting inspection of the data. The parameter estimates are given in the first part of Table 11.

As yet, in the consideration of psychoticism, the purely environmental hypothesis has not been examined. The presence of a marital correlation for psychoticism necessitated the extension of the basic environmental hypothesis. A regression model approach was employed in this instance, similar to that developed by Cavalli-Sforza and Feldman (1973). The covariance of juveniles was conceptualised as being partly due to their

Table 10. Expected variances and covariances of alternative genetical and environmental models specified separately for the adult and juvenile statistics

	Expectations	
	Genetical model	Environmental model
Adult statistics		
Total variance (V_p)*	$\frac{1}{2}\left(1 + \frac{A}{1-A}\right)D_{RA} + E_{1E}$	$E_{2A} + E_{1A}$
Covariance of MZ twins	$\frac{1}{2}\left(1 + \frac{A}{1-A}\right)D_{RA}$	E_{2A}
Covariance of DZ twins	$\frac{1}{2}\left(\frac{1}{2} + \frac{A}{1-A}\right)D_{RA}$	
Covariance of spouses (C_m)	$\frac{A(1-A)V_P^2}{\frac{1}{2}D_{RA}}$	μV_P
Juvenile statistics		
Total variance	$\frac{1}{2}\left(1 + \frac{A}{1-A}\right)\frac{D_{RC}^2}{D_{RA}} + \frac{1}{2}D_{RS} + E_{1J}$	$E_{2J} + 2z^2(1+\mu)V_P + E_{1J}$
Covariance of MZ twins	$\frac{1}{2}\left(1 + \frac{A}{1-A}\right)\frac{D_{RC}^2}{D_{RA}} + \frac{1}{2}D_{RS}$	$E_{2J} + 2z^2(1+\mu)V_P$
Covariance of DZ twins	$\frac{1}{2}\left(\frac{1}{2} + \frac{A}{1-A}\right)\frac{D_{RC}^2}{D_{RA}} + \frac{1}{4}D_{RS}$	$E_{2J} + 2z^2(1+\mu)V_P$
Covariance of parent–child†	$\frac{1}{4}\left(\frac{1+MU}{1-A}\right)D_{RC}$	$z(1+\mu)V_P$

* Some expectations are shown for clarity in terms of V_P, however, V_P was not estimated explicitly.
† $MU = C_m/V_P$.

regression onto the phenotypes of their parents and partly due to other factors uncorrelated with the parental psychoticism scores. If, for the moment, we ignore the sources of variation in the parents and concentrate on the parental phenotypes, P_1 and P_2, which are correlated to the extent, μ, we may visualise (Fig. 1) that variation in juvenile psychoticism scores is partly the result of environmental influences of the parental psychoticism phenotype, with regression coefficient z, partly the result of other environmental influences, Ec, shared by siblings but uncorrelated with the parental psychoticism phenotype, with variance E_{2J}, and thirdly resulting from specific environmental influences, E_S, with variance E_{1J}. Now, if we represent the variance of the parental (adult) phenotype by V_p, we may then derive expectations for the juvenile variance and covariances, which are presented in the second part of Table 10.

At no time in the formulation of the above environmental model for juvenile variation and covariation was any assumption made about the causes of variation in the psychoticism scores of the adults. Eaves and Eysenck (1977), in a prior analysis of a subset of this data, were able to reject a purely environmentally based model for the adult scores in

Table 11. Estimates of parameters for alternative genetical and environmental models fitted to psychoticism

	Genetic model		Environmental model	
	Parameter	Estimate	Parameter	Estimate
Adult model	E_{1A}	0.25 ± 0.02	E_{1A}	0.30 ± 0.02
	D_{RA}	0.42 ± 0.04	E_{2A}	0.18 ± 0.02
	A	0.09 ± 0.02	μ	0.18 ± 0.04
Juvenile model	E_{1J}	0.25 ± 0.03	E_{1J}	0.30 ± 0.02
	D_{RC}	0.12 ± 0.04	E_{2J}	0.13 ± 0.03
	D_{RS}	0.33 ± 0.07	z	0.07 ± 0.03

favour of the simple genotype–environmental hypothesis. Nevertheless, the two alternatives for the adult variation were fitted, together with the environmentally based model for the juvenile scores. The expectations relating to the alternative models for the adult variation are given in the upper part of Table 11. The estimates of the parameters of the environmental model for juvenile variation and covariation were unaffected by the model fitted to the adults and similarly the estimates of the parameters of the genotype–environmental model fitted to the adults were unaffected by the model fitted to the juveniles. The estimates of parameters not previously fitted are therefore presented in Table 11, alongside those of the six parameter genotype–environmental model considered earlier. The likelihood ratio tests for the two alternative models yielded chi-squares for 71 *df* of 65.56 and 55.17 respectively. Thus at least three models provide very good explanations of the observed variances and covariances and there is no wholly satisfactory method of deciding between them. However, adding E_{2A} to the five parameter gene–environment model

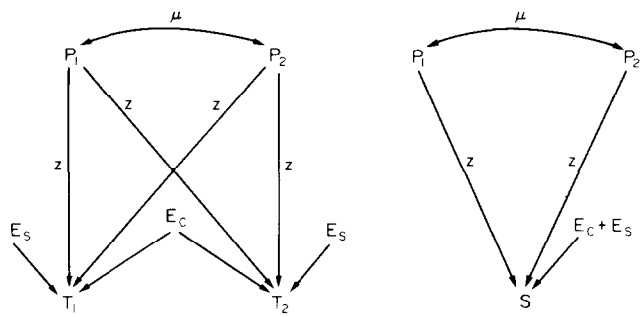

Fig. 1. Regression model for juvenile psychoticism scores. 1. Twin family. 2. Singleton family.

does not produce a significant reduction in χ^2 ($\chi^2_1 = 0.01$, $p > 0.9$), or a significant parameter estimate, but adding D_{RA} to the environmental model produces both results (e.g. $\chi^2_1 = 10.4$, $p \approx 0.001$).

Therefore, two alternative explanations are tentatively proposed for the variation and covariation of juvenile and adult psychoticism scores, acknowledging that we are unable, with the present data, to distinguish between the alternatives. Variation in adult scores is most likely to be the result of specific environmental influences and additive gene action, superimposed upon which are manifestations of the linkage disequilibrium resulting from the assortative mating ($\hat{\mu} = 0.18 \pm 0.04$). The estimated heritability of psychoticism in adults is, therefore,

$$\hat{h}^2_A = V_{GA}/(V_{GA} + E_{1A}) = 0.48 \pm 0.04, \text{ where } V_{GA} = \tfrac{1}{2}(1/1 - A)\, D_{RA}.$$

Considering firstly the purely environmental model for juvenile variation, slightly more than two-thirds of this variation may be related to specific environmental experiences not shared by members of a sibship, i.e.

$$[E_1/(E_{2J} + 2z^2(1 + \mu)\, V_p + E_{1J})].$$

Further, of the covariance of members of a twin pair only 4% may be traced to the influences of the parents' psychoticism scores, i.e.

$$[2z^2(1 + \mu)\, V_p/(E_{2J} + 2z^2(1 + \mu)\, V_p)],$$

providing little support for the contention that parental psychotic behaviour may be passed environmentally to adolescent children. However, this finding does not imply that other independent aspects of the parental phenotype are not developmentally significant. Such effects would of course contribute to the estimate of E_{2J} and are inseparable from those effects contributing to twin covariation, but which are unrelated to parental behaviour. Neither does it imply that the regression of parental phenotype onto the *adult* phenotype of the offspring may not be greater.

The genotype–environmental model for juvenile psychoticism scores may be summarised firstly by estimating the proportion of the expected variation attributable to gene effects,

$$\hat{h}_J^2 = V_{GJ}/(V_{GJ} + E_{1J}) = 0.42 \pm 0.07, \text{ where } V_{GJ} = \tfrac{1}{2}(1/1 - A)(D_{RC}^2/D_{RA}) + \tfrac{1}{2}D_{RS},$$

on the assumption that this model is correct and recognising that at this time it is only an assumption. Secondly, only about 10% of the expected juvenile variation apparently due to additive gene action, is the result of genes that in adulthood also contribute to variation in psychoticism scores, i.e.

$$[(V_{GJ} - \tfrac{1}{2}D_{RJ})/V_{GJ}].$$

Thus, whether or not genes are involved in variation in juvenile psychoticism scores, those scores will not be very good predictors of the later adult phenotype.

6.5. *The lie scale*

The lie scale was originally included in the EPQ to provide a measure of dissimulation that could be used to check the validity of scores obtained on the other scales. However, Eysenck and Wilson (1978) argue that the scale is also a measure of conformity. The lie scale provides the exception in this investigation, in that the simple five parameter genotype–environmental model (Tables 6 and 7) fails utterly to account for the observed variances and covariances ($\chi_{72}^2 = 145.47$, $p < 0.001$).

In an earlier section a highly significant marital correlation was described for the lie scale. However, although leading to a significant improvement in fit ($\chi_1^2 = 44.36$, $p \ll 0.001$), allowing the marital correlation to be greater than zero still does not yield an adequate model ($\chi_{71}^2 = 101.10$, $p = 0.01$). The equivalent six parameter environmental model that allows for both specific environmental influences (E_1) and environmental influences shared by siblings (E_2) is no better ($\chi_{71}^2 = 102.23$, $p = 0.01$). A combination of these two models still did not fit ($\chi_{69}^2 = 91.66$, $p = 0.035$) but showed that for the purpose of explaining the adult variation the contribution of additive genetic effects (D_R) was far more significant than those effects of the family environment (E_2). On the other hand, the reverse seemed to be the case for the juvenile variation where common environmental influences, or influences simulating these, appeared to play a somewhat greater role.

In preliminary investigations of the juvenile scales (Eaves *et al.*, 1978) the possibility that sibling cooperation might be influencing juvenile lie scale scores was considered. Sibling cooperation implies that the phenotype of each member of a sibship has an increasing effect upon the phenotype of the other members. The specification of cooperation, and the converse effect which we call competition, against a *genetical* background is considered by Eaves (1976), who illustrates the approach with adult psychoticism data obtained on an earlier scale than that used in the present study. We now develop a model in which the primary sources of variation are environmental, instead of genetical, and include the environmental influences of the parental phenotype. Upon those sources are superimposed the effects of juvenile cooperation.

Leaving aside the sources of adult variation for the moment, and proceeding in a manner similar to that taken for psychoticism, we let the parents have phenotype, P, with variance V_p and further let this have an effect environmentally upon the phenotype of the offspring, with regression coefficient z. Also let there be environmental sources (E_c) of juvenile twin covariation that are not correlated with the adult lie scale scores, represented by parameter E_{2J}, and a source (E_s) of environmental variation specific to individuals, represented by the parameter E_{1J}. Thus far, the model is the same as one specified for juvenile psychoticism. However, now let the sum of the environmental influences upon one twin (E_T) have not only a direct effect upon that twin's own phenotype but also an indirect effect upon that of his cotwin with regression coefficient c. There are of course, by definition, no sibling influences upon the phenotype of singletons. The model is represented diagramatically in Fig. 2. It may be shown by standard rules of regression

Table 12. Expected variances, expected covariances and parameter estimates of the environmental cooperation model for the lie scale scores of juveniles

Statistic	Expectation	
Variances		
Singletons	$E_{1J} + E_{2J} + 2z^2(1 + \mu)V_p$	
MZ and DZ twins	$(1 + c^2)E_{1J} + (1 + c)^2E_{2J} + 2z^2(1 + c)^2(1 + \mu)V_p$	
Covariances		
MZ and DZ twins	$2cE_{1J} + (1 + c)^2E_{2J} + 2z^2(1 + c)^2(1 + \mu)V_p$	
Singleton with parent	$z(1 + \mu)V_p$	
Twin with parent	$z(1 + c)(1 + \mu)V_p$	
Parameter	Full model Estimate	Adapted model Estimate
E_{1J}	0.031 + 0.006	0.035 + 0.002
E_{2J}	0.003 ± 0.005	
z	0.14 ± 0.02	0.14 ± 0.02
c	0.20 ± 0.08	0.24 ± 0.03

theory that the appropriate expectations for the juvenile variation and covariation are those of Table 12.

The model so far has been independent of the sources of variation in the adult lie scale scores. In the first instance let us consider a model specifying only environmental variation specific to individuals, E_{1J}; additive gene action, D_{RJ}; and the genetic correlation between spouses, A. This model for the adult variation and covariation is the basic genotype–environmental model with assortation previously considered for adult psychoticism (Table 10). The combination of this model for the adult scores and the full model of Table 12 for the juvenile scores was fitted to the mean squares and mean products matrices for the lie scale. The estimates of parameters of the full juvenile model are given in Table 12, whilst the estimates from the adult model were,

$$\hat{E}_{1A} = 0.021 \pm 0.001$$

$$\hat{D}_{RA} = 0.033 \pm 0.003$$

$$\hat{A} = 0.16 \pm 0.02$$

The model provided an adequate, although perhaps not good, explanation of the observed matrices ($\chi^2_{70} = 87.78$, $p = 0.07$). However, the estimate of E_{2J} is obviously not

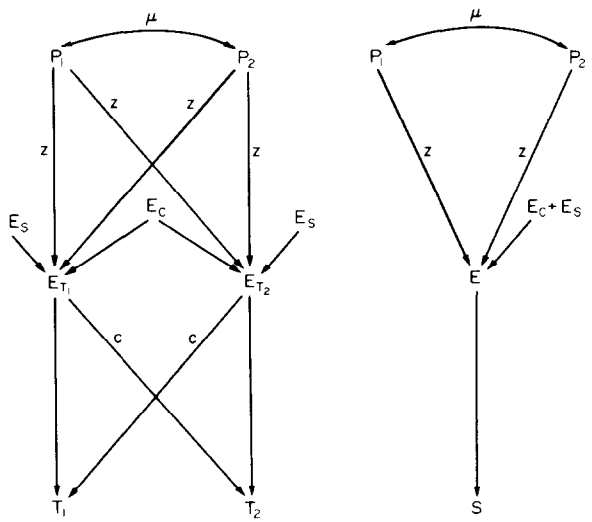

Fig. 2. Regression model for juvenile lie scale scores. 1. Twin family. 2. Singleton family.

significant and was discarded from the model, the remaining parameters being re-estimated (Table 12). The estimates of parameters contributing to adult variation and covariation did not alter. The fit of the resultant model ($\chi^2_{71} = 88.08$, $p = 0.08$) was no worse for the dropping of the superfluous parameter. Allowing for environmental sources of twin covariation in the adults, by fitting an E_{2A} parameter, did not produce a significant improvement in fit and the resulting parameter estimate was small and not significant.

The possibility that this apparent sibling cooperation could be genetically based was also considered. However, a suitable model had to account for three important aspects of the data: (i) additive gene action in the adults; (ii) a non-zero marital correlation; (iii) a correlation between juvenile and adult genetic expression in the range $0 < r_G \leqslant 1$. The model specified contained elements of two previous models considered with respect to extraversion and psychoticism. The sibling effects were specified by means of the parameter, b, employed in the investigation of extraversion. This model was combined with the basic genotype–environmental model with assortation previously considered for adult psychoticism (Table 10) and with the associated model for juveniles in which the additive genetical variation was separated into that also having expression in adulthood and that genetical variation specific to adolescence. The resultant model contained seven parameters, E_{1A} and E_{1J} representing environmental variation specific to individuals in adults and juveniles respectively; D_{RA}, additive genetic variation in adults; D_{RC}, the covariance of gene effects in adults and juveniles; D_{RS}, the genetical variation specific to juveniles; A, the additive genetic correlation between spouses; and b, to represent the effects of sibling cooperation. The expectations for the juvenile variation and covariation are presented in Table 13, whilst the expectations for the adult statistics have already been presented (Table 10). The fit of the model was quite good ($\chi^2_{70} = 84.55$, $p = 0.11$). However, two of the estimates (Table 14) were not significant, the sibling effects parameter, b, and the parameter estimating the juvenile specific genetical variation, D_{RS}. When fitting the environmental sibling effects model there was no evidence for twin covariation that could not be related to the parents. Therefore, D_{RS} was dropped from

Table 13. Expected variances and covariances of the genetical cooperation model for the lie scale scores of juveniles

Statistic	Expectation
Variances	
Singletons	$\frac{1}{2}\frac{D_{RC}^2}{D_{RA}} + \frac{1}{2}D_{RS} + E_{1J}$
MZ twins	$(1+b)^2\left[\frac{1}{2}\frac{1}{(1-A)}\frac{D_{RC}^2}{D_{RA}} + \frac{1}{2}D_{RS}\right] + E_{1J}$
DZ twins	$(1+b^2)\left[\frac{1}{2}\frac{1}{(1-A)}\frac{D_{RC}^2}{D_{RA}} + \frac{1}{2}D_{RS}\right] + 2b\left[\frac{1}{4}\frac{(1+A)}{(1-A)}\frac{D_{RC}^2}{D_{RA}} + \frac{1}{4}D_{RS}\right] + E_{1J}$
Covariances	
MZ twins	$(1+b)^2\left[\frac{1}{2}\frac{1}{(1-A)}\frac{D_{RC}^2}{D_{RA}} + \frac{1}{2}D_{RS}\right]$
DZ twins	$(1+b^2)\left[\frac{1}{4}\frac{(1+A)}{(1-A)}\frac{D_{RC}^2}{D_{RA}} + \frac{1}{4}D_{RS}\right] + 2b\left[\frac{1}{2}\frac{1}{(1-A)}\frac{D_{RC}^2}{D_{RA}} + \frac{1}{2}D_{RS}\right]$
Singleton with parent*	$\frac{1}{4}\left(\frac{1+\mu}{1-A}\right)D_{RC}$
Twin with parent*	$\frac{1}{4}\left(\frac{1+\mu}{1-A}\right)(1+b)D_{RC}$

* Where μ is the expected phenotypic correlation between spouses.

Table 14. Estimates of parameters of the genetical co-operation model for the lie scale scores of juveniles

Parameter	Full model Estimate	Adapted model Estimate
E_{1A}	0.021 ± 0.001	0.023 ± 0.002
D_{RA}	0.033 ± 0.003	0.031 ± 0.003
A	0.16 ± 0.02	0.15 ± 0.02
E_{1J}	0.021 ± 0.002	0.023 ± 0.002
D_{RC}	0.013 ± 0.005	0.014 ± 0.004
D_{RS}	0.007 ± 0.006	
h	1.02 ± 0.75	1.53 ± 0.67

the present model. This action resulted in a significant estimate of h (Table 14), but also failure of the model ($\chi^2_{71} = 93.38$, $p = 0.04$), both at the 5% significance level. Thus, while the evidence is by no means conclusive, the environmentally based explanation is an acceptable provisional hypothesis for the variation and covariation in adult and juvenile lie scale scores.

Several summary statistics might be derived from the environmental sibling effects model. Thus it was found: (i) that the expected variance of twins is approximately 10% greater than that of singletons, i.e.

$$[(1 + c^2)E_{1J} + 2z^2(1 + c)^2(1 + \mu)V_p]/[E_{1J} + 2z^2(1 + \mu)V_p] = 1.09 \pm 0.02;$$

(ii) that of the expected variance of twins 92%, i.e.

$$[(1 + c^2)E_{1J}]/[(1 + c^2)E_{1J} + 2z^2(1 + c)^2(1 + \mu)V_p] = 0.92 \pm 0.03$$

may be ultimately traced to environmental influences specific to individuals, and similarly for singletons, 94%, i.e.

$$E_{1J}/[E_{1J} + 2z^2(1 + \mu)V_p] = 0.94 \pm 0.02$$

may be traced to the same source; (iii) that the covariance of twins and their parents is approximately 25% greater than that of singletons and their parents, due to the 'cooperative' effects, i.e.

$$[z(1 + c)(1 + \mu)V_p]/[z(1 + \mu)V_p] = 1.24 \pm 0.03;$$

(iv) finally, that of the covariance of twins only about 15% may be traced to the shared influences of their parents, i.e.

$$[2z^2(1 + c)^2(1 + \mu)V_p]/[2cE_{1J} + 2z^2(1 + c)^2(1 + \mu)V_p] = 0.16 \pm 0.05,$$

the remainder being the result of each twins' specific environmental circumstances having an indirect 'cooperative' effect upon their cotwin. Nevertheless, the most important finding is the lack of evidence for primary sources of juvenile twin covariation (E_{2J}) that are not traceable to the environmental influences of the parental 'lie' behaviour.

The sources of variation in the adult lie scale scores seem to be clearer than those for the juvenile scores. As in the case of psychoticism the most important effects appear to be those due to specific environmental influences, contributing to E_{1A} and additive gene action, D_{RA}, ($h^2_A = 0.48 \pm 0.04$). We also recognise a fairly large spousal correlation ($\mu = 0.34 \pm 0.04$), which might suggest that the aspect of self-effacement measured by the adult lie scale may be important in consideration of a marriage partner.

7. DISCUSSION AND CONCLUSIONS

With the demise of conventional behaviour genetic studies, in which the emphasis is on estimation of the parameters of untested models from one or two different degrees of relationship, we would expect the search for paradigms of different mechanisms of determination, to focus on a few traits which reflect consistent and different causal mechanisms, rather than on a morass of traits chosen for little other reason than the fact

that someone has claimed they can be measured. Furthermore, the emphasis of the greater part of the model-fitting effort on the analysis and reanalysis of cognitive variation has perhaps obscured the great diversity of causal mechanisms which are awaiting discovery and analysis in man. The above analysis, albeit indecisive in many respects, has demonstrated how the model-fitting approach, far from being a sterile exercise in the juggling of numbers, may enliven the discussion of causal mechanisms in man by bringing to our attention many of the anomalies which can become the points of departure for a new and more flexible understanding of the significance of human variation.

In this paper a general model-fitting approach to the analysis of structured pedigree data, "the balanced pedigree method" (Eaves *et al.*, 1978), has been outlined. The application of the method to a particular data set should not be allowed to detract from its generality. There would appear to be a need in human behaviour genetics, with the collection of extended data sets becoming more common, for a maximum-likelihood method for the fitting of clearly defined expectations to a set of statistics irrespective of their independence.

In summarising the results of the model fitting to the EPQ data, the tentative nature of many of the conclusions must be stressed. Any hypothesis needs to be tested by the collection of fresh and more extensive data. The three major dimensions of the Eysenckian theory of personality appear on the whole, with the one possible exception of psychoticism in juveniles, to be characterised by variation resulting from specific life experiences and the additive effects of genetical polymorphism. The effects of test unreliability contribute to our estimate of the importance of the individual environmental experiences. The covariance of genetical effects as expressed in juveniles and adults has been shown to be reasonably high for extraversion and neuroticism, especially so in the latter case, for which trait there is a surprising degree of inter-generational consistency. However, in the case of psychoticism the covariance of parents and offspring is low, irrespective of its basis, genetical or environmental. These results suggest that the prediction of adult temperament in childhood may be quite successful along the dimension of anxiety–stability. However, the success of such prediction is not expected to be so great, if interest is in or related to the extraversion–introversion dimension, while the indications for the psychoticism dimension perhaps reflect its weaker theoretical basis, and the poor discriminating power in the normal range of variation of the present scales employed in its measurement.

The findings for the lie scale represent a striking departure from those expected on the basis of a simple genotype–environmental model, and suggest that social interactions between parents and offspring, reinforced by the interaction of siblings in the case of twin pairs, may play an important role in the manifestation of 'honesty' in juveniles, as measured by the lie scale of the JEPQ. It is remarkable that there is absolutely no evidence that the similarity of twins depends on any environmental factors which are not assessed by the lie scores of their immediate relatives. The possible detection of social interaction effects in the juvenile scores and the presence of a relatively high phenotypic correlation between spouses ($\mu = 0.34$) for the lie scale suggests that whether the junior version of the scale is measuring actual deceit, lack of insight, or genuine variation in "approved behaviour" (Eysenck *et al.*, 1971), the trait may well repay further examination as a paradigm of a trait for which social interactions, rather than genetical differences, are paramount determinants of individual variability.

The inclusion of singleton families in the study has highlighted the increased power of this design for detecting certain types of social interaction. This increase in power is witnessed by the detection of apparent transitory sibling effects in the variation and covariation of juvenile lie scale, and possibly extraversion, scores. The consequences of sibling effects could not be adequately specified in the parental generation since the characteristics of the families in which the parents were reared were unknown. The collection of such information and the data to estimate the variance of adult singleton scores would be desirable. In this treatment of sibling effects we have obviously only touched the surface of the subject. It would be interesting to test our models against data

collected on, say, unrelated individuals reared together or siblings raised in sibships of sizes greater than two. Factors in addition to sibship size such as birth interval, which collectively may be called "family density" effects (Eaves *et al.*, 1978), might also prove to be important.

This paper extends two basic concepts in the quantitative analysis of human personality. Firstly, the interaction of gene expression with age differences, and in addition, the formulation of explicit models for the influence of social interactions upon personality development. Although the idea that gene expression and interaction may be modified with age is largely accepted in the animal literature (e.g. Broadhurst and Jinks, 1966), its rigorous analysis in man is still far from realisation. The present experimental design has been able to show that a simple model which assumes consistency of gene action between adults and juveniles measured on similar personality scales cannot account for the degree of similarity between relatives. It is therefore quite likely that the traits measured in adults and juveniles differ in their causes of variation, at least in part, so that genetic effects manifest in juveniles are not expressed in the same individuals as adults and vice-versa. The fact that somewhat different scales were used in the assessment of adults and juveniles does not alter the general conclusion, since the scales were designed expressly to measure those aspects of behaviour which were factorially consistent in adults and juveniles. Other explanations of the data for the three primary dimensions of personality, extraversion, neuroticism and psychoticism, would have to invoke a substantial amount of genetical non-additivity to explain the findings, and would not be consistent with the general findings for infra-human genetical systems (e.g. Mather and Jinks, 1971). The inability of parental data to predict the findings for offspring, therefore, is perhaps more likely to be attributable to the interaction of genetical differences with age.

Acknowledgements We thank Mrs. J. Kasriel for collection of the data. The computations were conducted on the University of Manchester Regional Computer Centre ICL 1094S/CDC7600 and the University of Birmingham ICL 1906A computers. This study was supported by the British Medical Research Council. P. A. Young currently holds an SRC Research Studentship.

REFERENCES

BROADHURST P. L. and JINKS J. L. (1966) Stability and change in the inheritance of behaviour in rats: a further analysis of statistics from a diallel cross. *Proc. R. Soc. B* **165**, 450–472.

CAVALLI-SFORZA L. L. and FELDMAN M. W. (1973) Cultural versus biological inheritance: phenotypic transmission from parents to children. *Am. J. Hum. Genet.* **25**, 618–637.

CLARIDGE G., CANTER S. and HUME W. I. (1973) *Personality Differences and Biological Variations: A Study of Twins*. Pergamon Press, Oxford.

DAVIS P. J. and POLONSKY I. (1965) Numerical interpolation differentiation and integration. In *Handbook of Mathematical Functions* (Edited by M. ABRAMOWITZ and I. A. STEGAN) Dover, New York.

DWORKIN R. H., BURKE B. W., MAHER B. A. and GOTTESMAN I. I. (1976) A longitudinal study of the genetics of personality. *J. Pers. Soc. Psychol.* **34**, 510–518.

DWORKIN R. H., BURKE B. W., MAHER B. A. and GOTTESMAN I. I. (1977) Genetic influences on the organisation and development of personality. *Dev. Psychol.* **13**, 164–165.

EAVES L. J. (1976) A model for sibling effects in man. *Heredity* **36**, 205–214.

EAVES L. J. and EYSENCK H. J. (1975) The nature of extraversion: A genetical analysis. *J. Pers. soc. Psychol.* **32**, 102–112.

EAVES L. J. and EYSENCK H. J. (1976) Genetic and environmental components of inconsistency and unrepeatability in twins' responses to a neuroticism questionnaire. *Behav. Genet.* **6**, 145–160.

EAVES L. J. and EYSENCK H. J. (1976a) Genotype × age interaction for neuroticism. *Behav. Genet.* **6**, 359–362.

EAVES L. J. and EYSENCK H. J. (1977) A genotype environmental model for psychoticism. *Adv. Behav. Res. Ther.* **1**, 5–26.

EAVES L. J., LAST K. A., MARTIN N. G. and JINKS J. L. (1977) A progressive approach to non-additivity and genotype-environmental covariance in the analysis of human differences. *Br. J. math. stat. Psychol.* **30**, 1–42.

EAVES L. J., LAST K. A., YOUNG P. A. and MARTIN N. G. Model-fitting approaches to the analysis of human behaviour. *Heredity*, in press.

EYSENCK J. H. and WILSON G. D. (Editors) (1978) *The Psychological Basis of Ideology*. MTP, Lancaster.

EYSENCK S. B. G. (1969) Personality dimensions in children. In *Personality Structure and Measurement*. (Edited by H. J. EYSENCK and S. B. G. EYSENCK) Routledge & Kegan Paul, London.

EYSENCK S. B. G. and EYSENCK H. J. (1975) *Manual of the EPQ (Eysenck Personality Questionnaire)*. University of London Press, London.

EYSENCK S. B. G., NIAS D. K. B. and EYSENCK H. J. (1971) The interpretation of children's Lie Scale scores. *Br. J. educ. Psychol.* **41**, 23–31.

FISHER R. A. (1918) The correlation between relatives on the supposition of Mendelian inheritance. *Trans. R. Soc.* (Edinburgh) **52,** 399–433.

GOTTESMAN I. I. (1965) Personality and natural selection. In *Methods and Goals in Human Behavior Genetics* (Edited by S. G. VANDENBERG). Academic Press, New York.

GOTTESMAN I. I. (1966) Genetic variance in adaptive personality traits. *J. Child Pyschol. Child Psychiat.* **7,** 199–208.

HORN J. M., PLOMIN R. and ROSENMAN R. (1976) Heritability of personality traits in adult male twins. *Behav. Genet.* **6,** 17–30.

JÖRESKOG K. G. (1973) Analysis of covariance structures. In *Multivariate Analysis III* (Edited by P. R. KRISHNAIAH). Academic Press, New York.

KASRIEL J. and EAVES L. J. (1976) The zygosity of twins: Further evidence on the agreement between diagnosis by blood groups and written questionnaires. *J. biosoc. Sci.* **8,** 263–266.

KENDALL M. G. and STUART A. (1963) *The Advanced Theory of Statistics.* Charles Griffin, London.

LANGE K., WESTLAKE J. and SPENCE M. A. (1976) Extension to pedigree analysis. III. Variance components by the scoring method. *Ann. Hum. Genet. (London)* **39,** 485–491.

MATHER K. and JINKS J. L. (1971) *Biomet. Genet.* 2nd Edn. Chapman & Hall, London.

MOSTELLER F. and YOUTZ C. (1961) Tables of the Freeman–Tukey transformations for the binomial and Poisson distributions. *Biometrika* **48,** 433–440.

NUMERICAL ALGORITHMS GROUP (1974) E04HAF *N.A.G. Library Manual. Mark IV.* N.A.G. Central Office, Oxford University, Oxford.

PARTANEN J., BRUUN K. and MARKKANEN T. (1966) *Inheritance of Drinking Behavior: A Study on Intelligence, Personality, and Use of Alcohol of Adult Twins.* Finnish Foundation for Alcohol Study, Helsinki.

RACHMAN S. (1969) Extraversion and neuroticism in childhood. In *Personality Structure and Measurement* (Edited by H. J. EYSENCK and S. B. G. EYSENCK) Routledge & Kegan Paul, London.

REZNIKOFF M. and HONEYMAN M. S. (1967) MMPI profiles of monozygotic and dizygotic twin pairs. *J. consult. Psychol.* **31,** 100.

VANDENBERG S. G., STAFFORD R. E. and BROWN A. M. (1968) The Louisville twin study. In *Progress in Human Behavior Genetics* (Edited by S. G. VANDENBERG) Johns Hopkins Press, Baltimore.

WILSON R. S. (1972) Twins: early mental development. *Science* **175,** 914–917.

WILSON R. S. (1977) Twins and siblings: concordance for school-age mental development. *Child Devel.* **48,** 211–216.

YOUNG P. A. (1977) The causes of differences in juvenile personality. Unpublished M.Sc. Thesis, University of Birmingham.