A TWIN STUDY OF PERSONALITY, SOCIAL ATTITUDES AND DRINKING BEHAVIOUR

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by

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DECLARATION

This thesis contains no material which has been accepted for the award of any other degree or diploma at any University. Although the data used in this thesis were obtained through a National Health and Medical Research Council grant to Dr J.D. Matthews, University of Melbourne; Dr J.B. Gibson, Australian National University and Dr N.G. Martin, Australian National University (presently Medical College of Virginia), all the work reported herein is entirely my own.

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ABSTRACT

A genetic and environmental analysis of personality, social attitudes and drinking behaviour in 3810 pairs of adult twins is reported.

Individual differences in the personality traits of psychoticism, neuroticism and lie can be explained by the additive effects of genes and individual environmental differences. The contribution of these effects, however, depends on age and sex. For extraversion there is also evidence that either dominance or sibling competition is important. There is no evidence for the importance of shared family environment in determining variation in personality. It is possible to distinguish between major aspects of personality both factor-analytically and in terms of their patterns of variation, and this is reflected at both the item and trait level.

Extensive power calculations were conducted to examine the ability of different experimental designs to discriminate between the effects of genetic dominance and sibling interaction. Our results show that discrimination between these effects is unlikely to prove feasible by model-fitting techniques. Other methods for the resolution of these effects are discussed.

Analysis of the causes of covariation between the personality trait of neuroticism, and symptoms of anxiety and depression, shows that genetic variation in anxiety and depression is largely dependent on the same genes which determine variation in the trait of neuroticism. Furthermore, additive genetic factors are more important than individual environmental factors in the covariation of these measures. There is no

evidence for the importance of environmental experiences shared by cotwins, such as common family environment or social influences.

Similar to our results for personality traits and symptoms, our analyses of the causes of variation in conservatism, at both the item and trait level, provide strong support for the importance of the additive effects of genes. In contrast to personality measures, however, there is substantial assortative mating for conservatism which results in inflated estimates of the shared environmental effect in twin data. We show that by fitting models which directly estimate the effects of genetic and cultural inheritance in the presence of phenotypic assortative mating, there is no evidence for the importance of family environment.

Our analysis of data relating to drinking habits provides some support for the role of family environment in the development of normal drinking behaviour. We also find that there are significant genetic effects on twin resemblance in drinking behaviour, and that the effects of genes and environment depend significantly an age, sex, and for alcohol consumption in females, marital status. One method for modelling changes in the sources of variation with age is illustrated with data relating to alcohol consumption.

Possibilities for future research based on the results of each of these analyses are discussed.

CHAPTER 1 INTRODUCTION

1.1 The quantitative genetic approach to the study of human variation

The application of quantitative methods to human genetics is essential to our understanding of the causes of many medical disorders and behavioural differences. Yet, as Eaves (1983) has noted, "many geneticists still view biometrical genetics as an arcane pursuit, one that has been eclipsed by developments in molecular genetics" (p. 355). Such a view fails to recognise the unique contribution that quantitative genetics can make to our understanding of human variation and, further, that the field addresses fundamental biological issues that are beyond the scope of molecular genetics.

Thus, an efficient quantitative genetic analysis can provide information on the contribution of possible sources of variation such as the additive and non-additive effects of genes, the mating system, specific and familial environmental effects, the dependence of genetic and environmental effects on age and sex, and the interaction and covariation of genes and environment. Any attempt to fully describe variation in terms of the molecular and biochemical effects of individual genes must also take account of these complex aspects of the phenotype.

In humans, the classical twin method, which is based on the comparison of the degree of similarity of monozygotic (MZ) and dizygotic (DZ) twin pairs, is the most common procedure for estimating the relative importance of genetic and environmental contributions to 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 described in detail elsewhere (Jinks and Fulker, 1970). Suffice to say here that in the past 10 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 has become apparent.

In the case of continuous data, several hypothesis-testing approaches have been espoused including path analysis of familial correlations (Rao et al., 1976), 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). For discontinuous data, variation has been explained in terms of a major gene with or without reduced penetrance (Elston and Campbell, 1970), or by regarding discontinuity in the phenotype as a manifestation of an underlying continuous normal distribution of liability which may itself be affected by both genetic and environmental factors (Falconer, 1965). Each method has its strengths and weaknesses depending on the type of data involved, continuous or discontinuous, and the summary statistics that are available. However, 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. Consequently, these techniques have been applied exclusively throughout this thesis.

1.2 Advantages and validity of the twin design

The classical twin design does have recognised limitations such as the inability to distinguish between the effects of assortative mating and between-families environmental variation, its low power for the detection of dominance, the confounding of dominance with the effects of between-families environmental variation and assortative mating, and its inappropriateness for the resolution of competing models of assortative mating and cultural inheritance. However, the twin design can provide more information about patterns of variation than has generally been appreciated even by many exponents of the twin paradigm. Thus the twin method can provide necessary information about the presence of certain types of genotype-environment interaction and the need for rescaling, preliminary estimates of the relative contributions of the additive and dominant effects of genes and the effects of between- and within-families environmental variation, possible age and sex interactions of these effects, and the presence of sibling interaction. There is little doubt that the twin method, despite its weaknesses, has contributed more to our understanding of the causes of human variation than any other design. However, the utility of twins is frequently questioned by those who challenge the assumptions underlying the twin method (e.g. Lewontin, 1974; Kamin, 1974; Lewontin et al.,

1984). We briefly outline these assumptions and some of the criticisms below.

1.2.1 Assumptions of the twin method

1. There is no genotype-environment interaction

The presence of genotype-environment interaction implies that different genotypes respond in different ways to the same environmental influences. Several authors (e.g. Layzer, 1974; Feldman and Lewontin, 1975) have suggested that the presence of genotype-environment interaction precludes a worthwhile analysis of human individual differences. Although these critics have made no attempt to offer methods of detecting or quantifying the effects of genotype-environment interaction, Jinks and Fulker (1970) have shown that MZ twins provide a unique opportunity to test for one important type of such interaction.

MZ twins are genetically identical, thus the absolute difference between co-twins is a measure of the specific individual environmental influences (including errors of measurement) to which that pair has been subjected, while the pair sum is a measure of their genetic value and/or the environmental influences which they have shared and which make them different from other twin pairs. Thus regression of absolute within-pair differences on pair sums provides a test for any systematic interactions between genotype and individual environmental influences. Martin and Eysenck (1976) showed that such interactions could be detected with great sensitivity but they could nearly always be removed by a transformation of the scale of measurement which lessened departures from normality.

This approach of data transformation has been criticised on the grounds that such a choice of scale is arbitrary (Kamin, 1974). However, such a view fails to recognise that although different transformations may be chosen for the sake of convenience in a genetical analysis, they are no more arbitrary than the raw scores, given that we have no information about the precise relationship of the traits in question to evolutionary fitness (Eaves et al. 1977a). Consequently in this thesis we have chosen to fit models to scores which have been transformed to best minimise genotype-environment interaction.

2. There is no genotype-environment covariation

Genotype-environment covariation arises when genetic and environmental deviations are correlated. Eaves et al. (1977a) have distinguished between three possible sources of genotype-environment covariation (CovGE).

(i) A person's genotype influencing his environment That is, certain genotypes creating or seeking out environments compatible with their genetic deviations. As Eaves et al. (1977a) have noted, although it would be possible to formulate a model which distinguished the direct effects of loci from those which operated indirectly by modifying the environment, such effects would have equivalent expectations and hence be inevitably confounded. However, if the environmental effects to which an individual is exposed are under the direct control of that person's genotype, then these effects are best included in the genetic variance, as in fact occurs in the classical twin design.

(ii)Cultural transmission That is, an environmental effect of parental genotypes on their offspring. For example, parents with favourable genes tend to provide their children with stimulating home environments (books, music, enriched language environment etc.). In addition, however, parents with favourable genes usually have genetically advantaged children. Thus such children end up with a double advantage. Jinks and Fulker (1970) proposed comparing the total variances of twins and siblings reared together with that of twins and siblings reared apart as a simple test of this form of CovGE. If one finds that the variance of the fostered individuals is less than that of individuals reared together, then we have evidence of cultural transmission. Without this information, however, if one finds a large between-families environmental component of variation this may be attributable to cultural transmission, between-families environmental differences uncorrelated with genetic differences, or additional additive genetic variation due to assortative mating (Eaves et al., 1978).

(iii)Sibling effects This occurs when siblings reared together influence each other's development according to their genotypes. Cooperation exists when an allele which increases the expression of a character in one sibling exerts an increasing environmental effect on the other sibling. Competition occurs when an increasing allele exerts a decreasing environmental effect on a siblng. As these effects vary according to the genetic similarity between siblings, this form of CovGE will produce a difference in the total variances of MZ and DZ twins.

Competition would tend to make the total variance of MZ twins less than that of DZ twins, whilst for cooperation the reverse would be true. Both effects would tend to cause failure of the simple models we shall fit which assume equality of MZ and DZ total variances and, if this occurs, it would be possible to fit models which quantify the effects of sibling competition and cooperation (Eaves, 1976).

3. The environmental experiences for MZ and DZ twins are the same

Perhaps the most commonly voiced criticism of the twin method is that MZ twins have greater environmental similarity than DZ twins and that this completely invalidates the results of twin studies (e.g. Lewontin, 1975; Kamin, 1974; Lewontin et al. 1984). It is argued that if the environments of MZ twins are more alike than those of DZ twins, then it is possible that it is environmental rather than genetic similarity that is responsible for the greater similarity of MZ over DZ twins. In assessing this claim we must consider both the pre- and post-natal environments of twins.

With respect to the pre-natal environment, Price (1950) found that, on average, MZ twins have less similar environments than DZ twins. When an imbalance in the mutual circulation of monochorial (only MZ) foetuses occurs, the development of either or both foetuses is altered. Often there is a considerable difference in the birthweight of the twins and such large differences in birthweight are not often found in DZ twins. Price (1950) suggested that differences in achievement may be caused by this condition. Indeed, Willerman and Churchill

(1967) found that in MZ twins, the heavier birthweight twin is on average more intelligent than the co-twin. However, this study was based on only 27 pairs of twins, of which 14 pairs had been referred to a psychological clinic because of their poor school performance, and thus there was selection bias for differences in intelligence.* A study by Breland (1974) has failed to support Price's hypothesis. Breland (1974) studied 365 MZ twin pairs concordant for handedness and 117 MZ pairs discordant for handedness. She assumed that twins concordant for handedness were more likely to have developed with separate placenta and chorions, while those discordant for handedness had a higher probability of developing from the same placenta and chorion.** If an imbalance in mutual circulation affects achievement, then discordant twins should show larger differences in ability than those concordant for handedness. Breland (1974) found no significant differences in ability between the two groups.

After birth, a greater degree of intra-pair similarity among MZ than DZ twins has been found with respect to such factors as habits, activities, personal preferences, parental treatment and self-image (Smith, 1965). However, two questions need to be answered. First, does the greater similarity of MZ twins reflect conscious differential treatment by parents, siblings etc. who are aware that MZ twins are supposed to be alike and thus they emphasise these similarities or, alternatively, are people merely responding to MZ twins greater genetic similarity? Second, does the greater similarity of MZ twins with regard to physical appearance, dress, school experiences etc. account for their greater behavioural similarity?

Scarr (1969) attempted to answer the first question by interviewing mothers of twins who had incorrectly determined the zygosity of their children. On the basis of the mothers' ratings it was found that monozygotic twins considered dizygotic were more similar than dizygotic twins considered monozygotic. Lytton (1977) further clarified the relationship between social expectation and twin behaviour by direct observations of young twins and their parents in the home environment. He observed parent-child interaction and distinguished between two types of actions, parent-initiated actions and child-initiated actions. Parent-intiated actions were those actions not directly elicited by the child, while child-initiated actions were actions made in response to the child's immediately preceding behaviour. He found that while parents do treat MZ twins more alike than DZ twins in some respects, this was only true for child-initiated actions. Furthermore, the greater similarity in the treatment of MZ twins was in accordance with the twins' actual rather than perceived zygosity. He concluded that parents respond to, rather than create, differences between twins (see also Munsinger and Douglass, 1976; Matheny, 1979; Scarr and Carter-Saltzman, 1979 ; Dibble and Cohen, 1980). *

For example, the greater environmental similarity of MZ twins should be regarded as a form of CovGE, that of an individual's genotype influencing his environment. As we have noted above, this form of CovGE is best included in the estimates of the genetic variance, however, we shall consider the bias that this form of CovGE could introduce.

Loehlin and Nichols (1976, Ch.5) attempted to determine whether the greater environmental similarity of MZ twins produced any greater behavioural similarity on tests of personality and cognition. They analysed within-pair differences for MZ twins and found that there was essentially a zero correlation between how differently MZ twins were treated as young children (clothes, school attended, playing together etc.) and how differently they performed on tests of personality and cognition. A similar result was found by Matheny et al. (1976). Thus, unless one can show that differences in the environmental experiences of MZ and DZ twins are relevant to behavioural variation, the criticism of the twin method on the grounds of different environmental experiences of MZ and DZ twins seems unjustified. It should be noted that in the event that there are differences in the environmental experiences of MZ and DZ twins, this would lead to an inequality in total variances and tend to cause model failure. Only if this occurs do we need to be concerned about enviromental differences between MZ and DZ twins and the possibility that these differences may account for trait similarity.

1.3 Applications of quantitative genetics to the analysis of human variation

It is one of the aims of this thesis to show the variety of information that can be obtained from an efficient analysis of data on MZ and DZ twins. We have selected for study several variables that have considerable social and/or medical significance.

In Chapter 3 we consider the causes of variation in personality traits. There have been many developments in the genetic analysis of personality since Newman et al. (1937) discounted the contribution of genetic factors in this field, and while there is considerable evidence that genetic factors are in fact important to variation in personality (see section 3.1.1 for a review), the detailed causes of variation are still not completely understood. There are several reasons for this.

Early studies were often based on personality inventories whose reliability and validity were doubtful, and further these inventories were often chosen without regard to a clearly defined theory of personality. Although there has been debate about the number of dimensions of personality which are theoretically important, there is consistent evidence from many replicated studies for the existence of three major dimensions of personality (e.g. Royce, 1973). These major factors are best described in Eysenck's theory of personality which seeks to explain the structure of personality in terms of three major dimensions, extraversion, psychoticism and neuroticism, and a fourth scale, lie. Although this classification of personality is not without its critics (e.g. Hamilton, 1959a, 1959b), there is nevertheless considerable support for this economical view of personality structure (Cattell and Scheier, 1961; Royce, 1973 also section 3.1).

A further problem has been the inefficient use of data. While Jinks and Fulker (1970) outlined the inadequacies of classical approaches to the analysis of human variation, it is only recently that hypothesis-tesing approaches have been applied

to the analysis of variation in personality (e.g. Eaves and Eysenck, 1975; Eaves and Eysenck, 1976a; Eaves and Eysenck, 1977; Young et al., 1980). Finally, even when studies have been conducted within a sound theoretical framework and with proper consideration of the needs for efficient techniques of analysis, sample sizes have been too small to allow for reasonable discrimination between alternative models of variation. Consequently, we believe a further study on the causes of variation in personality traits is justified.

When studying personality it has been suggested that one should distinguish between relatively permanent traits and transient symptoms (Foulds 1965, 1974). Of particular interest are symptoms of anxiety and depression which are among the most common complaints seen in medical practice; at least 9% of the population being recognised as having a formal psychiatric disorder (Henderson et al., 1979; Bebbington et al., 1981). Despite the prevalence of anxiety and depression, relatively little is known about the aetiology of these symptoms. Previous studies have suffered from small sample sizes (see Shields, 1976), selection bias (cf Torgensen, 1983) and confusion between traits and symptoms (Young et al., 1971). Thus in Chapter 5 we attempt to determine the causes of variation in symptoms of anxiety and depression.

A third area of psychological interest, related to personality, is the field of social attitudes. In contrast to personality, however, attitudes refer to a particular object that is outside the persons own behaviour. Of the many dimensions of

social attitudes that have been identified (see Eysenck, 1954; Bagley, 1970; Eysenck, 1975; Feather, 1975), conservatism is seen as the general factor which underlies all social attitudes (Wilson, 1973a). Although it is often assumed that attitudes are learned (e.g. Fishbein and Ajzen, 1975), there is evidence from three relatively small twin studies that genetic factors are important to variation in conservatism (see Eaves et al., 1978). In Chapter 6 we present our results, from the largest twin study that has been done to date, on the relative importance of genetic and environmental factors in variation in conservatism.

The final variables chosen for study in this thesis relate to various aspects of drinking behaviour. The use of alcohol in our society is widespread. In the period 1979-1980, which was just prior to our data collection, the apparent per capita consumption of alcohol per year in the Australian population included 134.3 litres of beer, 17.4 litres of wine and 1.0 litres of spirits (Australian Bureau of Statistics, 1981). Although the positive aspects of alcohol consumption are not well documented, the negative aspects are. Thus alcohol consumption is associated with health problems (e.g. alcoholism and cirrhosis of the liver), may have serious psychological and economic effects, and may even lead to death (Seixas and Eggleston, 1976; Luce and Schweitzer, 1978). Although it can not be assumed that the relationship between alcohol consumption and psychological and physical health problems is necessarily causal, it is important to understand the causes of individual differences in alcohol

consumption before attempting to determine the sources of covariation between drinking and disease. Although there have been several studies investigating the causes of individual differences in drinking behaviour (e.g. Perry, 1973; Partanen et al., 1976; Kaprio et al., 1981) the results are far from clear. Thus in Chapter 7 we present data on the genetic and environmental causes of variation in drinking behaviour.

All the data reported in this thesis were collected from the same sample of twins. The method of data collection and features of the sample are described in the next chapter.

CHAPTER 2 ASCERTAINMENT AND STRUCTURE OF THE TWIN SAMPLE

2.1 Ascertainment of the twin sample

A questionnaire containing items concerning drinking and smoking behaviour, sleep patterns, general health, personality and attitudes (Appendix 1) was mailed to all twins aged 18 years and over who were enrolled on the Australian NH and 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% 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. However, we shall compare, were possible, the distribution of scores in this sample with those obtained in random samples in Australia to assess any effects of selection bias.

2.2 Pilot sample

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 the likely response rate and any problems in construction of the questionnaire. Completed responses were obtained by both members of 65 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 96 responses from the original pilot sample of 200 were obtained when the final questionnaire was mailed some months later. However, we thus have 96 individuals who completed the

entire questionnaire twice and whose duplicate responses have been used to assess the short-term repeatability of the various measures studied in this thesis.

2.3 Diagnosis of zygosity

Accurate diagnosis of zygosity is critical to twin research. Early twin studies often based zygosity diagnosis on the common misconception that MZ twins are invariably monochorionic, while DZ twins are dichorionic. However, while all DZ twins are in fact dichorionic, so too are one-third of MZ twins (MacGillvray et al., 1975).

Extensive blood grouping and enzyme typing provides one reliable method of zygosity diagnosis. If same-sex pairs are discordant for any loci then they must be DZ. Although identity for all loci studied may arise by chance, it is possible to calculate the probability that a pair of twins is MZ given that they are identical for particular loci (e.g. Smith and Penrose, 1955). The more loci studied the greater the probability that a pair of twins, identical at all loci, is MZ. The difficulty of such an approach, certainly in large-scale twin studies such as ours, is the need to obtain blood samples and the prohibitive cost of extensive typing.

We have diagnosed the zygosity of same-sex pairs based on their response to the following questions:

1. As children were you and your twin mistaken by people who knew you?
   (a)Frequently (b)Sometimes (c)Rarely
2. "Non-identical twins are no more alike than ordinary brothers and sisters. Identical twins on the other hand

have such a strong resemblance to each other in stature, colouring, features of the face, etc that people often mistake one for the other."
Having read the above statement, do you think you are?
(a)Identical (b)Non-identical

If twins differed in their response to these two 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% 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.

2.4 Summary of sample structure

i)Sex and zygosity distribution of the sample

The sex and zygosity distribution of the twin sample is shown in Table 2.1. As is usual in volunteer samples of this type, there was a bias towards female participants and more MZ than DZ twins (c.f. Lykken et al., 1978). Despite the nearly equal frequencies of MZ and DZ same-sex pairs in populations of European origin (Bulmer, 1970), in our sample 1.6 times as many MZ and DZ same-sex pairs were observed. Martin and Wilson (1982) argue that this inequality could result if sample selection were based on traits which were partly heritable. Thus for any trait where sampling is truncated, a greater proportion of MZ than DZ pairs will be included. It has also been suggested that motivational factors may contribute to the excess of MZ over DZ pairs, and that for social reasons MZ twins are more cooperative with twin research (Lykken et al., 1978).

Table 2.1 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

ii)Age structure of the sample

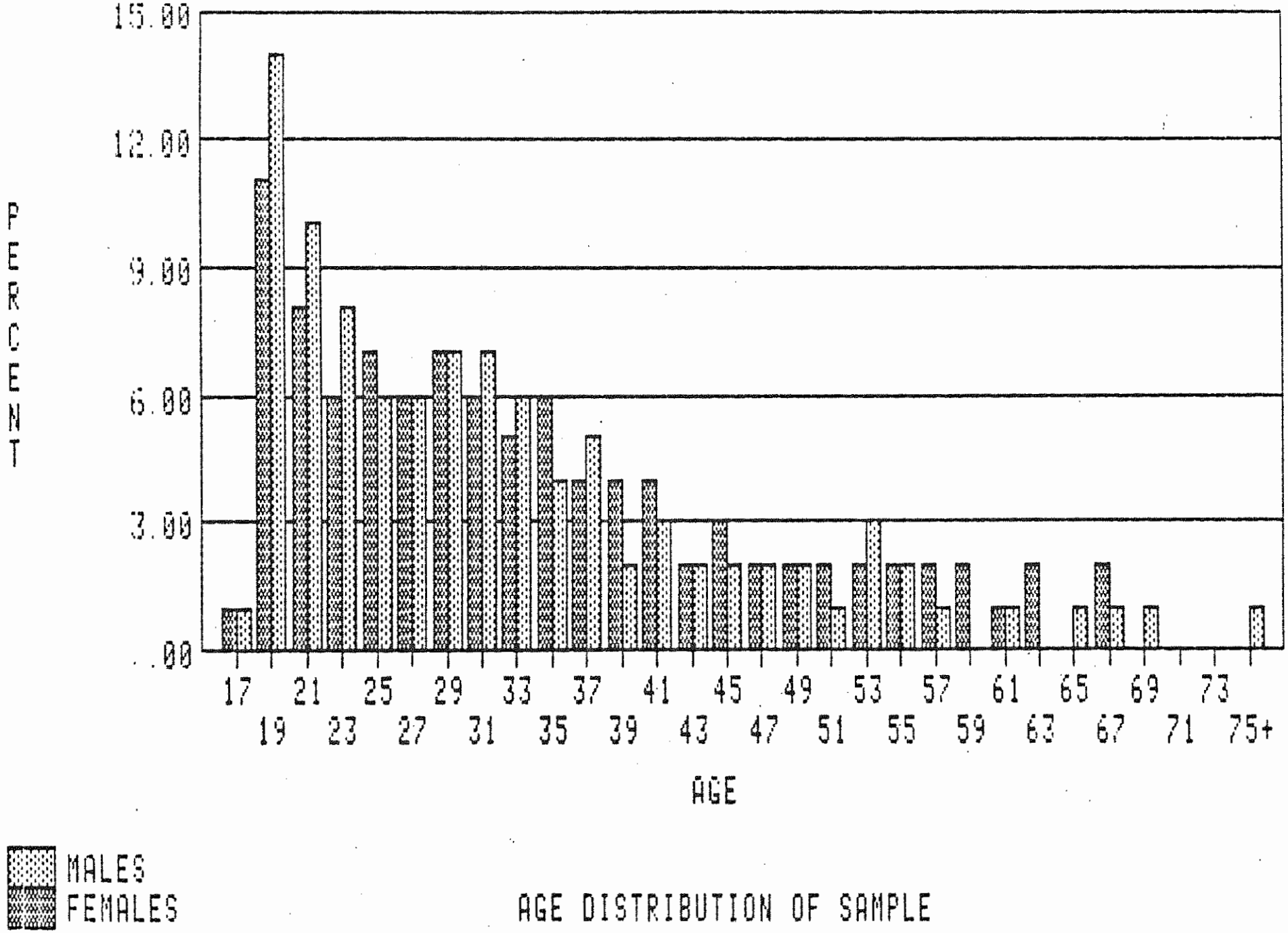
Table 2.1 gives the age distribution of the twin sample broken down by zygosity. The age of twins was calculated from their date of birth and a mean year of return of 1981 to avoid any discrepancies between cotwins who returned the questionnaire in different years.

There was a slight, although statistically significant, difference in the age distribution of the five zygosity groups ($F_{(4,7609)} = 16.01$, $p<.001$). There was a significant difference in the mean age of MZ and DZ males, with MZ males being older ($t_{1834} = 3.12$, $p<.01$), but not MZ and DZ females ($t_{3964} = 0.70$, $p>.05$).

Figure 2.1 shows the age distribution of the sample broken down by sex. Males were significantly older than females ($t_{7612} = 5.14$, $p<.001$), and we see that a disproportionate number of the respondents fall into the under-thirties age group.

iii)Educational level of the sample

Table 2.2 gives the educational level of the twin sample broken down by zygosity. There was a significant difference between the educational achievement of the five twin groups ($\chi^2_{24} = 451.95$, $p<.001$). There was a significant difference between MZ and DZ males ($\chi^2_6 = 15.29$, $p<.05$), but not females ($\chi^2_6 = 8.64$, $p>.05$). Males had a higher educational level than females ($\chi^2_6 = 427.50$, $p<.001$). Our sample also tends to be biased in favour of respondents who have completed schooling and continued with further education.



AGE DISTRIBUTION OF SAMPLE

Table 2.2 Educational level of the twin sample(individuals), broken down by zygosity.

	MZF	MZM	DZF	DZM	DZO
Less than 7 years schooling	53	13	36	9	39
8-10 years schooling	844	175	472	117	394
11-12 years schooling	701	242	447	189	536
Apprenticeship, diploma, certificate, etc.	369	288	261	163	380
Technical or teachers college	296	174	180	78	226
University first degree	148	163	73	111	171
University postgraduate	55	79	33	37	68

CHAPTER 3 THE CAUSES OF VARIATION IN PERSONALITY TRAITS

3.1 INTRODUCTION

Although the concept of personality as a persevering and consistent set of behaviour patterns is sometimes doubted (eg. Mischel, 1968), a number of factor-analytic studies have shown that it is possible to consistently identify factors important to variation in personality. There has been debate, however, over the number of factors which are conceptually important.

Guilford and Guilford (1934) analysing the factor structure of Jung's hypothesised introversion-extraversion dimension initially extracted four, and then five major factors (Guilford and Guilford, 1936), with subsequent analyses resulting in the establishment of 13 primary personality factors (Guilford and Zimmerman, 1956; Guilford, 1975).

Cattell (1945) reduced a pool of some 17000 trait names to 35 clusters from which he extracted 12 factors, with later analyses resulting in the identification of 16 factors (Cattell, 1956). A summary of the primary personality factors identified by Guilford and Cattell is given in Table 3.1.

Eysenck (1947), while he identified a large number of primary factors, placed greater emphasis upon higher order factors. He originally postulated that there were two independent dimensions of personality: extraversion-introversion (E) and neuroticism (N). Extraversion-introversion was based on the intercorrelations of such traits as sociability, impulsiveness and activity, while neuroticism was based on the intercorrelations between such traits as worry, anxiety and

Table 3.1 A summary of the primary personality factors identified by Guilford and Cattell.

Guilford's thirteen primary personality factors	Cattell's sixteen primary personality factors
G General activity	A Cyclothymia vs schizothymia
A Ascendence	B Intelligence
M Masculinity vs femininity	C Ego strength vs dissatisfied emotionality
I Confidence vs inferiority feelings	E Dominance vs submission
N Calmness, composure vs nervousness	F Surgency vs desurgency
S Sociablity	G Super ego strength vs lack of internal standards
T Reflectiveness	H Parmia vs threctia
D Depression	I Premsia vs harria
C_1 Emotionality	L Paranoid tendency vs relaxed security
R Restraint vs rhathymia	M Autia vs praxernia
O Objectivity	N Shrewdness vs naivete
Ag Agreeableness	O Guilt proneness vs confident adequacy
Co Cooperativeness, tolerance	Q_1 Radicalism vs conservatism
	Q_2 Self-sufficiency vs group dependency
	Q_3 High vs low self sentiment formation
	Q_4 High vs low ergic tension

emotionality. While there has been some disagreement about this scheme of personality (eg. Hamilton, 1959a, 1959b), the major alternative schemes of Cattell and Guilford have been shown to result in second-order factors which closely resemble those of Eysenck (Cattell and Scheier, 1961; White et al., 1969a).

Later, Eysenck (1952) postulated that there was a third dimension of personality which was independent of E and N. This factor, psychoticism (P), was based on the observed inter-correlations between such traits as emotional coldness, hostility and egocentricity. Royce (1973) in a review of factor-analytic studies of personality over the past fifty years also found evidence for the existence of three major factors which closely resemble those identified by Eysenck (1952), suggesting that the factors of extraversion-introversion, neuroticism and psychoticism can be used to adequately describe the major dimensions of personality. In addition to these three major factors, Eysenck also identified a fourth factor, lie, a measure of dissimulation or the tendency to "fake good" (Eysenck and Eysenck, 1964). This factor, in addition to measuring dissimulation, however, also seems to measure some degree of conformity or conventionality. While the precise nature of this factor is therefore in doubt, there is evidence that lie represents some stable personality attribute (see Eysenck and Eysenck, 1976). It is one of the aims of this present study to replicate the factor structure of personality postulated by Eysenck.

3.1.1 Previous work on the causes of individual differences in personality

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 (eg. Eysenck, 1967) 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, 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 the 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).

Data from adoption studies (Loehlin et al., 1981; Scarr et al., 1981) confirm some of the results from twin studies. Adoptive family members show little resemblance in personality, which is consistent with the finding that there is no effect of

family environment on personality. However, the correlations between biological relatives (sibling and parent-offspring) are lower than would be predicted from the results of twin studies. Thus, this disparity in correlations may be attributable to genotype x age interaction which is a problem inherent in the nuclear family design. Thus to the extent that genes are differentially expressed at different ages, parent-offspring and sibling correlations will be reduced in comparison to those of twins.

In general the results from these various studies 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) reanalysed their data from 12898 same-sex 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.

A superficial resemblance to dominance could however be generated if there were any sibling competition, that is if an increasing allele in one twin exerted a decreasing environmental influence on the co-twin (Eaves, 1976). There is some evidence that competition may be important for extraversion. Jinks and Fulker (1970) in a reanalysis of Shields' (1962) data on extraversion found that twins reared together tended to develop opposite characteristics and that this effect was based on genetic differences. Young et al. (1980) fitted the competition

model developed by Eaves (1976) to extraversion data collected from a juvenile sample and found evidence of a negative sibling influence. In this study we shall compare the ability of models incorporating dominance or competition to explain variation in extraversion.

The studies on the causes of individual differences in personality discussed so far were based on the analysis of test scores summed over a number of discrete item responses. While the use of composite scales is justified in terms of their validity and reliability, some loss of information may be involved. For example, although Eaves and Eysenck (1975) found that a simple genetic model could explain variation in extraversion, and the covariation of its component scales sociability and impulsiveness, there was some evidence that it was possible to distinguish genetically between the two components of extraversion. In contrast, Loehlin and Nichols (1976) were unable to distinguish either composite personality scales or individual items in terms of differences between MZ and DZ concordance rates. Their results, however, based on intraclass correlations, are possibly misleading and certainly far less informative than an explicit hypothesis testing approach (Jinks and Fulker, 1970). Indeed, Zonderman (1982) has shown that Loehlin and Nichols (1976) conclusions, based on their analysis of the composite scales, are not supported by the data.

In this study we shall first use factor-analytic methods in an attempt to distinguish major aspects of personality. We shall then examine the extent to which different genetic and

environmental sources of variation are important in determining variation in the personality traits of extraversion, psychoticism, neuroticism and lie, as well as examine the causes of variation in the individual items defining these scales. If an analysis of composite scores obscures item heterogeneity, it may be possible to define a relationship between items which have a predominantly genetic or environmental basis. Thus, we shall conclude our analysis by examining whether it is possible to define any consistent relationships between the results of our factor analysis and the genetic analysis of the individual items and composite test scores. We hope to determine whether one can statistically distinguish between major dimensions of personality, and if this is possible, whether these dimensions are characterised by differences in their genetic or social architecture at both the item and trait level.

3.2 MEASUREMENTS

3.2.1 Eysenck Personality Questionnaire (EPQ)

The EPQ (Eysenck and Eysenck, 1975) attempts to summarise 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 90 items of the Yes/No type. The items defining the scales of E, P, N and L are shown in Table 3.2. The factors are scored in the direction of naming, so for E, extraverted responses are scored +1, and introverted responses are scored 0. The range of scores are for E, 0-21; P, 0-25;

Table 3.2 Items defining the extraversion, psychoticism, neuroticism and lie scales of the EPQ.

Extraversion:

E1. Do you have many different hobbies?
E2. Are you a talkative person?
E3. Are you rather lively?
E4. Can you usually let yourself go and and enjoy yourself at a lively party?
E5. Do you enjoy meeting new people?
E6. Do you tend to keep in the background on social occasions?
E7. Do you like going out a lot?
E8. Do you prefer reading to meeting new people?
E9. Do you have many friends?
E10. Would you call yourself happy-go-lucky?
E11. Do you usually take the initiative in making new friends?
E12. Are you mostly quiet with with other people?
E13. Can you usually get some life into a rather dull party?
E14. Do you like telling jokes and funny stories to your friends?
E15. Do you like mixing with people?
E16. Do you nearly always have a "ready answer" when people talk to you?
E17. Do you like doing things in which you have to act quickly?
E18. Do you often take on more activities than you have time for?
E19. Can you get a party going?
E20. Do you like plenty of bustle and excitement around you?
E21. Do other people think of you as being rather lively?

Psychoticism:

P1. Do you stop to think things over before doing anything?
P2. Would being in debt worry you?
P3. Do you lock up your house carefully at night?
P4. Would it upset you to see a child or an animal suffer?
P5. Do you believe insurance schemes are a good idea?
P6. Would you take drugs which may have strange or dangerous effects?
P7. Do you enjoy hurting the people you love?
P8. Do you have enemies who want to harm you?
P9. Do you enjoy practical jokes that can sometimes really hurt people?
P10. Do good manners and cleanliness matter much to you?
P11. Do you think marriage is old-fashioned and should be done away with?
P12. Do people who drive carefully annoy you?
P13. Do most things taste the same to you?
P14. Does it worry you if you know there are mistakes in your work?
P15. Do you like to arrive at appointments in plenty of time?
P16. Is (or was) your mother a good woman?
P17. Are there several people who keep trying to avoid you?
P18. Do you think people spend too much time safeguarding their future with savings and insurances?
P19. Do you try not to be rude to people?
P20. When you catch a train do you often arrive at the last minute?
P21. Do your friendships break up easily without it being your fault?
P22. Do you sometimes like teasing animals?
P23. Would you like people to be afraid of you?
P24. Do people tell you a lot of lies?
P25. Would you feel sorry for an animal caught in a trap?

Table 3.2 cont'd

Neuroticism:

N1. Does your mood often go up and down?
N2. Do you ever feel "just miserable" for no reason?
N3. Do you often worry about things you should not have done or said?
N4. Are you an irritable person?
N5. Are your feelings easily hurt?
N6. Do you often feel "fed-up"?
N7. Are you often troubled by feelings of guilt?
N8. Would you call yourself a nervous person?
N9. Are you a worrier?
N10. Do you worry about awful things that might happen?
N11. Would you call yourself "higly-strung"?
N12. Do you worry about your health?
N13. Do you suffer from sleeplessness?
N14. Have you often felt listless and tired for no reason?
N15. Do you often feel life is very dull?
N16. Do you worry about your looks?
N17. Have you ever wished that you were dead?
N18. Do you worry too long after an embarassing experience?
N19. Do you suffer from nerves?
N20. Do you often feel lonely?
N21. Are you easily hurt when people find fault with you or the work you do?
N22. Are you sometimes bubbling over with energy and sometimes very sluggish?
N23. Are you touchy about some things?

Lie:

L1. Have you ever taken praise for something you knew someone else had really done?
L2. Were you ever gready by helping yourself to more than your share of anything?
L3. If you say you will do something, do you always keep your promise no matter how inconvenient it might be?
L4. Have you ever blamed someone for doing something you knew was really your fault?
L5. Are ALL your habits good and desirable ones?
L6. Have you ever taken a anything (even a pin or a button) that belonged to someone else?
L7. Do you sometimes talk about things you know nothing about?
L8. As a child did you do as you were told immediately and without grumbling?
L9. Have you ever broken or lost something belonging to someone else?
L10. Do you sometimes boast a little?
L11. Have you ever said anything bad or nasty about anyone?
L12. As a child were you ever cheeky to your parents?
L13. Do you always wash before a meal?
L14. Have you ever cheated at a game?
L15. Have you ever taken advantage of someone?
L16. Would you dodge paying taxes if you were sure you could never be found out?
L17. Have you ever insisted on having your own way?
L18. Do you always practice what you preach?
L19. Have you ever been late for an appointment or work?
L20. Do you sometimes put off until tomorrow what you ought to do today?
L21. Are you always willing to admit it when you have made a mistake?

N, 0-23 and L, 0-21. 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).

3.3 RESULTS

3.3.1 Distribution of item scores and sex differences

We shall first test whether the MZ and DZ groups are comparable. Chi-square tests were performed to determine if there were any significant differences between MZ and DZ endorsement frequencies for individual items, separately for females (Table 3.3) and males (Table 3.4). Of the 180 chi-square tests only 15 were significant at least at the 5% level and there was no consistent pattern in these differences. The groups appear to be comparable and thus the MZ and DZ classes were combined to examine the effect of sex on endorsement frequency.

Table 3.5 presents the percentage of individuals endorsing an item, broken down by sex. Chi-square tests were performed between male and female endorsement frequencies (Table 3.5). For extraversion, women give more extraverted responses for 7 of the items, while men give more extraverted responses for 8 of the items, the remaining 6 items showing no sex difference. For psychoticism, all of the items show significant sex differences and for only 2 items do women give more psychotic responses than men. In the case of neuroticism, with the exception of the two items showing no difference between the sexes, women give consistently more neurotic responses than men. For lie, women give more socially desirable responses for all the items showing

Table 3.3 Chi-squares (for one degree of freedom) testing the significance of differences between MZ and DZ endorsement frequencies for the EPQ items in females. Asterisks denote significant differences between MZ and DZ endorsement frequencies.

Item	Extraversion	Psychoticism	Neuroticism	Lie
1	0.05	0.09	2.81	1.40
2	0.28	1.24	0.00	0.21
3	3.42	7.64**	0.23	0.34
4	0.01	0.18	0.25	0.69
5	1.15	8.57**	2.99	0.00
6	0.61	1.49	1.75	2.02
7	1.99	0.33	0.00	3.44
8	0.01	0.67	1.28	0.20
9	2.27	0.06	0.11	0.02
10	1.20	0.00	0.13	0.47
11	0.19	1.03	0.20	0.59
12	1.30	1.04	0.89	0.10
13	8.14**	1.81	0.14	0.66
14	0.04	0.02	1.58	4.06*
15	1.64	1.44	1.29	2.11
16	0.03	3.01	0.35	2.18
17	0.17	0.11	3.21	0.39
18	6.99**	2.73	0.00	8.14**
19	1.25	12.00***	0.29	1.45
20	2.48	2.53	1.83	0.66
21	3.13	0.33	0.16	1.74
22		0.31	1.06	
23		1.01	0.51	
24		0.01		
25		0.37		

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

Table 3.4 Chi-squares (for one degree of freedom) testing the significance of differences in MZ and DZ endorsement frequencies for the EPQ items in males.

Item	Extraversion	Psychoticism	Neuroticism	Lie
1	0.08	3.37	0.81	0.56
2	0.37	0.00	3.41	1.12
3	0.60	1.13	0.00	5.50*
4	1.75	1.69	3.49	0.06
5	0.29	0.01	2.40	0.80
6	0.04	1.05	0.27	0.05
7	3.38	1.00	4.46*	6.02*
8	4.54*	0.05	0.08	0.00
9	0.69	1.35	0.10	1.26
10	0.79	2.16	1.14	1.40
11	1.47	2.67	0.35	0.19
12	1.48	0.15	2.00	0.02
13	0.48	1.05	0.70	0.02
14	0.01	0.87	0.55	3.16
15	0.03	2.18	1.37	0.00
16	0.36	0.95	0.52	0.73
17	11.57***	0.02	0.00	1.11
18	0.55	1.21	0.11	4.95*
19	2.35	2.57	0.70	0.00
20	4.78*	0.32	2.58	0.75
21	0.15	1.34	0.84	0.47
22		4.79*	0.74	
23		1.45	0.00	
24		0.00		
25		1.94		

Table 3.5 Percentage of individuals endorsing an EPQ item in the direction of naming, broken down by sex. Asterisks denote significant differences between male and female endorsement frequencies.

	Extraversion		Psychoticism		Neuroticism		Lie	
Item	Females	Males	Females	Males	Females	Males	Females	Males
1	52.6	49.2**	19.0	13.6***	58.1	50.1***	85.5	79.5***
2	58.8	49.1***	8.5	23.0***	51.1	26.9***	58.1	51.6***
3	70.7	68.5*	30.4	40.9***	80.8	71.9***	76.9	67.6***
4	67.2	74.1***	1.0	4.1***	23.2	22.1	69.6	67.6
5	84.3	82.4*	21.9	26.9***	74.9	58.1***	40.4	35.8***
6	45.3	50.4***	4.5	8.4***	48.8	37.1***	36.0	21.0***
7	62.0	58.2**	1.1	1.9***	37.8	34.2**	61.4	58.2**
8	77.5	79.3	3.3	7.9***	31.9	24.0***	36.6	33.6**
9	79.8	79.5	2.5	8.5***	60.0	46.5***	38.1	27.6***
10	50.4	51.9	3.7	8.7***	49.0	35.5***	38.8	27.6***
11	49.4	49.1	3.7	6.6***	24.8	18.0***	15.4	14.1
12	55.1	54.1	12.6	23.1***	41.8	43.9	41.0	40.5
13	29.4	36.9***	5.3	9.7***	21.6	15.5***	48.7	49.7
14	64.2	79.1***	8.6	13.0***	55.9	42.5***	57.8	42.3***
15	56.8	84.2**	9.3	14.5***	26.6	22.1***	68.8	51.6***
16	45.0	52.4***	2.2	1.3***	50.3	37.0***	54.7	33.0***
17	54.9	71.4***	5.7	10.4***	28.6	20.4***	17.8	18.6
18	50.6	54.7***	32.0	41.0***	64.7	47.5***	58.6	56.3
19	40.0	47.8***	6.2	11.0***	34.3	27.0***	64.8	71.4***
20	57.7	56.3	24.7	29.0***	32.5	25.9***	15.9	14.5
21	63.0	59.3**	7.0	9.3***	76.0	60.2***	74.1	72.2
22			10.9	27.5***	77.6	69.1***		
23			1.9	8.7***	81.7	74.4***		
24			8.4	15.2***				
25			2.9	14.4***				

sex differences. Thus out of 90 items, 74 show significant sex differences indicating that the effect of sex on endorsement frequency must be incorporated into our analyses.

3.3.2 Factor analysis of the Eysenck Personality Questionnaire

The 90 items of the EPQ were intercorrelated using product moment correlations (= phi coefficients), separately for males and females. The use of this coefficient has been criticised in the factor analysis of dichotomous variables because the estimates of the coefficients are not independent of the means of the variables when the latter are unequal (Maxwell, 1977). Although this may result in a spurious 'difficulty' factor (Cattell, 1952; Ferguson, 1941), this can generally be detected as it will have loadings closely proportional to the means of the variables (Maxwell,1977). Although tetrachoric correlations and a corrected phi coefficient, phi divided by maximum phi, have been preferred these often lead to unreasonably large communalities (sometimes greater than one), especially when there are unequal means, a situation where these alternative coefficients are supposed to be better (Comrey and Levonian, 1958). This problem has been examined empirically by Comrey and Levonian (1958) and they conclude "that the phi coefficient is the method of choice in point correlation work were factor analysis is to follow" (p. 753). Furthermore, they state that "if spurious factors exist with factor analysis of phi coefficients, they may be no less evident with phi-over-phi-max or tetrachoric coefficients" (p. 753). These results support our use of phi coefficients in this study.

Principal factoring with iteration, a procedure available in the Statistical Package for the Social Sciences (Nie et al., 1975), was performed on the EPQ item correlation matrices obtained separately for males and females. Although 24 factors had eigenvalues greater than one, only 12 could be interpreted with any confidence, that is, were defined by more than one or two items. As our primary interest was to test the model of personality postulated by Eysenck, we first extracted four orthogonal factors followed by varimax rotation, and compared this to a four factor solution obtained with oblique rotation. Because there is no one best solution in factor analysis, by using different methods of rotation we can determine to what extent our final solution is dependent on the method of analysis chosen. The factors were named in terms of the items having the highest loadings and for purposes of interpretation a loading of 0.25 or greater was considered large. In the tables of the resulting factor pattern matrices to follow, the order of factors is arbitrary and does not necessarily correspond to the sequence in which they were extracted.

Factor 1 (Table 3.6) is clearly an extraversion factor. In both males and females, only two items (E1, "Do you have many different hobbies?" and E18, "Do you often take on more activities than you have time for?") fail to load substantially on this factor. None of the items from the other scales define this factor. The loadings are similar in males and females suggesting agreement in the structure of extraversion between the sexes. Removing the constraint of orthogonality does not appear to alter the pattern of results markedly.

Table 3.6 Factor pattern coefficients of items defining factor 1, extraversion, from the four factor solution.

	Females Solution			Males Solution	
Item	Orthogonal	Oblique	Item	Orthogonal	Oblique
E2	.54	.54	E2	.54	.54
E3	.61	.61	E3	.59	.60
E4	.56	.56	E4	.51	.50
E5	.51	.51	E5	.50	.50
E6	-.62	-.62	E6	-.62	-.62
E7	.45	.45	E7	.47	.47
E8	-.48	-.48	E8	-.48	-.48
E9	.44	.45	E9	.44	.43
E10	.38	.38	E10	.35	.36
E11	.55	.56	E11	.56	.56
E12	-.58	-.57	E12	-.56	-.55
E13	.55	.56	E13	.61	.62
E14	.34	.33	E14	.35	.34
E15	.55	.55	E15	.55	.54
E16	.31	.32	E16	.31	.32
E17	.32	.31	E17	.33	.33
E19	.56	.57	E19	.60	.61
E20	.46	.46	E20	.50	.50
E21	.59	.60	E21	.60	.61

Factor 2 (Table 3.7) is not as well defined. In females, the orthogonal solution results in only three items, from three different scales, loading on this factor. The oblique solution results in one additional item (P18) contributing to this factor. In males the pattern is somewhat clearer. Of the 9 items defining this factor, 8 are from the P scale. This general trend can be seen more clearly in Table 3.8. Shown are the loadings of all P items on this tentative psychoticism factor. Of the 25 items defining P, 10 items in females, and 21 in males, have loadings in the predicted direction. While this factor is not particularly strong, it does seem identifiable as psychoticism, particularly in males. It has been suggested that an oblique solution may help to clarify weak factors (e.g. Eysenck et al., 1969), however, in our data there is little difference between the orthogonal and oblique solutions.

Factor 3 (Table 3.9) is easily identified as neuroticism. In both sexes all of the N items, and only these items, contribute substantially to this factor. The structure of neuroticism appears to be the same in males and females, and again removing the constraint of orthogonality appears to make little difference.

Factor 4 (Table 3.10) is easily identified as lie. Only one lie item in males (L16, "Would you dodge paying taxes if you were sure you could never be found out?") loads marginally on this factor (-0.22). With this exception, there is considerable agreement between the sexes and different methods of rotation.

Table 3.7 Factor pattern coefficients of items defining factor 2, psychoticism, from the four factor solution.

Females Solution				Males Solution		
Item	Orthogonal	Item	Oblique	Item	Orthogonal	Oblique
E13	.25	P8	.25	P4	-.26	-.26
N15	.26	E13	.25	P9	.31	.30
P18	.29	N15	.26	P10	-.35	-.34
		P18	.29	P14	-.33	-.33
				N15	.31	.30
				P18	.26	.26
				P19	-.28	-.28
				P23	.29	.27
				P24	.28	.28

Table 3.8 Factor pattern coefficients of all P items on factor 2, psychoticism, from the four factor solution.

	Females Solution		Males Solution	
Item	Orthogonal	Oblique	Orthogonal	Oblique
P1	-.18	-.18	-.23	-.23
P2	-.14	-.14	-.24	-.24
P3	-.15	-.14	-.20	-.19
P4	-.10	-.10	-.26	-.26
P5	-.23	-.24	-.20	-.21
P6	.24	.23	.17	.15
P7	.13	.13	.22	.22
P8	.24	.25	-.24	-.24
P9	.17	.16	.31	.30
P10	-.15	-.14	-.35	-.34
P11	.24	.24	.24	.24
P12	.16	.15	.20	.19
P13	.17	.18	.21	.22
P14	-.21	-.21	-.33	-.33
P15	-.09	-.07	-.15	-.14
P16	-.09	-.09	-.17	-.16
P17	.23	.24	.23	.22
P18	.29	.29	.26	.26
P19	-.14	-.15	-.28	-.28
P20	.06	.04	.08	.06
P21	.22	.22	.24	.24
P22	.14	.12	.20	.18
P23	.21	.21	.29	.27
P24	.23	.24	.28	.28
P25	-.09	-.08	-.23	-.23

Table 3.9 Factor pattern coefficients of items defining factor 3, neuroticism, from the four factor solution.

	Females Solution		Males Solution	
Item	Orthogonal	Oblique	Orthogonal	Oblique
N1	.52	.50	.48	.47
N2	.43	.41	.47	.46
N3	.44	.44	.47	.47
N4	.41	.39	.38	.37
N5	.46	.46	.50	.50
N6	.55	.52	.55	.54
N7	.47	.46	.50	.49
N8	.55	.54	.56	.57
N9	.61	.62	.64	.65
N10	.50	.50	.47	.48
N11	.49	.49	.49	.50
N12	.38	.39	.34	.35
N13	.35	.34	.32	.33
N14	.44	.43	.43	.42
N15	.41	.37	.40	.38
N16	.37	.38	.35	.35
N17	.29	.27	.32	.30
N18	.47	.47	.50	.49
N19	.56	.55	.50	.50
N20	.45	.42	.45	.44
N21	.41	.41	.45	.45
N22	.30	.30	.31	.30
N23	.38	.38	.38	.36

Table 3.10 Factor pattern coefficients of items defining factor 4, lie, from the four factor solution.

	Females Solution			Males Solution	
Item	Orthogonal	Oblique	Item	Orthogonal	Oblique
L1	-.37	-.37	L1	-.39	-.38
L2	-.56	-.58	L2	-.51	-.52
L3	.37	.37	L3	.34	.34
L4	-.42	-.42	L4	-.39	-.38
L5	.45	.46	L5	.41	.42
L6	-.48	-.49	L6	-.46	-.48
L7	-.36	-.35	L7	-.39	-.38
L8	.39	.39	L8	.35	.36
L9	-.43	-.44	L9	-.43	-.44
L10	-.40	-.38	L10	-.40	-.38
L11	-.46	-.47	L11	-.48	-.48
L12	-.39	-.38	L12	-.39	-.38
L13	.32	.32	L13	.32	.32
L14	-.49	-.48	L14	-.52	-.52
L15	-.51	-.50	L15	-.51	-.50
L16	-.28	-.27	L17	-.28	-.29
L17	-.31	-.31	L18	.47	.47
L18	.47	.48	L19	-.38	-.39
L19	-.37	-.38	L20	-.37	-.38
L20	-.30	-.31	L21	.34	.35
L21	.38	.39			

The correlations between the factors for the oblique solution are shown in Table 3.11. The factors do depart slightly from orthogonality, and the same pattern of relationships has been found using earlier versions of the E, P, N and L scales (Eysenck and Eysenck, 1968; Michaelis and Eysenck, 1971). The correlations are however fairly small, and the fact that the factor patterns are so similar for the orthogonal and oblique solutions suggests that the factors of E, P, N and L can be considered as relatively independent dimensions of personality.

The four principal factors account for 22.3% and 21.4% of the variance in items in males and females respectively. The strongest factor is E (7.8% in males, 7.6% in females) followed by N (7.2%, 7.1%), L (4.3%, 4.3%) then P (2.9%, 2.5%). With the exception of P, these percentages are slightly higher than those found by Michaelis and Eysenck (1971) using earlier versions of the four scales.

These results provide support for the existence of extraversion, neuroticism and lie, and to some extent psychoticism, as major dimensions of personality. As previous studies have shown that it is possible to identify other dimensions of personality (e.g. Guilford, 1975; Cattell, 1956; White et al., 1969b), we decided to examine more extensively the factor structure of the EPQ to determine whether we could isolate sub-factors of personality which were independent of E, P, N and L.

Using the method of principal factoring with iteration, we extracted 12 orthogonal factors with varimax

Table 3.11 Correlations between factors from the oblique four factor solution.

		Females			
		E	P	N	L
Males					
	E		.04	-.11	-.14
	P	.02		.09	-.18
	N	-.09	.06		-.09
	L	-.15	-.18	-.14	

rotation from the EPQ item correlation matrices obtained separately for males and females. We chose to extract this number of factors because, as mentioned previously, a prelimanary analysis had shown that only 12 factors could be identified with any confidence. Orthogonal rotation was employed to simplify the interpretation of the results. In the discussion to follow the order of factors does not correspond to the sequence of extraction.

Factor 1 (Table 3.12) is easily interpreted as a general extraversion factor. Similar to the four factor solution there is considerable agreement between the sexes, and items E1 and E18 are the only two extraversion items which fail to load substantially on this factor. Factor 2 (Table 3.13) is psychoticism. Although again this is not a strong factor, particularly in females, in contrast to the four factor solution only P items load on this factor. Factor 3 (Table 3.14), neuroticism, is more strongly identified in males than females, and clearly its expression in the two sexes is not identical. Similarly Factor 4 (Table 3.15), lie, shows some sex differences and it is more pronounced in females than males.

Sub-factors of personality also show differences in expression between the sexes. Two additional extraversion factors in males, and three in females were identified. In males, Factor 5 (Table 3.16) seems to represent sociability (enjoying meeting and mixing with people) but not gregariousness (can't get life into a dull party or get a party going). In females this factor is more strongly identified as sociability

Table 3.12 Factor pattern coefficients of items defining factor 1, extraversion, from the 12 factor solution.

Item	Females	Males
E2	.58	.52
E3	.61	.59
E4	.54	.51
E5	.41	.55
E6	-.66	-.60
E7	.41	.50
E8	-.40	-.52
E9	.38	.45
E10	.35	.36
E11	.53	.54
E12	-.65	-.54
E13	.58	.57
E14	.32	.35
E15	.44	.60
E16	.33	.27
E17	.30	.32
E19	.57	.57
E20	.43	.51
E21	.61	.59
Variance accounted for	7.6%	7.8%

Table 3.13 Factor pattern coefficients of items defining factor 2, psychoticism, from the 12 factor solution.

Item	Females	Item	Males
P17	.27	P4	-.33
P21	.25	P9	.35
P23	.26	P10	-.30
P24	.29	P19	-.31
		P22	.29
		P23	.34
		P25	-.33
Variance accounted for	1.8%		2.9%

Table 3.14 Factor pattern coefficients of items defining factor 3, neuroticism, from the 12 factor solution.

Item	Females	Item	Males
N1	.55	N1	.52
N2	.50	N2	.48
N3	.27	N3	.50
N4	.36	N4	.35
N6	.62	N5	.50
N7	.37	N6	.60
N8	.26	N7	.50
N9	.33	N8	.41
N10	.32	N9	.55
N11	.26	N10	.46
N13	.26	N11	.34
N14	.49	N12	.33
N15	.51	N13	.28
N17	.31	N14	.44
N18	.22	N15	.44
N19	.29	N16	.38
N20	.52	N17	.33
N22	.33	N18	.50
		N19	.35
		N20	.47
		N21	.46
		N22	.33
		N23	.35
Variance accounted for	4.3%		7.2%

Table 3.15 Factor pattern coefficients of items defining factor 4, lie, from the 12 factor solution.

Item	Females	Item	Males
L1	-.40	L1	-.37
L2	-.57	L2	-.50
L3	.34	L3	.29
L4	-.46	L4	-.40
L5	.44	L5	.42
L6	-.48	L6	-.48
L7	-.37	L7	-.39
L8	.33	L8	.36
L9	-.43	L9	-.44
L10	-.43	L10	-.40
L11	-.45	L11	-.50
L12	-.34	L12	-.41
L13	.28	L13	.29
L14	-.51	L14	-.54
L15	-.54	L15	-.53
L16	-.25	L17	-.30
L17	-.31	L18	.45
L18	.45	L19	-.35
L19	-.32	L20	-.33
L20	-.27	L21	.30
L21	.37		
Variance accounted for	7.1%		4.3%

Table 3.16 Factor pattern coefficients of items defining subfactors of extraversion, obtained from the 12 factor solution.

Factor 5: Sociability		
Item	Short description	Females
E5	Enjoy meeting new people	.59
E7	Like going out a lot	.25
E8	Prefer reading to meeting people	-.43
E15	Like mixing with people	.58
	Variance accounted for	1.8%
Item	Short description	Males
E5	Enjoy meeting new people	.29
E13	Get life into a dull party	-.47
E15	Like mixing with people	.28
E19	Get a party going	-.42
	Variance accounted for	1.5%
Factor 6: Females - Gregariousness		
Item	Short description	Coefficient
E13	Get life into a dull party	.46
E19	Get a party going	.47
	Variance accounted for	1.4%
Factor 6: Males - Social shyness		
Item	Short description	Coefficient
E2	Talkative person	-.26
E6	Keep in the background on social occasions	.32
E12	Quiet with other people	.38
	Variance accounted for	1.4%
Factor 7: Females - Liveliness, activity		
Item	Short description	Coefficient
E1	Many different hobbies	.27
E3	Rather lively	.25
E18	More activities than time for	.34
E21	People think of you as lively	.29
	Variance accounted for	1.5%

(preferring meeting people to reading, going out a lot, enjoying meeting and mixing with people), and it is interesting that the two items which have a substantial negative loading on this factor in males (E13, "Can you easily get some life into a dull party?" and E19, "Can you get a party going?") appear as a separate gregariousness factor in females (Factor 6, Table 3.16). However, as this factor is only defined by these two items it may be just a statistical artefact. The two remaining extraversion factors are completely different in males and females. Factor 6 in males, (Table 3.16) seems to represent shyness in social situations, (keeping in the background on social occasions, mostly quiet with other people), while factor 7 in females (Table 3.16) is defined by items concerning liveliness and activity.

Two sub-factors of psychoticism in females, and three in males were identified. Factor 7 in males (Table 3.17) seems to represent paranoia (enemies want to harm them, people avoid and lie to them). Factor 8 (Table 3.17) is easily identified as punctuality (arriving for appointments in plenty of time, never catching a train at the last minute) and is consistent across sexes. However, as this factor contains only one lie item, it is questionable as to whether it can be regarded as a true sub-factor of psychoticism. Similarly, Factor 9 (Table 3.17) is more suggestive of the social attitude of conservatism (attitudes to insurance, drug taking and marriage) rather than psychotic behaviour, and there are differences in the expression of this factor in males and females.

Table 3.17 Factor pattern coefficients of items defining subfactors of psychoticism, obtained from the 12 factor solution.

Factor 7: Males - Paranoia

Item	Short description	Coefficient
P8	Enemies want to harm you	.39
P17	People try to avoid you	.39
P21	Friendships break up, but not their fault	.26
P24	People lie to you	.34
	Variance accounted for	1.6%

Factor 8: Punctuality

Item	Short description	Females	Males
P15	Arrive for appointments in plenty of time	.51	.52
P20	Arrive at the last minute to catch a train	-.67	-.57
L19	Ever late for an appointment or work	-.37	-.37
	Variance accounted for	1.6%	1.7%

Factor 9: Conservatism

Item	Short description	Females
P5	Insurance schemes a good idea	.44
P6	Take drugs with strange or dangerous effects	-.26
P11	Marriage old-fashioned	-.26
P18	Too much time spent safeguarding future with insurances and savings	-.44
	Variance accounted for	1.6%

Item	Short description	Males
P5	Insurance schemes a good idea	.33
P18	Too much time spent safeguarding future with insurances and savings	-.40
	Variance accounted for	1.9%

Sex differences were also evident in the two sub-factors of neuroticism that were identified. Factor 10 (Table 3.18) is sensitivity (feelings easily hurt, touchy about some things, a worrier), and is more strongly identified in females than males. Similarly Factor 11 (Table 3.18), anxiety (nervous, tense), shows some difference in expression between the sexes.

Only one additional lie factor was obtained. While this factor (Factor 12, Table 3.19) seems to represent acquiescence as a child in females, it is not easily interpreted in males and we will not attempt to name it.

These 12 principal factors account for 35.3% and 34.5% of the variance in males and females respectively, indicating other factors may be involved. The proportions of variance accounted for by each factor are shown in the individual summary tables. Our results show marked differences in the expression and importance of factors in males and females. While it has been possible to identify several sub-factors of personality, clearly the most consistently identified factors are E, P, N and L.

3.3.3 Scaling

We shall now test some of the assumptions implicit in the twin method. 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 x E_1 interaction. Table 3.20 shows these regressions for MZ male and female twins for the raw scores and

Table 3.18 Factor pattern coefficients of items defining subfactors of neuroticism, obtained from the 12 factor solution.

Factor 10: Sensitivity		
Item	Short description	Females
N3	Worry about things not done or said	.38
N5	Feelings easily hurt	.58
N7	Troubled by feelings of guilt	.25
N9	Are you a worrier	.35
N10	Worry about awful things that might happen	.29
N16	Worry about looks	.32
N18	Worry too long after an embarassing experience	.48
N21	Hurt when people find fault with work or themselves	.61
N23	Touchy about some things	.35
	Variance accounted for	2.5%
Item	Short description	Males
N5	Feelings easily hurt	.30
N21	Hurt when people find fault with work or themselves	.35
	Variance accounted for	1.6%

Factor 11: Anxiety		
Item	Short description	Females
N8	A nervous person	.69
N9	Are you a worrier	.41
N11	Tense or "highly-strung"	.61
N13	Suffer from sleeplessness	.28
N19	Suffer from nerves	.67
	Variance accounted for	1.9%
Item	Short description	Males
N8	A nervous person	.66
N9	Are you a worrier	.34
N11	Tense or "highly-strung"	.56
N19	Suffer from nerves	.62
	Variance accounted for	2.0%

Table 3.19 Factor pattern coefficients of items defining a subfactor of lie, obtained from the 12 factor solution.

Factor 11: Not interpreted		
Item	Short description	Females
L8	Did what told immediately as a child	.50
L12	Ever cheeky to parents as a child	-.52
	Variance accounted for	1.4%
Item	Short description	Males
L1	Taken praise for something someone else had done	-.27
L4	Blamed someone for something that is your fault	-.29
L21	Always willing to admit mistakes	-.33
	Variance accounted for	1.4%

Table 3.20 Proportions of variance in absolute within-pair differences accounted for by regression on pair sums for the raw EPQ scores and various transformations. Linear (L) and quadratic components after the linear regression has been removed (Q) are shown.

		MZF		MZM	
		L	Q	L	Q
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

various transformations. Martin and Eysenck (1976) showed that such interactions could be detected with great sensitivity but they could nearly always be removed by a transformation of the scale of measurement which lessened departures from normality.*

The extraversion scale shows negative skewness (Figure 3.1) which produces significant linear and quadratic regression. The psychoticism scale shows positive skewness (Figure 3.2) producing linear regression. The neuroticism scale shows a reasonably symmetric distribution but with an appreciable number of observations at the extremes producing a "basement-ceiling" effect (Figure 3.3) which results in quadratic regression. The lie scale shows a slight tendency toward positive skewness (Figure 3.4) which results in quadratic regression. In all cases these regressions are best reduced by angular transformation $(\arcsin \sqrt{p})$[1] (Snedecor and Cochran, 1980). Although in most cases transformations to minimise G x 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 psychoticism scale, the results may differ markedly (Martin and Eysenck, 1976).

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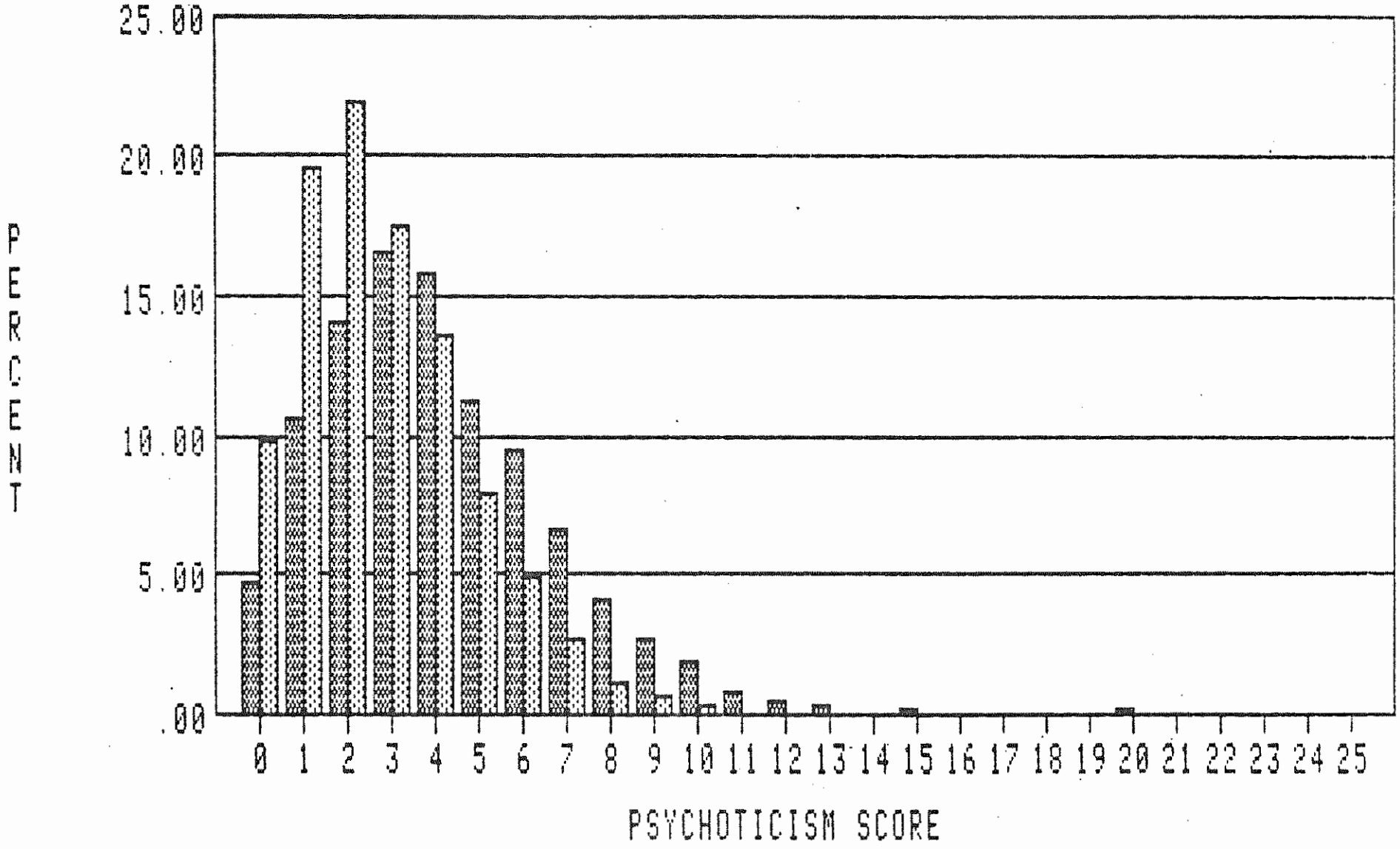
[1] If an individual scores x out of a maximum score of n, then let $p=x/n$ and $\theta(\text{radians})=\arcsin\sqrt{p}$. For $n<50$, if $p=0$ let $p=1/4n$ and if $p=1$ let $p=1-1/4n$ (Bartlett's improvement). We may express θ in degrees by multiplying by $180/\pi$. See Snedecor and Cochran (1980, p290) for details.

DISTRIBUTION OF E SCORES
FEMALES
MALES
PERCENT
00.00
02.00
04.00
06.00
08.00
10.00
EXTRAVERSION SCORE
0 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21



DISTRIBUTION OF P SCORES

DISTRIBUTION OF N SCORES
MALES
FEMALES
NEUROTICISM SCORE
0 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23
PERCENT
00.00
02.00
04.00
06.00
08.00
10.00

DISTRIBUTION OF L SCORES
FEMALES
MALES
PERCENT
00.00
02.00
04.00
06.00
08.00
10.00
LIE SCORE
0 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21

3.3.4 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 3.21 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 3.21). In the raw scores, 4 of the 8 t-tests and 2 of the 8 F-tests were significant at least at the 5% level. However, there was no consistent pattern in these differences, and they tended to be small 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 (see Chapter 1). 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 MZ's. 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 3.21 Means and variances of the twin sample for raw and transformed EPQ scores. Asterisks denote significant differences between MZ and DZ means and/or variances.

		MZF		MZM		DZF		DZM		DZO	
		Mean	Variance	Mean	Variance	Mean	Variance	Mean	Variance	Mean	Variance
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

Table 3.22 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 (Table 3.22)*. Females have signficantly higher neuroticism and lie scores and lower extraversion and psychoticism scores than males. The distributions of scores in the twin sample are similar to those obtained in a previous study using the EPQ in an Australian sample (Eysenck et al., 1980). 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 standardisation sample the pattern of differences was the same as we find here. 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 find that females are less variable in their psychoticism scores than males. These results are identical for both the raw and transformed scores.

3.3.5 Repeatability

Table 3.23 shows the distribution of age, and the raw and transformed personality and attitude scores for the 96 individuals who completed both the pilot and the main questionnaire. There were no significant differences in the age and distribution of scores of the pilot and total sample.

Estimates of repeatability (Table 3.23) were obtained by examining consistency of scores from the pilot and main

Table 3.22 Means and variances for raw and transformed EPQ scores. Asterisks denote significant differences between female and male means and/or variances.

		Females		Males	
		Mean	Variance	Mean	Variance
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

Table 3.23 Distribution of age, and raw and transformed EPQ 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	-	-
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

questionnaire. Separate analyses of variance were performed to obtain mean squares between (MS_b) and within (MS_w) individuals and repeatabilities (intraclass correlations) were calculated as $R_i = (MS_b - MS_w)/(MS_b + MS_w)$. Where there were significant differences between scores on the two occasions, corrected correlations were calculated by removing the between parts effects from the within individuals mean square. The within individual variance components are also shown in Table 3.23 and these are estimates of the portion of the total variance which is unrepeatable and will include measurement error.

The repeatabilities for the three EPQ scales are all high ranging from .70 to .92, and similar in males and females, which is consistent with previous results (Eysenck and Eysenck, 1975). As the time interval between the completion of the pilot and the main questionnaire ranged from 1-10 months ($\bar{X}$ = 3), it is unlikely that memory would be an important factor in these results.

3.3.6 Genetic and environmental analysis of variation in personality - Composite scores

We first fitted models to the total E, P, N and L scores, which had been transformed to best minimise G x E interaction, using the method of iterative weighted least squares. 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.

3.3.6.1 The basic model

The starting point for an analysis of twin data is an analysis of variance, which is used to compare the variation between and within each separate group of n twin pairs:

Source	Degrees of freedom	Expected mean squares
Between pairs	n-1	$\sigma_W^2 + 2\sigma_B^2$
Within pairs	n	σ_W^2

Where there is a sex difference in means, the within-pairs mean square (WMS) of DZ opposite-sex pairs is inflated by an amount $n/2(\bar{M} - \bar{F})^2$, where there are n pairs, $\bar{M}$ is the male mean and $\bar{F}$ is the female mean. Since significant sex differences were found for all variables (Table 3.22) we corrected for this by calculating the residual WMS (with n-1 df) given by $n/(n-1)[WMS - 1/2(\bar{M} - \bar{F})^2]$.

Where a variable is strongly age dependent this inflates the between-pairs mean square (BMS). The linear age correlations are shown in Table 3.24 separately for males and females. Although the correlations are significant in every case only for the lie scale is the correlation substantial. We corrected for age dependence in this variable by regressing within-pair sums on age and replacing the BMS with one-half the residual mean square (with n-2 df). The mean squares for the transformed variables and their degrees of freedom, corrected for sex differences and regression on age where appropriate are shown in Table 3.25.

Table 3.24 Two-tailed linear correlations of the angle transformed EPQ scores with age.

		Females	Males
Extraversion	Angle	$-.16^{***}$	$-.14^{***}$
Psychoticism	Angle	$-.20^{***}$	$-.28^{***}$
Neuroticism	Angle	$-.13^{***}$	$-.14^{***}$
Lie	Angle	$.36^{***}$	$.38^{***}$

Table 3.25 Observed mean squares for the angle transformed EPQ scores, and their degrees of freedom.

		Extraversion[a]		Psychoticism[a]		Neuroticism[a]		Lie[a,b]	
		df	Mean square	df	Mean square	df	Mean square	df	Mean square
MZF	Between	1232	368.00	1232	71.92	1232	330.85	1231	238.15
	Within	1233	112.46	1233	34.18	1233	105.38	1233	77.54
MZM	Between	565	347.84	565	98.61	565	315.18	564	220.57
	Within	566	116.46	566	37.98	566	115.65	566	98.59
DZF	Between	750	283.65	750	69.11	750	265.12	749	195.57
	Within	751	194.62	751	43.59	751	157.38	751	112.61
DZM	Between	350	292.18	350	86.66	350	263.16	349	186.59
	Within	351	224.60	351	52.05	351	183.94	351	105.36
DZO	Between	904	295.30	904	84.61	904	227.82	903	167.93
	Within	904	192.98	904	51.55	904	174.64	904	114.34

[a] Corrected for sex differences
[b] Corrected for regression on age

We may also examine whether twins become more or less similar with age by correlating absolute within-pair differences with age and these are shown in Table 3.26. For psychoticism only the DZ opposite-sex correlation is significant with opposite-sex pairs becoming more similar with advancing age. For neuroticism and lie the correlations are only significant for DZ females. This indicates that for females genetic differences become more pronounced with age but no such effect is apparent in males. Eaves and Eysenck (1976b) also found genetic differences in neuroticism become more pronounced with age but did not look for differences between the sexes in age-dependent gene expression. From these results it is clear that if environmental circumstances of co-twins become more different as they get older, these do not appear to produce any greater differences in any of the personality traits we have measured.

If genetic and environmental effects are acting independently and additively, then the expectations of the variance components for MZ and DZ twins have the following form:

MZ

$$\sigma_B^2 = E_2 + V_A(1 + (A/(1-A))) + V_D$$

$$\sigma_W^2 = E_1$$

DZ

$$\sigma_B^2 = E_2 + V_A(1 + (A/(1-A))) + 1/4V_D$$

$$\sigma_W^2 = E_1 + 1/2V_A + 3/4V_D$$

Table 3.26 Two-tailed linear correlations of absolute within-pair differences in the angle transformed EPQ scores with age.

	Extraversion	Psychoticism	Neuroticism	Lie
MZF	.03	-.02	.02	-.03
MZM	.03	-.04	.01	-.01
DZF	.07	-.03	.12***	.09*
DZM	-.04	-.01	.02	.03
DZO	.01	-.07*	.01	.05

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. 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 by $h^2\mu$ (h^2 is the heritability). V_D is the genetic variance due to dominant gene action.

Combining the terms of the expectations of the variance components with their coefficients in the analysis of variance gives the expectations of mean squares in terms of the main components of variation (Table 3.27).

A weighted least squares procedure is now used to estimate the parameters that will adequately describe the observed mean squares obtained from the analysis of variance of each separate group of twin pairs. The estimates of the parameters, denoted by a vector $\underline{\theta}$, are obtained by solving:

$$\hat{\underline{\theta}} = (\underline{\mathbb{B}}'\underline{W}\underline{\mathbb{B}})^{-1}\underline{\mathbb{B}}'\underline{W}\underline{x}$$

where $\mathbb{B}$ is the matrix of the expected coefficients of the parameters being estimated, $\underline{x}$ is the vector of the observed mean squares, and $\underline{W}$ is the diagonal matrix of weights. $\underline{W}$ is

Table 3.27 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

calculated as $w_i = n_i/2Ex_i^2$, where Ex_i is the expected value of the ith mean square with n_i degrees of freedom. As the Ex_i are only known after the estimates have been obtained, an iterative procedure using trial weights is used. When the estimates using these weights are obtained, they are used to calculate expected mean squares until successive parameter estimates converge to stable estimates. The covariance matrix of the stable estimates, $(\underline{B}'\underline{W}\underline{B})^{-1}$, can then be used to test the significance of the estimates by the normal deviate:

$$c_i = \hat{\theta}/\sigma_{\theta_i}$$

$$\text{where } \sigma_{\theta_i} = \sqrt{\text{diag}(\underline{B}\ \underline{W}\underline{B})^{-1}}$$

Providing that the observed statistics are normally distributed the estimates of $\underline{\theta}$ are maximum likelihood estimates and the overall fit of a given model can be tested by the residual chisquare calculated as:

$$\chi^2_{k-p} = (\underline{x} - E\underline{x})'W(\underline{x} - E\underline{x})$$

where there are k observed mean squares and p parameter estimates. / Although normality was not tested, it should be noted that transformation will normalise distributions (Martin et al., 1978).

In choosing the parameters we wish to estimate we want to provide the simplest description of the observed variation. Thus a sensible hierarchy of models is to 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 environmental 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 3.27) 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 will be completely confounded. It is thus more appropriate to rename E_2 as B (for extra between families variation) where

$$\hat{B} = E_2 + V_A(A/(1-A))$$

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 $\hat{B}$.

Eaves (1970a) showed that if the E_1BV_A model is fitted when both B and V_D are present then

$$\hat{V}_A = V_A + 3/2V_D$$

and

$$\hat{B} = B - 1/2V_D$$

If the $E_1V_AV_D$ model is fitted when both B and V_D are present then

$$\hat{V}_A = V_A + 3B$$

and

$$\hat{V}_D = V_D - 2B$$

Thus, if $B > 1/2V_D$, $\hat{V}_D$ will be negative, while if $B < 1/2V_D$, $\hat{B}$ will be negative. By fitting both three parameter models and comparing the signs of $\hat{B}$ and $\hat{V}_D$ we can make some inference about the presence of E_2 (or assortative mating) and V_D. Although the twin method is obviously a poor design for the detection of dominance, with the number of twin pairs available in the present study there is 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% heritability, complete dominance and no assortative mating or E_2 (ie B = 0), 3330 twin pairs would be sufficient to detect dominance at the 5% level with 95% probability, and our sample size is somewhat larger than this. However, the number of twin pairs required rises to over 30000 when there is only intermediate dominance. Even when significant estimates of V_D are obtained, it should be noted that the expectations for dominance and for additive x additive epistasis 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 mean squares 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 two male and female chisquares 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.

We shall discuss the results of model fitting separately for each factor. In every case a model (E_1) postulating that all variation was due to individual environmental differences 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.

3.3.6.2 Results of fitting models to extraversion scores

The results of fitting models to angle transformed extraversion scores are shown in Table 3.28. For females, although the E_1V_A model is able to adequately describe the data, addition of the parameter V_D results in a significant improvement in chi-square ($\chi^2_1 = 5.20$, $p<.05$). For males, the $E_1V_AV_D$ model also provides the best description of the data although, because of the large correlation between V_A and V_D in the classical twin design, the estimate of V_A is negative. There is no heterogeneity of fit of the $E_1V_AV_D$ model over sexes ($\chi^2_3 = 1.92$, $p>.05$) so we are entitled

Table 3.28 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	.52 ± .02	
$E_1E_2V_A$	112.4^{***}	-37.9	165.3^{***}	-	1	0.01		
$E_1V_AV_D$	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 & 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 & Male & 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

to fit this model to the joint male, female and opposite-sex data (Table 3.28). This gives an excellent fit to the data as well as yielding significant positive estimates of all parameters.

As discussed in the introduction to this chapter, a resemblance to dominance could be generated if there was any sibling competition. This model (Table 3.29) developed by Eaves (1976) can be described by three parameters; the additive genetic component V_A, V_A'' the environmental variance produced by the effects of those genes on the co-twin, and V_A' the covariance of the additive genetic effect on one twin and the environmental effect on his co-twin. V_A and V_A'' have the same expectations in twins (and any other relationships where individuals are reared in pairs) and so have to be estimated as one parameter. If there is sibling competition, the estimate of V_A' will be negative, and the model also predicts that the variance of DZ twins will be greater than the variance of MZ twins. In fact we found no significant differences between the variances of MZ and DZ twins (Table 3.21), however, when we fitted the competition model to the data (Table 3.30) there was little to choose in our Australian sample between a model that showed a substantial effect due to dominance and one with a small effect due to sibling competition.

In the extensive Swedish data of Floderus-Myhred et al. (1980) which we reanalysed (Table 3.30), we can reject the competition model in the larger sample of female twins, although there is still little discrimination between these alternative models in the male twins. Previous studies have suggested that either competition (Jinks and Fulker, 1970) or dominance (Eaves

Table 3.29 The specification of additive genetic effects in the presence of competition, omitting other environmental effects.

	Parameter		
Mean Square	Genetic V_A	"Environmental" V_A''	G x E Covariance V_A'
MZ Between	2	2	4
Within	0	0	0
DZ Between	3/2	3/2	3
Within	1/2	1/2	-1

$$h^2_{MZ} = \frac{V_A + V_A'' + V_A'}{E_1 + V_A + V_A'' + V_A'}$$

$$h^2_{DZ} = \frac{V_A + V_A'' + 1/2V_A'}{E_1 + V_A + V_A'' + 1/2V_A'}$$

Table 3.30 Summary of fitting models incorporating dominance or competition effects to extraversion.

	Parameter								
	E_1	$(V_A+V''_A)$	V'_A	V_A	V_D	χ^2	df	h^2	
Australian sample[a]									
Female & Male & Opposite-sex (n=3810 pairs)	114.4^{***}	-	-	50.9^{**}	76.4^{***}	5.42	7	h^2_N=0.21	h^2_B=0.53
	113.5^{***}	146.18^{***}	-12.5^{***}	-	-	5.20	7	h^2_{MZ}=0.54	h^2_{DZ}=0.55
Swedish sample[b]									
Female (n=6843 pairs)	2.29^{***}	-	-	1.49^{***}	1.12^{***}	1.38	1	h^2_N=0.31	h^2_B=0.53
	2.30^{***}	2.80^{***}	-0.14^{**}	-	-	6.15^{*}	1		
Male (n=5924 pairs)	2.46^{***}	-	-	1.51^{***}	0.66^{*}	0.02	1	h^2_N=0.33	h^2_B=0.47
	2.45^{***}	2.30^{***}	-0.10^{*}	-	-	0.94	1	h^2_{MZ}=0.47	h^2_{DZ}=0.48

[a] Angle transformed scores

[b] Raw scores

and Young, 1981) may effect the expression of extraversion. Although this is the first study to examine the ability of both models to explain variation in extraversion, obviously with these data alone it is difficult to determine which of these two models is most appropriate. In the Swedish data the dominance hypothesis is favoured slightly over the competition hypothesis. In Chapter 4 we shall consider the types of designs where competition and dominance are potentially separable and conduct power calculations to determine the sample sizes that would be required to discriminate, with a reasonable probability, between these alternative models.

3.3.6.3 Results of fitting models to psychoticism scores

The E_1E_2 model fails to adequately describe the variation in psychoticism in either males or females, while the E_1V_A model gives a good fit in both sexes (Table 3.31). The chi-square for the heterogeneity of fit over sexes is highly significant ($\chi^2_2 = 33.26$, $p<.001$) and inspection of the parameter estimates shows that there is a larger $\hat{V}_A$ component in males than females.

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 3.32. $V_{A_{MF}}$ is the covariance between the genetical effects in males and the genetical effects in females. If the genes affecting a trait in males are quite different from those affecting the trait in females then we expect $V_{A_{MF}}$ to be zero. If the genes acting in males and females are exactly the same but produce scalar

Table 3.31 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_1E_2	37.74^{***}	16.56^{***}	-	-	2	14.87^{***}	
E_1V_A	34.20^{***}	-	20.14^{***}	-	2	2.81	.37 ± .02
$E_1E_2V_A$	34.65^{***}	4.15	15.56^{***}	-	1	1.59	
$E_1V_AV_D$	34.65^{***}	-	28.00^{***}	-8.29	1	1.59	
Male							
E_1E_2	43.36^{***}	25.34^{***}	-	-	2	13.16^{**}	
E_1V_A	37.78^{***}	-	30.91^{***}	-	2	0.28	.45 ± .03
$E_1E_2V_A$	38.09^{***}	3.38	27.26^{***}	-	1	0.05	
$E_1V_AV_D$	38.09^{***}	-	37.41^{**}	-6.77	1	0.05	
Female & Male							
E_1E_2	39.52^{***}	19.33^{***}	-	-	6	63.69^{***}	
E_1V_A	35.32^{***}	-	23.56^{***}	-	6	36.35^{***}	
$E_1E_2V_A$	35.71^{***}	3.84	19.35^{***}	-	5	35.12^{***}	
$E_1V_AV_D$	35.71^{***}	-	30.86^{***}	-7.67	5	35.12^{***}	
Female & Male & Opposite-sex							
E_1E_2	42.37^{***}	18.67^{***}	-	-	8	88.22^{***}	
E_1V_A	35.80^{***}	-	25.37^{***}	-	8	48.39^{***}	
$E_1E_2V_A$	36.39^{***}	3.34	21.46^{***}	-	7	46.94^{***}	
$E_1V_AV_D$	36.39^{***}	-	31.47^{***}	-6.67	7	46.94^{***}	

Table 3.32 Model for twin mean squares 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

differences in the two sexes then we expect the correlation between the effects

$$r_{V_{A_{MF}}} = V_{A_{MF}} / \sqrt{V_{A_M} . V_{A_F}}$$

to be one. A similar argument applies to $E_{2_{MF}}$, 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 3.33. Fitting separate V_A parameters for males and females causes a significant reduction in chi-square ($\chi^2_2 = 36.31$, $p<.001$) the correlation $r_{V_{A_{MF}}} = 1.09$ indicating that the same V_A effects which act in males act in females but with smaller effect. Thus in females approximately 36% of the variation in psychoticism is due to additive genetic effects while in males this rises to approximately 50%. Eaves and Eysenck (1977) found that 49% of the variation in psychoticism is genetic in origin but did not look for differences in gene expression between the sexes.

We may also subtract the values of the residual mean square, obtained from the repeatability data, from the estimates of E_1 and so estimate the proportion of variance due to repeatable environmental differences and non-repeatable error (Table 3.34). In females, true individual environment accounts for a greater proportion of E_1 than error, while in males the reverse is true. 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).

Table 3.33 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 3.34 Sources of variance (%) for angle transformed psychoticism scores.

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

3.3.6.4 Results of fitting models to neuroticism scores

The results of fitting models to angle transformed neuroticism scores are shown in Table 3.35. In both males and females the E_1V_A model provides the best fit to the data. Although the chi-square for the heterogeneity of fit over sexes is non-significant ($\chi^2_2 = 3.71$, $p>.05$) we notice that there are smaller $\hat{E}_1$ and larger $\hat{V}_A$ components in females than males.

Fitting a model allowing different E_1 and V_A components in males and females (Table 3.36) results in a significant reduction in chi-square ($\chi^2_3 = 12.64$, $p<.01$), the correlation $r_{V_{A_{MF}}} = 0.58$ 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 3.37). 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. Our results are similar to those of Floderus-Myrhed et al. (1980). Eaves and Young (1981) reanalysed their data and found that both age and sex affected the expression of additive genetic and environmental differences in neuroticism.

3.3.6.5 Results of fitting models to lie scores

The results of fitting models to angle transformed and age corrected lie scores are shown in Table 3.38. In both males and females, the E_1V_A model is able to adequately describe the data, although in males there is some evidence that E_2 effects are also important. There is significant heterogeneity of fit of the E_1V_A

Table 3.35 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	.51 ± .02
$E_1E_2V_A$	104.8^{***}	1.2	109.2^{***}	-	1	0.42	
$E_1V_AV_D$	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 & 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 & Male & 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	12.26	
$E_1V_AV_D$	107.6^{***}	-	56.7^{**}	48.1^{**}	7	12.26	.27 ± .09

Table 3.36 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

$\chi^2_5 = 5.78$ ($p = .33$)

$h^2_{males} = 0.45 \pm .03$ $\quad$ $h^2_{females} = 0.51 \pm .02$

Table 3.37 Sources of variance (%) for angle transformed neuroticism scores.

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

Table 3.38 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 & 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 & Male & 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^{**}	

model over sexes (χ^2_2 = 12.73, p<.005) and we notice that there are larger $\hat{E}_1$ and smaller $\hat{V}_A$ components for males than females.

Fitting separate E_1 and V_A parameters for males and females (Table 3.39) results in a significant reduction in chi-square (χ^2_1 = 13.77, p<.01), the correlation $r_{V_{A_{MF}}}$ = 0.93 indicating that the same V_A effects which act in females act in males but with smaller effect. The correlation of age with absolute within-pair differences in DZ females does however indicate that genetic differences become more pronounced as females get older. Addition of an E_2 parameter in males results in a non-significant reduction in chi-square (χ^2_1 = 3.46, p>.05) indicating that this effect is not necessary to describe variation. The breakdown of the total variation into genetic and environmental components (Table 3.40) is similar to that obtained in previous studies of the lie scale (Martin and Eysenck, 1976; Eaves et al., 1978).

3.3.7 Genetic and environmental analysis of variation in personality - Individual items

Our analyses of the personality traits of extraversion, psychoticism, neuroticism and lie presented above suggest that population variance in these measures is due to the additive effects of genes and environmental factors unique to the individual. For extraversion either dominance or competition may also be important. There is no evidence for the importance of family environment. However, while genetic factors may contribute to item covariation, and hence the contribution of genetic factors will be enhanced when item responses are summed to obtain a total trait score, it is possible that environmental

Table 3.39 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

$\chi^2_5 = 11.13$ ($p = .05$)

$h^2_{males} = 0.38 \pm .03$ $\quad$ $h^2_{females} = 0.50 \pm .02$

Table 3.40 Sources of variance (%) for angle transformed and age corrected lie scores.

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

factors may be specific to individual items. These environmental factors may be unique to the individual, including error, or more importantly, discrete particles of shared environment which are still specific in their effect. On this model, composite test scores will have greater heritability than individual items. This is because the variance of the composite score includes the genetic component of the individual items and the covariance between them, but includes only the environmental components from specific items since there is no environmental communality between them. The following example illustrates this concept.

Suppose we combine N items into a composite test score by unweighted summation. Furthermore, let us suppose that the heritability of each item is a constant, h^2. The environmental component of each item is thus $e=1-h^2$. If we assume that the genes are the only source of communality between the items, then the inter-item correlations will all be equal to the heritability h^2. The total variance of the test scores will be

$$\sigma_T^2 = N + N(N-1)h^2.$$

The genetic component of variance will be N^2h^2, and the environmental component will be Ne. Thus, while the heritable variance in composite test scores increases as the square of the number of items, the environmental variance increases only linearly with the number of items. Consequently, if the family environment primarily determines the specific profile of item responses, we might expect that a genetic analysis of composite test scores would obscure the effects of this source of

variation. On this model, a genetic and environmental analysis of individual items will thus enhance our chances of detecting between-families environmental effects on personality.

3.3.7.1 The threshold model

The method of genetic analysis described in section 3.3.6.1 of this thesis is applicable to the analysis of continuous traits. In this section we consider the analysis of discontinuous traits such as the EPQ items.

The twins' responses to the individual EPQ items were summarized by 2x2 contingency tables containing the number of twin pairs whose joint responses fell into each of the four possible response categories. An example of a set of contingency tables is given in Table 3.41. For same-sex pairs the order of twin 1 and twin 2 was arbitrary, for opposite-sex pairs twin 1 was female, twin 2 was male. The full set of contingency tables is available from the author on request.

In general, the log likelihood of obtaining an mxm contingency table is

$$L = \sum_{i=1}^{i=m} \sum_{j=1}^{j=m} n_{ij} \ln p_{ij} + C$$

where n_{ij} and p_{ij} are the observed and expected number of twin pairs in cell ij respectively and C is a constant (Tallis, 1962). Since the contingency tables for the five twin groups are independent, the log likelihood of observing the set of contingency tables is simply the sum of the log likelihoods of the separate tables.

Table 3.41 Contingency tables for item E1, "Do you have many different hobbies?".

MZF

		Twin 2	
		No	Yes
	No	385	195
Twin 1			
	Yes	213	440

MZM

		Twin 2	
		No	Yes
	No	186	109
Twin 1			
	Yes	100	172

DZF

		Twin 2	
		No	Yes
	No	212	146
Twin 1			
	Yes	154	239

DZM

		Twin 2	
		No	Yes
	No	107	65
Twin 1			
	Yes	76	104

DZO

		Male	
		No	Yes
	No	226	181
Female			
	Yes	233	267

We assume that underlying a two-way mxm contingency table there are two continuous distributions of liability, in our case corresponding to the true personality types of twin 1 and twin 2 respectively, whose joint distribution is bivariate normal with correlation ρ. Superimposed on the distributions of liability are m-1 thresholds. We may also introduce n liability classes dependent on such factors as age and sex. Thus multiple thresholds are denoted by t_{ab}; a=0,1,...,m; b=0,1,...,n, where $t_{0n} = -\infty$ and $t_{mn} = +\infty$.

Assuming that our continuous scale of liability has zero mean and unit variance, and in the absence of differences in MZ and DZ total variances, the expected proportion of twin pairs (p_{ij}) in which the first and second twins fall in the i,jth cell of an mxm contingency table is given by

$$\int_{t_{i-1n}}^{t_{in}} \int_{t_{j-1n}}^{t_{jn}} \phi(x,y,\rho)\,dy\,dx$$

The t_{i-1n} and t_{j-1n} denote the lower thresholds, and the t_{in} and t_{jn} denote the upper thresholds of our continuous scale of liability delineating the ith row and jth column of a given contingency table. $\phi(x,y,\rho)$ is the standard bivariate normal distribution where x is the liability of twin 1, y is the liability of twin 2 and ρ is the correlation between the pair defined above. The value of ρ will depend on the causal model proposed for the similarity in liability between twins. The contributions of various genetic and environmental effects to the correlation of twins are shown in Table 3.42. The parameters

Table 3.42 The contribution of genetic and environmental sources of variation to the correlation in liability in twins.

	E_1	E_2	E_{2_M}	E_{2_F}	$E_{2_{MF}}$	V_A	V_{A_M}	V_{A_F}	$V_{A_{MF}}$	V_D
MZF	0	1	0	1	0	1	0	1	0	1
MZM	0	1	1	0	0	1	1	0	0	1
DZF	0	1	0	1	0	1/2	0	1/2	0	1/4
DZM	0	1	1	0	0	1/2	1/2	0	0	1/4
DZO	0	1	0	0	1	1/2	0	0	1/2	1/4

have been defined in section 3.3.6.1 of this thesis with the exception that E_1 is now not separately estimated but is calculated as a residual category. Thus in the case of the E_1V_A model, E_1 is calculated as

$$\hat{E}_1 = 1 - \hat{V}_A$$

Obviously, when we allow for either sex-limited genetic and/or environmental effects, E_1 will also differ between the sexes. Thus for the $E_{1_M}E_{1_F}V_{A_M}V_{A_F}V_{A_{MF}}$ model

$$\hat{E}_{1_M} = 1 - \hat{V}_{A_M}, \text{ and}$$

$$\hat{E}_{1_F} = 1 - \hat{V}_{A_F}.$$

Estimation involves the selection of parameter estimates which maximise the log likelihood of observing the set of contingency tables. We have minimised -L using a subroutine for unconstrained optimisation (E04JBF) from the Numerical Algorithms Group Library (1981). This procedure will yield maximum likelihood estimates of the genetic and environmental parameters and the threshold values that we choose to estimate.

There is no perfect test of goodness-of-fit with this approach, but an approximate test is given by

$$\chi^2_{K(m^2-1)p} = \sum_{i=1}^{i=m} \sum_{j=1}^{j=m} \sum_{k=1}^{k=K} (n_{ijk} - e_{ijk})^2/e_{ijk}$$

where K is the number of contingency tables, m the number of categories and p the number of parameters estimated. The observed and expected number of twin pairs in the ijth cell of the kth contingency table are n_{ijk} and e_{ijk} respectively. This test is sensitive to low expected cell frequencies which will result in the underestimation of the goodness-of-fit of a model

(Olsson, 1979). Furthermore, a significant value of chi-square may indicate either failure of the statistical assumption of bivariate normality in twins' liabilities (to respond) and/or the causal model proposed for twin similarity. An alternative approach, which we have used here, is to compare the fit of a specific model against the full model where we estimate separate correlations for each of the five twin groups. These maximum likelihood extimates of ρ (which we obtain) are the tetrachoric correlations (Pearson,1900) which give the correlation between twins for the hypothesised distribution of liability, not the discontinuous EPQ items. By the likelihood ratio criterion (Elston and Stewart, 1971) twice the difference in log likelihoods between the two models is distributed as chi-square with degrees of freedom equal to the difference in the number of parameters estimated. Since we wish to explain the observed variation with as few parameters as possible, we select as the best fitting model the simplest one, with positive estimates of our parameters, which is not rejected against the full model. However, in the case where more complex models which are themselves not rejected against the full model significantly improve the fit over the simplest non-rejected model, then that model is considered as most appropriate.

Models were fitted to the set of contingency tables of the five twin groups responses to the items of the EPQ. Obviously in the case of dichotomous traits one can only estimate one threshold. However, as the majority of items had shown significant differences in endorsement frequency between the

sexes we estimated separate thresholds for males and females for all the models that were fitted. In the case of the 16 items not showing significant sex differences in endorsement frequency (Table 3.5), after deciding on the best fitting model we also fitted that genetic model again but now estimated a common threshold for males and females. In every case this resulted in accepting the model with sex independent thresholds as the most appropriate and this will be obvious in the later tables of model fitting. We shall discuss the results of fitting models to the EPQ items separately for the items defining extraversion, psychoticism, neuroticism and lie.

3.3.7.2 Results of fitting models to extraversion items

The likelihood ratio test of specific models against the full model for the items defining extraversion are shown in Table 3.43. Table 3.44 gives, for the best fitting models, the values of the genetic and environmental parameters estimated and the position of the thresholds on the underlying distribution of liability. To illustrate the method of selecting the best fitting model we shall discuss in detail the results for item E2: "Are you a talkative person?".

By the likelihood ratio test the E_1E_2, E_1V_A and the $E_{1_M}E_{1_F}V_{A_M}V_{A_F}V_{A_{MF}}$ models are all rejected against the full model. As in the case of fitting models to variance components, both the $E_1E_2V_A$ and $E_1V_AV_D$ models yield identical results and neither is rejected against the full model. However, the $E_1E_2V_A$ model yielded a negative estimate of E_2 while the $E_1V_AV_D$ model resulted in postive estimates of all parameters. Thus we accept the

Table 3.43 Likelihood ratio test of specific models as compared to the full model for extraversion items.†

Model	df	E1	E2	E3	E4	E5	E6	E7	E8	E9
E_2	4	35.30***	79.94***	82.48***	71.80***	12.60*	42.54***	16.02**	23.16***	37.72***
V_A	4	8.56	17.72**	18.54***	13.00*	1.34	4.92	3.90	1.78	6.36
E_2V_A	3	8.56	6.32	4.90	3.20	0.66	3.78	0.94	1.24	4.44
V_AV_D	3	8.56	6.32	4.90	3.20	0.66	3.78	0.94	1.24	4.44
$V_{A_M}V_{A_F}V_{A_{MF}}$	2	2.58	16.26***	17.02***	7.06*	2.00	3.16	2.00	1.24	4.44
- log likelihood of full model		5157.80	5072.30	4530.65	4525.03	3317.34	5140.85	4973.77	3929.27	3755.81

Model	df	E10	E11	E12	E13	E14	E15	E16	E17	E18
E_2	4	62.70***	68.92***	65.58***	53.90***	31.66***	24.68***	13.02*	23.72***	30.94***
V_A	4	17.46**	29.04***	8.64	3.22	1.34	3.04	1.24	4.00	9.58*
E_2V_A	3	7.70	20.34***	2.32	1.22	1.12	0.74	1.22	3.24	8.70*
V_AV_D	3	7.70	20.34***	2.32	1.22	1.12	0.74	1.22	3.24	8.70*
$V_{A_M}V_{A_F}V_{A_{MF}}$	2	5.84	10.72***	5.66	0.20	0.28	2.78	0.18	0.04	1.28
- log likelihood of full model		5182.54	5188.12	5110.26	4610.96	4437.48	3047.71	5201.47	4935.38	5198.00

Model	df	E19	E20	E21
E_2	4	30.38***	32.04***	39.76***
V_A	4	1.54	2.06	4.58
E_2V_A	3	1.52	1.10	1.24
V_AV_D	3	1.52	1.10	1.24
$V_{A_M}V_{A_F}V_{A_{MF}}$	2	0.08	1.92	3.64
- log likelihood of full model		5049.77	5106.77	4976.13

† Where twice the difference in log likelihood of the models under comparison has χ^2 distribution with degrees of freedom equal to the difference in the number of parameters in the two models. Values for the best fitting model are underlined.

Table 3.44 Parameter estimates and position of the threshold for best fitting models for extraversion items.†

Item	T_M	T_F	E_1	E_{1_M}	E_{1_F}	V_A	V_{A_M}	V_{A_F}	$V_{A_{MF}}$	V_D
E1	.023	-.068	.528	-	-	.472	-	-	-	-
E2	.026	-.225	.456	-	-	.037	-	-	-	.507
E4	-.651	-.447	.434	-	-	.055	-	-	-	.511
E5	-.928	-1.008	.488	-	-	.512	-	-	-	-
E6	.005	-.114	.517	-	-	.483	-	-	-	-
E7	-.205	-.306	.492	-	-	.508	-	-	-	-
E8	.777		.550	-	-	.450	-	-	-	-
E9	-.830		.495	-	-	.505	-	-	-	-
E10	-.022		-	.616	.517	-	.384	.483	.122	-
E12	.120		.456	-	-	.169	-	-	-	.375
E13	.330	.546	.437	-	-	.563	-	-	-	-
E14	-.803	-.370	.428	-	-	.572	-	-	-	-
E15	-1.000	-1.115	.535	-	-	.465	-	-	-	-
E16	-.059	.126	.671	-	-	.329	-	-	-	-
E17	-.558	-.123	.627	-	-	.373	-	-	-	-
E18	-.117	-.013	-	.766	.568	-	.234	.432	.359	-
E19	.051	.255	.492	-	-	.508	-	-	-	-
E20	-.181		.560	-	-	.440	-	-	-	-
E21	-.236	-.330	.564	-	-	.436	-	-	-	-

† T_M is the threshold for males, T_F is the theshold for females.

$E_1V_AV_D$ model as the best fitting model for this item. It should be noted, however, that we did not attempt to fit a competition model to the data because of the lack of discrimination between dominance and competition in the total scale scores.

For the extraversion items, in two cases it was not possible to provide an adequate description of the data. Of the remaining items, the $E_1V_AV_D$ model provides the best fit for 3 items, while the E_1V_A model is most appropriate for 14 items. The $E_{1_M}E_{1_F}V_{A_M}V_{A_F}V_{A_{MF}}$ model gives a good fit to two items (E10 and E18). For item E10 ("Would you call yourself happy-go-lucky?") the correlation between additive genetic effects in males and females ($r_{V_{A_{MF}}}$) is 0.29 suggesting that different genes are acting in males and females. For item E18 ("Do you often take on more activities than you have time for?") $r_{V_{A_{MF}}} = 1.13$ indicating that the same genes which act in females act in males but with a smaller effect on the variance.* Between 23% and 57% of the variance in the extraversion items can be ascribed to genetic effects, the remaining variance due to the effects of individual environment and error. Similar to our results for the total scale score we find no evidence for the importance of family environment.

3.3.7.3 Results of fitting models to psychoticism items

The likelihood ratio test of specific models against the full model, and the estimates for the best fitting models for psychoticism items are given in Tables 3.45 and 3.46 respectively. A simple genetic model is most appropriate for 20 of the items, and for only one item is a purely environmental

Table 3.45 Likelihood ratio test of specific models as compared to the full model for psychoticism items.

Model	df	P1	P2	P3	P4	P5	P6	P7	P8	P9
E_2	4	15.38**	7.12	10.28*	3.30	12.28*	26.58***	4.40	2.96	4.18
V_A	4	8.54	5.88	8.32	1.30	12.30*	8.94	4.30	1.48	5.18
E_2V_A	3	8.48*	5.10	2.76	1.28	6.32	8.94*	3.94	1.40	3.66
V_AV_D	3	8.48*	5.10	2.76	1.28	6.32	8.94*	3.94	1.40	3.66
$V_{A_M}V_{A_F}V_{A_{MF}}$	2	4.40	4.38	5.66	0.78	10.08**	6.50*	0.04	1.16	4.26
- log likelihood of full model		3427.94	2876.55	4741.40	725.28	4058.77	1622.56	549.75	1453.86	1353.10
Model	df	P10	P11	P12	P13	P14	P15	P16	P17	P18
E_2	4	12.82*	6.60	10.50*	18.38**	11.00*	11.64*	14.80**	6.28	11.34*
V_A	4	5.96	3.10	3.30	8.86	3.44	4.46	13.98**	4.44	8.60
E_2V_A	3	5.96	2.70	3.20	5.76	0.30	4.32	9.66*	4.06	6.72
V_AV_D	3	5.96	2.70	3.20	5.76	0.30	4.32	9.66*	4.06	6.72
$V_{A_M}V_{A_F}V_{A_{MF}}$	2	4.76	2.54	1.30	5.22	2.36	2.02	8.26*	3.04	7.48*
$E_{2_M}E_{2_F}E_{2_{MF}}V_{A_F}$	1	-	-	-	-	-	-	2.70	-	-
- log likelihood of full model		1557.53	1400.80	3281.29	1859.32	2475.28	2599.86	633.15	1959.10	4856.85
Model	df	P19	P20	P21	P22	P23	P24	P25		
E_2	4	10.48*	44.76***	12.16*	11.60*	5.36	6.60	8.20		
V_A	4	6.98	4.12	1.62	8.90	1.86	2.42	0.68		
E_2V_A	3	6.92	2.16	0.58	4.58	1.80	1.16	0.66		
V_AV_D	3	6.92	2.16	0.58	4.58	1.80	1.16	0.66		
$V_{A_M}V_{A_F}V_{A_{MF}}$	2	2.74	2.14	0.18	8.72*	1.36	2.30	0.40		
- log likelihood of full model		2071.51	4263.41	2059.49	3199.56	1266.56	2521.30	1734.01		

Table 3.46 Parameter estimates and position of the threshold for best fitting models for psychoticism items.

Item	T_M	T_F	E_1	E_{1_M}	E_{1_F}	E_2	E_{2_M}	E_{2_F}	$E_{2_{MF}}$	V_A	V_{A_F}
P1	-1.094	-.881	.683	-	-	-	-	-	-	.317	-
P2	-.739	-1.369	.666	-	-	-	-	-	-	.334	-
P3	-.224	-.515	.553	-	-	.181	-	-	-	.226	-
P4	-1.735	-2.331	.409	-	-	-	-	-	-	.591	-
P5	-.606	-.780	.535	-	-	.206	-	-	-	.259	-
P6	1.365	1.691	.295	-	-	-	-	-	-	.705	-
P7	2.055	2.297	.491	-	-	-	-	-	-	.509	-
P8	1.409	1.840	.634	-	-	-	-	-	-	.366	-
P9	1.370	1.958	.643	-	-	.357	-	-	-	-	-
P10	-1.357	-1.783	.476	-	-	-	-	-	-	.524	-
P11	1.509	1.791	.459	-	-	-	-	-	-	.541	-
P12	.731	1.152	.604	-	-	-	-	-	-	.396	-
P13	1.301	1.614	.615	-	-	-	-	-	-	.385	-
P14	-1.123	-1.367	.724	-	-	-	-	-	-	.276	-
P15	-1.058	-1.320	.540	-	-	-	-	-	-	.460	-
P16	-2.220	-2.020	-	.455	.108	-	.545	.484	.242	-	.408
P17	1.261	1.580	.610	-	-	-	-	-	-	.390	-
P18	.218	.470	.650	-	-	-	-	-	-	.350	-
P19	-1.223	-1.533	.673	-	-	-	-	-	-	.327	-
P20	.558	.677	.467	-	-	-	-	-	-	.533	-
P21	1.320	1.475	.562	-	-	-	-	-	-	.438	-
P22	.583	1.240	.479	-	-	.202	-	-	-	.319	-
P23	1.353	2.065	.567	-	-	-	-	-	-	.433	-
P24	1.021	1.384	.502	-	-	-	-	-	-	.498	-
P25	-1.061	-1.901	.492	-	-	-	-	-	-	.508	-

model adequate. The $E_1E_2V_A$ model provides the best description of the data for 3 items, while the $E_{1_M}E_{1_F}E_{2_M}E_{2_F}E_{2_{MF}}V_{A_F}$ model provides the best fit for one item. For this item (P16, "Is (or was) your mother a good woman?"), the correlation between family environmental effects in males and females ($r_{E_{2_{MF}}}$) is 0.47 indicating that there are some differences in the between family environmental effects acting in males and females. Where additive genetic effects are present they account for between 26% and 71% of the variance , while when common family environment is present it accounts for 18% to 36% of the variance. The detection of E_2 for at least some of the psychoticism items is in contrast to our results for the total score where we found no evidence for the importance of family environment.

3.3.7.4 Results of fitting models to neuroticism items

The results of fitting models to neuroticism items are shown in Table 3.47. For one item it was not possible to adequately describe the data. Of the remaining items, the E_1V_A model provides the best description of the data for 13 items, while 5 items are best fit by the $E_{1_M}E_{1_F}V_{A_M}V_{A_F}V_{A_{MF}}$ model. For 4 items a model incorporating dominance is most appropriate although this is almost certainly an artefact of sex-limited gene expression. Much of the evidence for dominance comes from the reduction of the DZ correlations relative to their MZ counterparts. A substantial proportion of DZ twins are of opposite-sex and any sex differences in gene expression will reduce the DZ opposite-sex correlation and hence mimic the effects of dominance when analysing the data pooled over the five twin groups.

Table 3.47 Likelihood ratio test of specific models as compared to the full model for neuroticism items, DZ opposite-sex pairs included.

Model	df	N1	N2	N3	N4	N5	N6	N7	N8	N9
E_2	4	34.20***	27.04***	10.54*	25.56***	23.36***	35.16***	26.96***	60.84***	48.32***
V_A	4	7.22	7.28	2.20	6.20	6.24	5.04	2.70	13.98**	10.56*
E_2V_A	3	5.00	6.74	1.94	4.70	5.72	3.96	2.34	0.28	5.26
V_AV_D	3	5.00	6.74	1.94	4.70	5.72	3.96	2.34	0.28	5.26
$V_{A_M}V_{A_F}V_{A_{MF}}$	2	4.52	1.10	2.12	0.08	3.76	0.44	2.20	6.72*	2.20
- log likelihood of full model		5141.69	4908.74	3958.09	4038.46	4553.63	5092.42	4909.27	4473.87	5086.59

Model	df	N10	N11	N12	N13	N14	N15	N16	N17	N18
E_2	4	34.94***	37.80***	17.06**	12.70*	12.04*	12.18*	33.60***	53.88***	44.14***
V_A	4	8.92	6.32	0.60	1.82	3.82	2.90	5.58	11.98*	10.02*
E_2V_A	3	7.40	1.78	0.56	1.80	3.16	1.90	5.04	1.74	4.16
V_AV_D	3	7.40	1.78	0.56	1.80	3.16	1.90	5.04	1.74	4.16
$V_{A_M}V_{A_F}V_{A_{MF}}$	2	0.22	0.92	0.22	0.70	2.04	2.76	4.08	3.26	7.08*
- log likelihood of full model		5085.20	3949.37	5126.18	3685.35	5150.75	4195.55	5034.96	4223.84	4985.60

Model	df	N19	N20	N21	N22	N23
E_2	4	39.72***	26.48***	11.38*	10.42*	26.30**
V_A	4	8.00	7.32	0.60	6.26	4.90
E_2V_A	3	3.86	6.46	0.56	4.58	2.88
V_AV_D	3	3.86	6.46	0.56	4.58	2.88
$V_{A_M}V_{A_F}V_{A_{MF}}$	2	2.18	1.26	0.46	3.74	2.60
- log likelihood of full model		4656.78	4582.62	4489.47	4227.92	3824.51

Consequently, we decided to reanalyse the data for neuroticism omitting the DZ opposite-sex pairs.

The results of fitting models to neuroticism items omitting the opposite-sex pairs, and the corresponding parameter estimates for the best fitting models are shown in Tables 3.48 and 3.49 respectively. Now only one item is best described by a model incorporating dominance, which supports our interpretation that the evidence for dominance in the pooled data was an artefact of sex-limitation. Again it should be noted, however, that we are unable to discriminate between dominance and competition in these data. For the remaining 22 items, a simple genetic model is most appropriate; between 33% and 54% of the variance is genetic in origin, with the remaining variance due to individual environmental experiences and error.

3.3.7.5 Results of fitting models to lie items

The results of fitting models to lie items and the estimates for the best fitting models are shown in Tables 3.50 and 3.51 respectively. For one item it was not possible to adequately describe the data. Of the remaining items the simple genetic model is most appropriate for the majority of items (14), while a purely environmental model is adequate for only one lie item. The $E_1E_2V_A$ model provides the best fit for 2 items while the $E_{1_M}E_{1_F}V_{A_M}V_{A_F}V_{A_{MF}}$ model is most appropriate for 3 items (L2, L8 and L14). For items L2 ("Were you ever greedy by helping yourself to more than your share of anything?"), L8 ("As a child did you do as you were told immediately and without grumbling?") and L14 ("Have you ever cheated at a game?") the correlations

Table 3.48 Likelihood ratio test of specific models as compared to the full model for neuroticism items, excluding DZ opposite-sex pairs.

Model	df	N1	N2	N3	N4	N5	N6	N7	N8	N9
E_2	3	25.96***	6.50	8.16*	12.92**	16.34***	20.12***	18.24***	41.34***	32.00***
V_A	3	6.38	1.12	2.14	3.02	5.28	2.84	2.28	6.78	6.64
E_2V_A	2	4.38	0.08	1.96	3.00	5.22	2.84	2.24	0.04	4.82
V_AV_D	2	4.38	0.08	1.96	3.00	5.22	2.84	2.24	0.04	4.82
- log likelihood of full model		3904.07	3749.17	3012.58	3035.64	3455.16	3856.32	3714.17	3411.91	3848.78
Model	df	N10	N11	N12	N13	N14	N15	N16	N17	N18
E_2	3	23.78***	21.32***	9.62*	4.76	8.30*	6.28	20.30***	30.96***	34.76***
V_A	3	7.44	1.68	0.26	0.70	3.88	2.90	5.48	3.74	8.38*
E_2V_A	2	7.20*	0.76	0.16	0.20	3.06	1.10	4.06	0.72	4.08
V_AV_D	2	7.20*	0.76	0.16	0.20	3.06	1.10	4.06	0.72	4.08
- log likelihood of full model		3855.10	2992.32	3889.74	2794.91	3913.47	3185.50	3814.11	3151.25	3779.59
Model	df	N19	N20	N21	N22	N23				
E_2	3	21.44***	7.44	10.04*	9.28*	18.20***				
V_A	3	2.72	1.36	0.46	5.12	3.48				
E_2V_A	2	2.16	0.76	0.12	4.44	2.60				
V_AV_D	2	2.16	0.76	0.12	4.44	2.60				
- log likelihood of full model		3550.32	3479.40	3394.28	3230.37	2892.28				

Table 3.49 Parameter estimates and position of the threshold for best fitting models for neuroticism items, excluding DZ opposite-sex pairs.

Item	T_M	T_F	E_1	V_A	V_D
N1	.041	-.205	.605	.395	-
N2	.617	-.020	.596	.404	-
N3	-.565	-.857	.608	.392	-
N4	.756		.591	.409	-
N5	-.167	-.657	.618	.382	-
N6	.364	.018	.550	.450	-
N7	.419	.324	.564	.436	-
N8	.714	.461	.526	.474	-
N9	.090	-.258	.560	.440	-
N10	.402	.024	.592	.408	-
N11	.949	.678	.528	.472	-
N12	.196		.616	.384	-
N13	1.025	.796	.601	.399	-
N14	.214	-.143	.639	.361	-
N15	.781	.627	.554	.446	-
N16	.347	.007	.456	.544	-
N17	.862	.599	.518	.482	-
N18	.070	-.369	.566	.014	.420
N19	.619	.397	.562	.438	-
N20	.676	.445	.598	.402	-
N21	-.217	-.706	.667	.333	-
N22	-.470	-.745	.612	.388	-
N23	-.618	-.911	.577	.423	-

Table 3.50 Likelihood ratio test of specific models as compared to the full model for lie items.

Model	df	L1	L2	L3	L4	L5	L6	L7	L8	L9
E_2	4	4.76	31.06***	18.08**	20.12***	43.20***	21.28***	6.16	44.26***	11.32*
V_A	4	1.58	9.70*	1.24	7.86	2.90	2.88	7.32	8.80	1.86
E_2V_A	3	1.04	9.32*	1.16	7.84*	2.36	2.54	3.68	7.00	1.02
V_AV_D	3	1.04	9.32*	1.16	7.84*	2.36	2.54	3.68	7.00	1.02
$V_{A_M}V_{A_F}V_{A_{MF}}$	2	0.74	1.82	0.18	3.16	2.36	0.38	5.96	1.14	1.76
- log likelihood of full model		3379.99	5093.81	4300.44	4669.07	4940.12	4491.50	5061.62	4848.82	4780.84

Model	df	L10	L11	L12	L13	L14	L15	L16	L17	L18
E_2	4	13.78**	12.86*	25.22***	22.80***	33.20***	25.74***	24.36***	4.68	24.34***
V_A	4	1.50	11.82*	6.96	6.62	13.90**	4.02	2.42	1.98	15.60**
E_2V_A	3	1.50	7.32	5.32	2.62	13.24**	4.02	1.02	0.60	14.52**
V_AV_D	3	1.50	7.32	5.32	2.62	13.24**	4.00	1.02	0.60	14.52**
$V_{A_M}V_{A_F}V_{A_{MF}}$	2	0.32	7.72	1.62	4.94	5.92	1.88	0.10	0.76	7.50*
- log likelihood of full model		4816.74	3114.85	5012.31	5089.62	5066.08	4825.95	4949.65	3552.65	5096.74

Model	df	L19	L20	L21
E_2	4	25.34***	14.50**	6.94
V_A	4	5.78	2.04	3.10
E_2V_A	3	3.62	1.58	1.96
V_AV_D	3	3.62	1.58	1.96
$V_{A_M}V_{A_F}V_{A_{MF}}$	2	3.82	0.10	2.44
- log likelihood of full model		4653.39	3228.77	4360.30

Table 3.51 Parameter estimates and position of threshold for best fitting models for lie items.

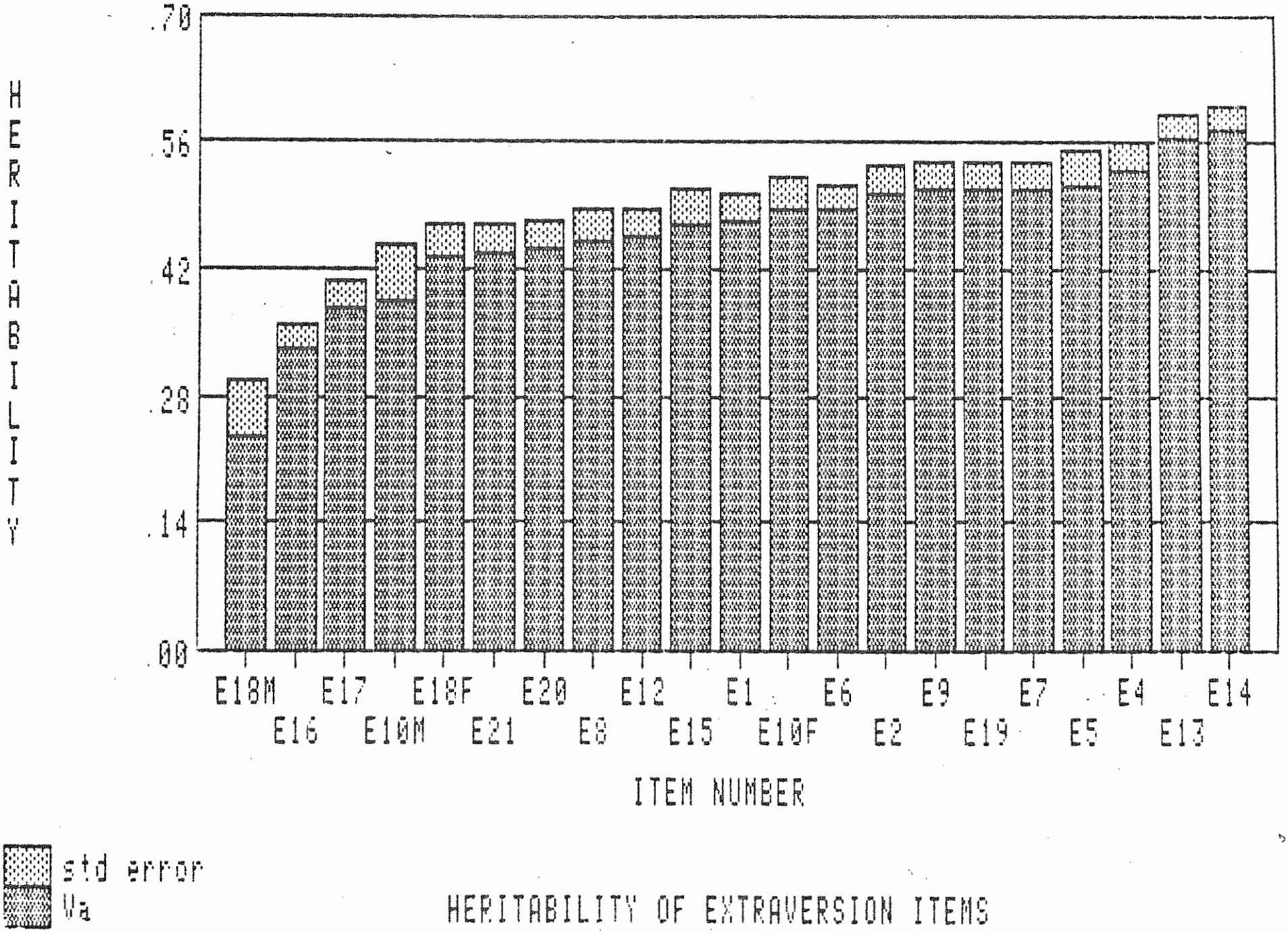
Item	T_M	T_F	E_1	E_{1_M}	E_{1_F}	E_2	V_A	V_{A_M}	V_{A_F}	$V_{A_{MF}}$
L1	.827	1.055	.656	-	-	-	.344	-	-	-
L2	.041	.021	-	.632	.458	-	-	.368	.542	.423
L3	-.451	-.734	.585	-	-	-	.415	-	-	-
L4	.492		.650	-	-	-	.350	-	-	-
L5	.363	.246	.473	-	-	-	.527	-	-	-
L6	-.804	-.359	.510	-	-	-	.490	-	-	-
L7	.204	.291	.737	-	-	.263	-	-	-	-
L8	.424	.341	-	.650	.471	-	-	.350	.529	.388
L9	-.596	-.304	.592	-	-	-	.408	-	-	-
L10	-.595	-.285	.646	-	-	-	.354	-	-	-
L11	-1.043		.495	-	-	.218	.287	-	-	-
L12	-.234		.482	-	-	-	.518	-	-	-
L13	.024		.438	-	-	.164	.398	-	-	-
L14	-.194	.189	-	.619	.456	-	-	.381	.544	.371
L15	.034	.492	.536	-	-	-	.464	-	-	-
L16	-.439	.114	.459	-	-	-	.541	-	-	-
L17	-.913		.611	-	-	-	.389	-	-	-
L19	-.563	-.380	.438	-	-	-	.562	-	-	-
L20	-1.021		.605	-	-	-	.395	-	-	-
L21	-.625		.645	-	-	-	.355	-	-	-

between additive genetic effects in males and females are 0.95, 0.90 and 0.82 respectively indicating that the same genes which act in females act in males but with a smaller effect on the variance. When present, additive genetic effects account for between 29% and 56% of the variance. Although for the total score it was not necessary to include E_2 effects when analysing the pooled data, there was some evidence for the importance of family environment in males.

3.3.7.6 The relationship between items and factors

We conclude our analysis by comparing the results from our factor analysis and the genetic analysis of the individual items and the composite scores.

We first examine if there is any heterogeneity in the contribution of genetic factors to variation in the individual items. The heritabilities of the EPQ items, and their standard errors, are shown separately for items defining extraversion (Figure 3.5), psychoticism (Figure 3.6), neuroticism (Figure 3.7) and lie (Figure 3.8). Just as models of variation can be fitted to mean squares, it is possible to fit models to means, or in our case heritabilities, using the method of iterative weighted least squares (section 3.3.6.1). We therefore decided, as a test of heterogeneity, to fit a model that assumes that the heritabilities of the items defining a scale are equal. The results of fitting this model to heritabilities (weighted by the inverse of their standard errors) for items defining the scales of E, P, N and L are shown in Table 3.52. In all cases this model fails badly indicating that there is significant



HERITABILITY OF EXTRAVERSION ITEMS

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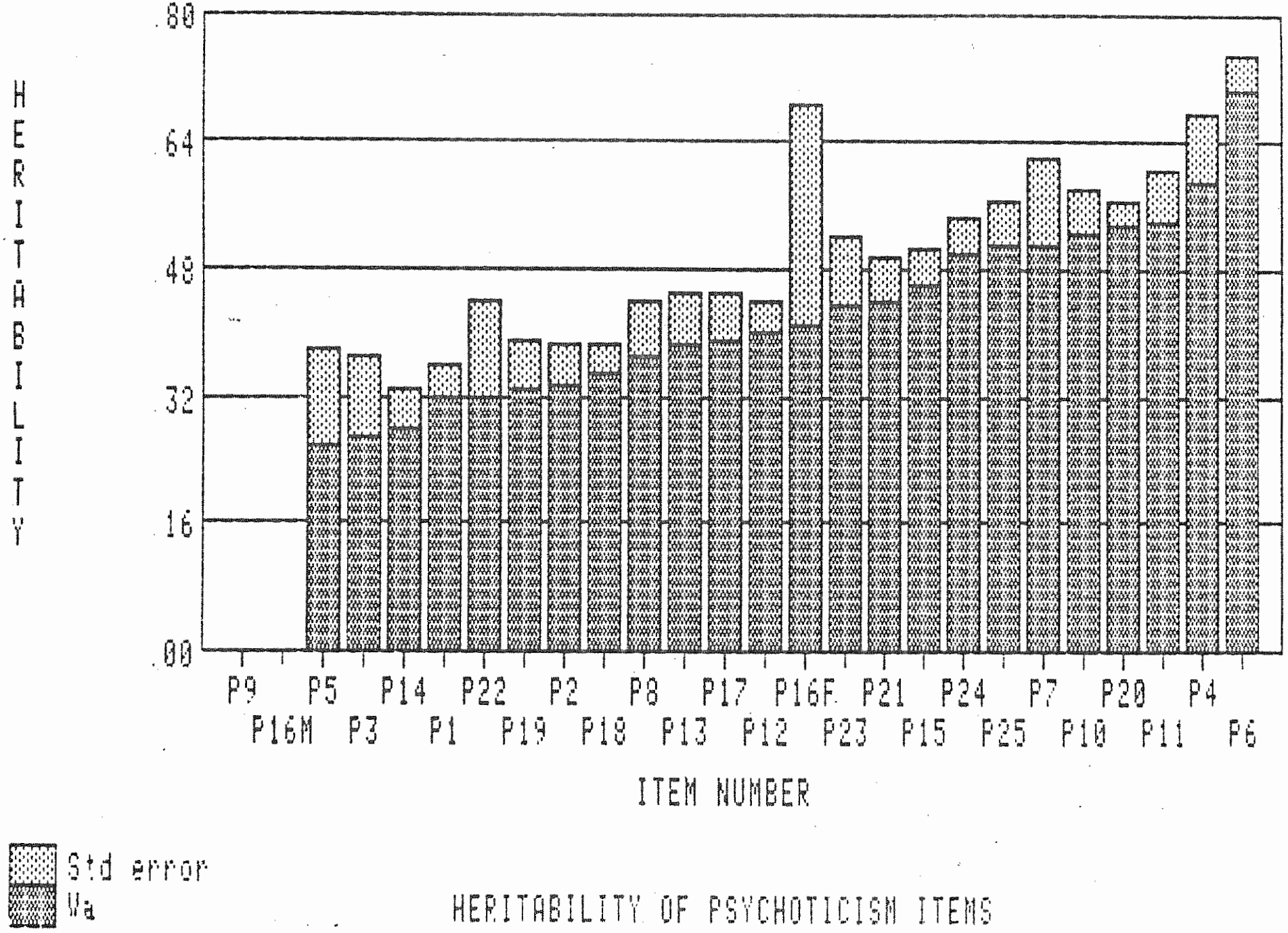
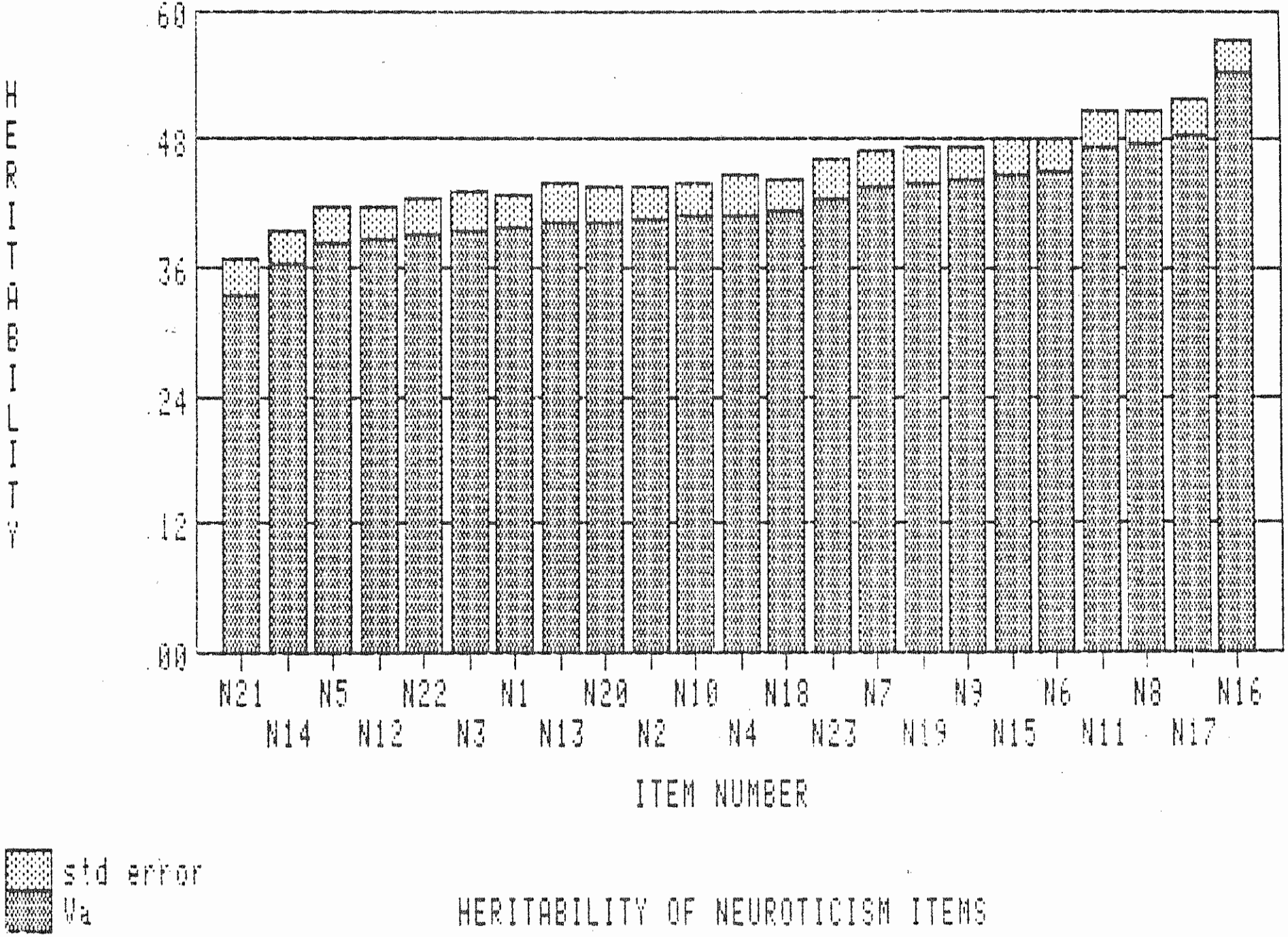
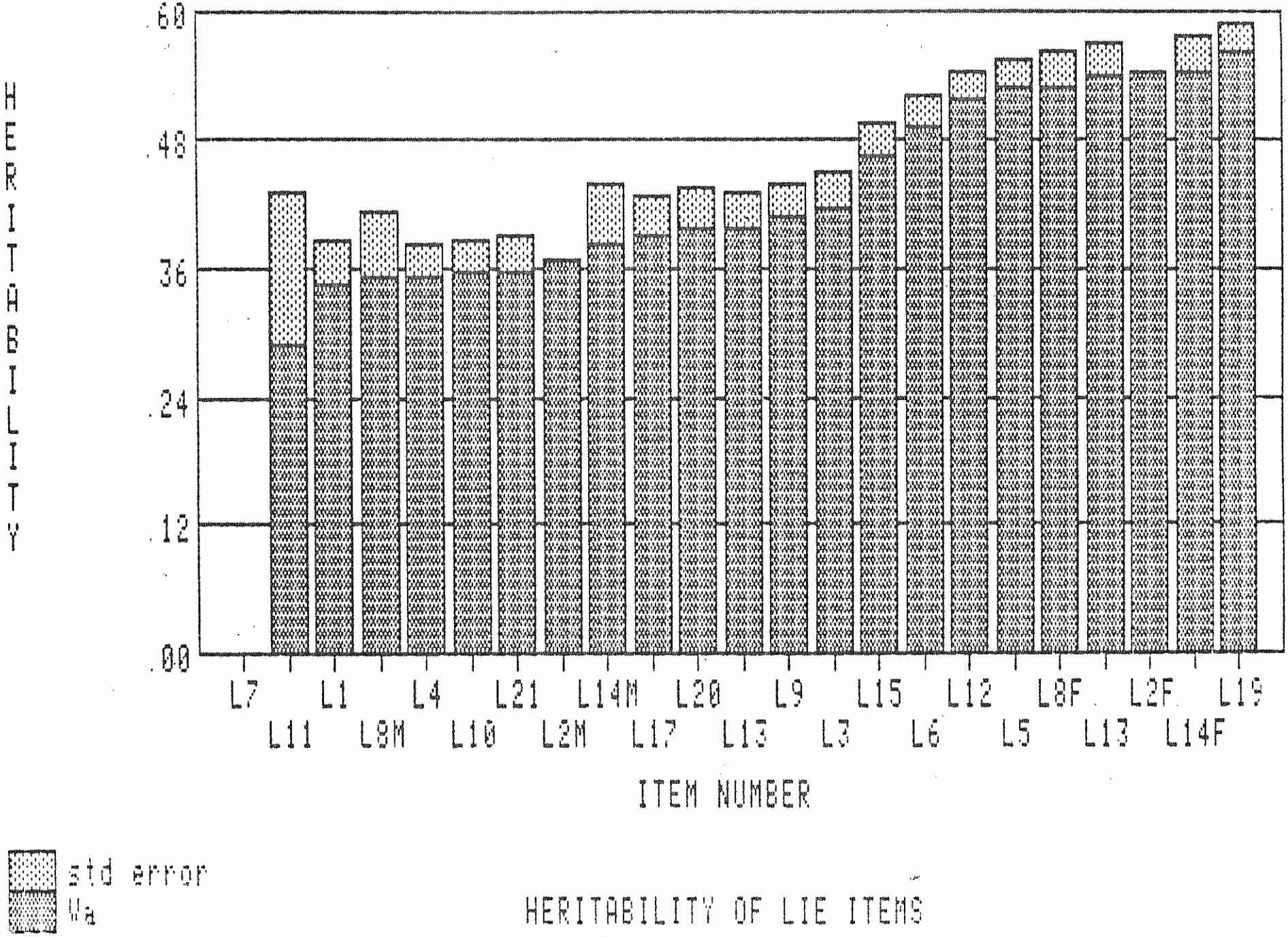


HERITABILITY OF PSYCHOTICISM ITEMS



HERITABILITY OF NEUROTICISM ITEMS



HERITABILITY OF LIE ITEMS

Table 3.52 Results of fitting a model (to the items defining the scales of E, P, N, and L) that assumes that the heritabilities of the items defining a scale are equal.

Items	Mean h^2	df	χ^2
Extraversion	.47***	20	86.70***
Psychoticism	.44***	23	99.66***
Neuroticism	.42***	22	45.68**
Lie	.46***	19	101.12***

heterogeneity in the contribution of genetic and environmental influences in the items defining these scales.

One possible explanation of this heterogeneity is that there are differences in sampling error in the individual items. However, some of the heterogeneity may reflect the relationship between the individual items and the underlying factors. Thus, if the factor is substantially genetic, the heritability of an item will depend partly on the loading of the item on the factor. Using the results obtained from the four orthogonal principal component solution (see section 3.3.2), we therefore correlated $\hat{V}_A$, estimated from the E_1V_A model, with the factor loadings of items defining a scale on the factors identified as E, P, N and L (Table 3.53). The DZ opposite-sex pairs were omitted from the model fitting analysis of the neuroticism items. The correlations between the estimates of the additive genetic variance and factor loadings are significant and positive for the extraversion items in both sexes, and significant and negative for psychoticism items in males.

We also examined whether there was any relationship between estimates of dominance variance obtained from the fit of the $E_1V_AV_D$ model and the factor loadings of all EPQ items on the factors identified as E, P, N and L (Table 3.53) because of the suggestion that dominance may be important to variation in extraversion. For both males and females, there is a highly significant and positive correlation between estimates of dominance and the factor loadings of the EPQ items on the extraversion factor. This suggests that the dominance we observe

Table 3.53 Correlations of V_A and V_D with factor loadings obtained from the four factor orthogonal solution. See text for further explanation.

	Extraversion[a]	Psychoticism[a]	Neuroticism[b]	Lie[a]
V_A				
Males	.44*	-.40*	.04	.02
Females	.48*	-.26	.08	.10
V_D				
Males	.41***	-.24*	.10	-.19
Females	.43***	-.10	.10	-.16

V_A estimated from the E_1V_A model

V_D estimated from the $E_1V_AV_D$ model

[a] Opposite-sex pairs included

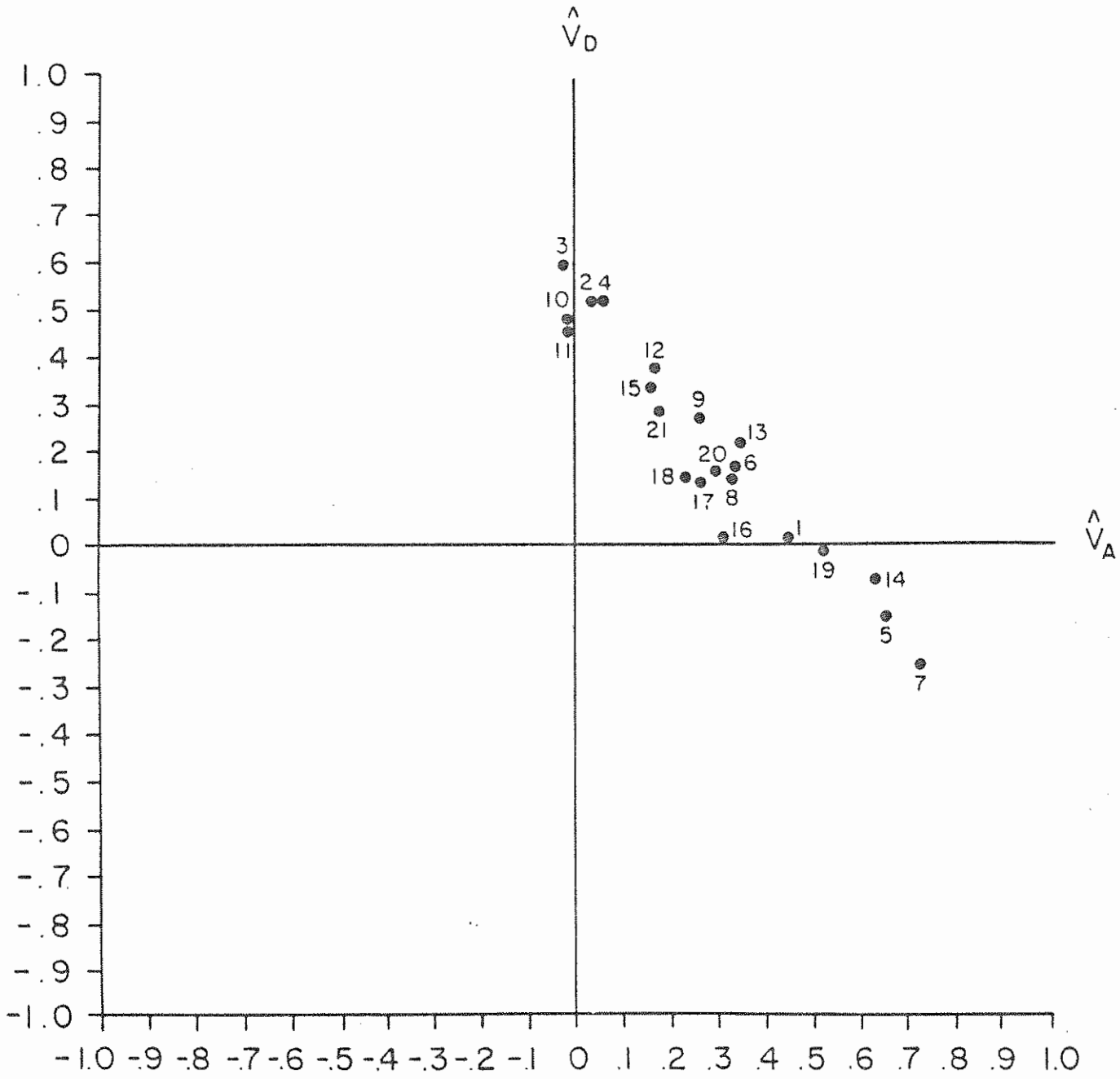
[b] Opposite-sex pairs omitted

at the factor level is also expressed at the item level for those items which load more highly on the extraversion factor. For psychoticism, the correlations are significant and negative only for males.

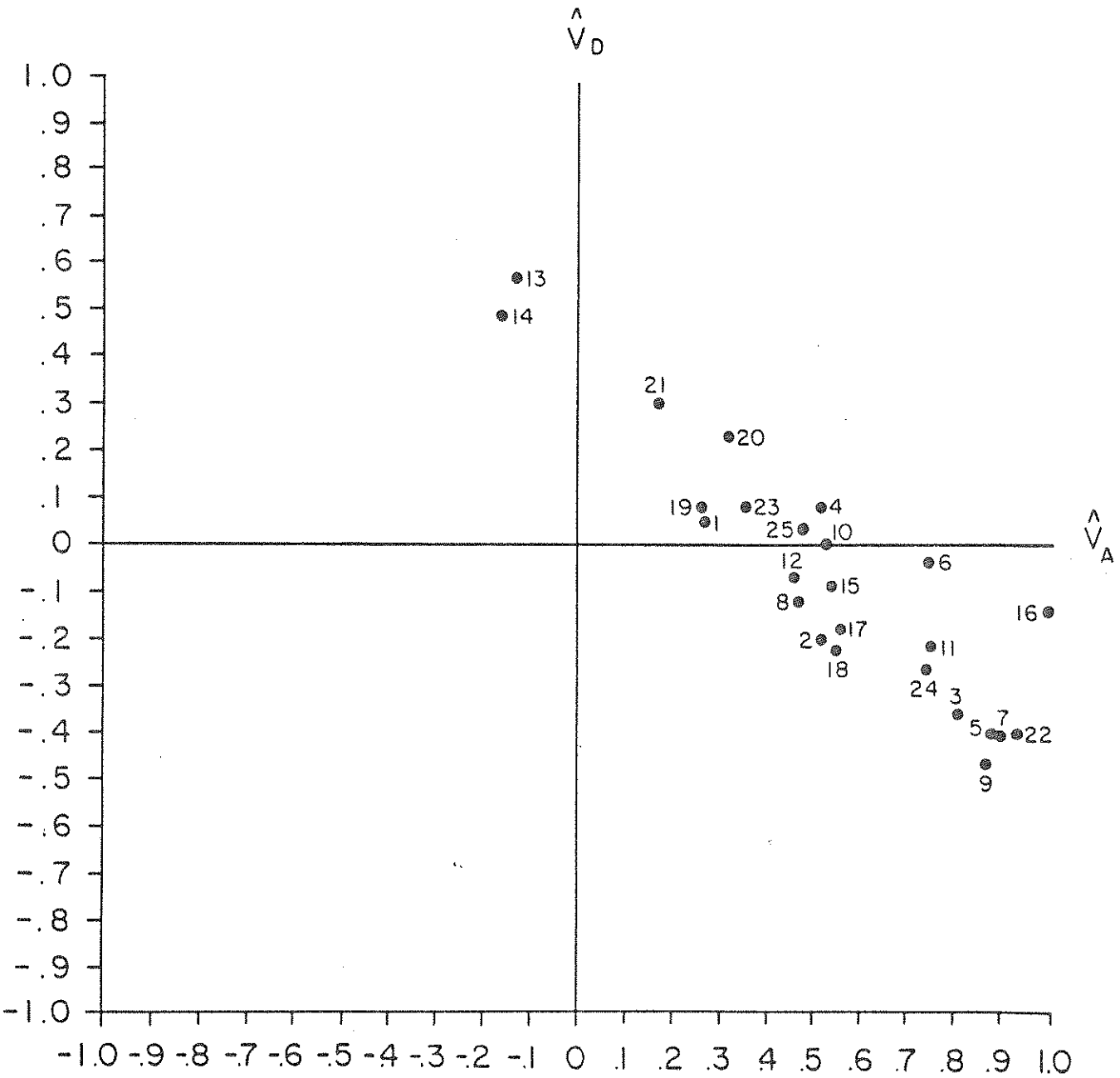
To further examine the relationship between the estimates of additive and dominance variance for each factor, we decided for each item to plot $\hat{V}_A$ against $\hat{V}_D$, both estimated from the fit of the $E_1V_AV_D$ model. However, due to the confounding of V_D and E_2 in twin studies, a negative estimate of V_D will also indicate the presence of E_2 (Eaves, 1970a). By considering the sign of V_D we are therefore able to make some inference about the pattern of variation of items defining a scale.

The plots of $\hat{V}_A$ against $\hat{V}_D$ are shown separately for items defining extraversion (Figure 3.9), psychoticism (Figure 3.10), neuroticism (Figure 3.11) and lie (Figure 3.12). For extraversion, 17 of the 21 items show positive estimates of V_D, which confirms the same trend for dominance or competition found for the total score. For neuroticism, about one-half of the items (13 out of 23) have positive estimates of V_D. This is what one would expect if there was no consistent trend for dominance or family environment in the items, and is consistent with the results found for the trait of neuroticism. For psychoticism, slightly more than half of the items (15 out of 25) have negative estimates of V_D, suggesting some effect due to family environment. In contrast, the majority of lie items (15 out of 21) show evidence of between-families environmental effects, and for the total score there was some evidence of E_2 but only for

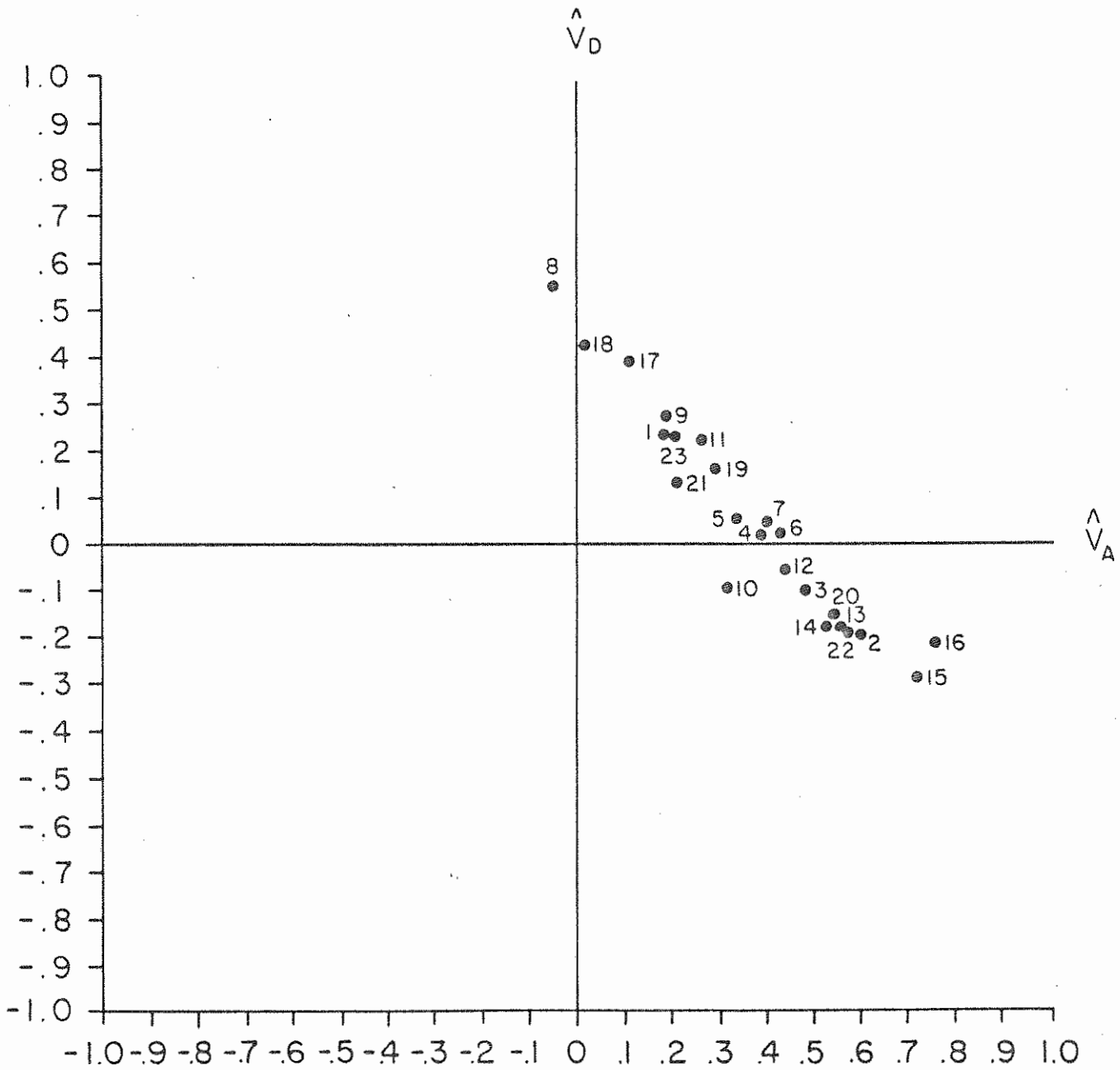
EXTRAVERSION

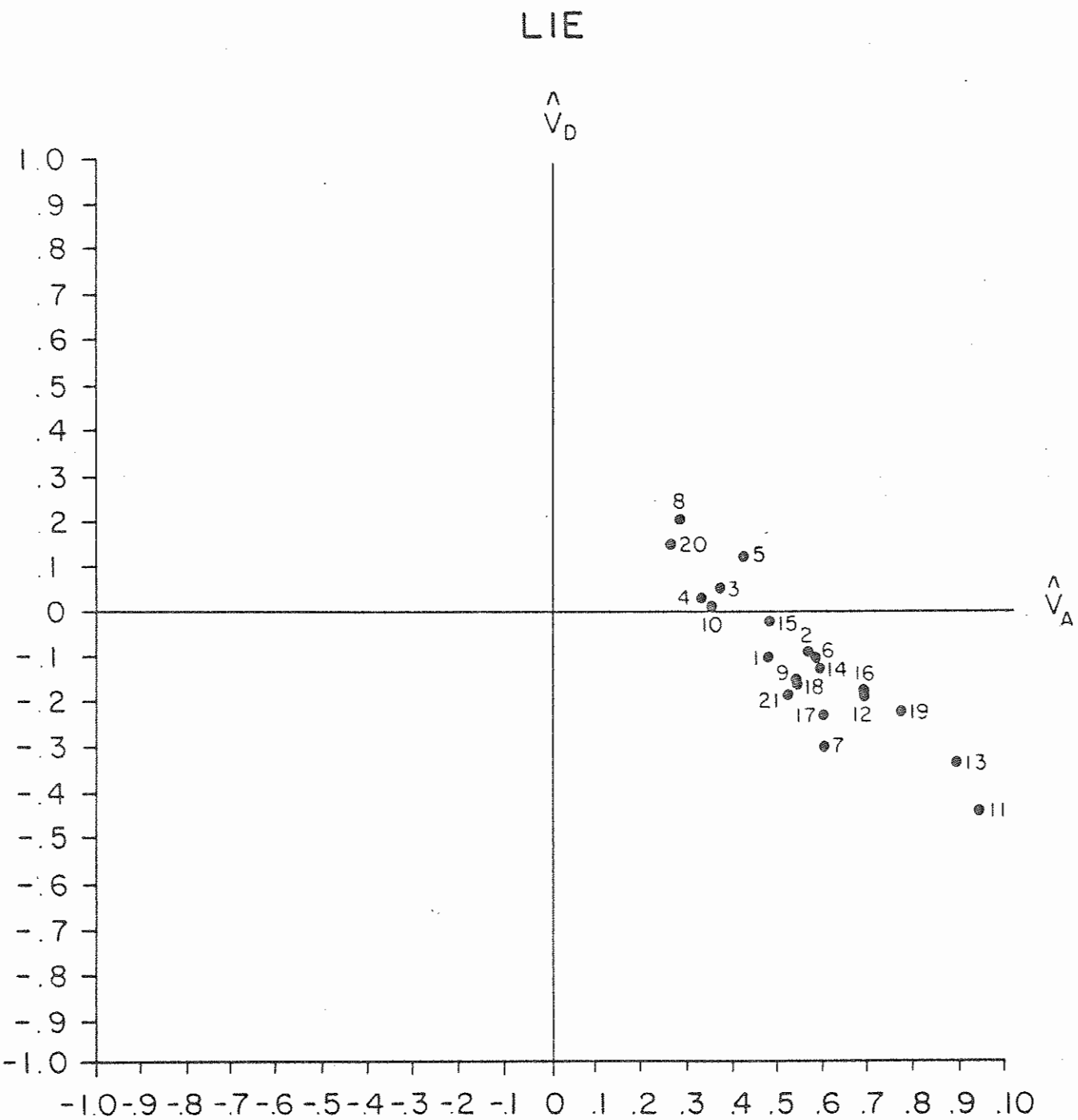


PSYCHOTICISM



NEUROTICISM




LIE
$\hat{V}_D$
$\hat{V}_A$
1.0
.9
.8
.7
.6
.5
.4
.3
.2
.1
0
-.1
-.2
-.3
-.4
-.5
-.6
-.7
-.8
-.9
-1.0
-1.0 -.9 -.8 -.7 -.6 -.5 -.4 -.3 -.2 -.1 0 .1 .2 .3 .4 .5 .6 .7 .8 .9 .10
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11

males. Our results suggest that it is possible to distinguish personality traits in terms of their patterns of variation, and that this is reflected in the individual items.

3.4 DISCUSSION

Our results support a view of personality which stresses the importance of the independent dimensions of extraversion, neuroticism and to a lesser extent psychoticism, and a fourth scale of social desirability or lie. In what is certainly one of the largest factorial studies of the EPQ, the factor of psychoticism is not as well defined as has been found previously (e.g. Eysenck and Eysenck, 1968; Michaelis and Eysenck, 1971). These studies, however, used earlier versions of the P scale and, as Clardige (1981) has pointed out, there has been a decline in the face validity of the P scale. He notes that several items of a distinctly psychotic kind (e.g. "Do you let your dreams guide or warn you?"; "When you are in a crowded place do you worry about dangers of infection?") had some of the highest loadings in the original studies, but were subsequently dropped to improve the distribution of P scores. Nevertheless, despite the removal of these items, the distribution of P scores in our sample is clearly non-normal; only 7 out of 25 of the P items in males, and 4 in females, have endorsement frequencies in the 20% to 80% range recommended by Maxwell (1971) for data that are to be factor analysed. * However, in view of these results it is not surprising that we find the P factor more strongly identified in males than females.

Other workers have questionned the construct validity of the P scale. Although it has been shown that the P scores of psychotic patients are correlated with symptom ratings and objective measures of performance deterioration (Verma and Eysenck, 1973), Bishop (1977) notes that psychotics do not have significantly different scores from groups who do not show signs of psychosis; prisoners, drug addicts, alcoholics or those with personality disorders or sex problems (Eysenck and Eysenck, 1975). Eysenck (1977) countered this by arguing that psychotics also tend to get high lie scores which in turn depresses their P scores. As Block (1977) points out, the P scores of psychotics whose L scores are in the normal range should be higher than those of psychotics with high L scores. However, although in the predicted direction, the differences for males are less than one scale point, for females less than three (Eysenck and Eysenck, 1975). While the P scale does describe individual differences in socially deviant behaviour, more work is needed to determine its ability to predict a predisposition to psychosis.

Sub-factors of personality are not well identified, and furthermore show marked differences between the sexes. Eysenck and Eysenck (1969) have argued that one cannot claim the existence of a factor unless it is shown to be invariant with respect to such factors as age, sex, education etc. Our results suggest that a description of personality in terms of the major dimensions of E, P, N and L is likely to be more useful than a description based on minor factors. Although Eysenck's original concept of extraversion was that of a factor made up of two

components of impulsivity and sociability (Eysenck and Eysenck, 1969) the E scale is now almost entirely a sociability scale, the impulsivity component now partly enclosed in psychoticism (Claridge, 1981). While we found some suggestion of a separate sociability factor, there was no evidence for an impulsivity component.

Factor-analytic studies cannot in themselves determine which is the most appropriate description of personality since different methods of rotation lead to different solutions which are mathematically interchangeable. However, Eysenck has always sought causal explanantions of the factors he describes. Thus he hypothesised that extraversion was characterised by differences in levels of activity in the corticorecticular loop, which modulates cortical excitation and inhibition, with extraverts characterised by low levels of activity. Neuroticism was linked with differences in the level of activity in the limbic system, with neurotics being over-reactive (Eysenck, 1967). A major problem in the theory is that as yet no causal models have been proposed for psychoticism and lie comparable to those of extraversion and neuroticism. Implicit, however, in Eysenck's theory is that these major factors should show a substantial genetic component.

Our results show that individual differences in psychoticism, neuroticism and lie can be explained by the additive effects of genes and individual environmental experiences. For extraversion there is also evidence that dominance or competition is important.

The single most striking finding from this study is the complete lack of evidence for the effect of shared environmental factors in shaping variation in personality. Power calculations by Martin et al. (1978) show that if the true model were $E_1E_2V_A$ (0.5 E_1, 0.1 E_2 and 0.4 V_A), and with the proportion of twins available in the present study (approximately 0.5 MZ, 0.5 DZ), 940 twin pairs would be required to reject the E_1E_2 model, and 11458 pairs to reject the E_1V_A model with 95% power at the 5% level of significance. That is, if E_2 accounts for 10% of the variation our sample size is sufficient to reject the E_1E_2 model, but not the E_1V_A model. However, if the true proportions of variance were 0.5 E_1, 0.3 E_2 and 0.2 V_A, 3268 pairs would be required to reject the E_1E_2 model and 1233 pairs to reject the E_1V_A model. That is, we have sufficient power to correctly reject both the E_1E_2 and E_1V_A models if E_2 accounts for at least 30% of the variance. Of course, 95% power is an extremely stringent requirement. From the method given above (and Pearson and Hartley, 1972, Vol. 2, Table 25) we see that for 80-85% power we would need about two-thirds this sample size, and for 50-60% power about one-third of this size. Thus, unless shared family environment is making a relatively minor contribution to variation in personality, our sample size is sufficient to detect its presence.

The major personality theorists have often suggested that genetic factors make some contribution to variation in personality. However, their role has generally been minimised in favour of the effects of family environmental and cultural

influences (see Hall and Lindzey, 1967; Hergenhahn, 1980 for reviews). Thus, in the psychoanalytic tradition, while Freud believed that all aspects of human personality were derived from biological instincts, and that a child goes through a series of developmental stages, he argued that a child's experiences during these stages determined his adult personality characteristics. Similarly, Jung argued that although there was an inherited predisposition to respond to certain aspects of the world, how one responded depended on one's life circumstances.

Social psychological theorists such as Adler and Erikson developed theories of personality which, while describing innate needs or genetically programmed developmental stages, gave critical importance to the nature of a child's interaction with his mother or the effects of the social environment. Horney did not consider the role of genetic factors at all, and stressed the importance of early parent-child relationships in her theory of personality development.

Learning theorists such as Skinner, and Dollard and Miller, believed that all human behaviour is learned. Skinner stressed the importance of classical conditioning and, more particularly, operant conditioning as the causal mechanisms in the development of personality. Dollard and Miller developed a theory of personality which combined traditional Hullian learning theory with Freudian concepts. Thus they described the development of personality in terms of the acquistion of habits, or cue-response bonds, that were formed as result of drive reduction (reinforcement in Hullian terms). In particular, they argued

that there were critical training periods in childhood, and that the way parents handled these critical periods determined subsequent personality development.

All of these theorists have argued that family environment is critical in the development of personality. None of them has attempted to test their theories. Our results show no evidence for the importance of family environment in variation in the personality traits of E, P, N and L.

Trait theorists such as Cattell, and particularly Eysenck, stand alone as personality theorists who have attempted to test their theories. Eysenck has consistently been interested in the possibility that observed differences in personality might be inherited, and that from genetic studies might ultimately come an understanding of their physiological basis and their evolutionary significance. His hypotheses concerning the nature and origin of individual differences in personality have been subjected to increasingly stringent tests, of which the present study is the most exacting, and have passed them well.

Our results show that the same genes are acting in males and females to produce variation in psychoticism and lie although there are scalar differences between the two sexes: genetic differences are more pronounced in males than females for psychoticism, while for lie the reverse is true. The correlation of age with absolute within-pair differences in lie scores in DZ females does, however, indicate that genetic differences become more pronounced as females get older. 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.

There are slight differences between the sexes in environmental and genetic contributions to variation in neuroticism, but these are not surprising in view of the striking evidence we find for the action of different genes on neuroticism in males than females. The correlation of age with absolute within-pair differences in DZ females also indicates that genetic differences in neuroticism become more pronounced as females get older; a similar result has been found by Eaves and Eysenck (1976b). Our results for neuroticism are similar to those of Floderus-Myrhed et al. (1980). Eaves and Young (1981) reanalysed their data and found that both age and sex affected the expression of additive genetic and environmental differences.

The results for extraversion are compatible with either a significant and substantial component of variation due to dominance, indicating that extraversion is a character which has been subject to selection, or a significant but small effect due to sibling competition. We shall consider in Chapter 4 of this thesis the types of designs in which these effects are potentially separable and the sample sizes that would be required to discriminate between them. Regardless of the model accepted, the detection of 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 individual differences in personality are better understood in terms of sensitivity to reward and sensitivity to punishment rather than extraversion and neuroticism. This corresponds to a 45° rotation of Eysenck's extraversion and neuroticism dimensions. Thus, under Gray's model, sensitivity to reward (impulsivity) is measured by the vector E+N, while sensitivity to punishment (anxiety) is represented by E-N. Eaves (personal communication) has suggested that neuroticism is an augmenting factor which amplifies the effect of both rewarding and punishing stimuli, while extraversion reflects the relative salience of rewarding and punishing events to the individual. 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. However, we are still left to question why there should be a difference in the genetic architecture of these two dimensions.

We speculate that the stimulus amplification reflected in neuroticism ought to be under stabilising selection. That is, intermediate expression of the trait would be adaptively superior because it offers an appropriate balance between approach and avoidance. If intermediate levels of a trait are adaptively

superior, then genetic variation is expected to be predominantly additive, with any genetical non-additivity expected to be weak and ambidirectional (Mather, 1973). There was no evidence of dominance for neuroticism.

In contrast, if extraversion is viewed as an exploration factor with extraverts at a selective advantage over introverts, one would expect dominance to evolve in the favour of increased extraversion. This hypothesis could be tested with inbreeding data, since we would expect inbreeding depression, and therefore lower extraversion scores, in the offspring of consanguineous marriages.

This model for extraversion also leads to predictions about the effects of sibling competition. If there were sibling competition, there would be mutual advantage if siblings, and especially twins, capitalised on different aspects of the environment. We would thus predict that sibling competition would be a function of the age difference in siblings since the greater the age difference the greater the range of environments available for individual exploitation. We shall consider these issues in more detail in the next chapter, when we consider experimental designs that could discriminate between the effects of dominance and competitive sibling interaction.

Our analysis of the EPQ items shows that in the majority of cases a simple genetic model is most appropriate, while 4 items are best fitted by a model incorporating dominance. Although the specification of between-families environmental effects is required for 8 items, generally at the level of individual items

there is little more support for an environmental hypothesis of family resemblance than there was for the composite scale scores. We found significant heterogeneity in items defining the scales of E, P, N and L. However, there was a consistent trend for extraversion items with a high genetic component (both additive and dominance) to load more highly on the E factor, while P items with a low genetic component (both additive and dominance) loaded more highly on P. Our results suggest that it is possible to distinguish major aspects of personality both factor-analytically and in terms of their patterns of variation (see also Royce, 1973) and that this is reflected at both the item and trait level.

CHAPTER 4 THE DETECTION AND RESOLUTION OF GENETIC DOMINANCE AND THE EFFECTS OF SIBLING INTERACTION

4.1 INTRODUCTION

In the previous chapter we demonstrated that individual differences in extraversion could be explained by either a significant and substantial component of variation due to genetic dominance, or a significant but small effect due to sibling competition. Although these effects are potentially separable with information on MZ and DZ twins, it would seem that in our sample, and the Swedish sample of male twins which we reanalysed, the sample sizes were too small to discriminate between these alternatives. This is the problem of power, the probability of correctly rejecting the null hypothesis when it is false.

Several approaches have been used to determine appropriate sample sizes for human quantitative genetic studies. Klein (1974) calculated standard errors of heritabilities as estimated by four relationships (regression of offspring on midparent, regression of offspring on single parent values, intraclass correlation of full-sibs, and intraclass correlation of half-sibs) to determine minimal sample sizes necessary to detect genetic components of given magnitude. Eaves (1969, 1970b, 1972) estimated the sampling covariance matrix for a range of true parameter values, to determine the optimal proportions of relatives of different types for the separation of V_A and V_D and E_1 and E_2, and to determine the sample sizes necessary to give parameter estimates which are significantly different from zero when tested against their standard errors. In our case, the most suitable approach

is that developed by Martin et al. (1978), based on the non-central chi-square distribution. With this method we are able to calculate sample sizes required to reject, with given probability models of variation when they are "wrong".

We shall examine the relative efficiency of different experimental designs to discriminate between the effects of sibling interaction and dominance. We shall consider the case where we have information on twins, twins and half-siblings, and twins and unrelated individuals reared together. We shall not examine parent-offspring data because of the inevitable confounding of genetic and environmental effects in this design.

4.2 METHODS

4.2.1 The model for sibling effects

Following Eaves (1976), we consider the direct and indirect effects of a single locus with two alleles A and a of population frequency u and v respectively, where u+v=1. Our model for the genotypic effects is as follows:

Genotype	AA	Aa	aa
Direct effect on phenotype	$+d_a$	h_a	$-d_a$
Indirect effect on phenotype	$+d'_a$	h'_a	$-d'_a$

The direct and indirect effects need not have the same sign. Thus, if an allele increases the expression of a trait in its carrier and creates an advantageous environment for a sibling we have cooperation. If an allele increases the expression of a trait in its carrier but creates a disadvantageous environment for a sibling we have competition.

Considering only pairs of individuals, and assuming no genotype x environment interaction, Eaves (1976) derived the expected frequencies of all possible pair genotypes for three kinds of relationship; MZ twins, DZ twins (full siblings) and unrelated individuals reared in the same family. We have extended these calculations to include the frequencies of pairs of half-siblings reared in the same family (Table 4.1). We give the expected frequencies given both equal and unequal allele frequencies.

Using these results, we may derive the expected total variances, and the variances between and within families, for different kinds of relationships. Eaves (1976) has calculated these for MZ and DZ twins, and unrelated individuals reared together. We now derive the expectations for half-siblings reared in the same family.

The total variance of half-sibs is calculated as

$$\sigma^2_{THS} = 12/128(d_a+d'_a)^2 + 16/128(d_a+h'_a)^2 + \ldots.$$
$$+ 12/128(-d_a - d'_a)^2 - (1/2h_a + 1/2h'_a)^2$$
$$= 1/2(d_a^2 + 1/2d_a d'_a + d'^2_a) + 1/4(h_a^2 + h'^2_a).$$

Summing over many loci, assuming the loci are in linkage equilibrium and there is no epistasis, we get

$$\sigma^2_{THS} = 1/2D + 1/4D' + 1/2D'' + 1/4H + 1/4H''$$

where

$$D = \Sigma_a d_a^2,\ D' = \Sigma_a d_a d'_a,\ D'' = \Sigma_a d'^2_a,$$
$$H = \Sigma_a h_a^2 \text{ and } H'' = \Sigma_a h'^2_a.$$

Table 4.1 The contribution of a single locus with two alleles, A, a, to the phenotypic expression of individuals reared in pairs.

	Phenotypic effect		Equal allele frequencies			
Pair genotype	First sib	Second Sib	MZ twins	DZ twins	Unrelated	Half-sibs
AA AA	$d_a + d'_a$	$d_a + d'_a$	1/4	9/64	1/16	12/128
AA Aa	$d_a + h'_a$	$h_a + d'_a$	-	3/32	1/8	16/128
AA aa	$d_a - d'_a$	$-d_a + d'_a$	-	1/64	1/16	4/128
Aa AA	$h_a + d'_a$	$d_a + h'_a$	-	3/32	1/8	16/128
Aa Aa	$h_a + h'_a$	$h_a + h'_a$	1/2	5/16	1/4	32/128
Aa aa	$h_a - d'_a$	$-d_a + h'_a$	-	3/32	1/8	16/128
aa AA	$-d_a + d'_a$	$d_a - d'_a$	-	1/64	1/16	4/128
aa Aa	$-d_a + h'_a$	$h_a - d'_a$	-	3/32	1/8	16/128
aa aa	$-d_a - d'_a$	$-d_a - d'_a$	1/4	9/64	1/16	12/128

	Phenotypic effect		Unequal allele frequencies			
Pair genotype	First sib	Second Sib	MZ twins	DZ twins	Unrelated	Half-sibs
AA AA	$d_a + d'_a$	$d_a + d'_a$	u_a^2	$u_a^4+u_a^3v_a+1/4u_a^2v_a^2$	u_a^4	$u_a^6+5/2u_a^5v_a+2u_a^4v_a^2+1/2u_a^3v_a^3$
AA Aa	$d_a + h'_a$	$h_a + d'_a$	-	$u_a^3v_a+1/2u_a^2v_a^2$	$2u_a^3v_a$	$3/2u_a^5v_a+7/2u_a^4v_a^2+5/2u_a^3v_a^3+1/2u_a^2v_a^2$
AA aa	$d_a - d'_a$	$-d_a + d'_a$	-	$1/4u_a^2v_a^2$	$u_a^2v_a^2$	$1/2u_a^4v_a^2+u_a^3v_a^3+1/2u_a^2v_a^4$
Aa AA	$h_a + d'_a$	$d_a + h'_a$	-	$u_a^3v_a+1/2u_a^2v_a^2$	$2u_a^3v_a$	$3/2u_a^5v_a+7/2u_a^4v_a^2+5/2u_a^3v_a^3+1/2u_a^2v_a^4$
Aa Aa	$h_a + h'_a$	$h_a + h'_a$	$2u_av_a$	$u_a^3v_a+3u_a^2v_a^2+u_av_a^3$	$4u_a^2v_a^2$	$1/2u_a^5v_a+4u_a^4v_a^2+7u_a^3v_a^3+4u_a^2v_a^4+1/2u_av_a^5$
Aa aa	$h_a - d'_a$	$-d_a + h'_a$	-	$1/2u_a^2v_a^2+u_av_a^3$	$2u_av_a^3$	$1/2u_a^4v_a^2+5/2u_a^3v_a^3+7/2u_a^2v_a^4+3/2u_av_a^5$
aa AA	$-d_a + d'_a$	$d_a - d'_a$	-	$1/4u_a^2v_a^2$	$u_a^2v_a^2$	$1/2u_a^4v_a^2+u_a^3v_a^3+1/2u_a^2v_a^4$
aa Aa	$-d_a + h'_a$	$h_a - d'_a$	-	$1/2u_a^2v_a^2+u_av_a^3$	$2u_av_a^3$	$1/2u_a^4v_a^2+5/2u_a^3v_a^3+7/2u_a^2v_a^4+3/2u_av_a^5$
aa aa	$-d_a - d'_a$	$-d_a - d'_a$	v_a^2	$1/4u_a^2v_a^2+u_av_a^3+v_a^4$	v_a^4	$1/2u_a^3v_a^3+2u_a^2v_a^4+5/2u_av_a^5+v_a^6$

The between-pairs variance is calculated as

$$\sigma^2_{BHS} = 12/128(d_a+d'_a)(d_a+d'_a) + 16/128(d_a+h'_a)(h_a+d'_a) + \ldots$$
$$+ 12/128(-d_a-d'_a)(-d_a-d'_a)-(1/2h_a+1/2h'_a)(1/2h_a+1/2h'_a)$$
$$= 1/8(d_a^2 + 8d_a d'_a + d'^2_a) + 1/2h_a h'_a.$$

Summing over many loci we get

$$\sigma^2_{BHS} = 1/8D + D' + 1/8D'' + 1/2H''$$

where $H' = \Sigma_a h_a h'_a$.

The within-pairs variance is calculated as

$$\sigma^2_{WHS} = 12/128(d_a+d'_a-d_a-d'_a)^2 + 16/128(d_a+h'_a-h'_a-d'_a)^2 + \ldots.$$
$$+ 12/128(-d_a-d'_a+d_a+d'_a)^2$$
$$= 3/8(d_a^2 - 2d_a d'_a + d'^2_a) + 1/4(h_a^2 - 2h_a h'_a + h'^2_a).$$

Summing over many loci we obtain

$$\sigma^2_{WHS} = 3/8D - 3/4D' + 3/8D'' + 1/4H - 1/2H' + 1/4H''.$$

Although we have assumed that the allele frequencies are equal at all loci, we may generalise our expectations to the case of unequal gene frequencies.

We put

$$V_A = \Sigma_a 2u_a v_a[d_a + (v_a - u_a)h_a]^2$$
$$V'_A = \Sigma_a 2u_a v_a[d_a + (v_a - u_a)h_a][d'_a + (v_a - u_a)h'_a]$$
$$V''_A = \Sigma_a 2u_a v_a[d'_a + (v_a - u_a)h'_a]^2$$
$$V_D = \Sigma_a 4u_a^2 v_a^2 h_a^2$$
$$V'_D = \Sigma_a 4u_a^2 v_a^2 h_a h'_a$$
$$V''_D = \Sigma_a u_a^2 v_a^2 h'^2_a$$

and substitute V_A, V'_A, V''_A, V_D, V'_D and V''_D for 1/2D, 1/2D', 1/2D", 1/4H, 1/4H' and 1/4H" respectively in the expectations for the variances above.

Combining the terms of the expectations of the variance components (including those of Eaves, 1976) with their coefficients in the analysis of variance (section 3.3.6.1) gives the expectations of the mean squares for twins, half-siblings and unrelated individuals reared in pairs in terms of the components of variation (Table 4.2).

4.2.2 Calculation of sample sizes required to discriminate between alternative models of variation

The application of the method of weighted least squares to the calculation of sample sizes required to reject, with given probability, models of variation is discussed in detail by Martin et al. (1978). Following their procedure, based on the non-central chi-square distribution, we wish to calculate sample sizes required to be 95% sure of rejecting alternative wrong models of variation at the 5% level of significance. Thus we

i)Take the "correct" model of variation and calculate the expected "observed mean squares", O_i, for given values of the genetic and environmental parameters contributing to the population variance.

ii)Obtain weighted least squares solutions (see section 3.3.6.1), assuming unit total sample size, for the parameters of a "wrong" model. From these we can calculate the "expected mean squares", $\underset{\sim}{E}_i$, on the basis of the "wrong" model.

iii)Calculate the weighted residual sum of squares

$$\Sigma_i v_i (O_i - \underset{\sim}{E}_i)^2 / 2\underset{\sim}{E}_i^2 \qquad (v_i \text{ degrees of freedom})$$

for the fit of the "wrong" model and use this as a non-centrality parameter λ' for s-p degrees of freedom where there are s statistics and p parameters in the "wrong" model.

Table 4.2 Contributions of within-family environment and genetic sources of variation in the presence of competition, to twin, half-sibling (HS) and unrelated individuals reared together (UT) mean squares.

Mean square	E_1	V_A	V_A''	V_A'	V_D	V_D''	V_D'
MZ Between	1	2	2	4	2	2	4
Within	1	0	0	0	0	0	0
DZ Between	1	3/2	3/2	3	5/4	5/4	5/2
Within	1	1/2	1/2	-1	3/4	3/4	-3/2
HS Between	1	5/4	5/4	5/2	1	1	2
Within	1	3/4	3/4	-3/2	1	1	-2
UT Between	1	1	1	2	1	1	2
Within	1	1	1	-2	1	1	-2

iv)Look up the non-centrality parameter (Pearson and Hartley, 1972; Vol. 2, Table 25) for the required 95% power at the 5% level of significance, i.e. $\lambda_{i(0.05,0.95,s-p)}$.

v)Calculate the required sample size as

$$N = \lambda/\lambda'.$$

To illustrate this method we shall work through a specific example.

Let us suppose that the real components of variation are

$$\underset{\sim}{\phi} = \begin{bmatrix} E_1 \\ V_A \\ V_D \end{bmatrix} = \begin{bmatrix} 0.5 \\ 0.1 \\ 0.4 \end{bmatrix}$$

i.e. 50% E_1, 10% V_A and 40% V_D. Considering only the classical twin design, and assuming that the total sample size is half MZ and half DZ, that is

$$N_{MZ}/N = N_{DZ}/N = 0.5$$

then each initial trial weight

$$w_{ii} = v_i/2x_i^2 = 0.5N/2x_i^2 = n/4x_i^2.$$

The "right" model matrix is

$$\underset{\sim}{B} = \begin{bmatrix} 1 & 2 & 2 \\ 1 & 0 & 0 \\ 1 & 1.5 & 1.25 \\ 1 & 0.5 & 0.75 \end{bmatrix}$$

and the expected "observed" statistics

$$\underset{\sim}{x} = \underset{\sim}{B}\underset{\sim}{\phi} = \begin{bmatrix} 1.5 \\ 0.5 \\ 1.15 \\ 0.85 \end{bmatrix}$$

We now fit the "wrong" $E_1(V_A+V_A")V_A'$ model to the data

$$\underset{\sim}{A} = \begin{bmatrix} 1 & 2 & 4 \\ 1 & 0 & 0 \\ 1 & 1.5 & 3 \\ 1 & 0.5 & -1 \end{bmatrix}$$

and after two iterations, the final solution

$$\underset{\sim}{\theta} = (\underset{\sim}{A}'\underset{\sim}{W}\underset{\sim}{A})^{-1}\underset{\sim}{A}\underset{\sim}{W}\underset{\sim}{x} = (\underset{\sim}{A}'\underset{\sim}{W}\underset{\sim}{A})^{-1}\underset{\sim}{A}\underset{\sim}{W}\underset{\sim}{B}\underset{\sim}{\phi} = \begin{bmatrix} E_1 \\ V_A + V_A" \\ V_A' \end{bmatrix} = \begin{bmatrix} 0.498 \\ 0.588 \\ -0.058 \end{bmatrix}$$

from which we can calculate the expected mean squares and weighted residual sum of squares as follows

Mean Square	N_i/N	Weight	"Observed"	"Expected"
MZ_b	0.5	0.1214	1.50	1.440947
MZ_w	0.5	1.0097	0.50	0.497634
DZ_b	0.5	0.1734	1.15	1.205119
DZ_w	0.5	0.3460	0.85	0.850000

$$"\chi_1^2" = \lambda_1' = 0.000956$$

Thus for unit total sample size the non-centrality parameter is $\lambda' = 0.000956$. From the table we find the non-centrality parameter

$$\lambda_{(0.05,0.95,1)} = 12.995$$

Therefore the sample size required for 95% power of rejecting the "wrong" model at the 5% level of significance is

$$N = \lambda/\lambda' = 12.995/0.000956 = 13598.$$

When considering the case where our sample comprises only MZ and DZ twins, we shall calculate sample sizes required to reject false hypotheses in the following combination of "true" and "false" models of population variance.

"True" model	"False" model
$E_1(V_A+V_A'')V_A'$	E_1
	E_1E_2
	E_1V_A
	$E_1V_AV_D$
$E_1V_AV_D$	$E_1(V_A+V_A'')V_A'$

Martin et al. (1978) have already presented extensive power calculations concerning the detection of dominance. Calculations will be made for samples comprising a proportion of MZ (pMZ) twins in the range 0.1-0.9(in steps of 0.2).

We shall also consider two other experimental designs: twins with half-siblings, and twins with pairs of unrelated individuals reared together. In these cases we shall only consider the following combination of models:

"True" model	"False" model
$E_1(V_A+V_A'')V_A'$	$E_1V_AV_D$
$E_1V_AV_D$	$E_1(V_A+V_A'')V_A'$

We shall calculate sample sizes for pMZ=0.1-0.7(0.2) and pDZ=0.1-0.7(0.2) with the proportion of either half-siblings or unrelated individuals reared together forming the non-zero remainder.

For the "true" $E_1V_AV_D$ model we shall calculate sample sizes for E_1=0.1-0.7(0.2) and V_A=0.1-0.7(0.2) with V_D forming the non-zero remainder. We shall also consider as a special case sample

sizes for 0.5 E_1, 0.2 V_A and 0.3 V_D, proportions which closely approximate the breakdown of variation found for extraversion (section 3.6.6.2).

To calculate appropriate parameter values for the "true" sibling effect model, we remember that it is possible, instead of parameterising competition and cooperation in terms of the direct and indirect effects of loci, to represent the indirect effects by their regression onto the direct effects (Young et al., 1980). Our genetic model thus becomes

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

Assuming b is the same at all loci, it can be shown that $V_A' = bV_A$ and $V_A'' = b^2V_A$. If there is cooperation b will be positive, if there is competition b will be negative. On this model, and remembering that V_A and V_A'' have the same expectations where individuals are reared in pairs, it can be shown that

$V_{A\ Twins} = (1 + b^2)V_{A\ Singletons}$ and

$V_{A\ Twins}' = 2bV_{A\ Singletons}$.

Assuming that the real components of variation in singletons are 0.5 E_1 and 0.5 V_A, for the "true" $E_1(V_A+V_A'')V_A'$ model we shall calculate sample sizes for absolute values of b of 0.3, 0.1, 0.05, 0.04 and 0.02. We shall consider both competition (where b is negative) and cooperation (where b is positive). It should be noted that the case where the "true" components of variation are 0.5 E_1 and b = -0.04 approximates that found for extraversion (section 3.3.6.2).

4.3 RESULTS

4.3.1 Sample sizes required to discriminate between dominance and sibling interaction

The required sample sizes of twins, twins plus half-siblings and twins plus unrelated individuals reared together for the "true" $E_1(V_A+V''_A)V'_A$ model form Tables 4.3-4.5 respectively. Those for the "true" $E_1V_AV_D$ model form Tables 4.6-4.8 respectively.

For samples comprising twin pairs alone, where the "true" model is $E_1(V_A+V''_A)V'_A$, the sample sizes required to reject the E_1 model are not prohibitively large. The rejection of both two parameter models is more difficult, especially the E_1V_A model. When the sibling effect is small (less than 5% of the variance), it is unlikely to be feasible (Table 4.3).

When the "false" model is E_1, it is easier to reject the cooperation model than the competition model. For a false E_1E_2 model, E_1V_A model when pMZ>0.3, or $E_1V_AV_D$ model, it is easier to reject the competition model than cooperation.

Where the competition effect is large (greater than 30% of the variance), sample sizes required to reject the $E_1V_AV_D$ model are feasible (211 pairs, 70% DZ). However, if cooperation accounts for 30% of the variance, 1880 twin pairs (50% MZ) are required to reject the $E_1V_AV_D$ model.

Where sibling effects account for less than 30% of the variance, sample sizes required to reject the $E_1V_AV_D$ model are prohibitively large. In the case where b=-0.04, which approximates the breakdown of variation found for extraversion, we would need 32331 twin pairs (50% MZ) to reject the $E_1V_AV_D$ model (Table 4.3).

Table 4.3 Total number of twin pairs required for 95% power of rejection of false hypotheses at the 5% level of significance when the true model is $E_1(V_A+V''_A)V'_A$. Sample sizes are shown for both competition and cooperation. See text for futher explanation.

True model				False model: E_1 pMZ									
E_1	$(V_A+V''_A)$	V'_A=b		0.1		0.3		0.5		0.7		0.9	
.5	.5450	-.30	(.30)	84	(40)†	78	(38)	79	(37)	93	(36)	180	(36)
	.5050	-.10	(.10)	1085	(91)	412	(76)	250	(65)	176	(58)	135	(53)
	.5013	-.05	(.05)	419	(131)	233	(101)	160	(82)	121	(70)	96	(62)
	.5008	-.04	(.04)	358	(142)	210	(107)	147	(87)	113	(73)	91	(64)
	.5002	-.02	(.02)	270	(171)	173	(124)	127	(97)	100	(80)	82	(69)
								E_1E_2					
.5	.5450	-.30	(.30)	1065	(5146)	361	(2424)	231	(2227)	202	(2888)	324	(7310)
	.5050	-.10	(.10)	1630	(3230)	599	(1294)	421	(1110)	410	(1266)	758	(2792)
	.5013	-.05	(.05)	1864	(2621)	705	(1061)	512	(829)	516	(903)	992	(1895)
	.5008	-.04	(.04)	1920	(2521)	730	(1013)	534	(785)	542	(848)	1051	(1763)
	.5002	-.02	(.02)	2042	(2340)	787	(926)	583	(707)	601	(751)	1185	(1534)
								E_1V_A					
.5	.5450	-.30	(.30)	332	(*)	186	(141)	141	(195)	135	(325)	244	(982)
	.5050	-.10	(.10)	2384	(1128)	1156	(1073)	1101	(1332)	1441	(2049)	3578	(5771)
	.5013	-.05	(.05)	7445	(5031)	4338	(4217)	4528	(5006)	6260	(7482)	16201	(20581)
	.5008	-.04	(.04)	11088	(8091)	6722	(6577)	7129	(7731)	9951	(11480)	25942	(31416)
	.5002	-.02	(.02)	40456	(34536)	26555	(26287)	29020	(30228)	41226	(44287)	108938	(119884)
								$E_1V_AV_D$					
.5	.5450	-.30	(.30)	862	(4327)	235	(2041)	217	(1880)	211	(2446)	395	(6216)
	.5050	-.10	(.10)	13370	(23337)	5324	(10142)	4077	(8643)	4554	(10444)	9786	(27752)
	.5013	-.05	(.05)	61798	(81581)	25123	(34666)	19911	(28869)	22474	(34069)	49448	(78797)
	.5008	-.04	(.04)	99336	(124043)	40558	(52473)	32331	(43417)	36616	(51082)	80954	(117550)
	.5002	-.02	(.02)	420349	(469702)	173128	(196918)	139496	(161634)	159228	(188082)	355506	(428457)

* Sample size could not be calculated

† Values in parentheses correspond to positive values of b, that is cooperation.

Table 4.4 Total number of twin pairs and half-siblings required for 95% power of rejection of the $E_1V_AV_D$ model at the 5% level of significance when the true model is $E_1(V_A+V_A'')V_A'$. Sample sizes are shown for both competition and cooperation.

True model					pDZ							
E_1	(V_A+V_A'')	$V_A'=b$		pMZ	0.1		0.3		0.5		0.7	
.5	.5450	-.30	(.30)	0.1	639	(748)	587	(618)	598	(730)	692	(1177)
	.5050	-.10	(.10)		7990	(9227)	7396	(7668)	7799	(8259)	9723	(11440)
	.5013	-.05	(.05)		33881	(36572)	30767	(31392)	32551	(33506)	41497	(45008)
	.5008	-.04	(.04)		53487	(56883)	48348	(49141)	51182	(52381)	65559	(69958)
	.5002	-.02	(.02)		218060	(224935)	195139	(196757)	206856	(209267)	267563	(276389)
				0.3								
.5	.5450	-.30	(.30)		244	(547)	253	(575)	290	(876)		
	.5050	-.10	(.10)		3480	(5035)	3818	(5285)	4664	(6916)		
	.5013	-.05	(.05)		15464	(18668)	16935	(19989)	20999	(25615)		
	.5008	-.04	(.04)		24657	(28674)	26975	(30809)	33550	(39335)		
	.5002	-.02	(.02)		102624	(110689)	111944	(119660)	140095	(151609)		
				0.5								
.5	.5450	-.30	(.30)		169	(583)	197	(809)				
	.5050	-.10	(.10)		2791	(4652)	3502	(5885)				
	.5013	-.05	(.05)		12747	(16632)	16242	(21025)				
	.5008	-.04	(.04)		20518	(25362)	26110	(32128)				
	.5002	-.02	(.02)		86878	(96410)	110308	(122574)				
				0.7								
.5	.5450	-.30	(.30)		165	(880)						
	.5050	-.10	(.10)		3286	(6227)						
	.5013	-.05	(.05)		15638	(21625)						
	.5008	-.04	(.04)		25277	(32774)						
	.5002	-.02	(.02)		108112	(123139)						

Table 4.5 Total number of twin pairs and unrelated individuals reared together required for 95% power of rejection of the $E_1V_AV_D$ model at the 5% level of significance when the true model is $E_1(V_A+V_A'')V_A'$. Sample sizes are shown for both competition and cooperation.

True model					pDZ							
E_1	(V_A+V_A'')	V_A'=b		pMZ	0.1		0.3		0.5		0.7	
.5	.5450	-.30	(.30)	0.1	50	(68)	56	(95)	69	(148)	108	(305)
	.5050	-.10	(.10)		461	(516)	560	(679)	761	(1001)	1347	(1930)
	.5013	-.05	(.05)		1886	(1920)	2341	(2579)	3252	(3733)	5895	(7059)
	.5008	-.04	(.04)		2962	(3097)	3691	(3989)	5151	(5752)	9378	(10833)
	.5002	-.02	(.02)		11968	(12239)	15044	(15640)	21173	(22377)	38890	(41798)
				0.3								
.5	.5450	-.30	(.30)		43	(93)	52	(144)	77	(288)		
	.5050	-.10	(.10)		487	(632)	652	(922)	1108	(1719)		
	.5013	-.05	(.05)		2076	(2365)	2846	(3385)	4966	(6182)		
	.5008	-.04	(.04)		3286	(3647)	4525	(5198)	7934	(9454)		
	.5002	-.02	(.02)		13486	(14208)	18741	(20087)	33175	(36212)		
				0.5								
.5	.5450	-.30	(.30)		40	(144)	59	(286)				
	.5050	-.10	(.10)		576	(878)	970	(1626)				
	.5013	-.05	(.05)		2573	(3173)	4448	(5753)				
	.5008	-.04	(.04)		4108	(4858)	7136	(8768)				
	.5002	-.02	(.02)		17148	(18646)	30087	(33347)				
				0.7								
.5	.5450	-.30	(.30)		47	(295)						
	.5050	-.10	(.10)		893	(1614)						
	.5013	-.05	(.05)		4193	(5630)						
	.5008	-.04	(.04)		6758	(8552)						
	.5002	-.02	(.02)		28722	(32309)						

Table 4.6 Total number of twin pairs required for 95% power of rejection of the $E_1(V_A+V'_A)V'_A$ model at the 5% level of significance, when the true model is $E_1V_AV_D$.

True model			pMZ				
E_1	V_A	V_D	0.1	0.3	0.5	0.7	0.9
0.1	0.1	0.8	11254	5184	4664	5928	14726
	0.3	0.6	21468	9715	8597	10762	26356
	0.5	0.4	51706	23009	19941	24723	59703
	0.7	0.2	220905	96765	82935	100946	240422
0.3	0.1	0.6	17037	7905	7044	9114	22682
	0.3	0.4	41324	18793	16588	20947	51380
	0.5	0.2	177732	79322	69159	85573	206911
0.5	0.1	0.4	32318	15086	13598	17527	43716
	0.2	0.3	59817	27606	24727	31496	77980
	0.3	0.2	140023	63914	56837	71625	176048
0.7	0.1	0.2	107775	50534	45965	59110	147830

Table 4.7 Total number of twin pairs and half-siblings required for 95% power of rejection of the $E_1(V_A+V_A'')V_A'$ model at the 5% level of significance, when the true model is $E_1V_AV_D$.

True model			pMZ	pDZ			
E_1	V_A	V_D		0.1	0.3	0.5	0.7
0.1	0.1	0.8	0.1	8011	6709	6829	8429
	0.3	0.6		14886	11997	12041	14961
	0.5	0.4		34655	26778	26465	33086
	0.7	0.2		141841	104688	101690	127776
			0.3				
0.1	0.1	0.8		4324	4265	4874	
	0.3	0.6		8026	7746	8832	
	0.5	0.4		18758	17653	20068	
	0.7	0.2		77476	70818	80168	
			0.5				
0.1	0.1	0.8		4101	4460		
	0.3	0.6		7490	7936		
	0.5	0.4		17256	18133		
	0.7	0.2		70278	72905		
			0.7				
0.1	0.1	0.8		5477			
	0.3	0.6		9823			
	0.5	0.4		22234			
	0.7	0.2		89143			
			0.1				
0.3	0.1	0.6		13076	11268	11363	13563
	0.3	0.4		31134	25947	25779	30837
	0.5	0.2		130700	105009	102619	122885
			0.3				
0.3	0.1	0.6		7000	6934	7747	
	0.3	0.4		16523	16062	17876	
	0.5	0.2		69062	65695	72742	
			0.5				
0.3	0.1	0.6		6557	7044		
	0.3	0.4		15358	16278		
	0.5	0.2		63491	66298		
			0.7				
0.3	0.1	0.6		8928			
	0.3	0.4		20317			
	0.5	0.2		82017			

Table 4.7 cont'd

True model			pMZ	pDZ			
E_1	V_A	V_D		0.1	0.3	0.5	0.7
0.5	0.1	0.4	0.1	26611	23417	23495	27214
	0.2	0.3		48909	42479	42342	49029
	0.3	0.2		113589	97312	96334	111476
			0.3				
0.5	0.1	0.4		14208	14069	15384	
	0.2	0.3		25915	25473	27797	
	0.3	0.2		59780	58298	63468	
			0.5				
0.5	0.1	0.4		13618	14347		
	0.2	0.3		24478	25680		
	0.3	0.2		56077	58512		
			0.7				
0.5	0.1	0.4		18227			
	0.2	0.3		32619			
	0.3	0.2		73856			
0.7	0.1	0.2	0.1	95327	84649	84443	95255
			0.3	50906	50031	53494	
			0.5	48954	50471		
			0.7	65446			

Table 4.8 Total number of twin pairs and unrelated individuals reared together required for 95% power of rejection of the $E_1(V_A+V'_A)V'_A$ model at the 5% level of significance, when the true model is $E_1V_AV_D$.

True model			pMZ	pDZ			
E_1	V_A	V_D		0.1	0.3	0.5	0.7
0.1	0.1	0.8	0.1	2683	1297	1243	1820
	0.3	0.6		4702	2215	2079	2999
	0.5	0.4		10425	4780	4377	6188
	0.7	0.2		41084	18304	16190	22456
			0.3				
0.1	0.1	0.8		2778	1503	1815	
	0.3	0.6		4852	2542	3014	
	0.5	0.4		10720	5428	6280	
	0.7	0.2		42099	20553	23057	
			0.5				
0.1	0.1	0.8		3007	1938		
	0.3	0.6		5214	3513		
	0.5	0.4		11436	7385		
	0.7	0.2		44578	27442		
			0.7				
0.1	0.1	0.8		3678			
	0.3	0.6		6280			
	0.5	0.4		13552			
	0.7	0.2		51919			
			0.1				
0.3	0.1	0.6		6234	2938	2572	3244
	0.3	0.4		13837	6374	5501	6861
	0.5	0.2		54602	24553	20735	25577
			0.3				
0.3	0.1	0.6		6065	2898	3075	
	0.3	0.4		13495	6304	6542	
	0.5	0.2		53367	24336	24590	
			0.5				
0.3	0.1	0.6		6306	3661		
	0.3	0.4		13973	7696		
	0.5	0.2		55031	29649		
			0.7				
0.3	0.1	0.6		7163			
	0.3	0.4		15688			
	0.5	0.2		61052			

Table 4.8 cont'd

True model			pMZ	pDZ			
E_1	V_A	V_D		0.1	0.3	0.5	0.7
0.5	0.1	0.4	0.1	17480	8389	6952	7826
	0.2	0.3		30873	14655	12169	13615
	0.3	0.2		69006	32402	26892	29918
			0.3				
0.5	0.1	0.4		16133	7195	6759	
	0.2	0.3		28578	12657	11788	
	0.3	0.2		64069	28169	26000	
			0.5				
0.5	0.1	0.4		16300	8093		
	0.2	0.3		28845	14243		
	0.3	0.2		64604	31659		
			0.7				
0.5	0.1	0.4		17567			
	0.2	0.3		30970			
	0.3	0.2		69097			
0.7	0.1	0.2	0.1	81034	39145	32258	33302
			0.3	72300	31017	26484	
			0.5	71697	32648		
			0.7	74672			

Where the classical twin design is extended to include half-siblings, we see that the sample sizes required to reject the $E_1V_AV_D$ model, while less than those comprising only twins, are still not feasible when sibling effects account for less than 30% of the variance (Table 4.4). Sample sizes are drastically reduced when the sample comprises twins and unrelated individuals reared together (Table 4.5). For both these extended twin designs it is easier to reject competition than cooperation.

For both competition and cooperation, sample sizes required to reject the $E_1V_AV_D$ model are smallest if the sample comprises twins and unrelated individuals reared together, followed by samples comprising twins and half-siblings. Sample sizes are largest if we only have information on twins.

Where the "true" model is $E_1V_AV_D$, the sample sizes required to reject the sibling effect model are prohibitive, even when dominance accounts for 80% of the variance. This is true for the classical twin design (Table 4.6), and samples comprising either twins and half-siblings (Table 4.7) or twins and unrelated individuals reared together (Table 4.8).

Table 4.9 summarizes the best solutions for the rejection of the $E_1(V_A+V_A'')V_A'$ and $E_1V_AV_D$ models using the parameter values found for extraversion, that is, 0.5 E_1, b= -0.04 and 0.5 E_1, 0.2 V_A, 0.3 V_D, for the three experimental designs. The optimal proportion of twin pairs varies with the "right" and "wrong" model, and the experimental design. However, we see that experimental designs using twins or twins plus half-siblings have prohibitively large sample sizes. Although the sample sizes

Table 4.9 Optimal total sample sizes required for 95% power of rejection of the $E_1V_AV_D$ and $E_1(V_A+V_A'')V_A'$ models at the 5% level of significance. Sample sizes were calculated assuming a breakdown of variation which corresponds to that found for extraversion. See text for futher explanation.

Sample	True model	False model	Sample size	Sample breakdown
Twins	$E_1(V_A+V_A'')V_A'$	$E_1V_AV_D$	32331	.5MZ, .5DZ
	$E_1V_AV_D$	$E_1(V_A+V_A'')V_A'$	24727	.5MZ, .5DZ
Twins &				
Half-sibs	$E_1(V_A+V_A'')V_A'$	$E_1V_AV_D$	20518	.5MZ, .1DZ, .4HS
	$E_1V_AV_D$	$E_1(V_A+V_A'')V_A'$	24478	.5MZ. .1DZ, .4HS
Twins &				
Unrelateds	$E_1(V_A+V_A'')V_A'$	$E_1V_AV_D$	2962	.1MZ, .1DZ, .8UT
	$E_1V_AV_D$	$E_1(V_A+V_A'')V_A'$	11788	.3MZ, .5DZ, .2UT

required when we have twins and unrelated individuals reared together are more reasonable, over 2500 pairs of unrelated individuals reared together would be required to discriminate between the alternative models. As noted in the previous chapter, we would need two-thirds of this sample size for 80-85% power, one-third of this for 50-60% power. Despite this, it would still seem unlikely that by model-fitting techniques alone, we will ever be able to discriminate between the effects of sibling competition and dominance.

4.3.2 Test for genetical non-additivity and sibling interaction

As our results show that the discrimination between dominance and sibling interaction is likely to prove infeasible by model-fitting, we now consider other methods for the resolution of these effects.

The finding of dominance for extraversion would suggest that this character has been subject to strong directional selection, rather than stabilising selection, during the course of human evolution (Mather, 1973). For a character under directional selection one would expect, as well as directional dominance, polygenic determination, some additive genetic variance and duplicate gene action, that is additive x additive, additive x dominance and dominance x dominance epistasis (Mather, 1973, 1974). These expectations lead to several predictions that could be used to detect the presence of these effects.

Two tests based on first degree statistics have been suggested for the detection of directional dominance. The first of these is based on the comparison of the means of offspring

from genetically unrelated and consanguineous matings. If the distributions of scores are the same in the genetically related and unrelated parents, then, if there is dominance in the increasing direction, inbreeding depression will occur. That is, the mean of the distribution of the children of consanguineous matings will be lower than that of the children of unrelated matings. However, it is critical that the distributions of the parental populations be the same. For example, in a study of intelligence, Schull and Neel (1965) found that inbreeding is associated with low social status, although the reverse was found by Bashi (1977). That is, any association depends on the community examine

The second test is based on the interracial cross (Morton et al., 1967; Mather and Jinks, 1971). Suppose we have a randomly mating population composed of several groups with different trait means. If there is no dominance, and assuming that there are no between-families environmental effects, then the mean of the offspring of between-group matings should be equal to the mean of the parental distributions, and also equal to the average of the means of the offspring of within-group matings for the same two groups. However, if there is dominance then the offspring of between-group matings should more closely resemble the parent of the higher scoring group. Unfortunately, the effects of assortative mating and between-families environment may mimic the effects of dominance. Thus, any tendency for individuals in the upper tails of each group to 'marry out' would mimic dominance, and if there were environmental influences of parents on their offspring it would not be unexpected to find that the parent with

the higher score had a stronger influence on the development of the offspring, especially for example if we were dealing with cognitive traits.

In addition to the comparison of means, several tests for genetical non-additivity based on third degree statistics have been suggested. Fisher et al. (1932) have shown that directional dominance in the increasing direction will produce negative sample skewness. However, sample skewness may also be produced by epistasis, genotype x environment interaction and unequal gene frequencies (Fisher et al., 1932; Martin et al., 1978). Martin et al. (1978) have shown that in the absence of mean-variance regressions in MZ twins, the sum-difference regression test (Jinks and Fulker, 1970) can be used in DZ twins to detect directional non-additivity (dominance or epistasis) or unequal gene frequencies, thus enabling one to eliminate the possibility of genotype x environment interaction. Furthermore, the mean skewness within family means, for families of three or more offspring, provides a test for genetical non-additivity (dominance, epistasis and genotype x environment interaction) which is not confounded with unequal gene frequencies (Fisher et al., 1932; Jinks and Fulker, 1970). Thus, sum-difference regressions in DZ twins, in the absence of regressions in MZ twins, plus within-family skewness in sibships greater than three, would provide evidence for dominance and/or epistasis. As we have noted, both dominance and epistasis will often coexist for a trait which is under directional selection (Mather, 1973) and thus if we wish to test the hypothesis that a trait is under

directional selection, our inability to discriminate between these alternatives is unimportant.

Tests have also been suggested for the detection of sibling interaction. Eaves (1976) has argued that the single most powerful test for sibling effects would be the expected ranking of total variances for different groups. Following his argument, in the presence of cooperation we would expect

$$\sigma^2_{MZ} > \sigma^2_{DZ} > \sigma^2_{HS} > \sigma^2_{UT} > \sigma^2_{Singletons}.$$

If there was competition we would expect

$$\sigma^2_{MZ} < \sigma^2_{DZ} < \sigma^2_{HS} < \sigma^2_{UT}$$

and that

$$\sigma^2_{UT} > \sigma^2_{Singletons}.$$

The relationship between $\sigma^2_{Singletons}$ and the total variances of twins and half-siblings will depend on, and be a guide to, the relative magnitudes of the direct and indirect effects of the genes affecting the trait. However, as we have already seen, although we found no difference in the variance of MZ and DZ twins for extraversion, we were still not able to reject the competition model.

As we mentioned in the previous chapter, if there was sibling competition, it would be mutually advantageous if siblings, and especially twins, capitalised on different aspects of the environment. We would thus expect that sibling competition would decrease as the age difference of siblings increased. Although it would be possible to test this hypothesis, the level of competition found for extraversion, while significant, is small. Thus, in practice, it may be difficult to detect changes in competition as a function of age.

4.4 DISCUSSION

The results of our power calculations show that the sample sizes required to discriminate between dominance and sibling interaction are almost certainly prohibitively large. Thus unless the competition effect is large (greater than 30% of the variance), or dominance accounts for at least 80% of the variance, the resolution of dominance and competitive sibling interaction by model-fitting is unlikely to prove feasible. This is true whether one considers the classical twin design, or samples comprising either twins and half-siblings, or twins and unrelated individuals reared together.

Although we have suggested other tests which may be used to detect genetical non-additivity and sibling interaction, they are not without their weaknesses. Thus, the validity of using inbreeding data for the detection of dominance depends on the distributions in the parental populations being the same. The interracial cross assumes that there is no assortative mating or between-families environmental effects. There is evidence that this assumption is valid for extraversion within populations (Eaves and Young, 1981). However, if there are cultural differences between groups, it is possible that the more varied environment experienced by offspring of mixed marriages may produce a higher mean score in these offspring than would otherwise occur, and hence mimic the effects of dominance.

Sum-difference regressions in DZ twins, in the absence of regressions in MZ twins, plus within-family skewness in sibships greater than three, would provide evidence for genetical non-

additivity. When the heritability is high and dominance is complete, the power of the regression test in DZ twins is very high (Martin et al., 1978). However, for intermediate heritability, even when there is complete dominance, the power is little above chance level (Martin et al., 1978). As yet, the power of the skewness test in sibships greater than three has not been established, so we are unable to comment on its practical use. This is obviously an area worthy of further investigation. While it is possible that families with three or more offspring may be atypical of families in the general population, I know of no data regarding this point.

More work is needed to determine the power of tests for sibling interaction. As we have noted, the presence of sibling interaction leads to predictions about the expected ranking of total variances. The conditions under which the ranking of total variances leads to the detection of sibling effects of given size needs to be determined. Similarly, our ability to detect changes in sibling interaction as a function of the age difference of siblings needs to be established.

Our results show that more detailed theoretical analyses, of the type we have suggested, need to be completed before one can make any conclusions about the optimal experimental design, and sample sizes required, to resolve the effects of dominance and sibling interaction.

CHAPTER 5 THE CAUSES OF VARIATION IN SYMPTOMS OF ANXIETY AND DEPRESSION AND COVARIATION WITH THE TRAIT OF NEUROTICISM

5.1 INTRODUCTION

In Chapter 3 we examined the causes of variation in personality traits. However, when studying personality it has been suggested (e.g. Foulds, 1965, 1974) that one should distinguish between traits such as extraversion, psychoticism, neuroticism and lie, and symptoms. While there are no absolute criteria by which one can make this distinction, guidelines have been suggested (e.g. Foulds, 1971). In general, a trait is seen as a relatively permanent and broad reaction tendency, while a symptom is seen as a transient condition or mood (see also Cattell, 1965). Of particular interest in this thesis are symptoms of anxiety and depression. This group of disorders is prevalent in all adult age groups; at least 9% of the population being recognised as having a formal psychiatric disorder (Henderson et al., 1979; Bebbington et al., 1981). In 6% of the population these symptoms will persist for a median of six months, and 3% of the population will be incapacitated by overwhelming feelings of anxiety and depression (Henderson et al., 1981; Andrews et al., 1981).

Despite the prevalence of anxiety and depression, relatively little is known about the aetiology of these symptoms. The dominant theories of causation have been overwhelmingly concerned with the effect of environment. For example, it has been argued that anxiety and depression are attributable to emotional detachment, that is, the failure to make strong

emotional bonds, especially with parents (Bowlby, 1977). Indeed, Parker (1979a, 1979b) did find that subjects who regarded their parents as lacking in care and/or being overprotective had higher levels of anxiety and depression.

Other environmental theories have stressed the importance of life events. For example, events such as parental loss before the age of 17, a poor non-confiding marriage and lack of full or part-time employment have been shown to be associated with depression (Brown et al., 1975, 1977; Roy, 1978, 1981). Finlay-Jones and Brown (1981) found that the degree of loss associated with stressful life events was related to depression, while the degree of danger associated with such events was related to anxiety. However, while life events have been shown to predict the occurence of later psychological symptoms, prior symptoms are a better predictor than previous life events (Andrews, 1981). Furthermore, it has been shown that personality traits such as neuroticism or trait anxiety are the best long term predictors of neurotic states (Henderson et al., 1981).

While we know that there is a substantial genetic component to variation in neuroticism, genetic studies of anxiety and depression have given conflicting results. Thus, there is evidence of a substantial genetic component in variation in symptoms of anxiety, but much less clear evidence for symptoms of depression (Slater and Shields, 1969; Young et al., 1971; Shields, 1976). In the study by Slater and Shields (1969), there were 62 MZ and 84 DZ twins, at least one in each pair having reached a psychiatric consultation. There was striking concordance in MZ twins for anxiety neurosis and

personality disorder, but for other diagnoses, mainly consisting of minor or neurotic depression, there was little agreement. Schepank (1971, 1973) tried to avoid the diagnostic problem by considering instead the concordance for individual symptoms. In a clinical series of 21 MZ and 29 DZ pairs, he found evidence for a genetic component in symptoms of both anxiety and depression. A much larger study based on 587 pairs of twins has also found evidence for a substantial genetic component in both these symptoms (Eaves and Young, 1981). 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.

We shall first examine the causes of variation in symptoms of anxiety and depression. Then, since neuroticism is known to be closely associated with neurotic symptoms under stress, we shall examine the extent to which genetic and environmental effects on anxiety and depression can be explained in terms of neuroticism.

5.2 Measurements

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

The DSSI/sAD (Bedford et al., 1976) consists of 7 state of anxiety and 7 state of depression items. The items defining the

scales of anxiety and depression are shown in Table 5.1. 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% of men and 3.5% of women had scores of 7 or more for depression, and only 1.0% and 5.6% for anxiety. It has been used here as an appropriate instrument for measuring symptoms by self-report in a large postal survey.

5.3 RESULTS

5.3.1 Scaling

As a test of additivity of genetic and environmental effects, we regressed absolute within-pair differences in MZ twins on pair sums.* The anxiety and depression scales both show extreme "reverse-J" shape distributions (Figures 5.1 and 5.2) which produce significant and substantial linear regressions (Table 5.2) These are best reduced by logarithmic transformation and although this results in an increase in quadratic regression components, more extreme transformation [$\log_{10}(\log_{10}(x+1)+1)$ or

Table 5.1 Items defining the anxiety and depression scales of the DSSI/sAD.

Anxiety

A1. Recently I have worried about every little thing.

A2. Recently I have been breathless OR had a pounding of my heart.

A3. Recently I have been so 'worked up' that I couldn't sit still.

A4. Recently for no good reason, I have had feelings of panic.

A5. Recently I have had a pain OR tense feeling in my neck or head.

A6. Recently worrying has kept me awake at night.

A7. Recently I have been so anxious that I couldn't make up my mind about the simplest thing.

Depression

D1. Recently I have been so miserable that I have had difficulty with my sleep.

D2. Recently I have been depressed without knowing why.

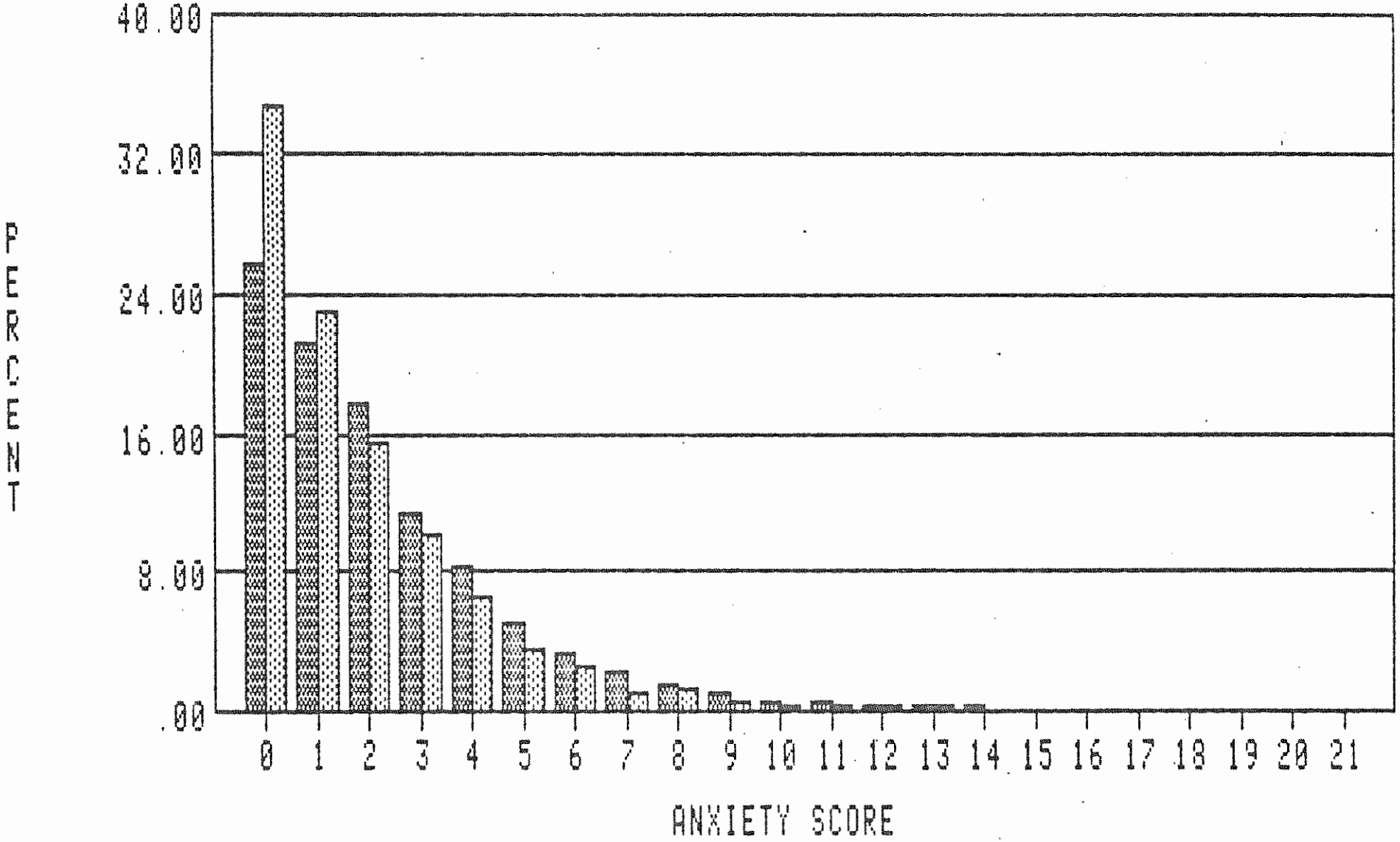
D3. Recently I have gone to bed not caring if I never woke up.

D4. Recently I have been so low in spirits that I have sat for ages doing absolutely nothing.

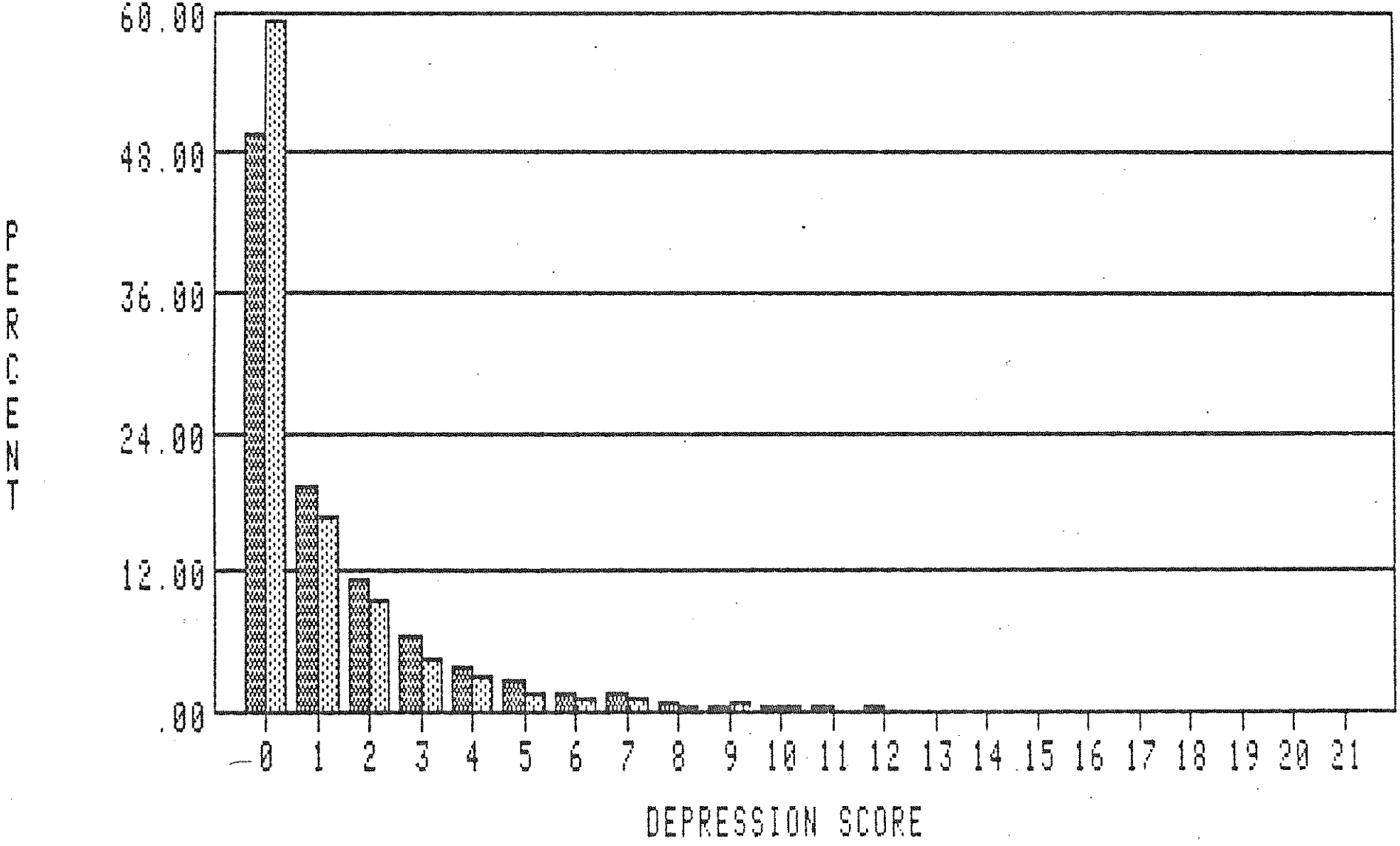
D5. Recently the future has seemed hopeless.

D6. Recently I have lost interest in just about EVERYTHING.

D7. Recently I have been so depressed that I have thought of doing away with myself.



DISTRIBUTION OF ANXIETY SCORES



DISTRIBUTION OF DEPRESSION SCORES

Table 5.2 Proportions of variance in absolute within-pair differences accounted for by regression on pair sums for raw and transformed DSSI/sAD scores and various transformations. Linear (L) and quadratic components after the linear regression has been removed (Q) are shown.

	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	$.33^{***}$	$.05^{***}$	$.49^{***}$	$.05^{***}$
$\sqrt{x+1}$	$.30^{***}$	$.09^{***}$	$.46^{***}$	$.08^{***}$
$\log_{10}(x+1)$	$.15^{***}$	$.21^{***}$	$.30^{***}$	$.21^{***}$

$\sqrt{\log_{10}(x+1)}$] produces no greater improvement so we regard the $\log_{10}(x+1)$ as most appropriate for both scales.

5.3.2 Distribution of scores and sex differences

As a test of sampling, two-tailed t-tests were performed between MZ and DZ means, and variance ratio tests between total variances, separately for males and females (Table 5.3). The only significant difference we observe is that the variance of raw depression scores is significantly greater in DZ females than MZ females. However, this difference becomes non-significant after transformation.

Examination of differences between sexes (Table 5.4) shows that females have significantly greater means and variances than males for both anxiety and depression. These inequalities are observed for both the raw and transformed scores. The distributions of anxiety and depression scores in the twin sample are similar to those observed in a random sample of Canberra electors (Henderson et al., 1981).*

5.3.3 Repeatability

The 96 individuals who completed both the pilot and the main questionnaire were typical of the total sample in distribution of anxiety and depression scores.

Separate analyses of variance of anxiety and depression scores to obtain mean squares between and within individuals enabled calculation of repeatabilities (Table 5.5). The repeatabilities of the anxiety and depression scales range from .55 ~ .67 and are no lower than one would expect of symptoms which fluctuate in their severity. In a longitudinal study of a

Table 5.3 Means and variances of the twin sample for raw and transformed anxiety and depression scores.

	Anxiety		Depression	
	Raw	$\log_{10}(x+1)$	Raw	$\log_{10}(x+1)$
MZF				
Mean	2.37	0.42	1.46	0.26
Variance	6.90	0.10	5.90*	0.09
MZM				
Mean	1.76	0.33	1.05	0.19
Variance	5.04	0.09	4.33	0.08
DZF				
Mean	2.37	0.41	1.54	0.27
Variance	6.99	0.10	6.99	0.10
DZM				
Mean	1.75	0.34	1.03	0.21
Variance	4.05	0.09	2.92	0.08
DZO				
Mean	2.15	0.39	1.41	0.25
Variance	6.09	0.10	6.23	0.10

* Difference between MZ female and DZ female variance significant at the 5% level.

Table 5.4 Means and variances for raw and transformed anxiety and depression scores separately for males and females. Asterisks denote significant differences between female and male means and/or variances.

	Females		Males	
	Mean	Variance	Mean	Variance
Anxiety				
Raw	2.37^{***}	6.92^{***}	1.82	4.88
$\log_{10}(x+1)$	0.42^{***}	0.10^{*}	0.34	0.09
Depression				
Raw	1.50^{***}	6.40^{***}	1.12	4.41
$\log_{10}(x+1)$	0.26^{***}	0.10^{***}	0.21	0.08

Table 5.5 Distribution of raw and transformed anxiety and depression scores for individuals who completed both the pilot and main questionnaire.

	Anxiety		Depression	
	Raw	$\log_{10}(x+1)$	Raw	$\log_{10}(x+1)$
Females				
Mean	2.55	0.44	1.75	0.29
Variance	6.85	0.10	9.17	0.10
Repeatability	0.67	0.63	0.55	0.66
S_W^2	2.29	0.04	4.09	0.03
Males				
Mean	1.67	0.30	0.94	0.18
Variance	5.30	0.10	3.23	0.07
Repeatability	0.61	0.62	0.58	0.58
S_W^2	2.15	0.04	1.35	0.03

general population sample (N=230) Henderson et al. (1981) administered the DSSI/sAD on two occasions three months apart. The anxiety scores correlated .62 and the depression .54. This sensitivity to change has also been reported by Bedford et al. (1976).

5.3.4 Genetic and environmental analysis of variation in symptoms of anxiety and depression

Alternative models of trait variation were fitted to between- and within-pairs mean squares by iterative weighted least squares (section 3.3.6.1). Models were fitted to the log transformed scores of anxiety and depression since this transformation best minimised G x E interaction. As there was a significant difference between male and female means for both anxiety and depression (Table 5.3), we corrected for this by replacing the within-pairs mean square with the residual pairs mean square corrected for sex differences in means (section 3.3.6.1).

Where a variable is strongly age dependent, this inflates the between-pairs mean square. The linear age correlations with anxiety (-0.06 in females, -0.09 in males) and depression (-0.14, -0.17) are in fact significant. This indicates that individuals become less anxious and depressed with increasing age. However, even the largest of these (-0.17 with depression in males) would produce only a trivial inflation of the between-pairs mean square so we have made no correction for this source of variance. The ten mean squares and their degrees of freedom, corrected for sex differences are shown for anxiety and depression scores in Table 5.6.

Table 5.6 Observed mean squares for log transformed anxiety and depression scores and their degrees of freedom.

		Anxiety		Depression	
		df	mean square	df	mean square
MZF	Between	1229	0.134	1229	0.128
	Within	1230	0.060	1230	0.061
MZM	Between	566	0.122	566	0.103
	Within	567	0.057	567	0.054
DZF	Between	749	0.117	749	0.118
	Within	750	0.081	750	0.081
DZM	Between	351	0.097	351	0.091
	Within	352	0.076	352	0.059
DZO	Between	901	0.106	901	0.108
	Within[a]	901	0.083	901	0.082

[a] Corrected for sex differences in means.

We may also examine whether twins become more or less similar with age by correlating absolute within-pair differences with age (Table 5.7). All correlations are small and non-significant for anxiety. For depression, both MZ and DZ males, and the DZ opposite-sex pairs, become more similar with advancing age, but no such effect is apparent in females. While this latter finding is open to a number of interpretations, it is obvious that if there are differences in the environmental circumstances of co-twins as they get older, these do not produce differences in symptoms of anxiety and depression.

We shall discuss the results of model fitting separately for anxiety and depression.

5.3.4.1 Results of fitting models to anxiety scores

The results of fitting models to log transformed anxiety scores are shown in Table 5.8. A purely environmental model (E_1E_2) fails to adequately 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 chi-square were seen with addition of extra parameters. When the E_1V_A model is fitted to the combined male and female data, the chi-square for the heterogeneity of fit over sexes is non-significant ($\chi^2_2 = 5.18$, $p>.05$). 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 $\hat{V}_A$ component for females than males.

The results of fitting a model that specifies a common E_1 parameter but different sized V_A effects in males and females is shown in Table 5.9. Fitting separate V_A parameters for males and

Table 5.7 Two-tailed correlations of absolute within-pair differences in log transformed anxiety and depression scores with age.

	MZF	MZM	DZF	DZM	DZO
Anxiety	.02	-.03	-.01	-.06	-.01
Depression	-.04	-.18***	-.01	-.13*	-.14**

Table 5.8 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 & Male							
E_1E_2	$.067^{***}$	$.028^{***}$	-	-	6	45.37^{***}	
E_1V_A	$.060^{***}$	-	$.035^{***}$	-	6	6.70	.37 ±.02
$E_1E_2V_A$	$.059^{***}$	-.004	$.040^{***}$	-	5	6.23	
$E_1V_AV_D$	$.059^{***}$	-	$.027^{**}$	.009	5	6.23	
Female & Male & Opposite-sex							
E_1E_2	$.071^{***}$	$.024^{***}$	-	-	8	67.68^{***}	
E_1V_A	$.060^{***}$	-	$.034^{***}$	-	8	10.36	.36 ± .02
$E_1E_2V_A$	$.059^{***}$	-.008	$.043^{***}$	-	7	7.33	
$E_1V_AV_D$	$.059^{***}$	-	$.020^{*}$	$.016^{*}$	7	7.33	

Table 5.9 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

$\chi^2_6 = 2.15$ ($p = .91$)

$h^2_{males} = 0.33 \pm .03$ $h^2_{females} = 0.39 \pm .02$

females causes a significant reduction in chi-square (χ^2_1 = 5.18, p>.05). The correlation $r_{V_{A_{MF}}}$ = 0.67 is not significantly different from unity and indicates that the same V_A effects which act in females also act in males, but with a smaller effect on the variance. Thus in males approximately 33% of the variation in anxiety is genetic in origin while in females this rises to approximately 39%, with the remaining variance due to individual environmental differences and error. We may subtract the values of the residual mean square, 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 5.10).

5.3.4.2 Results of fitting models to depression scores

As in the case of anxiety, in both males and females, the E_1V_A model best describes the data, although in males there is some evidence that E_2 effects are also important (Table 5.11). The chi-square for the heterogeneity of fit over sexes is highly significant (χ^2_2 = 27.26, p<.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 5.12) causes a significant reduction in chi-square (χ^2_3 = 24.97, p>.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 chi-square (χ^2_1 =

Tables 5.10 Sources of variance (%) for log transformed anxiety scores.

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

Table 5.11 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	$.36 \pm .02$
$E_1E_2V_A$	$.062^{***}$	-.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	$.32 \pm .04$
$E_1E_2V_A$	$.053^{***}$	.010	.013	-	1	0.46	
$E_1V_AV_D$	$.053^{***}$	-	$.044^{**}$	-.021	1	0.46	
Female & 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 & Male & 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 5.12 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

$\chi^2_5 = 8.73$ $(p = .12)$

$h^2_{males} = 0.33 \pm .03$ $\qquad$ $h^2_{females} = 0.37 \pm .02$

1.40, $p>.05$) indicating that this effect is not necessary to describe variation. The breakdown of the total variation into genetic and environmental components separately for males and females is shown in Table 5.13. 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.

5.3.5 Correlations of anxiety and depression with personality and attitude scores

Partial correlations, controlling for age, between the transformed symptoms of anxiety and depression and the other transformed personality and attitudes scores measured in this study are shown in Table 5.14. 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. There is also a slight tendency for more liberal men to be more anxious and depressed. While many of these correlations are statistically significant, they are however quite low with the exception of the correlations 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 trait variation in both sexes, although there are differences in the importance of these effects in males and females. We now ask whether these two sources of variation are responsible for trait covariation using the genetical analysis of covariance structures

Tables 5.13 Sources of variance (%) 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

Table 5.14 Partial correlations, controlling for age, between the transformed personality and attitude variables separately for females and males.

		Anxiety log(x+1)	Depression log(x+1)	Extraversion Angle	Psychoticism Angle	Neuroticism Angle	Lie Angle	Conservatism Raw
Females								
Anxiety	log(x+1)	-	.66***	-.08***	.15***	.61***	-.12***	-.01
Depression	log(x+1)	.66***	-	-.10***	.18***	.57***	-.09***	-.03*
Males								
Anxiety	log(x+1)	-	.60***	-.12***	.12***	.60***	-.11***	-.05*
Depression	log(x+1)	.60***	-	-.16***	.16***	.55***	-.08***	-.05**

developed by Martin and Eaves (1977). 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. (1981a) so only a brief account will be given.

5.3.5.1 Genetic and environmental analysis of covariation between symptoms of anxiety and depression and the trait of neuroticism

The genetical analysis of covariance structures is a technique that tests simultaneously hypotheses about both the sources and the structure of covariation. Just as univariate models were fitted to mean squares, multivariate models are fitted to the between- and within-mean products matrices shown in Table 5.15.

In general, we write the expectation for a mean-products matrix:

$$\Sigma_i = \sum_{j=1}^{p} c_{ij}[B_j(\Delta_j \Phi_j \Delta_j')B_j' + \theta_j^2]$$

where there are p sources of variation and c_{ij} is the coefficient from the univariate model for the i-th mean square and j-th source. For the j-th source Δ_j is the matrix of the factor loadings and θ_j^2 the diagonal matrix of specific variance components. Note, however, that we may complicate the model by introducing correlations between the factors in Φ_j, or relate the factor structures of different sources by a simpler scalar held in the diagonal matrix B.

Table 5.15 Mean products matrices for the transformed anxiety, depression and neuroticism scores.

		MZ females between 1228 df		
		330.30	4.4175	4.0954
Neuroticism	Angle	104.96	.13371	.09509
Anxiety	log(x+1)	1.1982	.05983	.12820
Depression	log(x+1)	1.1647	.03232	.06063
		MZ females within 1229 df		
		MZ males between 565 df		
		315.18	4.1190	3.4697
		115.65	.12219	.07395
		1.1616	.05706	.10310
		1.1665	.02764	.05397
		MZ males within 566 df		
		DZ females between 749 df		
		264.70	3.6399	3.5327
		157.57	.11675	.08109
		1.9503	.08064	.11795
		1.8274	.04999	.08120
		DZ females witnin 750 df		
		DZ males between 350 df		
		263.16	2.9354	2.8184
		183.94	.09708	.05346
		1.9195	.07612	.09118
		1.5822	.03385	.05875
		DZ males within 351 df		
		DZ opposite-sex between 899 df		
		228.45	3.1143	2.9562
		174.26	.10568	.07114
		2.3166	.08276	.10786
		2.0929	.05045	.08259

Having specified the sources of variation and the factor structures of a model, we test the fit of the model as follows: Generally, the data will consist of k matrices of mean products. We write $\underline{S}_i$ for the i-th matrix, having N_i degrees of freedom. Given some model for the $\underline{S}_i$, we compute the expected values Σ_i, being positive definite, for particular values of the parameters of the model. When the observations are multivariate normal, the log likelihood of obtaining the k observed independent $\underline{S}_i$ is:

$$\log L = -\tfrac{1}{2} \sum_{i=1}^{i=k} N_i [\log|\Sigma_i| + tr(\underline{S}_i \Sigma_i^{-1})]$$

(omitting the constant term).

For a given model we require the parameter estimates that maximise Log L. Given maximum-likelihood estimates of our parameters, the hypothesis that a less restricting model (ie. one involving more parameters) does not significantly improve the fit can be tested by computing:

$$\chi^2 = 2(L_0 - L_1),$$

where L_1 is the log likelihood obtained under the restricted hypothesis (H_1) and L_0 is the log likelihood obtained under the less demanding hypothesis (H_0). The H_0 we use is that which assumes that as many parameters are required to explain the data as there are independent mean squares and mean-products in the first place ; ie. $\Sigma_i = \underline{S}_i$. In this case we have simply:

$$L_0 = \tfrac{1}{2} \sum_{i=1}^{i=k} N_i [\log|\underline{S}_i| + p]$$

When there are k matrices the χ^2 has 1/2kp(p+1)-m df, where m denotes the number of parameters estimated under H_1 and p is the number of variables.

The log likelihood is maximised by attempting to minimise -Log L for a given model. We have minimised -Log L using the Fortran routine E04JAF from the Numerical Algorithms Group Library (1981).

The simplest E_1V_A model involves a single general factor causing covariation between anxiety, depression and neuroticism plus a variance component specific to each trait for both the E_1 and V_A sources of covariation. For each source then, we estimate 3 factor loadings and 3 specific variance components, or 12 parameters in all. Each mean products matrix contributes 3 mean squares from the diagonal and 3 off-diagonal mean products, making 24 unique statistics from the four between- and within-pairs matrices of MZ and DZ twins of the same sex. We are thus left with 12 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 5.16. 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

Table 5.16 Results of fitting a multivariate E_1V_A model to transformed anxiety depression and neuroticism scores. Results are in terms of the proportion of variance accounted for by each source.

		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)	$.33^{***}$	$.31^{***}$	$.30^{***}$	$.06^{***}$
			$\chi^2_{12} = 6.90$ ($p = .86$)		
Males					
Neuroticism	Angle	$.22^{***}$	$.32^{***}$	$.34^{***}$	$.12^{***}$
Anxiety	log(x+1)	$.31^{***}$	$.35^{***}$	$.30^{***}$	$.04^{**}$
Depression	log(x+1)	$.33^{***}$	$.35^{***}$	$.23^{***}$	$.09^{***}$
			$\chi^2_{12} = 12.52$ ($p = .40$)		

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. The proportion of variance due to error or fluctuating environment in anxiety and depression (Tables 5.10 and 5.13) 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 5.17. 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 5.17 Genetic and environmental correlations between transformed anxiety, depression and neuroticism scores. Females upper triangle, 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	-

5.3.6 Evidence for item heterogeneity

As we noted in the previous section, genetic variation in anxiety and depression is largely due to the effects of the same genes which determine variation in the trait of neuroticism. However, there is still some genetic variance specific to symptoms of anxiety and depression. This may indicate that there is heterogeneity in the aetiology of specific symptoms of anxiety and depression, and Kendler et al., (1985) have recently examined the causes of variation in the responses of our twin sample to the individual items of the anxiety and depression scales, with a view to detecting and characterising such heterogeneity. Although the author was not involved in the analysis of these data, the main results of Kendler et al. (1985) are briefly presented for completeness.

Kendler et al. (1985) fitted alternative models of variation to the five contingency tables derived from the twins' responses to each of the fourteen DSSI/sAD items using the technique of threshold analysis (section 3.3.7.1). As the procedure of Kendler et al. (1985) was essentially the same as that used in our analysis of the EPQ items (Chapter 3), we shall only present their final model-fitting results. The parameter estimates of the best-fitting models are given separately for the anxiety (Table 5.18) and depression (Table 5.19) items.

For one item (D7) it was not possible to find an adequate description of the data. Of the remaining items, the E_1V_A model provides the best description of the data for seven of the symptoms (A1, A3, A5 and A7; D3, D4 and D5). The heritability of

Table 5.18 Parameter estimates for best fitting models for anxiety items.

	Parameter							
Item	E_1	E_{1_M}	E_{1_F}	E_{2_M}	E_{2_F}	$E_{2_{MF}}$	V_A	V_D
A1	.656	-	-	-	-	-	.344	-
A2	.667	-	-	-	-	-	-.149	.482
A3	.642	-	-	-	-	-	.358	-
A4	.536	-	-	-	-	-	-.144	.608
A5	.649	-	-	-	-	-	.351	-
A6	.667	-	-	-	-	-	.333	-
	-	.728	.707	.272	.293	.125	-	-
A7	.563	-	-	-	-	-	.437	-

Table 5.19 Parameter estimates for best fitting models for depression items.

	Parameter								
Item	E_1	E_{1_M}	E_{1_F}	E_{2_M}	E_{2_F}	$E_{2_{MF}}$	V_A	V_{A_M}	V_{A_F}
D1	-	.751	.586	-	-	-	-	.249	.414
	-	.773	.651	.227	.349	.109	-	-	-
D2	.666	-	-	-	-	-	.334	-	-
	-	.674	.718	.326	.282	.116	-	-	-
D3	.555	-	-	-	-	-	.445	-	-
D4	.541	-	-	-	-	-	.459	-	-
D5	.615	-	-	-	-	-	.385	-	-
D6	.602	-	-	-	-	-	.398	-	-
	-	.697	.638	.303	.362	.092	-	-	-

these items ranged from 0.34 to 0.46. For two items (A2 and A4) a model incorporating dominance (the $E_1V_AV_D$ model) provided an adequate description of the data. Because of the large correlation between V_A and V_D in the classical twin design, the estimate of V_A was negative in both cases. Although it is thus difficult to determine precise values of V_D for these items, the broad heritabilities are in the range 0.35 to 0.45. For four items, it was not possible to choose definitively between two possible best fitting models, one of which was always the $E_{1_M}E_{1_F}E_{2_M}E_{2_F}E_{2_{MF}}$ model. In three of the items (A6, D2 and D6) the other possible best fitting model was the E_1V_A model, and the heritabilities ranged from 0.33 to 0.40. For the remaining item (D1), the other best fitting model was the $E_{1_M}E_{1_F}V_{A_M}V_{A_F}$ model. This model assumes that the same genes are acting in both sexes, although the estimated heritability was greater in females (0.41) than males (0.25). When we consider the results of fitting the $E_{1_M}E_{1_F}E_{2_M}E_{2_F}E_{2_{MF}}$ model to these items, we see that the correlation between family-environmental effects in males and females ranges from 0.28 (D6) to 0.44 (A6), indicating that there are some differences in between-families environmental effects in the two sexes. For three items (A6, D1 and D6) between-families environmental effects are more important in females than males, while for D2 the reverse is true.

As Kendler et al. (1985) note, there is some common theme between items for which alternatives to the E_1V_A model could be found. The items for which dominance may play some role were both "panic-like" symptoms (A2 - breathless or heart pounding; A4

- feelings of panic"). The authors suggest that if confirmed, the results may indicate that the genetic basis of "panic-like" anxiety may differ from that of "cognitive" or "physical-tension-like" anxiety (e.g. as typified by items A1 and A7, and A3 and A5 respectively).

The family-environmental effect was most likely for two "insomnia" items (A6 - worrying kept me awake; D1 - miserable, difficulty with sleep), and two core symptoms of depression (D2 - depressed without knowing why; D6 - lost interest in everything). If replicated, the results suggest that there may be genuine aetiological heterogeneity in specific symptoms of anxiety and depression.

5.4 DISCUSSION

While previous studies on the aetiology of neuroses and minor depression have yielded conflicting results (Slater and Shields, 1969; 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 find 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 family environmental experiences are a major influence on anxiety and depression in adulthood (Bowlby,1977; Parker, 1979a, 1979b, 1981a, 1981b) must recognise 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).

Our results also have important implications for theorists who emphasise the importance of life events in the development of anxiety and depression. While it has been shown that life events are related to symptoms of distress such as anxiety and depression (e.g. Brown et al., 1977; Roy, 1981; Finlay-Jones and Brown, 1981) the direction of causality has not been determined. That is, while life events may result in symptoms of distress, distress could also result in life events. Since our results show that genetic factors are important to variation in anxiety and depression, it is possible that genetic factors could also act to increase an individual's predisposition to experience life events.

Furthermore, it has been shown that life events are associated with symptoms of distress in only some individuals (e.g. Akiskal and McKinney, 1975). To explain this variability, it has been suggested that other variables act as modifiers of the relationship between life events and symptoms of anxiety and depression. One class of modifier variables reflects social support, that is the perception that support is available from other people if needed. The results of Henderson et al. (1981),

however, indicate that the effect of social support on symptoms of anxiety and depression can be explained by the trait of neuroticism. They argue that neuroticism is the real variable which modifies the relationship between life events and neurotic symptoms. This result is particularly interesting in view of our results which show that genetic variation in symptoms of anxiety and depression is largely dependent on the same genes which determine variation in the trait of neuroticism. Our results, in conjunction with those of Henderson et al. (1981), suggest that in the presence of life events, genes influencing neuroticism will have a greater impact on levels of anxiety and depression than they do in the absence of a life event. This is obviously an important area for future research.

Our analysis of the causes of genetical and environmental covariation of anxiety, depression and neuroticism 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% of the total in females, 12% in males) which are independent of the two symptoms we have measured. It is

interesting to speculate that the general factor and specific components of genetic variance that we observe for anxiety and depression may distinguish between different forms of these symptoms which differ in their aetiology. The results of Kendler et al. (1985) do in fact indicate that there is heterogeneity in symptoms of anxiety and depression. Although for six of the symptoms a simple genetic model was most appropriate, there was evidence that dominance may be important for "panic-like" symptoms, while common-family environment may affect variation in "insomnia" and core symptoms of depression. If replicated these results may prove useful in characterising forms of anxiety and depression which differ in their aetiology.

CHAPTER 6 THE CAUSES OF VARIATION IN CONSERVATISM

6.1 INTRODUCTION

In previous chapters of this thesis we examined the causes of variation in personality traits and symptoms. Related to personality is the concept of social attitudes. Whereas personality refers to a general predisposition to behave, attitudes have particularity; that is they have as a particular object something or someone that is outside a persons own behaviour (Eysenck, 1954). Theories on the structure of social attitudes have had a similar history to theories on the structure of personality, with considerable debate over the number of dimensions of social attitudes that are important.

Thurstone (1934) analysing the factor structure of 11 attitude scales and one intelligence test extracted two major factors: nationalism and conservatism. Carlson (1934) in a factor analysis of five attitude scales and one intelligence test extracted three major factors which he labelled intelligence, conservatism and religious. Ferguson in a series of experiments on the structure of social attitudes isolated two (Ferguson, 1939, 1940, 1941), and then three major factors (Ferguson, 1942), which he called religionism, humanitarianism and nationalism.

Eysenck (1944) attempted to synthesise the findings of Thurstone, Carlson and Ferguson, as well as his own from an analysis of a 32 item attitude questionnaire. He found that if one considered the original two centroid axes in each of the various analyses, the various solutions were mathematically equivalent in that one solution could be converted into another

by simple orthogonal rotation through 45°. He argued that the different solutions could be best described by reference to two orthogonal factors which he labelled radicalism-conservatism and toughmindedness-tendermindedness. Conservatism was seen as a contrast between religious, nationalistic and punitive views on the one hand, with those that are more permissive, liberal and reforming on the other. Toughmindedness was not considered as a dimension of social attitudes in its own right, but rather was viewed as a projection of personality variables on to the radical-conservative continuum. Tendermindedness was considered as the projection of introverted personality traits, toughmindedness as the projection of extraverted personality traits. Thus only one factor, radicalism-conservatism, was seen to underlie all social attitudes (Eysenck, 1954; Wilson, 1973a; Eysenck and Wilson, 1978). In the present study, we shall attempt to test this model of the structure of social attitudes.

As with personality, it is often assumed that individual differences in conservatism are due mainly to the socialising influence of the family (e.g. Feather, 1978). Certainly attitudes show substantial family resemblance (Cavalli-Sforza et al., 1982; Insel, 1974; Feather, 1978) and secular changes in attitudes are so rapid (Jahoda and Warren, 1966) that frequent revision of test instruments is necessary. While such findings are consistent with a cultural model for family resemblance, the majority of studies so far have not attempted to test this assumption. For example, Cavalli-Sforza et al. (1982) studied interests and attitudes in a small sample of nuclear families and

pairs of friends. Although they found that attitudes were mostly determined within the family, the authors themselves admitted that the nuclear family design, comprising only parents and children, is incapable of resolving cultural and biological inheritance.

There is evidence, however, from three independent twin studies (see Eaves et al., 1978 for a summary) which suggests that genetic factors are important to variation in conservatism. The three studies showed remarkable consistency in assigning approximately equal proportions of variance to additive genetic effects, within-family environmental effects and a between-family component (B). When corrected for the effects of assortative mating the heritabilites were around 50%, while cultural effects accounted for less than 20% of the total variation. In this study, which is the largest twin study of conservatism that has been done to date, we have the opportunity to test the generality of these findings.

The fact that attitudes are however, 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. 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 if there are any indivdual attitude items which 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.

For example, Feingold (1984) in a study of 825 twin pairs found that attitude items most affected by family environment and cultural influences related to socialism and prejudice. Thus, as well as examining the sources of variation in the trait of conservatism, we shall also examine the causes of variation in the individual items and attempt to determine if there are any items which have a predominantly genetic or environmental basis.

6.2 MEASUREMENTS

6.2.1 The C-Scale

The C-Scale (Wilson and Patterson, 1968) was developed to measure the general personality dimension of authoritarianism with specific reference to "resistance to change" (Patterson and Wilson, 1969; Wilson, 1970). The scale consists of 50 items (Table 6.1) 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". Answers to odd-numbered items are scored such that Yes reflects a conservative response and No a liberal response, while Yes to even-numbered items reflects a liberal response and No a conservative response. The items are scored 0, 1 or 2 according to whether the response is liberal, ambiguous or conservative, so that total conservatism scores could range from 0 to 100 in the direction of increasing conservatism. The scale has been used to demonstrate the relationship between conservatism and art preference (Wilson, 1973a), projective aggression (Wilson, 1973b) and child-rearing practices (Thomas, 1975).

Table 6.1 The C-Scale.

1 Death penalty	26 Computer music
2 Evolution theory	27 Chastity
3 School uniforms	28 Fluoridation
4 Striptease shows	29 Royalty
5 Sabbath observance	30 Women judges
6 Hippies	31 Conventional clothes
7 Patriotism	32 Teenage drivers
8 Modern Art	33 Apartheid
9 Self-denial	34 Nudist camps
10 Working mothers	35 Church authority
11 Horoscopes	36 Disarmament
12 Birth control	37 Censorship
13 Military drill	38 White lies
14 Co-education	39 Caning
15 Divine law	40 Mixed marriage
16 Socialism	41 Strict rules
17 White superiority	42 Jazz
18 Cousin marriage	43 Strait-jackets
19 Moral training	44 Casual living
20 Suicide	45 Learning Latin
21 Chaperones	46 Divorce
22 Legalised abortion	47 Inborn conscience
23 Empire-building	48 Coloured immigration
24 Student pranks	49 Bible truth
25 Licensing laws	50 Pyjama parties

6.3 RESULTS

6.3.1 Distribution of item scores and sex differences

Chi-square tests were performed to determine if there were any significant differences between MZ and DZ endorsement frequencies for individual items, separately for females (Table 6.2) and males (Table 6.3). Of the 100 chi-square tests, only 9 were significant at least at the 5% level which is scarcely above chance level. As the groups appear to be comparable, MZ and DZ classes were combined to examine the effect of sex on endorsement frequency.

Table 6.4 gives the percentage of individuals giving a liberal, ambiguous or conservative response to an item, broken down by sex. Chi-square tests were performed between male and female endorsement frequencies (Table 6.4). There is evidence of highly significant sex differences in endorsement pattern for all but 5 items, indicating that the effect of sex on response frequency must be incorporated into our analyses.

6.3.2 Factor analysis of the C-Scale

The 50 items of the C-Scale were intercorrelated using product moment correlations, separately for males and females. Principal factoring with iteration was carried out on the item correlation matrices obtained separately for males and females. Although 11 factors had eigenvalues greater than one, only 8 could be easily interpreted. As we wished to test the hypothesis that a general factor of conservatism underlies all social attitudes, we first extracted only one principal factor. If the uni-dimensional hypothesis of social attitudes

Table 6.2 Chi-squares (for two degrees of freedom) testing the significance of differences between MZ and DZ endorsement frequencies for conservatism items in females.

Item	χ^2	Item	χ^2
1	0.46	26	3.31
2	0.93	27	13.38**
3	0.55	28	2.20
4	1.54	29	1.39
5	2.33	30	0.14
6	0.61	31	4.79
7	1.66	32	1.77
8	0.43	33	1.96
9	1.94	34	3.22
10	1.37	35	3.42
11	0.52	36	12.95**
12	4.56	37	3.95
13	2.74	38	0.08
14	2.84	39	5.64
15	2.26	40	1.80
16	0.92	41	1.47
17	0.50	42	0.99
18	2.33	43	1.46
19	0.19	44	1.00
20	3.84	45	0.08
21	2.23	46	4.31
22	0.27	47	2.57
23	1.44	48	0.06
24	4.58	49	0.55
25	0.32	50	1.09

Table 6.3 Chi-squares (for two degrees of freedom) testing the significance of differences between MZ and DZ endorsement frequencies in conservatism items in males.

Item	χ^2	Item	χ^2
1	0.58	26	2.14
2	0.22	27	3.82
3	3.14	28	0.57
4	0.15	29	2.04
5	1.53	30	1.15
6	6.19*	31	0.98
7	1.15	32	0.34
8	0.08	33	1.63
9	0.55	34	1.85
10	2.03	35	0.06
11	3.06	36	2.75
12	8.59*	37	1.54
13	7.62*	38	0.21
14	13.48**	39	4.73
15	1.78	40	2.64
16	0.62	41	0.28
17	0.07	42	7.51*
18	0.61	43	0.62
19	0.00	44	2.29
20	12.46**	45	0.26
21	6.05*	46	4.35
22	1.50	47	2.38
23	0.70	48	2.22
24	2.89	49	2.69
25	2.32	50	2.21

Table 6.4 Percentage of individuals giving a liberal, ambiguous or conservative response, broken down by sex. Asterisks denote significant differences between male and female response frequencies.

	Liberal		Ambiguous		Conservative		
Item	Female	Male	Female	Male	Female	Male	χ^2_2
1	33.6	30.4	19.5	10.3	47.0	59.4	149.78***
2	28.3	20.6	34.0	23.7	21.8	19.1	128.28***
3	8.1	24.0	3.3	6.7	88.7	69.3	447.12***
4	22.6	57.8	20.6	15.2	56.8	27.0	979.87***
5	26.9	36.9	23.1	23.5	49.9	39.6	97.85***
6	28.4	34.4	26.3	21.0	45.3	44.6	40.75***
7	9.3	12.0	16.5	12.6	74.2	75.4	30.10***
8	48.2	44.1	21.4	21.2	30.5	34.8	16.41***
9	26.1	30.6	26.6	24.8	47.3	44.6	17.44***
10	55.4	52.9	13.9	13.4	30.7	33.7	7.65*
11	52.0	72.0	16.7	12.2	31.3	15.8	306.63***
12	92.3	87.9	3.1	4.7	4.6	7.5	41.09***
13	18.2	26.9	21.4	13.8	60.4	59.2	116.92***
14	91.5	92.3	5.3	5.1	3.1	2.6	1.76
15	18.9	30.4	38.2	30.8	42.9	38.8	134.50***
16	24.0	29.1	30.6	22.6	45.5	48.3	61.07***
17	83.6	81.6	9.3	7.7	7.1	10.7	32.86***
18	14.0	19.1	20.3	24.2	65.8	56.7	65.85***
19	4.0	7.1	9.9	12.9	86.1	80.0	54.22***
20	8.8	11.3	9.8	9.8	81.4	78.9	12.34**
21	56.7	55.1	20.0	20.0	23.2	24.9	2.89
22	61.4	63.6	10.8	11.2	27.8	25.2	5.95
23	43.8	50.5	34.4	22.2	21.7	27.3	126.85***
24	28.2	44.3	19.1	15.9	52.6	39.8	203.31***
25	6.6	13.1	12.9	11.5	80.4	75.4	89.30***
26	28.9	33.4	31.5	24.0	39.6	42.5	49.35***
27	32.9	40.6	26.4	24.3	40.7	35.1	46.66***
28	59.7	58.6	18.6	20.0	21.7	21.4	2.20
29	17.3	30.7	13.3	12.6	69.4	56.6	186.95***
30	94.4	85.3	3.2	6.3	2.4	8.4	191.05***

Table 6.4 cont'd

	Liberal		Ambiguous		Conservative		
Item	Female	Male	Female	Male	Female	Male	χ^2_2
31	14.2	11.9	15.2	14.5	70.6	73.6	9.70**
32	62.7	70.6	12.3	9.7	25.0	19.7	48.69***
33	65.3	72.2	28.8	19.8	5.9	8.0	79.69***
34	40.9	63.5	18.8	15.2	40.3	21.3	385.46***
35	40.9	50.5	24.4	20.8	34.7	28.7	65.29***
36	40.3	56.2	35.9	17.7	23.8	26.0	297.73***
37	17.3	32.6	13.8	13.3	68.9	54.1	243.08***
38	55.6	51.7	16.2	17.6	28.1	30.7	10.96**
39	54.5	39.7	18.5	15.5	27.1	44.8	252.02***
40	70.7	69.7	12.5	11.5	16.7	18.8	6.37*
41	28.8	33.5	21.4	19.7	49.8	46.8	18.40***
42	69.7	64.2	12.2	14.2	18.1	21.6	24.40***
43	50.9	43.0	29.2	26.2	19.9	30.8	114.55***
44	70.6	79.2	12.1	9.7	17.3	11.1	71.33***
45	48.2	58.9	26.9	22.3	24.9	18.8	81.90***
46	61.9	62.6	12.5	13.4	25.6	24.0	2.98
47	7.7	11.7	29.1	31.0	63.2	57.3	42.39***
48	57.9	54.4	22.0	16.3	20.1	29.3	96.11***
49	20.3	31.6	28.0	26.0	51.7	42.4	126.43***
50	46.5	49.2	20.5	21.0	33.0	29.8	8.59*

is correct, then we would expect that all of the odd-numbered items should have substantial positive loadings, and the even-numbered items substantial negative loadings, on this first general factor.

In both males and females, with the exception of three items (items 11, 28 and 45), the attitude items do in fact load in the predicted direction (Table 6.5). However, 20 of the items have marginal loadings (less than 0.25). These items range from the comparatively trivial (attitudes to computer music, jazz and learning Latin) to extremely sensitive and topical issues (attitudes to apartheid, the death penalty and disarmament). This first principal component accounts for 12.8% and 13.0% of the variance in items in females and males respectively. While these percentages are slightly lower than those found in other studies of the C-Scale in England (18.7%), the Netherlands (15.3%) and New Zealand (14.0%) (see Wilson, 1973a), they are similar to the value of 12.6% found by Feather (1975) in a study of 575 Australian children and adults aged 14 years and over. Thus, while in general our results support the hypothesis of a general conservatism factor, more work is needed to determine the relative salience of particular items in the Australian population.

As previous research had indicated that it is possible to identify other broad attitude dimensions, independent from conservatism, which are of psychological siginificance, e.g. Eysenck's toughmindedness-tendermindedness factor, we decided to examine more extensively the factor structure of the C-Scale. We

Table 6.5 Factor pattern coefficients for the first principal component of the C-Scale.

Item	Females	Males	Item	Females	Males
1	.08	.19	26	-.06	-.12
2	-.43	-.43	27	.48	.48
3	.30	.41	28	.02	.04
4	-.47	-.46	29	.37	.33
5	.50	.53	30	-.16	-.12
6	-.52	-.49	31	.31	.30
7	.22	.24	32	-.10	-.07
8	-.34	-.29	33	.13	.14
9	.29	.27	34	-.63	-.58
10	-.33	-.34	35	.51	.53
11	-.18	-.15	36	-.22	-.23
12	-.24	-.22	37	.35	.45
13	.38	.43	38	-.25	-.20
14	-.15	-.13	39	.17	.25
15	.53	.58	40	-.31	-.28
16	-.31	-.32	41	.41	.42
17	.18	.14	42	-.16	-.12
18	-.30	-.28	43	.04	.04
19	.32	.36	44	-.44	-.41
20	-.38	-.34	45	-.11	-.03
21	.31	.32	46	-.45	-.45
22	-.45	-.42	47	.11	.10
23	.17	.18	48	-.21	-.19
24	-.38	-.36	49	.60	.61
25	.10	.20	50	-.47	-.45

chose to extract 8 orthogonal factors, using the method of principal factoring with iteration and varimax rotation, since a preliminary analysis had shown that only 8 factors could be easily interpreted. Orthogonal rotation was employed to simplify the interpretation of results. In the discussion to follow, for the purposes of interpretation a loading of 0.25 or greater was considered large. Also, the order of factors does not necessarily correspond to the sequence of extraction. The factor pattern coefficients of items defining the sub-factors of the C-Scale are shown in Table 6.6.

Factor 1 has high loadings on items concerning military strength, harsh punishment and strict rules and has therefore been labelled militarism-punitiveness. While several of the items have similar loadings in males and females, more strongly identified in males than females. One cannot compute the correlation between factor loadings in males and females as a test of factor similarity, and I know of no other available method of comparison. However, it appears that this factor is

Factor 2 has been labelled religion. It contrasts sabbath observance, divine law and church authority on the one hand, with evolution theory, legalized abortion and divorce on the other. While there is some similarity between the sexes, this factor is more pronounced in females than males.

A second religion factor was identified and this has been labelled religious fundamentalism (Factor 4). In particular, this factor is defined by items concerned with the role of women as agents of reproduction (birth control, legalized abortion, divorce) and as such seems to be characteristic of the more dogmatic beliefs associated with the Roman Catholic church and fundamentalist protestant sects.

Table 6.6 Factor pattern coefficients of items defining sub-factors of the C-Scale.

Factor 1: Militarism-punitiveness			
Item	Females	Item	Males
School uniforms	.35	Death penalty	.30
Sabbath observance	.25	Hippies	-.26
Patriotism	.52	Patriotism	.43
Military drill	.41	Military drill	.50
Socialism	-.37	Socialism	-.37
Moral training	.38	Moral training	.39
Licensing laws	.26	Licensing laws	.36
Royalty	.41	Royalty	.42
Conventional clothes	.25	Conventional clothes	.35
Censorship	.35	Censorship	.42
Inborn conscience	.27	Church authority	.25
		Caning	.35
		Strict rules	.41
		Bible truth	.28
Variance accounted for	5.2%		13.0%
Factor 2: Religion			
Item	Females	Item	Males
Evolution theory	-.37	Evolution theory	-.38
Sabbath observance	.52	Sabbath observance	.56
Divine law	.62	Divine law	.64
Legalised abortion	-.31	Legalised abortion	-.33
Chastity	.33	Chastity	.32
Church authority	.59	Church authority	.59
Bible truth	.71	Bible truth	.69
Divorce	-.25		
Variance accounted for	12.8%		5.7%

Table 6.6 cont'd.

Factor 3: Religious fundamentalism

Item	Females	Item	Males
Birth control	-.40	Birth control	-.45
Legalised abortion	-.64	Legalised abortion	-.57
Nudist camps	-.28	Nudist camps	-.25
Divorce	-.54	Divorce	-.46
Evolution theory	-.25		
Variance accounted for	2.7%		2.4%

Factor 4: Permissiveness

Item	Females	Item	Males
Striptease shows	.51	Striptease shows	.59
Hippies	.48	Hippies	.31
Student pranks	.32	Student pranks	.30
Nudist camps	.59	Nudist camps	.59
Pyjama parties	.49	Pyjama parties	.59
Casual living	.32	Chastity	-.36
Variance accounted for	5.1%		5.6%

Factor 5: Out-group hostility and anti-art

Item	Females	Item	Males
Hippies	-.31	Hippies	-.38
Modern art	-.52	Modern art	-.56
Computer music	-.30	Computer music	-.31
Jazz	-.39	Jazz	-.38
Learning Latin	-.35		
Working mothers	-.26		
Student pranks	-.25		
Coloured immigration	-.30		
Pyjama parties	-.29		
Variance accounted for	3.4%		2.8%

Table 6.6 cont'd.

Factor 6: Racialism-prejudice

Item	Females	Item	Males
White superiority	.52	White superiority	.65
Empire building	.36	Empire building	.31
Apartheid	.46	Apartheid	.49
Mixed marriage	-.35	Mixed marriage	-.41
Coloured immigration	-.46	Coloured immigration	-.58
Military drill	.25	Women judges	-.29
Variance accounted for	2.6%		3.5%

Factor 7: Corporal punishment

Item	Females	Item	Males
Death penalty	.34	Death penalty	.29
Caning	.49	Caning	.37
Strait jackets	.38	Strait jackets	.30
Strict rules	.37	Suicide	.26
Variance acounted for	2.3%		2.3%

Factor 8: Not interpreted

Item	Females	Item	Males
Student pranks	.35	Self-denial	.26
		Suicide	.26
Variance accounted for	2.3%		2.3%

Factor 5 suggests opposition to modern art forms. In females, this factor is also associated with hostility towards other groups and cultures (e.g. hippies) and has been labelled out-group hostility and anti-art.

Factor 6 is dominated by items concerning racial issues and is thus easily identified as racialism-prejudice. This factor is more strongly identified in males than females, and it is interesting that in males an item concerning the status of women (women judges) also loads on this factor.

Factor 7, although defined by only a few items, is obviously concerned with corporal punishment. Factor 8 is defined by only one item in females (student pranks) and two items in males (self-denial and suicide). This factor is not easily interpreted, and in view of the small number of items loading on it, may be just a statistical artefact.

These 8 principal factors account for 36.4% and 37.7% of the variance in items in females and males respectively, and the proportions of variance accounted for by each factor are shown in Table 6.6. Our factors are similar to those identified in previous studies of the C-Scale (e.g. Wilson, 1973a, Feather, 1975), and it is interesting that in none of these studies is there strong evidence for Eysenck's toughmindedness-tendemindedness factor. Wilson (1973a) has argued that this may be attributable to the format of the C-Scale. He notes that the toughmindedness-tendermindedness factor is the projection of extraversion-introversion onto the attitude field, while the content of the attitudes of these different personality types is

determined by their position on the radicalism-conservatism axis. Thus, he argues that radicalism-conservatism is a function of attitude content, while toughmindedness-tendermindedness is a function of item context. Since context effects were deliberately excluded in the format of the C-Scale, one would not necessarily expect this factor to emerge. While we have identified several other sub-factors of social attitudes, clearly conservatism is a general factor of almost overwhelming importance compared to second and subsequent factors.

6.3.3 Scaling

As a test of additivity of genetic and environmental effects, we regressed absolute within-pair differences in MZ twins on pair sums. The proportions of variance in absolute differences within pairs accounted for by variation in pair sums are shown below.

	MZ females	MZ males
Linear	.00	.01*
Quadratic (after the linear regression was removed)	.00	.00

Both linear and quadratic components are shown and it can be seen that in MZ twins only the linear regression in males is significant and even then it only accounts for a trivial proportion of the within-pairs variance.* Thus, this lack of regression in MZ pairs reflects the almost perfect normality of the

distribution of C-scores (Figure 6.1) and indicates that the scale has uniform discriminating properties across the range, at least to the level of second order effects.

6.3.4 Distribution of conservatism scores and sex differences

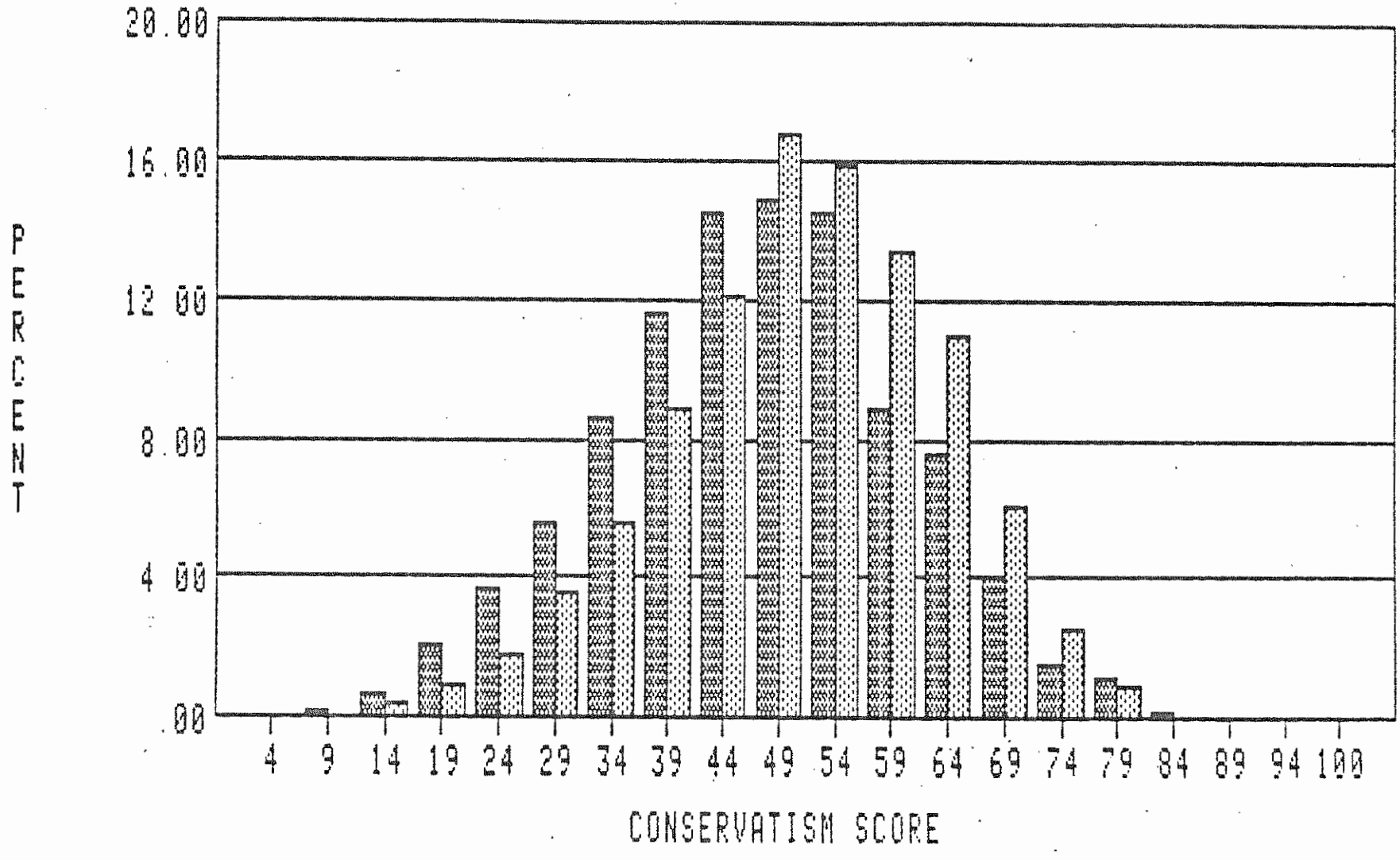
As a test of sampling, two-tailed t-tests were performed between MZ and DZ means and variance ratio tests between the total variances, separately for males and females (Table 6.7). There are no differences in means or variances between MZ and DZ male twins and the MZ and DZ female variances. The MZ female mean conservatism score is significantly greater than the DZ female mean but the difference is trivial and only significant because of the very large numbers available.

However, examination of differences between sexes shows that females ($\bar{X}$ = 49.00) are more conservative than males ($\bar{X}$ = 45.21) ($t_{5351.34}$ = 12.32, $p<.001$), which confirms previous results using the C-Scale in an Australian sample (Feather, 1977, 1978). Males also have a greater variance in C-scores than females ($F_{(2743,4874)}$ = 1.15, $P<0.001$).

6.3.5 Repeatability

Analysis of the C-Scores of the 96 individuals who completed both the pilot and the main questionnaire (Table 6.8) shows that they were typical of the total sample in distribution of C-scores except that the males have significantly lower C-scores than those of the total sample although the variances are equal.*

Analysis of variance of C-scores to obtain meansquares between and within individuals enabled calculation of the repeatability. These are very high for females (0.86) and males



DISTRIBUTION OF CONSERVATISM SCORES

Table 6.7 Means and variances of the twin sample for conservatism scores.

	Mean	Variance
MZ females	49.53**	148.61
MZ males	45.32	175.27
DZ females	49.23	151.45
DZ males	45.08	192.22
DZ opposite-sex	46.17	158.85

** Difference between MZ female and DZ female means significant at the 1% level.

Table 6.8 Distribution of conservatism scores from pilot study for individuals who also completed the main questionaire. Repeatability and within individual variance (S_W^2) are also shown.

	Females	Males
Mean	49.27	39.61
Variance	153.87	169.48
Repeatability	0.86	0.92
S_W^2	21.14	13.52

(0.92) and indicate that conservatism scores obtained from the C-Scale are very repeatable, at least over the short term. The within pair variance components ($S_W^2=MS_W$) are also shown in Table 6.8 and these are estimates of the portion of the total variance which is unrepeatable and will include measurement error. Although S_W^2 is higher in females than males, this difference is not significant.

6.3.6 Genetic and environmental analysis of variation in conservatism scores

Alternative models of variation were fitted to between- and within-pairs mean squares using the method of iterative weighted least squares (see section 3.3.6.1). As there was a significant difference between male and female means, we corrected for this by replacing the within-pairs mean square with the residual within-pairs mean square corrected for sex differences in means (section 3.3.6.1).

When a variable is age dependent, this inflates the between-pairs mean square. The linear age correlation with conservatism is 0.37 for males and 0.44 for females and both are highly significant. We corrected for this age dependence (section 3.3.6.1) and the ten mean squares and their degrees of freedom, corrected for sex differences and regression on age, are shown in Table 6.9.

We may also examine whether twins become less similar with age by correlating absolute pair differences with age and these are shown in Table 6.10. Correlations are small and non significant for the two MZ groups and for DZ females but are

Table 6.9 Observed mean squares for conservatism

Statistic		Degrees of freedom	Mean Square
MZ Female	Between[a]	1231	200.39
	Within	1233	43.66
MZ Male	Between[a]	564	250.49
	Within	566	62.44
DZ Female	Between[a]	749	175.62
	Within	751	64.25
DZ Male	Between[a]	350	238.67
	Within	352	85.06
DZ Opposite sex	Between[a]	904	179.92
	Within[b]	905	76.05

[a] Corrected for regression on age

[b] Corrected for sex differences in means

Table 6.10 Correlation of absolute differences in conservatism scores with age.

MZF	MZM	DZF	DZM	DZO
0.05	0.00	0.04	0.20***	0.12***

highly significant for DZ males and opposite sex pairs. This indicates that in males, genetic differences for conservatism become more pronounced with age, but no such effect is apparent in females. If environmental circumstances of co-twins become more different with age, these do not appear to produce greater differences in conservatism scores.

The results of fitting models of variation to conservatism scores are shown in Table 6.11. Both the E_1E_2 and E_1V_A model fail to adequately describe the data in either males or females but a model including all three sources of variation ($E_1E_2V_A$) gives excellent fits in both sexes. However, when this model is fitted to the combined male and female data it fails badly and the chi-square for heterogeneity of fit over sexes is highly significant ($\chi^2_3 = 60.66$, $p<.001$). Inspection of the parameter estimates reveals that there are larger $\hat{E}_1$ and $\hat{E}_2$ components for males than females but a similar estimate of V_A in both sexes.

Fitting separate E_1 and E_2 parameters for males and females (Table 6.12) causes a significant reduction in chi-square ($\chi^2_3 = 60.01$, $p<.001$). The correlation $R_{E_{2_{MF}}} = 0.90$ (the correlation between E_2 effects in males and females) is not significantly different from unity and indicates that the same E_2 effects which act in males also 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 indicating that in males genetic differences for conservatism become more pronounced with age.

As described in section 3.3.6.1 our estimate of E_2 can be better described by a parameter B which may be attributable to

Table 6.11 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 & 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 & Male & 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 6.12 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

$\chi^2_4 = 4.40$ ($p = .35$)

$h^2_{males} = 0.27 \pm .04$ $h^2_{females} = 0.35 \pm .05$

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 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

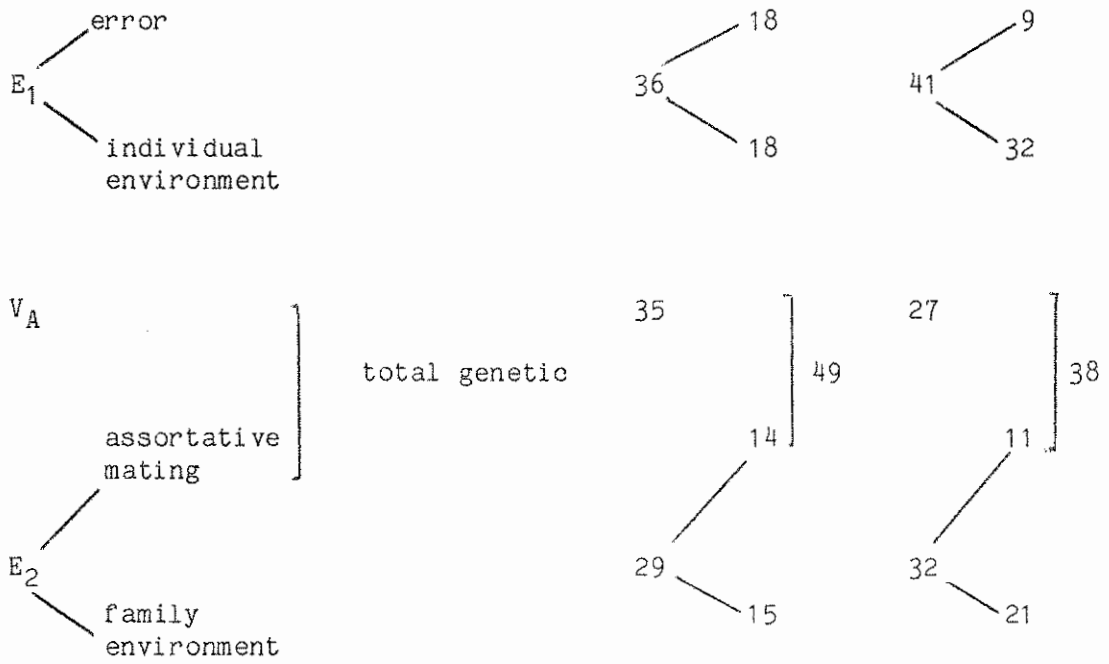
$$A = h^2\mu$$

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

(where $V_T = E_1 + B + V_A$) in 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 the value V_T, we obtain the breakdown of B into E_2 and AM as shown in Table 6.13. Thus approximately 38% of the variation in conservatism in males is genetic in origin and in females this rises to approximately 49%. Cultural influences and parental transmission account for about 21% and 14% of the variation in males and females respectively, the remaining variance due to non-

Table 6.13 Sources of variance (%) for age-corrected Conservatism scores.

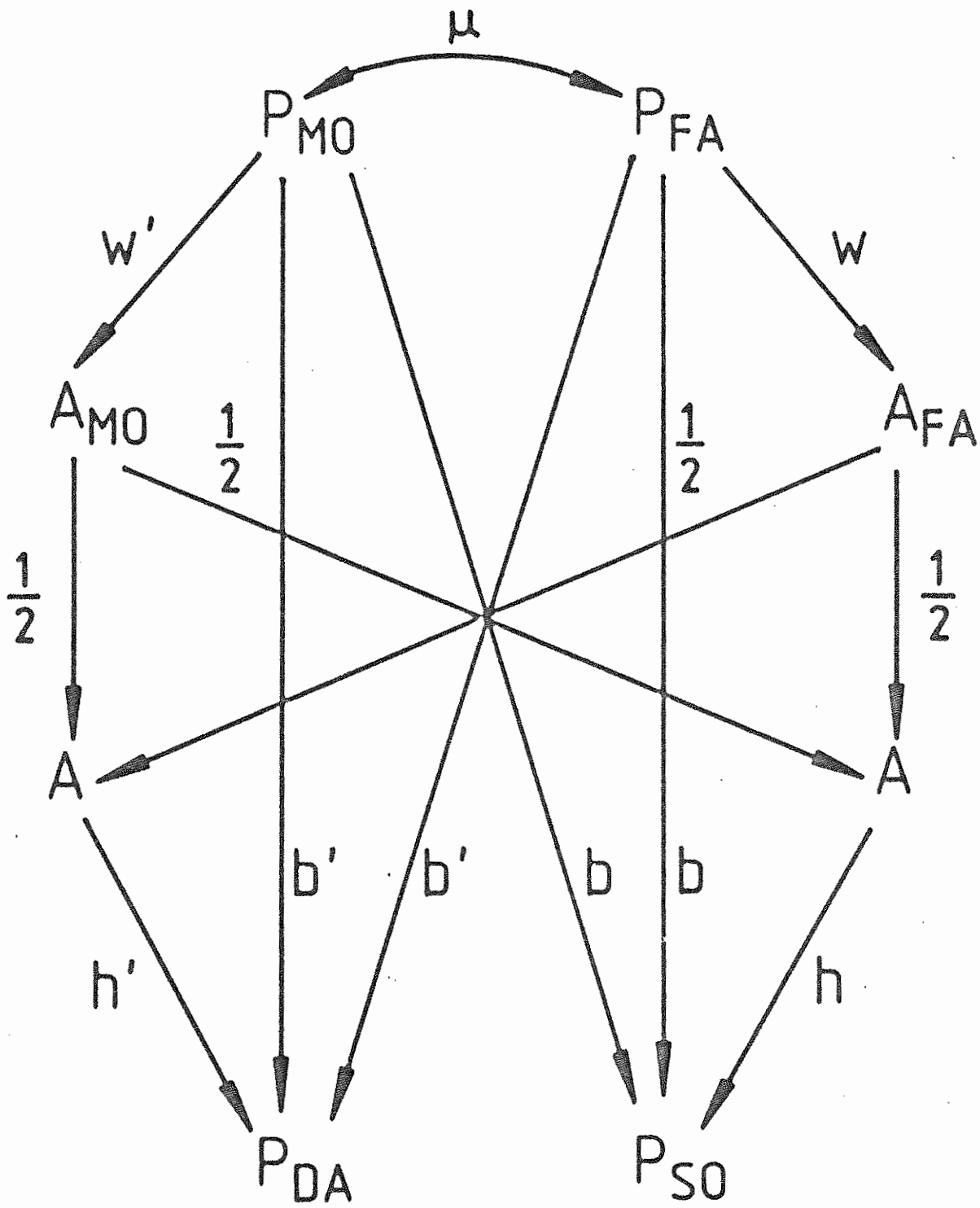


repeatable "error" and that due to repeatable individual environmental differences.

6.3.6.1 An alternative approach to the resolution of assortative mating and cultural transmission

In the previous section we demonstrated one method for partitioning the effects of assortative mating from cultural inheritance. However, such an approach does not enable us to estimate directly the importance of these effects. We now illustrate an alternative approach, based on path analysis of familial correlations, which enables us to obtain direct estimates of these parameters.

In Figure 6.2 we present a path diagram which represents genetic and cultural inheritance in nuclear families in the presence of phenotypic assortative mating. The model assumes transmission of additive genetic differences, A, and a direct cultural effect of parental phenotype, P, on the phenotype of offspring. The regression of phenotype on genotype is h in males, h' in females and the partial regression of offspring phenotype on parental phenotpye is b in males and b' in females. The parameter b, therefore, represents cultural inheritance in the model. The phenotypic correlation between mates is μ. Under the rules of path analysis, the paths from phenotype of parent to additive genetic effect (w and w') can be expressed as functions of h, h', b, b' and μ at equilibrium under cultural transmission and assortative mating. For readers unfamiliar with the technique of path analysis, a brief description follows.


μ
P_{MO}
P_{FA}
w'
w
A_{MO}
A_{FA}
$\frac{1}{2}$
$\frac{1}{2}$
$\frac{1}{2}$
$\frac{1}{2}$
A
A
b'
b'
b
b
h'
h
P_{DA}
P_{SO}

Basic to path analysis is the path diagram (e.g. Figure 6.2), in which every latent or measured variable is represented by paths which may be determined by either independent variables ('ultimate factors') or other variables which may in turn have a common causal basis. Two-headed paths are used to connect ultimate factors and indicate unanalysed correlations. That is, the cause of covariation is left ambiguous (causal or spurious) and no directionality is implied. A single-headed path from an ultimate (or dependent) variable to another dependent variable implies a direct causal influence, and thus indicates a regression on the dependent variable. It is assumed that all measured and latent variables have been standarised to zero mean and unit variance. On this model, the expectation for the correlation between two variables in terms of the path coefficients in the path diagram may be derived using the following rules (Wright, 1968):

i)Locate the two variables of interest and trace all possible rules between them. Note, however, that it is possible to reverse direction once, but only once; and that it is not possible, in a single route, to pass through a variable more than once.

ii)Calculate the contribution of each route traced as the product of the path coefficients along which the route was traced.

iii)Calculate the expected correlation between two variables as the sum of the contributions of all possible routes.

Thus, following these rules, the expectation of w in terms of h, b and μ (assuming equilibrium) can be calculated by tracing the following routes:

A to P_{SO},

A to A_{FA} to P_{FA} to P_{SO},

A to A_{FA} to P_{FA} to P_{MO} to P_{SO},

A to A_{MO} to P_{MO} to P_{SO} and

A to A_{MO} to P_{MO} to P_{FA} to P_{SO}.

These routes make contributions to the path between phenotype of parent to additive genetic effect, h, 1/2bw, 1/2μbw, 1/2bw' and 1/2μbw' and summing these gives

$$w = h + 1/2b(w + w')(1 + \mu).$$

Similarly, the rules of path analysis permit the expected correlations of twins and spouses to be calculated as shown in Table 6.14.

Having derived expected correlations, it is now possible to fit models to the observed correlations for conservatism (Table 6.15) using the method of non-linear weighted least squares (Rao et al., 1977). Estimation involves the selection of parameter estimates which minimise the function

$$C = (\underset{\sim}{z} - \bar{\underset{\sim}{z}})' \underset{\sim}{E}^{-1} (\underset{\sim}{z} - \bar{\underset{\sim}{z}})$$

where $\underset{\sim}{z}$ is the column vector of z-transformed observed correlations[2], $\bar{\underset{\sim}{z}}$ the corresponding vector of z-transformed

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[2] If we have an observed correlation r, then the z-transform is calculated as $z = 1/2[\log_e(1+r) - \log_e(1-r)]$ with standard error $1/\sqrt{(N - 3)}$, where N is the number of observations on which r was calculated.

Table 6.14 The expected correlations of twins and spouses in the presence of phenotypic assortative mating.

Relationship	Expected correlation
MZ Female	$h'^2 + 2b'^2(1+\mu) + b'h'(w+w')(1+\mu)$
MZ Male	$h^2 + 2b^2(1+\mu) + bh(w+w')(1+\mu)$
DZ Female	$1/2h'^2(1+\mu ww') + 2b'^2(1+\mu) + b'h'(w+w')(1+\mu)$
DZ Male	$1/2h^2(1+\mu ww') + 2b^2(1+\mu) + bh(w+w')(1+\mu)$
DZ Opposite-sex	$1/2hh'(1+\mu ww') + 2bb'(1+\mu) + 1/2(bh'+b'h)(w+w')(1+\mu)$
Spouses	μ

where $w = h + 1/2b(w+w')(1+\mu)$ and

$w' = h' + 1/2b'(w+w')(1+\mu)$.

Table 6.15 Age-corrected correlations for twins conservatism scores.

MZF	MZM	DZF	DZM	DZO
0.6429	0.6028	0.4642	0.4729	0.4064

expected correlations, $\underline{E}$ is diagonal with elements $E_{ii} = 1/(N_i - 3)$, and N_i is the number of observations from which the observed correlations were calculated. We have minimised C using a subroutine for constrained optimisation (E04UAF) from the Numerical Algorithms Group Library (1981). This procedure will yield maximum likelihood estimates of the parameters we choose to estimate. Since the $\underline{z}$'s are normally distributed and independent, the sum of weighted residuals (i.e the minimum value of C) is approximately chi-square for n-p degrees of freedom, where n is the number of observed correlations and p is the number of free parameters estimated in the model.

Models were fitted to the age-corrected correlations of the five twin groups conservatism scores (Table 6.15), with the marital correlation (μ) fixed at 0.675 (Feather, 1978). Because this correlation was based on relatively small numbers (n=103) and had not been age-corrected, we also repeated our analysis specifying a value of $\mu = 0.400$ which would be a reasonable lower bound of the marital correlation. The results of model-fitting are shown in Table 6.16.

The results for $\mu = 0.675$ show that models allowing only for cultural inheritance (models 1 and 2) give an extremely poor fit to the data. In contrast, a model allowing genetic inheritance, with the same genetic effects in males and females (model 3), gives an excellent fit to the data. No further reductions in chi-square were seen by either allowing different genetic effects in males and females (model 4), or including the effects of cultural inheritance (models 5 and 6). Although it could be argued that a marital correlation of 0.675 is extremely high, and

Table 6.16 Results of fitting models specifying genetic and cultural inheritance in the presence of phenotypic assortative mating to twin and spouse correlations for conservatism scores.

Model	h	h'	b	b'	μ	df	χ^2
1. bμ	-	-	.40	-	.675	4	73.38***
2. bb'μ	-	-	.38	.41	.675	3	68.68***
3. hμ	.79	-	-	-	.675	4	4.61
4. hh'μ	.78	.80	-	-	.675	3	2.84
5. hbμ	.81	-	-.02	-	.675	3	4.46
6. hh'bb'μ	.75	.83	.02	-.02	.675	1	2.70
1. bμ	-	-	.44	-	.400	4	73.38***
2. bb'μ	-	-	.42	.45	.400	3	68.68***
3. hμ	.80	-	-	-	.400	4	7.31
4. hh'μ	.79	.81	-	-	.400	3	6.04
5. hbμ	.71	-	.07	-	.400	3	4.46
6. hh'bb'μ	.67	.73	.09	.07	.400	1	2.71

that it might arise from convergence after marriage rather than being an initial correlation at the time of mate selection, our conservative estimate of $\mu = 0.400$ leaves the conclusions of model-fitting unaltered. Furthermore, in an earlier study, Martin (1978) found no correlation between absolute differences in radicalism scores of spouses and the number of years they had been married. Thus we have no evidence that resemblance between mates is due to convergence after marriage. Our results show that when we specify explicitly the effects of genetic and cultural inheritance, we have no evidence that family environment effects variation in the trait of conservatism.

6.3.7 Genetic and environmental analysis of variation in conservatism items

The twins' responses to the 50 C-Scale items were summarised by 3x3 contingency tables containing the number of twin pairs whose joint responses fell into each of the nine possible reponse categories. The set of 50 3x3 contingency tables is available from the author on request. Alternative models of variation were fitted to these contingency tables using the technique of threshold analysis (section 3.3.7.1), except that, having three response categories, there were two threshold values to be estimated. As the majority of items had shown significant differences in endorsement frequency between the sexes we estimated separate thresholds for males and females for all the models that were fitted. In the case of the five items not showing significant sex differences in endorsement frequency (Table 6.4), after deciding on the best fitting model, we fitted

that model again estimating a common threshold for males and females. In every case this resulted in the model with sex independent thresholds being accepted as most appropriate and this will be obvious in the table of model-fitting.

The likelihood ratio tests of specific models against the full model for conservatism items are shown in Table 6.17. Table 6.18 gives, for the best fitting model, the values of the genetic and environmental parameters estimated and the positions of the thresholds on the underlying distribution of liability.

For two items it was not possible to find an adequate description of the data. Of the remaining items, only 3 were best fit by a purely environmental model, while a simple genetic model is adequate for 21 of the items. The $E_1E_2V_A$ model provides the best fit for 15 items.

For the remaining 9 items it was necessary to fit models specifying sex-limited genetic and/or environmental effects. The $V_{A_M}V_{A_F}V_{A_{MF}}$ model is appropriate for one item (item 23). For this item ("Empire building") the correlation between additive genetic effects in males and females is 0.59, indicating that there are some differences in the genes acting in males and females. For items 3 and 25, the $E_{2_M}E_{2_F}E_{2_{MF}}V_{A_M}$ model is most appropriate. For items 3 ("School uniforms") and 25 ("Licensing laws") the correlations between family environmental effects in males and females ($r_{E_{2_{MF}}}$) are 0.61 and 0.07 respectively. This indicates that for item 3 there are some different, and for item 25 completely different, family environmental effects in males and females. For items 12 ("Birth control") and 48 ("Coloured

Table 6.17 Likelihood ratio test of specific models as compared to the full model for conservatism items.†

		Item											
Model	df	1	2	3	4	5	6	7	8	9	10	11	12
E_2	4	48.56***	24.82***	15.72**	26.78***	24.46***	18.62***	19.18***	30.40***	14.36**	23.08***	27.08***	17.12**
V_A	4	1.52	24.56***	11.22*	18.66***	9.58*	23.16***	7.90	13.60**	3.24	6.36	8.44	19.04***
E_2V_A	3	1.26	7.84*	7.36	7.50	0.58	2.94	5.88	10.52*	2.20	4.62	6.68	11.78**
$E_{2_M}E_{2_F}E_{2_{MF}}V_{A_M}$	1	–	–	0.18	–	–	–	–	–	–	–	–	–
$E_{2_M}E_{2_F}E_{2_{MF}}V_{A_F}$	1	–	–	–	–	–	–	–	–	–	–	–	0.14
$E_{2_M}E_{2_F}V_{A_M}V_{A_F}$	1	–	–	–	–	–	–	0.04	0.02	–	–	0.06	–
- log likelihood of full model		7387.42	7594.02	4092.47	7192.87	7781.48	7830.87	5496.80	7828.75	7994.93	7237.62	6900.73	2715.49

Model	df	13	14	15	16	17	18	19	20	21	22	23	24
E_2	4	25.56***	1.32	13.76**	16.62**	16.56**	17.04**	8.76	21.36***	28.82***	23.06***	36.46***	20.48***
V_A	4	3.76	8.00	24.02***	16.34**	4.54	1.14	6.86	18.72***	7.56	31.66***	7.46	14.20**
E_2V_A	3	0.78	1.12	3.74	4.10	3.70	0.74	2.18	13.78**	7.52	3.06	6.04	4.68
$V_{A_M}V_{A_F}V_{A_{MF}}$	2	–	–	–	–	–	–	–	–	–	–	0.50	–
- log likelihood of full model		6999.49	2524.24	7869.40	7857.26	4285.37	6858.23	4013.64	4688.22	7418.33	6453.76	7918.40	7513.77

Model	df	25	26	27	28	29	30	31	32	33	34	35	36
E_2	4	9.54*	25.12***	39.06***	17.74**	31.98***	7.96	28.76***	8.10	27.38***	18.62***	18.56***	25.94***
V_A	4	9.22	4.58	27.36***	2.52	6.74	6.36	6.64	2.74	2.48	30.32***	13.60**	1.52
E_2V_A	3	8.60*	1.46	21.32***	0.48	2.66	5.26	4.44	0.36	2.08	1.84	2.16	1.52
$E_{2_M}E_{2_F}E_{2_{MF}}V_{A_M}$	1	0.56	–	–	–	–	–	–	–	–	–	–	–
$E_{2_M}E_{2_F}E_{2_{MF}}V_{A_F}$	1	–	–	2.34	–	–	–	–	–	–	–	–	–
- log likelihood of full model		5002.13	8206.09	8050.61	7163.38	6412.57	2613.48	5964.67	6463.18	5867.25	7265.21	7863.00	7832.61

Table 6.17 cont'd.

		Item											
Model	df	37	38	39	40	41	42	43	44	45	46	47	48
E_2	4	21.64^{***}	17.62^{***}	12.02^{*}	18.00^{**}	16.14^{**}	27.72^{***}	4.16	13.42^{**}	10.52^{*}	26.16^{***}	26.30^{***}	23.88^{***}
V_A	4	0.70	1.08	11.84^{*}	7.76	3.38	2.66	7.12	9.82^{*}	1.98	6.00	8.58	18.76^{***}
E_2V_A	3	0.56	0.42	5.28	5.10	1.78	2.60	3.08	3.68	1.08	2.86	8.50^{*}	12.64^{*}
$E_{2_M}E_{2_F}E_{2_{MF}}V_{A_F}$	1	-	-	-	-	-	-	-	-	-	-	-	0.16
- log likelihood of full model		6607.31	7432.18	7531.24	6038.66	7797.76	6312.52	7906.57	5585.47	7687.74	6726.49	6616.11	7298.50

Model	df	49	50
E_2	4	19.98^{***}	3.60
V_A	4	42.42^{***}	58.94^{***}
E_2V_A	3	4.66	2.18
- log likelihood of full model		7588.14	7657.81

† Where twice the difference in log likelihood of the models under comparison has χ^2 distribution with degrees of freedom equal to the differences in the number of parameters in the two models. Values for the best fitting model are underlined.

Table 6.18 Parameter estimates and position of the thresholds for best fitting models for conservatism items.

Item	T_{1_M}	T_{1_F}	T_{2_M}	T_{2_F}	E_1	E_{1_M}	E_{1_F}	E_2	E_{2_M}	E_{2_F}	$E_{2_{MF}}$	V_A	V_{A_M}	V_{A_F}	$V_{A_{MF}}$
1	-0.521	-0.423	-0.242	0.076	0.491	-	-	-	-	-	-	0.509	-	-	-
3	-0.705	-1.397	-0.503	-1.206	-	0.519	0.485	-	0.288	0.515	0.236	-	0.193	-	-
4	-0.614	0.170	-0.193	0.746	0.462	-	-	0.209	-	-	-	0.329	-	-	-
5	-0.347	-0.615	0.248	0.000	0.472	-	-	0.178	-	-	-	0.350	-	-	-
6	-0.113	-0.127	0.419	0.562	0.465	-	-	0.261	-	-	-	0.274	-	-	-
7	-1.175	-1.318	-0.688	-0.648	-	0.115	0.588	-	0.366	0.248	-	-	0.519	0.164	-
8	-0.388	-0.514	0.152	0.042	-	0.626	0.514	-	0.039	0.257	-	-	0.335	0.229	-
9	-0.511	-0.640	0.129	0.068	0.642	-	-	-	-	-	-	0.358	-	-	-
10	-0.414	-0.509	-0.071	-0.138	0.540	-	-	-	-	-	-	0.460	-	-	-
11	0.580	0.052	1.001	0.490	-	0.627	0.504	-	0.030	0.195	-	-	0.343	0.301	-
12	-1.440	-1.681	-1.168	-1.427	-	0.515	0.338	-	0.485	0.465	0.272	-	-	0.197	-
13	-0.626	-0.898	-0.242	-0.256	0.475	-	-	-	-	-	-	0.525	-	-	-
14	-1.889		-1.392		0.612	-	-	0.388	-	-	-	-	-	-	-
15	-0.535	-0.865	0.264	0.191	0.524	-	-	0.257	-	-	-	0.219	-	-	-
16	-0.025	-0.124	0.565	0.698	0.533	-	-	0.210	-	-	-	0.257	-	-	-
17	0.897	0.981	1.241	1.471	0.499	-	-	-	-	-	-	0.501	-	-	-
18	0.173	0.406	0.877	1.082	0.599	-	-	-	-	-	-	0.401	-	-	-
19	-1.473	-1.743	-0.853	-1.079	0.519	-	-	0.194	-	-	-	0.287	-	-	-
21	0.155		0.712		0.599	-	-	-	-	-	-	0.401	-	-	-
22	-0.615		-0.309		0.351	-	-	0.324	-	-	-	0.325	-	-	-
23	0.009	-1.516	0.600	0.782	-	0.711	0.603	-	-	-	-	-	0.289	0.397	0.201
24	-0.252	0.060	0.151	0.567	0.506	-	-	0.192	-	-	-	0.302	-	-	-
25	-1.122	-1.502	-0.687	-0.856	-	0.687	0.855	-	0.135	0.145	0.041	-	0.178	-	-
26	-0.187	-0.264	0.430	0.555	0.740	-	-	-	-	-	-	0.260	-	-	-

Table 6.18 cont'd.

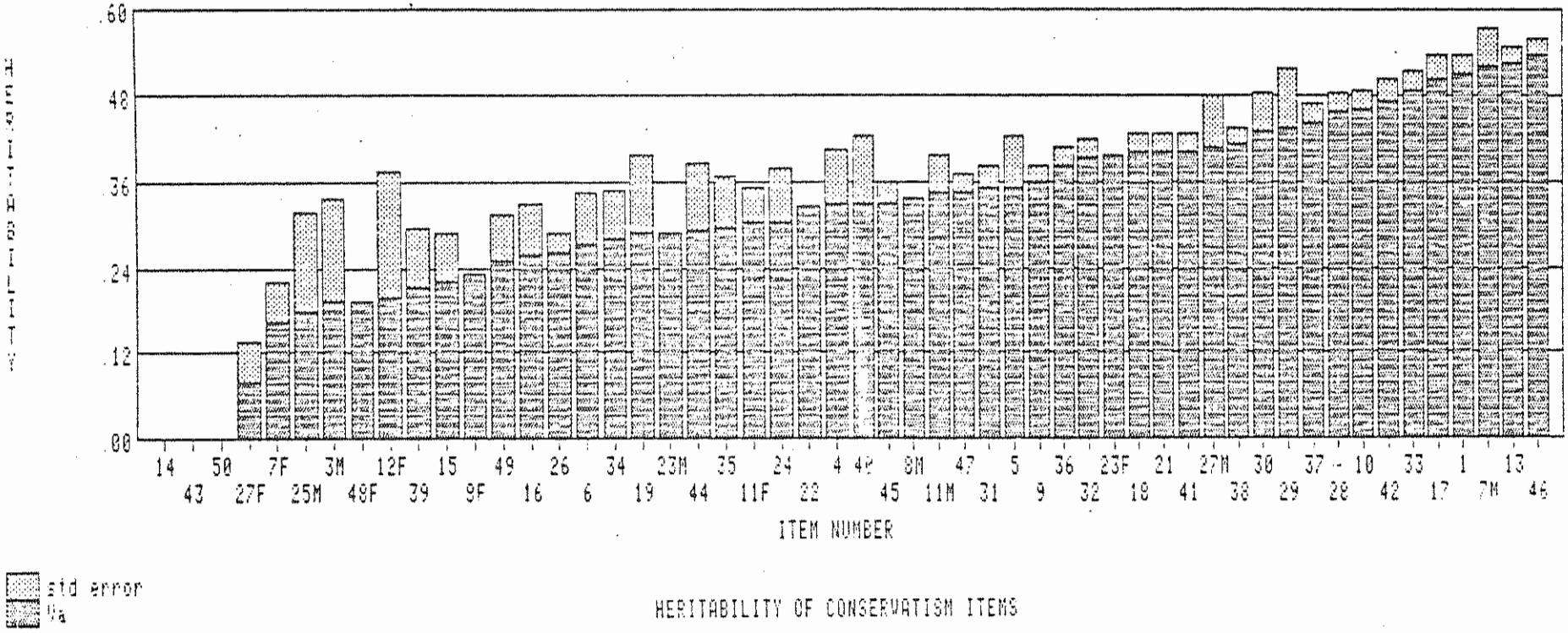
Item	T_{1_M}	T_{1_F}	T_{2_M}	T_{2_F}	E_1	E_{1_M}	E_{1_F}	E_2	E_{2_M}	E_{2_F}	$E_{2_{MF}}$	V_A	V_{A_M}	V_{A_F}	$V_{A_{MF}}$
27	-0.237	-0.444	0.383	0.234	-	0.572	0.521	-	0.022	0.398	-	-	0.406	0.081	-
28	-0.783		-0.235		0.544	-	-	-	-	-	-	0.456	-	-	-
29	-0.506	-0.940	-0.172	-0.505	0.429	-	-	0.135	-	-	-	0.436	-	-	-
30	-1.372	-1.978	-1.046	-1.589	0.568	-	-	-	-	-	-	0.432	-	-	-
31	-1.183	-1.070	-0.636	-0.542	0.650	-	-	-	-	-	-	0.350	-	-	-
32	-0.853	-0.676	-0.543	-0.325	0.608	-	-	-	-	-	-	0.392	-	-	-
33	0.586	0.402	1.401	1.556	0.514	-	-	-	-	-	-	0.486	-	-	-
34	-0.789	-0.252	-0.336	0.223	0.402	-	-	0.317	-	-	-	0.281	-	-	-
35	0.000	-0.225	0.548	0.399	0.503	-	-	0.203	-	-	-	0.294	-	-	-
36	-0.635	-0.714	-0.150	0.244	0.617	-	-	-	-	-	-	0.383	-	-	-
37	-0.451	-0.941	-0.105	-0.490	0.560	-	-	-	-	-	-	0.440	-	-	-
38	-0.501	-0.581	-0.042	-0.142	0.590	-	-	-	-	-	-	0.410	-	-	-
39	-0.260	0.112	0.133	0.611	0.617	-	-	0.170	-	-	-	0.213	-	-	-
40	-0.874	-0.970	-0.510	-0.547	0.548	-	-	0.122	-	-	-	0.330	-	-	-
41	-0.428	-0.557	0.075	0.007	0.599	-	-	-	-	-	-	0.401	-	-	-
42	-0.783	-0.916	-0.365	-0.520	0.527	-	-	-	-	-	-	0.473	-	-	-
43	-0.176	0.022	0.500	0.844	0.806	-	-	0.194	-	-	-	-	-	-	-
44	-1.225	-0.942	-0.820	-0.542	0.516	-	-	0.193	-	-	-	0.291	-	-	-
45	0.223	-0.044	0.882	0.680	0.670	-	-	-	-	-	-	0.330	-	-	-
46	-0.675		-0.310		0.466	-	-	-	-	-	-	0.534	-	-	-
47	-1.185	-1.426	-0.187	-0.339	0.656	-	-	-	-	-	-	0.334	-	-	-
48	-0.538	-0.844	-0.105	-0.205	-	0.549	0.570	-	0.451	0.235	0.192	-	-	0.195	-
49	-0.498	-0.819	0.166	-0.033	0.411	-	-	0.338	-	-	-	0.251	-	-	-
50	-0.514	-0.444	0.031	0.085	0.502	-	-	0.498	-	-	-	-	-	-	-

immigration") the $E_{2_M}E_{2_F}E_{2_{MF}}V_{A_F}$ model is most appropriate, and $r_{E_{2_{MF}}}$ is 0.57 and 0.59 respectively, indicating that there are some differences in family environmental effects in males and females. For the remaining 4 items the $E_{2_M}E_{2_F}V_{A_M}V_{A_F}$ model is most appropriate. While this model assumes that the same genetic and environmental effects act in males and females, for items 8 ("Modern art"), 11 ("Horoscopes") and 27 ("Chastity") V_A has a greater, and E_2 a smaller, effect on the variance in males than females. For item 7 ("Patriotism") both E_2 and V_A have a greater effect on the variance in males than females. Our analysis of the conservatism items shows that when additive genetic effects are present they account for between 8% and 53% of the variance, while when common family environment is present it accounts for 3% to 52% of the variance, the remaining variance due to the effects of individual environment and error.*

6.3.7.1 The relationship between items and factors

We conclude our analysis by comparing the results from our factor analysis and the genetic analysis of the trait of conservatism and the individual items.

We first examine whether there is any heterogeneity in the contribution of genetic factors to individual items. The heritabilities of the conservatism items, and their standard errors, are shown in Figure 6.3. As in our analysis of the EPQ items (section 3.3.7.6), as a test of heterogeneity we fitted a model that assumed that the heritabilities of the conservatism items were equal, using the method of iterative weighted least squares. The mean heritability was calculated as 0.40±0.005, and



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this model resulted in a highly significant heterogeneity chi-square (χ^2_{43} = 226.67, $p<.001$), indicating that there are significant differences in the proportion of variance attributable to genetic factors in the individual items.

As we argued in section 3.3.7.6, while some of this heterogeneity may reflect inter-item differences in reliability, it may also reflect the relationship between individual items and the factor of conservatism. Using the results from the first principal component solution (section 6.3.2), and recognising that there is evidence that both genetic and cultural factors are important to item variation, we correlated $\hat{E}_2$ and $\hat{V}_A$, estimated from the fit of the $E_1E_2V_A$ model, with the the factor loadings of items on the general factor of conservatism (Table 6.19).

The results are surprising indeed. In neither sex does the estimate of V_A for an item correlate with its loading on the general conservatism factor. However, in both sexes, there is a highly significant and positive correlation between the between-families environmental component of an item and its loading on the conservatism factor. That is, items which have the greatest loading on the general factor of conservatism display, on average, the greatest cultural component. This suggests that while genetic factors contribute to specific attitudes, cultural factors contribute to the correlations between these attitudes.

At first glance, these findings seem to contradict the results of our genetic analysis of the trait of conservatism. The composite score of conservatism includes information on the covariation between items, and yet we found no evidence for

Table 6.19 Correlations of E_2 and V_A with factor loadings from the first principal component solution of the C-Scale†.

	Females	Males
E_2	0.58^{***}	0.52^{***}
V_A	-0.08	-0.06

† E_2 and V_A estimated from the fit of the $E_1E_2V_A$ model

cultural inheritance for the trait of conservatism. However, as we have noted previously, the trait of conservatism shows remarkably high correlations between spouses. If such correlations reflect association between cultural and genetic determinants of attitudes rather than convergence in opinions after marriage, they will generate parental and sibling correlations in excess of those predicted under random mating. Thus, if the spousal correlation of attitudes is reflected at the item level, then assortative mating will simulate the effects of cultural resemblance between relatives even when vertical transmission is purely genetic. We suggest that it is in fact assortative mating that generates the significant covariation between attitude items, and that the correlation between mates predicts very well the increased similarity of DZ twins which we have attributed to cultural inheritance in our analysis of the individual items.

6.4 DISCUSSION

Our results support a view of the structure of social attitudes which stresses the importance of an underlying general factor of conservatism. However, while our results are similar to those of Feather (1975) in his study of conservatism in an Australian population, in both studies the factor of conservatism is not as clearly defined as has been found in other studies of the C-Scale in Western populations (see Wilson, 1973a). As we have noted, secular changes in attitudes demand frequent revision of test instruments. Certainly, a study of the salience of particular attitude items in the Australian population is needed.

While we have been able to identify sub-factors of social attitudes, the general factor of conservatism is considerably more important than these subsequent factors. Furthermore, there is some evidence that these factors differ between the sexes. While Eysenck (1954) found that radicalism-conservatism and toughmindedness-tendermindedness were of approximately equal importance in terms of the amount of variance in social attitudes that they accounted for, we found no evidence for a toughmindedness-tendermindedness component. If as Wilson (1973a) argues this is because context effects have been eliminated in the format of the C-Scale, it would be interesting to see if we could identify a toughmindedness-tendermindedness factor in the Australian population using a scale which does not eliminate context effects and purports to measure toughmindedness-tendermindedness (e.g. Eysenck's Public Opinion Inventory; Eysenck, 1954).

It has been argued 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 purely social model which includes only individual and family environmental effects is inadequate to explain variation in conservatism. In contrast to Eaves and Eysenck (1974) we also find that a simple individual environmental and additive genetic model is also inappropriate.

Our results, based on fitting models to mean squares, are similar to those of three independent twin studies (see Eaves et

al., 1978 for a summary) that measured conservatism by three different scales. In every case approximately equal proportions of variance were assigned to additive genetic effects, within-family environment effects and a between-family component (B), which may be due to cultural inheritance or assortative mating. When corrected for the effects of assortative mating, the results suggested that approximately half of the variance due to B was attributable to cultural inheritance. However, the method used provided no test of this aspect of the model.

In contrast to these earlier twin studies, we did fit models that estimated directly the effects of genetic and cultural inheritance in the presence of phenotypic assortative mating. We found no evidence for the importance of family environment. This result is similar to that obtained by Eaves et al. (1978) in their study of conservatism based 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, 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.

The final test of the validity of making generalisations from twin data about the causes of variation in the general population must be its ability to make predictions about other twin and non-twin relatives. We predict a zero correlation between foster-parent and adult foster-child for conservatism. Our model (Table 6.16, model 3; $h=0.79$, $\mu=0.675$) predicts a parent-offspring correlation of $1/2h^2(1+\mu)=0.52$, correlations of $1/4h^2(1+h^2\mu)^2=0.32$ for the offspring of monozygotic twins and $h^2=0.62$ for separated monozygotic twins. If our model of assortative mating is correct we predict that the spouses of sibs should show a correlation of $1/2h^2\mu^2(1+h^2\mu)=0.20$. The correlation between the spouse of one MZ twin and the co-twin is not expected to differ significantly from $h^2\mu=0.42$. All these data are potentially obtainable and can yield further tests of our model.*

Our analysis of the individual conservatism items indicates that 21 items show marked support for genetic inheritance, but not cultural transmission. For 15 items there is evidence of both genetic and social components of family resemblance, and for an additional 9 items there is strong indication of sex differences in these effects. Only 3 items show significant cultural transmission but no genetic transmission.

We found significant heterogeneity in conservatism items. However, there was a consistent trend for items with a strong cultural component to load more highly on the general factor of conservatism. It should be noted, however, that random mating was assumed in our analysis of the individual items. Positive

assortative mating will result in inflated estimates of the shared environmental effect in twin data and this will give false support for cultural transmission even when inheritance is purely genetic.

In fact, there is remarkable resemblance between spouses for conservatism (Feather, 1978). The correlation of 0.675 is amongst the highest marital correlations for any character, physical or behavioural (Spuhler, 1968; Vandenberg, 1972). It might be objected that such a high correlation arises from convergence of attitudes after marriage rather than being an initial marital correlation. We know of no direct evidence to support or contradict this view. However, Martin (1978) found no correlation between the absolute difference in radicalism scores of spouses and the number of years they had been married. Similarly, Eaves (personal communication) studying a sample of 301 spouse pairs in Virginia, USA with a 42 year range in the duration of marriage, found a correlation of -0.11 between duration of marriage and absolute spouse differences in radicalism, which does not support the argument that a spouse correlation of 0.675 is due to convergence. The apparent lack of divergence between the C-scores of MZ co-twins with age (Table 6.10) is also not what one would expect if attitudes tended to converge towards those of spouses, although a high correlation between spouses of MZ co-twins might vitiate this test.

The evidence so far suggests that it is unlikely that the resemblance between mates is simply due to convergence after marriage, although as Cavalli-Sforza et al. (1982) suggest, a longitudinal study of spouses would be necessary to determine if

there is any spousal interaction. Rather, it appears that assortative mating is primary, and that this generates a significant association between the causes of variation in the next generation. Thus we suggest that at both the item and trait level, the correlation between mates predicts very well the excess similarity of DZ twins for conservatism.

Although the high correlation between spouses for conservatism is well documented, the evolutionary significance of assortative mating needs to be assessed. Species are likely to invest resources in the selection of mates for traits that are especially adaptive. Certainly, the role of attitude concordance in mate selection and marital success needs further investigation.

CHAPTER 7 THE CAUSES OF VARIATION IN DRINKING BEHAVIOUR

7.1 INTRODUCTION

The existence of large variations between people in the use of alcohol has long been established and although a number of studies have investigated the contribution of genetic and environmental factors to variation in alcohol use, the results show little agreement.

Perry (1973) in a study of 84 pairs of twins found a substantial heritability of .56 for the amount of alcohol consumed per week. In contrast, Conterio and Chiarelli (1962) studying drinking behaviour in a sample of 77 male twins found that MZ twins were not significantly more alike than DZ twins with respect to a drinking/non-drinking dichotomy or a wine drinking/wine non-drinking dichotomy. Also, there was no difference between MZ and DZ concordance for quantity of wine consumed. While both these studies were based on small samples, larger studies have also failed to give conclusive results.

Partanen et al. (1966) studied drinking behaviour in 902 pairs of male twins aged between 28 and 37 years and derived three measures of alcohol consumption based on a factor analysis of drinking items. The first, Density, measured the frequency of alcohol consumption. The second, Amount, measured both the quantity of alcohol consumed and the duration of drinking during the last reported drinking occasion. The third, Lack of Control, measured the individual's control over his consumption. The results showed that genetic factors were an important determinant of a persons drinking behaviour, the heritability estimates for

Density and Amount being .39 and .36 respectively. Lack of Control had a lower heritability of .14. An interesting difference was however found in the comparison of younger (28-33) and older (34-37) pairs. The heritabilities were much higher for younger than older twins for both Density (.52 vs .31) and Lack of Control (.54 vs -.07). The heritability for Amount was .37 for both age groups.

Kaprio et al. (1981) in a study of 5044 male twins aged 18 and over found a heritability of .51 for alcohol use. However, when they analysed the data separately for 10 year age cohorts, the heritability was close to zero for those older than 60. These results and those of Partanen et al. (1966) suggest that age is an important factor which needs to be considered in genetic studies of drinking behaviour.

Different results on the causes of variation in drinking behaviour are also obtained when one considers males and females separately. Cederlof et al. (1977) studied alcohol consumption in 13000 pairs of twins and found higher coincidence rates in MZ and DZ twins for drinking and particularly excessive drinking, but that this higher coincidence was much more pronounced in females than males. Clifford et al. (1981) in a study of 399 twin pairs found that genetic influences seemed to be important in alcohol consumption in both males and females although for females there was evidence of a competition effect as well, where the consumption of one twin, whether high or low, influenced the other twin to drink in the opposite manner.

Overall, the results of these various studies suggest that there are genetic factors contributing to variation in alcohol consumption but their importance depends on the age and sex of the individual.

In this chapter we shall assess the relative importance of genetic and environmental factors in determining variation in drinking behaviour. We shall examine concordance with respect to a drinking/non-drinking dichotomy and the frequency of alcohol consumption, as well as determine the causes of variation in the amount of alcohol consumed (not alcoholism).

7.2 MEASURES OF DRINKING BEHAVIOUR

The items relating to alcohol use from the entire twin questionnaire are given in Appendix I. We have looked at several measures of alcohol consumption based on these items:

1. Drinking status - based on item 33 each twin was classified as either a drinker or non-drinker.

2. Frequency of alcohol consumption - based on items 33 and 35, the frequency of alcohol consumption over the previous year was recorded. Non-drinkers were also included and were given an additional code of "never", giving seven categories of consumption.

3. Normal weekly alcohol consumption - based on items 35-39 we have calculated an average of weekly alcohol consumption which reflects usual drinking behaviour.

4. Alcohol consumption last week - based on item 40 we have calculated the actual amount of alcohol consumed over the course of the previous week. These last two measures were calculated as the total number of standard drinks (beer, wine, spirits etc)

consumed. A comparison of the two measures allows us to assess the variability of consumption and/or response.

We shall first present our analysis of the categorical data concerning drinking status and the frequency of drinking. This will be followed by our analysis of the continuous measures of alcohol consumption.

7.3 Drinking status and the frequency of alcohol consumption

7.3.1 Distribution of responses and sex differences

As in the case of continuous data, before fitting models to explain trait variation, it is important to test whether the MZ and DZ groups are comparable. There were no significant differences between MZ and DZ endorsement frequencies for either the drinking/non-drinking dichotomy (χ^2_1 = 0.004 in males, χ^2_1 = 0.000 in females) or the frequency of alcohol consumption (χ^2_6 = 9.82 in males, χ^2_6 = 4.73 in females). Thus, the MZ and DZ classes were combined to examine the effect of sex on response frequency.

Table 7.1 presents the percentage of drinkers/non-drinkers and the frequency of alcohol consumption in the sample, broken down by sex. Chi-square tests (Table 7.1) show that there are more male drinkers than female drinkers, and that males drink more frequently than females. Thus the effect of sex on response frequency must be incorporated into our genetic analyses. *

7.3.2 Fitting models to data on drinking status

The twins' drinking status was summarised by 2x2 contingency tables containing the number of twin pairs whose joint responses fell into each of the four possible reponse categories (Table 7.2). ** Models were fitted to the set of contingency tables of the

Table 7.1. The percentage of drinkers/non-drinkers and the frequency of alcohol consumption in the sample, broken down by sex.

Drinking status

	Males	Females
Drinker	91	86
Non-drinker	9	14

$\chi^2_1 = 40.95^{***}$

Frequency of alcohol consumption

	Males	Females
Every day	13	8
3-4 times each week	20	10
About twice a week	17	12
About once a week	15	16
Once or twice a month	13	17
Less often	12	22
Never	9	14

$\chi^2_6 = 339.51^{***}$

Table 7.2. Contingency tables for drinking status.

MZF

		Twin 2	
		Drinker	Non-drinker
	Drinker	978	82
Twin 1			
	Non-drinker	69	104

MZM

		Twin 2	
		Drinker	Non-drinker
	Drinker	489	24
Twin 1			
	Non-drinker	17	37

DZF

		Twin 2	
		Drinker	Non-drinker
	Drinker	576	64
Twin 1			
	Non-drinker	67	44

DZM

		Twin 2	
		Drinker	Non-drinker
	Drinker	298	16
Twin 1			
	Non-drinker	20	18

DZO

		Male	
		Drinker	Non-drinker
	Drinker	745	39
Female			
	Non-drinker	93	30

five twin groups response to the item on drinking status using the threshold technique described in section 3.3.7.1 of this thesis. As there were significant sex differences in response to this item (Table 7.1), separate thresholds were estimated for males and females.

The result of fitting models to data on drinking status are shown in Table 7.3. The fit of alternative models was compared by both the likelihood ratio criterion and approximate chi-squares based on observed and expected cell frequencies (see section 3.3.7.1). By the likelihood ratio test, both the E_1E_2 and E_1V_A models are rejected against the full model, while the $E_1E_2V_A$ model is able to adequately describe the data. However, a model allowing scalar sex-limited E_2 and V_A effects (the $E_{1_M}E_{1_F}E_{2_M}E_{2_F}V_{A_M}V_{A_F}$ model) results in a significant improvement in chi-square over the $E_1E_2V_A$ model ($\chi^2_2 = 6.26$, $p<.05$). The same conclusions are reached if we compare alternative models by chi-squares calculated from observed and expected frequencies. Thus, by both criteria, we accept the $E_{1_M}E_{1_F}E_{2_M}E_{2_F}V_{A_M}V_{A_F}$ model as the best description of the data. The same additive genetic and between-family environmental effects are acting in males and females, although they have a different effect on the variance. Thus, approximately 26% of the variation in drinking status in males, and 55% in females, is due to the additive effects of genes, while 60% of the variance in males, and 26% in females, is attributable to common family environment, the remaining variance due to within-family environment and error.

Table 7.3 Results of fitting models to data on drinking status.

Model	T_M	T_F	E_2	E_{2_M}	E_{2_F}	V_A	V_{A_M}	V_{A_F}
E_2	1.334	1.058	0.694	-	-	-	-	-
V_A	1.343	1.062	-	-	-	0.819	-	-
E_2V_A	1.334	1.058	0.334	-	-	0.465	-	-
$E_{2_M}E_{2_F}V_{A_M}V_{A_F}$	1.333	1.061	-	0.603	0.221	-	0.255	0.554

	Likelihood ratios		Goodness of fit†	
Model	χ^2	df	χ^2	df
E_2	32.24^{***}	4	38.82^{***}	12
V_A	20.36^{***}	4	30.44^{**}	12
E_2V_A	6.82	3	14.53	11
$E_{2_M}E_{2_F}V_{A_M}V_{A_F}$	0.56	1	7.72	9

- log likelihood of full model 2607.95

† Based on observed and expected frequencies

7.3.2.1 Comparison of older and younger pairs

Since the results of previous studies (Kaprio et al., 1981; Partanen et al., 1966) had indicated that the relative contribution of genetic and environmental factors to variation in drinking behaviour might depend on the age of the individual, we decided to fit models to the data on drinking status separately for older and younger pairs. We divided the twin pairs into two groups, those 30 and under, and those over 30. The contingency tables are shown in Table 7.4.

The results of fitting models to data on drinking status are shown separately for younger (Table 7.5) and older (Table 7.6) pairs. For younger pairs, by the likelihood ratio test the E_1E_2, E_1V_A and $E_1E_2V_A$ models are all rejected against the full model. The scalar sex-limited $E_{1_M}E_{1_F}E_{2_M}E_{2_F}V_{A_M}V_{A_F}$ model, while not significantly different from the full model, results in a negative estimate of V_{A_M}. The $E_{1_M}E_{1_F}E_{2_M}E_{2_F}E_{2_{MF}}V_{A_F}$ model is able to adequately describe the data as well as yielding positive estimates of all parameters. This model is also the most appropriate if we consider the deviations from observed and expected frequencies, thus, we accept this as the final model. Approximately 89% of the variation in drinking status in males is due to the effects of common family environment, while in females only 54% of the variation is due to E_2. The correlation between E_2 effects in males and females ($r_{E_{2_{MF}}}$) is 0.63, indicating that there are some differences in common family environmental effects between the sexes. In females, additive genetic effects are also important, accounting for 34% of the variance.

Table 7.4. Contingency tables for drinking status, separately for younger and older pairs.

		Younger		Older	
MZF					
		Twin 2		Twin 2	
		Drinker	Non-drinker	Drinker	Non-drinker
	Drinker	491	24	487	58
Twin 1					
	Non-drinker	16	39	53	65
MZM					
		Twin 2		Twin 2	
		Drinker	Non-drinker	Drinker	Non-drinker
	Drinker	243	9	246	15
Twin 1					
	Non-drinker	6	16	11	21
DZF					
		Twin 2		Twin 2	
		Drinker	Non-drinker	Drinker	Non-drinker
	Drinker	301	17	275	47
Twin 1					
	Non-drinker	18	15	49	29
DZM					
		Twin 2		Twin 2	
		Drinker	Non-drinker	Drinker	Non-drinker
	Drinker	185	7	113	9
Twin 1					
	Non-drinker	4	10	16	8
DZO					
		Male		Male	
		Drinker	Non-drinker	Drinker	Non-drinker
	Drinker	443	22	302	17
Female					
	Non-drinker	36	9	57	21

Table 7.5 Results of fitting models to data on drinking status for younger pairs.

Model	T_M	T_F	E_2	E_{2_M}	E_{2_F}	$E_{2_{MF}}$	V_A	V_{A_M}	V_{A_F}
E_2	1.452	1.296	0.783	-	-	-	-	-	-
V_A	1.470	1.307	-	-	-	-	0.897	-	-
E_2V_A	1.460	1.298	0.431	-	-	-	0.451	-	-
$E_{2_M}E_{2_F}V_{A_M}V_{A_F}$	1.456	1.303	-	0.889	0.311	-	-	-0.011	0.572
$E_{2_M}E_{2_F}E_{2_{MF}}V_{A_F}$	1.456	1.302	-	0.892	0.539	0.443	-	-	0.337

	Likelihood ratios		Goodness of fit	
Model	χ^2	df	χ^2	df
E_2	25.02^{***}	4	28.66^{**}	12
V_A	21.04^{***}	4	29.16^{**}	12
E_2V_A	10.54^{*}	3	15.32	11
$E_{2_M}E_{2_F}V_{A_M}V_{A_F}$	2.06	1	7.76	9
$E_{2_M}E_{2_F}E_{2_{MF}}V_{A_F}$	0.02	1	5.86	9

- log likelihood of full model 993.58

Table 7.6 Results of fitting models to data on drinking status for older pairs.

Model	T_M	T_F	E_2	E_{2_M}	E_{2_F}	V_A	V_{A_M}	V_{A_F}	$V_{A_{MF}}$
E_2	1.223	0.877	0.616	-	-	-	-	-	-
V_A	1.220	0.879	-	-	-	0.748	-	-	-
E_2V_A	1.221	0.877	0.222	-	-	0.507	-	-	-
$V_{A_M}V_{A_F}V_{A_{MF}}$	1.215	0.880	-	-	-	-	0.840	0.698	1.000†
$E_{2_M}E_{2_F}V_{A_M}V_{A_F}$	1.215	0.880	-	0.281	0.188	-	0.555	0.501	-

Model	Likelihood ratios χ^2	df	Goodness of fit† χ^2	df
E_2	18.28**	4	25.78*	12
V_A	7.60	4	14.39	12
E_2V_A	4.50	3	11.33	11
$V_{A_M}V_{A_F}V_{A_{MF}}$	1.16	2	8.20	10
$E_{2_M}E_{2_F}E_{2_{MF}}V_{A_F}$	0.64	1	7.68	9

- log likelihood of full model 1562.62

† Parameter went to upper bound

For older pairs, the E_1E_2 model is rejected against the full model by the likelihood ratio test, while the E_1V_A model is able to adequately describe the data (Table 7.6). Either no further reductions in chi-square were seen with the addition of extra parameters or parameters went to upper bounds. By both the likelihood ratio test and the chi-square test of goodness of fit, the E_1V_A model is most appropriate. Approximately 75% of the variation in drinking status in older twins is due to the additive effects of genes, with the remaining variance due to within-family environment and error.

Our results thus show that there are marked differences in the causes of variation in drinking status when one considers different age cohorts, and further, that within age cohorts there may be differences in the causes of variation between the sexes. It is perhaps not surprising that our results contradict those of Conterio and Chiarelli (1962) who found no evidence for a genetic component to drinking status in a small sample of male twins who were heterogeneous in age.

7.3.3 Fitting models to data on the frequency of alcohol consumption

The set of 7x7 contingency tables of the five twin groups response to the item on the frequency of alcohol consumption are shown in Table 7.7. As in the previous section, alternative models were fitted to the set of contingency tables using the technique of threshold analysis, except that, having seven response categories, there were six threshold values to be

Table 7.7. Contingency tables for frequency of alcohol consumption.

MZF		Twin 2						
		1†	2	3	4	5	6	7
T	1	104	29	15	10	6	5	3
w	2	47	127	45	24	15	14	7
i	3	16	56	56	42	21	13	8
n	4	8	34	29	63	28	22	10
	5	5	18	22	29	42	18	8
1	6	3	13	12	16	25	34	26
	7	3	8	4	4	8	22	55

MZM		Twin 2						
		1	2	3	4	5	6	7
T	1	37	9	5	0	3	0	0
w	2	9	25	15	10	6	2	3
i	3	6	19	13	22	12	8	1
n	4	1	11	12	34	15	13	5
	5	2	4	12	16	24	21	4
1	6	2	8	6	16	22	50	7
	7	4	1	2	2	5	21	42

DZF		Twin 2						
		1	2	3	4	5	6	7
T	1	44	28	17	6	10	5	1
w	2	22	54	36	19	14	16	8
i	3	12	25	29	24	13	13	6
n	4	13	13	24	26	26	9	7
	5	7	6	13	21	17	13	10
1	6	7	14	12	14	11	16	11
	7	3	10	10	6	9	7	14

DZM		Twin 2						
		1	2	3	4	5	6	7
T	1	18	6	6	0	2	5	1
w	2	6	10	6	3	4	4	2
i	3	2	8	17	8	7	4	4
n	4	4	3	9	11	12	12	3
	5	1	9	5	11	21	13	3
1	6	1	2	8	8	18	23	12
	7	2	2	3	2	6	8	17

DZO		Male						
		1	2	3	4	5	6	7
F	1	30	31	13	12	12	15	10
e	2	13	29	24	30	36	34	23
m	3	9	19	33	27	35	38	20
a	4	10	8	19	24	31	34	19
l	5	5	13	14	23	25	27	14
e	6	2	3	6	10	21	22	24
	7	0	2	6	4	10	18	20

† Frequency code: 1 = Never, 2 = Less than once or twice a month, 3 = Once or twice a month, 4 = About once a week, 5 = About twice a week, 6 = 3-4 times each week, 7 = Every day

estimated. As there were significant sex differences in response to this item (Table 7.1), separate thresholds were estimated for males and females.

By the likelihood ratio criterion, the E_1E_2 (model I), E_1V_A (model II) and $E_1E_2V_A$ (model III) models are all rejected against the full model, while the $E_{1_M}E_{1_F}E_{2_M}V_{A_M}V_{A_F}V_{A_{MF}}$ model (model IV) is able to adequately describe the data (Table 7.8). However, when one examines the tests of goodness of fit, the deviations between observed and expected cell frequencies are highly significant. Although, as we have noted, the chi-square test is approximate when there are low expected cell frequencies (Olsson, 1979), it is unlikely that such highly significant residuals are due simply to our approximate test of significance. Thus, we shall now consider some of the possible explanations for these very large residuals.

7.3.3.1 Comparison of older and younger pairs

One possible source of model failure is that the causes of variation in the frequency of alcohol consumption are dependent on age, and thus fitting models to a heterogeneous age sample is inappropriate. Therefore, we again decided to divide the twin sample into two groups, those 30 and under, and those over 30. The contingency tables are shown in Table 7.9.

The results of fitting models to the data on the frequency of alcohol consumption are shown separately for younger (Table 7.10) and older (Table 7.11) pairs. For younger pairs, by the likelihood ratio test, the E_1E_2 (model I), E_1V_A (model II) and $E_1E_2V_A$ (model III) models are all rejected against the full

Table 7.8. Results of fitting models to data on frequency of alcohol consumption.

	Model			
Parameter	I	II	III	IV
T_{1_M}	-1.350	-1.360	-1.358	-1.334
T_{2_M}	-0.810	-0.816	-0.816	-0.802
T_{3_M}	-0.403	-0.408	-0.409	-0.401
T_{4_M}	-0.012	-0.014	-0.016	-0.014
T_{5_M}	0.426	0.432	0.426	0.421
T_{6_M}	1.110	1.130	1.120	1.105
T_{1_F}	-1.046	-1.050	-1.048	-1.055
T_{2_F}	-0.330	-0.334	-0.334	-0.336
T_{3_F}	0.103	0.094	0.098	0.100
T_{4_F}	0.508	0.505	0.501	0.508
T_{5_F}	0.894	0.891	0.887	0.897
T_{6_F}	1.394	1.393	1.387	1.401
E_2	0.523	-	0.143	-
E_{2_M}	-	-	-	0.383
E_{2_F}	-	-	-	0.066
V_A	-	0.660	0.506	-
V_{A_M}	-	-	-	0.305
V_{A_F}	-	-	-	0.566
Goodness of fit				
χ^2	652.9^{***}	741.4^{***}	669.7^{***}	777.4^{***}
df	227	227	226	224
Likelihood ratios				
χ^2	135.0^{***}	26.4^{***}	15.6^{**}	1.4
df	4	4	3	1
- log likelihood of full model		13948.7		

Table 7.9. Contingency tables for frequency of alcohol consumption, separately for younger and older pairs.

		Younger							Older						
MZF		Twin 2							Twin 2						
		1	2	3	4	5	6	7	1	2	3	4	5	6	7
T	1	39	7	3	4	1	0	1	65	22	13	6	5	5	2
w	2	13	58	26	15	5	3	1	34	69	19	9	10	11	6
i	3	6	28	43	26	14	2	1	10	28	13	16	7	11	7
n	4	2	17	21	45	22	14	0	6	17	8	18	6	8	10
	5	3	9	13	22	34	11	1	2	9	9	7	8	7	7
1	6	0	2	3	7	18	12	8	3	11	9	9	7	22	18
	7	0	0	0	1	3	4	2	3	8	4	3	5	18	53
MZM		Twin 2							Twin 2						
		1	2	3	4	5	6	7	1	2	3	4	5	6	7
T	1	16	2	3	0	1	0	0	21	7	2	0	2	0	0
w	2	2	13	6	6	1	1	0	7	12	9	4	5	1	3
i	3	3	10	7	14	6	2	0	3	9	6	8	6	6	1
n	4	1	5	9	25	10	10	0	0	6	3	9	5	3	5
	5	1	3	4	6	20	9	1	1	1	8	10	4	12	3
1	6	1	3	1	9	12	28	1	1	5	5	7	10	22	6
	7	1	0	0	1	0	6	14	3	1	2	1	5	15	28
DZF		Twin 2							Twin 2						
		1	2	3	4	5	6	7	1	2	3	4	5	6	7
T	1	15	5	4	2	6	1	0	29	23	13	4	4	4	1
w	2	5	30	21	12	8	5	0	17	24	15	7	6	11	8
i	3	2	14	17	19	6	6	1	10	11	12	5	7	7	5
n	4	5	6	16	23	20	6	2	8	7	8	3	6	3	5
	5	2	3	8	14	15	7	1	5	3	5	7	2	6	9
1	6	2	4	7	10	8	3	1	5	10	5	4	3	13	10
	7	1	2	1	1	2	0	2	2	8	9	5	7	7	12
DZM		Twin 2							Twin 2						
		1	2	3	4	5	6	7	1	2	3	4	5	6	7
T	1	10	0	3	0	0	1	0	8	6	3	0	2	4	1
w	2	2	5	5	1	2	3	0	4	5	1	2	2	1	2
i	3	1	4	11	6	5	3	1	1	4	6	2	2	1	3
n	4	1	1	8	9	10	8	1	3	2	1	2	2	4	2
	5	1	9	4	10	19	8	0	0	0	1	1	2	5	3
1	6	0	1	6	5	13	13	5	1	1	2	3	5	10	7
	7	2	1	0	0	1	4	3	0	1	3	2	5	4	14
DZO		Male							Male						
		1	2	3	4	5	6	7	1	2	3	4	5	6	7
F	1	9	14	4	9	2	6	1	21	17	9	3	10	9	9
e	2	5	16	19	17	25	17	6	8	13	5	13	11	17	17
m	3	7	11	26	20	29	23	6	2	8	7	7	6	15	14
a	4	7	5	13	17	26	27	7	3	3	6	7	5	7	12
l	5	3	7	9	18	19	20	3	2	6	5	5	6	7	11
e	6	0	1	2	6	10	15	10	2	2	4	4	11	7	14
	7	0	0	2	1	3	6	1	0	2	4	3	7	12	19

Table 7.10 Results of fitting models to data on the frequency of of alcohol consumption for younger pairs.

	Model			
Parameter	I	II	III	IV
T_{1_M}	-1.468	-1.478	-1.476	-1.454
T_{2_M}	-0.916	-0.926	-0.924	-0.907
T_{3_M}	-0.447	-0.456	-0.455	-0.446
T_{4_M}	0.039	0.036	0.036	0.035
T_{5_M}	0.593	0.597	0.596	0.586
T_{6_M}	1.515	1.541	1.537	1.518
T_{1_F}	-1.292	-1.294	-1.292	-1.304
T_{2_F}	-0.487	-0.489	-0.488	-0.495
T_{3_F}	0.053	0.049	0.049	0.047
T_{4_F}	0.614	0.608	0.608	0.611
T_{5_F}	1.224	1.219	1.218	1.227
T_{6_F}	1.971	1.967	1.964	1.979
E_2	0.513	-	0.054	-
E_{2_M}	-	-	-	0.294
E_{2_F}	-	-	-	0.013
V_A	-	0.685	0.626	-
V_{A_M}	-	-	-	0.429
V_{A_F}	-	-	-	0.647
Goodness of fit				
χ^2	699.3^{***}	2047.4^{***}	1843.2^{***}	2456.8^{***}
df	227	227	226	224
Likelihood ratios				
χ^2	103.2^{***}	10.0^{*}	9.2^{*}	0.1
df	4	4	3	1
- log likelihood of full model		6665.08		

Table 7.11 Results of fitting models to data on the frequency of of alcohol consumption for older pairs.

	Model			
Parameter	I	II	III	IV
T_{1_M}	-1.241	-1.243	-1.242	-1.224
T_{2_M}	-0.703	-0.703	-0.703	-0.693
T_{3_M}	-0.357	-0.357	-0.357	-0.352
T_{4_M}	-0.074	-0.073	-0.074	-0.074
T_{5_M}	0.248	0.252	0.250	0.244
T_{6_M}	0.805	0.817	0.812	0.796
T_{1_F}	-0.867	-0.869	-0.865	-0.874
T_{2_F}	-0.192	-0.196	-0.194	-0.197
T_{3_F}	0.153	0.149	0.150	0.150
T_{4_F}	0.418	0.414	0.414	0.417
T_{5_F}	0.659	0.654	0.654	0.659
T_{6_F}	1.105	1.102	1.100	1.109
E_2	0.529	-	0.155	-
E_{2_M}	-	-	-	0.337
E_{2_F}	-	-	-	0.082
V_A	-	0.660	0.494	-
V_{A_M}	-	-	-	0.358
V_{A_F}	-	-	-	0.547
Goodness of fit				
χ^2	293.1**	265.4*	253.2	272.4*
df	227	227	226	224
Likelihood ratios				
χ^2	59.5***	12.1*	6.1	0.8
df	4	4	3	1
- log likelihood of full model		6936.58		

model, while the $E_{1_M}E_{1_F}E_{2_M}E_{2_F}V_{A_M}V_{A_F}$ model (model IV) is able to adequately describe the data. However, the deviations between observed and expected cell frequencies are still highly significant, suggesting that age heterogeneity is not the only cause of model failure in younger twins.

In contrast, for older twins, although by both criteria the E_1E_2 and E_1V_A models (models I and II) fail to adequately describe the data, the $E_1E_2V_A$ model (model III) is neither rejected against the full model nor results in significant deviation between observed and expected frequencies. By neither test of significance is a model allowing scalar sex-limited E_2 and V_A effects considered more appropriate. Thus, for older twins, 49% of the variance in the frequency of alcohol consumption is due to the additive effects of genes, while approximately 16% is due to the effects of common family environment, the remaining variance due to environmental factors unique to the individual and error.

7.3.3.2 An alternative scaling approach

As we noted in the previous section, although for older twin pairs we were able to find an adequate description of the data, for younger twin pairs the deviations between observed and expected frequencies were highly significant. This suggests that, for younger twin pairs at least, there is a problem with our scale of measurement.

An assumption implicit in our threshold analysis of the frequency of alcohol consumption data is that, underlying our observed categories, there is a continuous and normal

distribution of liability to drinking behaviour. However, the ordering of responses may be different from that presupposed in the model or, indeed, any ordering of responses along a single dimension may be inappropriate. We shall now consider one approach to this problem, and attempt to determine if there is more than one dimension of twin similarity that determines the twins' responses. In particular, we shall attempt to determine whether the same dimension of variation which distinguishes light drinkers from heavy drinkers, also differentiates non-drinkers from drinkers. If this is not the case, this may explain the failure of the threshold model in younger twins.

As a first step, we decided to exclude twin pairs where either one or both of the twins was a non-drinker, and see what effect this had on the results of model-fitting. The results are shown separately for younger (Table 7.12) and older (Table 7.13) pairs.

For younger pairs, by the likelihood ratio test, both the E_1E_2 (model I) and $E_1E_2V_A$ (model III) models are rejected against the full model, while the E_1V_A model (model II) is able to adequately describe the data. No further reductions in chi-square were seen with the addition of extra parameters. However, while the deviations between observed and expected frequencies are less extreme than those including non-drinkers, they are still significant.

For older pairs, by both the likelihood ratio and goodness of fit test, the E_1E_2 model (model I) fails to adequately describe the data, while the E_1V_A model (model II) gives a good

Table 7.12 Results of fitting a model to data on the frequency of alcohol consumption for younger pairs, excluding non-drinkers.

	Model			
Parameter	I	II	III	IV
T_{1_M}	-1.259	-1.265	-1.266	-1.252
T_{2_M}	-0.656	-0.662	-0.663	-0.658
T_{3_M}	-0.102	-0.104	-0.104	-0.106
T_{4_M}	0.507	0.511	0.511	0.502
T_{5_M}	1.489	1.509	1.510	1.500
T_{1_F}	-0.743	-0.741	-0.741	-0.748
T_{2_F}	-0.099	-0.102	-0.102	-0.104
T_{3_F}	0.517	0.512	0.512	0.514
T_{4_F}	1.159	1.152	1.153	1.158
T_{5_F}	1.950	1.944	1.945	1.955
E_2	0.452	-	-0.018	-
E_{2_M}	-	-	-	-0.000
E_{2_F}	-	-	-	0.144
V_A	-	0.625	0.645	-
V_{A_M}	-	-	-	0.685
V_{A_F}	-	-	-	0.443
Goodness of fit				
χ^2	304.3^{***}	225.4^{**}	225.9^{***}	218.1^{**}
df	164	164	163	161
Likelihood ratios				
χ^2	74.3^{***}	8.1	8.1^{*}	2.3
df	4	4	3	1
- log likelihood of full model		5369.30		

Table 7.13 Results of fitting models to data on the frequency of of alcohol consumption for older pairs, excluding non-drinkers.

	Model			
Parameter	I	II	III	IV
T_{1_M}	-1.170	-1.171	-1.171	-1.161
T_{2_M}	-0.682	-0.686	-0.687	-0.680
T_{3_M}	-0.311	-0.316	-0.317	-0.312
T_{4_M}	0.061	0.056	0.056	0.057
T_{5_M}	0.683	0.684	0.685	0.681
T_{1_F}	-0.628	-0.631	-0.632	-0.636
T_{2_F}	-0.178	-0.182	-0.183	-0.185
T_{3_F}	0.141	0.135	0.134	0.134
T_{4_F}	0.416	0.411	0.410	0.411
T_{5_F}	0.911	0.907	0.907	0.910
E_2	0.419	-	-0.076	-
E_{2_M}	-	-	-	0.321
E_{2_F}	-	-	-	-0.083
V_A	-	0.557	0.640	-
V_{A_M}	-	-	-	0.261
V_{A_F}	-	-	-	0.633
Goodness of fit				
χ^2	200.7*	157.1	156.9	151.9
df	164	164	163	161
Likelihood ratios				
χ^2	49.6***	6.2	5.4	0.2
df	4	4	3	1
- log likelihood of full model		4851.07		

fit. By neither test of significance do models allowing additional parameters result in a significant improvement in fit over this model. Again, the deviations between observed and expected frequencies are less than those where non-drinkers are included.

Thus, for both younger and older pairs, the deviations between observed and expected frequencies are reduced when one excludes non-drinkers, giving some support to the notion that non-drinkers may not lie on the same continuum of drinking behaviour as drinkers. Although the residuals are still significant in younger twins, suggesting that this is not the only cause of model failure in this cohort, we decided to see what effect allowing a bi-dimensional liability to drinking behaviour had on the results of model fitting to the 7x7 contingency tables of drinking frequency. Our model is as follows.

Consider a 7x7 contingency table relating to frequency of alcohol consumption. The expected proportions of twin pairs in the table may be denoted by a square matrix $\underset{\sim}{A}$. The elements of $\underset{\sim}{A}$ may be calculated from the expected proportions of twins in the 2x2 contingency table of non-drinker vs drinker ($\underset{\sim}{B}$), and the 6x6 contingency table of frequency of alcohol consumption given that both twins are drinkers ($\underset{\sim}{C}$).

Thus, if both twins are non-drinkers

$$A_{11} = B_{11}.$$

In the case where twin 1 is a drinker, twin 2 a non-drinker

$$A_{1j} = B_{12} \cdot \sum_{m=1}^{m=6} C_{mn}$$

where $j=2,3,\ldots,7$ and $n=j-1$.

Similarly, where twin 2 is a drinker, twin 1 a non-drinker

$$A_{i1} = B_{21} \cdot \sum_{n=1}^{n=6} C_{mn}$$

where $i=2,3,\ldots,7$ and $m=i-1$.

Where both twins are drinkers

$$A_{ij} = B_{22} \cdot C_{mn}$$

where $i=2,3,\ldots,7$, $j=2,3,\ldots,7$, $m=i-1$ and $n=j-1$.

On this model, alternative models of variation may be fitted to the set of 7x7 contingency tables of twins' responses using the technique of threshold analysis. However, as shown above, expected proportions are now calculated in terms of the expected proportions of twins in the 2x2 contingency tables of non-drinkers vs drinkers, and the 6x6 contingency tables of frequency of alcohol consumption given that both twins are drinkers. It should be noted that an important assumption of the model is that the determinants of drinking status, and the determinants of frequency of alcohol consumption (in those who drink), are completely independent.

For younger twin pairs, the $E_{1_M}E_{1_F}E_{2_M}E_{2_F}E_{2_{MF}}V_{A_F}$ model was most appropriate for the 2x2 contingency tables of drinkers vs non-drinkers, while for the 6x6 contingency tables of frequency of alcohol consumption the E_1V_A model was most appropriate. For older twin pairs, the E_1V_A model was most appropriate for both the dichotomous (2x2) and polychotomous (6x6) contingency tables. The results of fitting these models to the set of 7x7 contingency tables of frequency of alcohol consumption are shown in Table 7.14, separately for younger and older pairs. In this table, two sets of thresholds and estimated sources of variance

Table 7.14 Results of fitting a bi-dimensional threshold model to data on the frequency of alcohol consumption, separately for younger and older twin pairs.

Parameter	Younger	Older
Dichotomous		
T_M	-1.356	-1.220
T_F	-1.359	-0.879
$E2_M$	0.819	-
$E2_F$	0.885	-
$E2_{MF}$	0.510	-
V_A	-	0.748
V_{A_F}	0.018	-
Polychotomous		
$T1_M$	-0.870	-1.036
$T2_M$	-0.278	-0.577
$T3_M$	0.297	-0.239
$T4_M$	0.906	0.129
$T5_M$	1.720	0.744
$T1_F$	-0.900	-0.544
$T2_F$	-0.282	-0.092
$T3_F$	0.269	0.224
$T4_F$	0.843	0.498
$T5_F$	1.757	0.990
V_A	0.642	0.565
Goodness of fit		
χ^2	568.07^{***}	323.41^{***}
df	223	226

are shown. The first (headed dichotomous in the table) gives the position of the threshold, and the parameter estimates, relating to variation in drinking status. The second (headed polychotomous in the table) gives the positions of the thresholds, and parameter estimates, relating to variation in the frequency of alcohol consumption.

Strictly, no likelihood ratio comparison of the unidimensional and bidimensional model is possible, since the latter model is not a special case of the former. However, for younger twin pairs the bidimensional model gives a substantially better fit than the previous best fitting model to the set of 7x7 contingency tables (the $E_{1_M}E_{1_F}E_{2_M}E_{2_F}V_{A_M}V_{A_F}$ model), although it still fails badly. For older twins, the bidimensional model is worse than any of the models previously fitted and also fails to adequately describe the data. Overall, these results suggest that while there may be some differences in the causes of variation in drinking status and, given that one is a drinker, how frequently one drinks, the causes of variation in these two measures are not entirely independent.

7.4 Amount of alcohol consumed

7.4.1 Scaling

As a test of genotype-environment interaction (Jinks and Fulker, 1970), absolute within-pair differences were regressed onto pair sums, for both normal weekly consumption and consumption last week. Both raw measures of alcohol consumption show significant and substantial linear regressions and these are considerably reduced by logarithmic transformation (Table

7.15). Although this results in an increase in the quadratic components further transformation [$\log_{10}(x+3)$ or $\log_{10}(\log_{10}(x+1))$] gave no greater improvement. Consequently, our genetical analysis was based on the log transformed measures of alcohol consumption.

7.4.2 Sampling

Means and variances of MZ and DZ twins were compared as a test of sampling. Table 7.16 lists the means and variances of the two alcohol measures for the five twin groups. Two-tailed t-tests and variance ratio tests were performed between MZ and DZ means and total variances, separately for males and females. For the raw scores, the variances of both consumption measures are significantly greater in MZ females than DZ females, and the variance of normal weekly consumption is significantly greater in MZ males than DZ males. However, these differences all become non-significant after transformation. Since the groups appear to be comparable, the MZ and DZ classes were combined in the examination of sex differences.

Table 7.17 presents the mean and variances of the consumption measures separately for males and females. Two-tailed t-tests and variance ratio tests were performed between male and female means and total variances (Table 7.17). The means of both consumption measures are significantly greater in males than females, which confirms previous results from surveys of alcohol consumption in Australian samples (Australian Bureau of Statistics, 1978; Reynolds et al., 1977). Males also have a greater variance in consumption than females. These results are

Table 7.15 Proportions of variance in absolute-within pair differences accounted for by regression on pair sums for raw and transformed measures of alcohol consumption. Linear (L) and quadratic components after the linear regression has been removed (Q) are shown.

	MZ Females		MZ Males	
	L	Q	L	Q
Normal weekly consumption				
Raw	.60***	.03***	.50***	.00
$\log_{10}(x+1)$	.13***	.20***	.02***	.22***
Consumption last week				
Raw	.41***	.09***	.45***	.01***
$\log_{10}(x+1)$	.04***	.23***	.00	.20***

Table 7.16 Means and variances of the twin sample for raw and transformed measures of alcohol consumption. Asterisks denote significant differences between MZ and DZ variances.

	Normal weekly consumption		Consumption last week	
	Raw	$\log_{10}(x+1)$	Raw	$\log_{10}(x+1)$
MZ Females				
Mean	5.12	0.453	5.20	0.501
Variance	137.47***	0.239	73.91**	0.239
MZ Males				
Mean	12.21	0.725	13.54	0.818
Variance	391.06*	0.361	324.92	0.358
DZ Females				
Mean	4.68	0.440	4.89	0.494
Variance	86.10	0.226	63.87	0.229
DZ Males				
Mean	12.48	0.763	13.92	0.837
Variance	337.04	0.354	321.20	0.357
DZ Opposite-sex				
Mean	9.58	0.624	10.43	0.697
Variance	316.61	0.331	275.22	0.331

Table 7.17 Means and variances of raw and transformed measures of alcohol consumption separately for males and females. Asterisks denote significant differences between male and female means and/or variances.

	Females		Males	
	Mean	Variance	Mean	Variance
Normal weekly consumption				
Raw	4.91***	115.75***	13.02	407.36
$\log_{10}(x+1)$	0.444***	0.232***	0.764	0.362
Consumption last week				
Raw	5.10***	68.94***	14.35	359.72
$\log_{10}(x+1)$	0.501***	0.234***	0.843	0.360

identical for both the raw and transformed scores. In both males and females the two measures of consumption correlate highly for both the raw scores (0.70 in females, 0.82 in males), and the transformed scores (0.83, 0.86), but normal weekly consumption tends to provide a lower estimate of alcohol consumption than consumption last week.

Since the purpose of a genetical analysis of twin data is to make inferences about the causes of variation in the population it is also important that twins are comparable with non-twin subjects. As a result of the Australian Bureau of Statistics, survey of drinking and smoking habits carried out in 1977 (ABS, 1978), information about the alcohol consumption patterns of Australian adults aged 18 years and over is available, although it is known that such survey estimates of consumption significantly underreport estimates of alcohol consumption based on sales statistics (Pernanen, 1974; Fitzgerald and Mulford, 1978; Popham and Schmidt, 1981). A comparison of the stated daily alcohol consumption of the twin respondents (calculated from consumption last week) with expected consumption based on ABS (1978) data showed that our male twins had similar drinking patterns to the general population (Table 7.18). However, our female twins showed a greater proportion of drinkers than the national average (Table 7.19). Also, our twin sample tended to be more moderate in their drinking behaviour than the general population, the average daily alcohol consumption of both male and female "drinkers" being less than the national average (Table 7.20), although these differences may reflect a change in

Table 7.18 A comparison of the average daily alcohol consumption of the male twin sample with expected numbers in each category calculated from Australian Bureau of Statistics (ABS) data.

Average daily alcohol consumption (gm alcohol)

18-24 year olds

	0	1-9	10-19	20-29	30-39	40-49	50-59	60-69	70-79	80+	
ABS	235	217	135	111	68	67	23	25	19	39	
Twins	240	213	169	113	59	40	22	31	13	39	$\chi^2_9 = 12.80$

25-44 year olds

	0	1-9	10-19	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99	100-109	110-119	120-149	150-199	200+	
ABS	252	292	203	175	93	74	62	33	23	13	14	7	9	10	5	3	
Twins	254	264	230	179	111	68	46	40	20	19	6	10	5	10	5	1	$\chi^2_{15} = 15.24$

45-64 year olds

	0	1-9	10-19	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99	100+	
ABS	107	91	56	48	30	23	18	14	6	5	3	10	
Twins	127	82	70	44	18	17	15	10	8	7	4	9	$\chi^2_{11} = 9.56$

65 years and over

	0	1-9	10-19	20-29	30-39	40-49	50-59	60+	
ABS	52	34	16	8	5	3	3	3	
Twins	50	29	12	8	9	7	1	8	$\chi^2_7 = 7.02$

Total

	0	1-9	10-19	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99	100-109	110-119	120-149	150-199	200+	
ABS	683	634	404	330	190	155	110	74	47	25	22	14	14	20	14	6	
Twins	671	588	481	344	197	132	84	83	43	37	18	18	11	22	10	3	$\chi^2_{15} = 20.32$

Table 7.19 A comparison of the average daily alcohol consumption of the female twin sample with expected numbers in each category calculated from Australian Bureau of Statistics (ABS) data.

Average daily alcohol consumption (gm alcohol)									
				18-24 years old					
	0	1-9	10-19	20-29	30-39	40+			
ABS	635	505	136	54	26	17			
Twins	519	489	208	82	41	32			$\chi^2_5 = 41.38^{***}$
				25-44 years old					
	0	1-9	10-19	20-29	30-39	40-49	50+		
ABS	1042	840	296	87	38	24	26		
Twins	818	918	358	147	55	20	36		$\chi^2_6 = 56.82^{***}$
				45-64 years old					
	0	1-9	10-19	20-29	30-39	40-49	50-59	60+	
ABS	503	265	88	39	24	7	6	5	
Twins	426	264	117	57	32	25	9	7	$\chi^2_7 = 27.64^{***}$
				60 years and over					
	0	1-9	10-19	20-29	30+				
ABS	148	48	9	4	3				
Twins	127	43	21	11	10				$\chi^2_4 = 13.66^{**}$
				Total					
	0	1-9	10-19	20-29	30-39	40-49	50-59	60+	
ABS	2484	1554	487	179	88	34	18	29	
Twins	1890	1714	704	297	132	68	31	36	$\chi^2_7 = 181.79^{***}$

Table 7.20 Average daily alcohol consumption (gm alcohol) of the twin sample and the ABS (1978) sample by age and sex and drinking status.

		Age group				
		18-24	25-44	45-64	65 and over	Total
Per female drinker	Twins	8.13	8.29	9.76	8.42	8.50
	ABS	9.36	10.04	11.79	10.79	10.46
Per female	Twins	7.25	7.38	7.54	5.32	7.28
	ABS	5.03	5.59	5.46	3.27	5.13
Per male drinker	Twins	21.23	23.81	22.35	21.06	22.61
	ABS	28.50	28.95	29.14	17.87	28.05
Per male	Twins	19.46	22.14	19.42	15.29	20.51
	ABS	21.36	23.22	21.59	10.25	21.06
Per drinker	Twins	13.55	13.88	13.92	13.50	13.77
	ABS	20.54	21.34	22.45	14.85	20.98
Per person	Twins	12.21	12.55	11.16	9.01	12.04
	ABS	13.23	14.54	13.51	6.16	12.98

drinking habits over the three years since the ABS (1978) data was collected (see Bungey and Winter, 1986)

7.4.3 Repeatability

Analysis of the alcohol consumption of the 96 individuals who completed both the pilot and main questionnaire (Table 7.21) shows that they were somewhat atypical in their consumption patterns. Both males and females have higher normal weekly consumption and lower consumption last week than the total sample. Also, for both sexes, the variance of the two alcohol measures is greater in the pilot sample than the total sample, and this is more pronounced for the measure of normal weekly consumption.

Separate analyses of variance of the alcohol consumption measures to obtain mean squares between and within individuals enabled calculation of repeatabilities. In females, for the raw scores, consumption last week is more reliable than normal weekly consumption, although this pattern is reversed when one considers the log transformed scores. In males, for both raw and transformed scores, consumption last week is more reliable than normal weekly consumption. With the exception of consumption last week in males, log transformation improves reliability and S_w^2, the proportion of variance which is unrepeatable, is higher in males than females. The reliabilities are also higher in males than females, except in the case of log transformed normal weekly consumption.

Table 7.21 Distribution of alcohol consumption measures from the pilot study for individuals who also completed the main questionnaire. Repeatability and within-individual variance (S_w^2) are also shown.

	Females				Males			
	Mean	Variance	Repeatability	S_w^2	Mean	Variance	Repeatability	S_w^2
Normal weekly consumption								
Raw	6.29	867.67	0.29	614.03	17.83	1535.37	0.40	930.43
$\log_{10}(x+1)$	0.403	0.231	0.84	0.038	0.833	0.375	0.65	0.133
Consumption last week								
Raw	4.95	83.79	0.49	43.19	13.77	399.36	0.89	44.98
$\log_{10}(x+1)$	0.464	0.235	0.55	0.107	0.872	0.282	0.81	0.053

7.4.4 Correlations with personality variables

The partial correlations, controlling for age, between the measures of alcohol consumption and the transformed personality and attitude scales described in previous chapters of this thesis are shown in Table 7.22. Individuals with higher levels of consumption tend to be more anxious, depressed, extraverted, psychotic and neurotic, and score lower on the lie and conservatism scales.* These results are similar to previous studies which have demonstrated a relationship between alcohol consumption and extraversion (Partanen et al., 1966; Langinvainio et al., 1981), anxiety (Partanen et al., 1966; Smart, 1968) and neuroticism (Partanen et al., 1966; Langinvainio et al, 1981).

7.4.5 Results of fitting models to data on the amount of alcohol consumed

Alternative models of trait variation were fitted to between- and within-pairs mean squares by iterative weighted least squares (see section 3.3.6.1).

The within-pairs mean square of DZ opposite-sex pairs was corrected for the significant sex differences in means that were found for both measures of consumption (Table 7.17).

Where a variable is strongly age dependent this inflates the between-pairs mean square. The linear age correlations with the log transformed measures of normal weekly consumption (-.06 in females, -.06 in males) and consumption last week (-.05, -.04) while significant in every case are not substantial, so we have not corrected for this age dependence. The mean squares for the consumption measures and their degrees of freedom, both corrected for sex differences, are shown in Table 7.23.

Table 7.22 Partial correlations, controlling for age, of raw and transformed measures of alcohol consumption with transformed personality and attitudes scales.

	Normal weekly consumption				Consumption last week			
	Raw		$\log_{10}(x+1)$		Raw		$\log_{10}(x+1)$	
	Females	Males	Females	Males	Females	Males	Females	Males
Anxiety	.04**	.07**	.05***	.07***	.06***	.05*	.06***	.06**
Depression	.04**	.07**	.04**	.04*	.07***	.06**	.05**	.04*
Extraversion	.14***	.15***	.20***	.21***	.16***	.16***	.19***	.20***
Psychoticism	.12***	.21***	.14***	.17***	.14***	.18***	.14***	.14***
Neuroticism	.03*	.09***	.05**	.09***	.05***	.08***	.05**	.08***
Lie	-.10***	-.14***	-.16***	-.16***	-.13***	-.14***	-.16***	-.16***
Conservatism	-.15***	-.12***	-.25***	-.19***	-.20***	-.13***	-.26***	-.19***

Table 7.23 Observed mean squares and their degrees of freedom, corrected for sex differences, for the measures of alcohol consumption.

		Normal weekly Consumption		Consumption last week	
		df	Mean square	df	Mean square
MZ Female	Between	1232	0.3742	1232	0.3710
	Within	1233	0.1032	1233	0.1075
MZ Male	Between	566	0.5722	566	0.5548
	Within	567	0.1510	567	0.1607
DZ Female	Between	750	0.2933	750	0.2905
	Within	751	0.1597	751	0.1688
DZ Male	Between	351	0.5141	351	0.5002
	Within	352	0.1948	352	0.2146
DZ Opposite-sex	Between	906	0.3578	906	0.3699
	Within	906	0.2318	906	0.2248

We may also examine whether twins become more or less similar with age by correlating absolute within-pair differences with age and these are shown in Table 7.24. The correlations are small and non-significant for both measures of consumption and indicate that if environmental circumstances of co-twins become more different as they get older, these do not appear to produce any greater differences in alcohol consumption.

The results of fitting models to mean squares for normal weekly consumption and consumption last week are shown in Tables 7.25 and 7.26 respectively. In every case a model (E_1) postulating that all variation was due to individual environmental experiences failed badly and is omitted from summary tables.

For both measures of consumption, in females the E_1V_A model is most appropriate, while in males the $E_1E_2V_A$ model provides a better fit. Inspection of the parameter estimates also reveals that there is a larger $\hat{E}_1$ component for males than females but a similar estimate of V_A in both sexes.

The results of fitting a model which specifies a common V_A parameter, an E_2 component in males and different E_1 effects in males and females, to both measures of consumption, is shown in Table 7.27. In both these measures about 55% of the variation in consumption between females and 36% between males is due to the additive effects of genes. In males, approximately 20% of the variation is due to environmental influences shared by brothers but there is no evidence of family environmental factors influencing alcohol consumption in females. We may also subtract

Table 7.24 Correlation of absolute within-pair differences in log transformed measures of alcohol consumption with age.

	Normal weekly consumption	Consumption last week
MZ Females	0.05	-0.05
MZ Males	0.04	0.03
DZ Females	0.06	0.02
DZ Males	0.04	0.06
DZ Opposite-sex	-0.01	-0.02

Table 7.25 Summary of model fitting for log transformed normal weekly consumption.

	$\hat{E}_1$	$\hat{E}_2$	$\hat{V}_A$	df	χ^2	h^2
Female						
E_1E_2	$.125^{***}$	$.110^{***}$	-	2	60.76^{***}	
E_1V_A	$.102^{***}$	-	$.131^{***}$	2	1.50	.56±.02
$E_1E_2V_A$	$.103^{***}$	.010	$.121^{***}$	1	1.09	
Male						
E_1E_2	$.168^{***}$	$.191^{***}$	-	2	8.62^{*}	
E_1V_A	$.143^{***}$	-	$.211^{***}$	2	11.56^{**}	
$E_1E_2V_A$	$.151^{***}$	$.118^{***}$	$.091^{**}$	1	0.07	.25±.09
Female & Male						
E_1E_2	$.138^{***}$	$.135^{***}$	-	6	171.51^{***}	
E_1V_A	$.115^{***}$	-	$.157^{***}$	6	135.65^{***}	
$E_1E_2V_A$	$.118^{***}$	$.045^{**}$	$.111^{***}$	5	120.80^{***}	
Female & Male & Opposite-sex						
E_1E_2	$.161^{***}$	$.118^{***}$	-	8	279.41^{***}	
E_1V_A	$.118^{***}$	-	$.160^{***}$	8	143.60^{***}	
$E_1E_2V_A$	$.119^{***}$	.001	$.159^{***}$	7	143.50^{***}	

Table 7.26 Summary of model fitting for log transformed consumption last week.

	$\hat{E}_1$	$\hat{E}_2$	$\hat{V}_A$	df	χ^2	h^2
Female						
E_1E_2	$.130^{***}$	$.105^{***}$	-	2	61.63^{***}	
E_1V_A	$.107^{***}$	-	$.128^{***}$	2	0.84	.55±.02
$E_1E_2V_A$	$.107^{***}$	.002	$.126^{***}$	1	0.82	
Male						
E_1E_2	$.181^{***}$	$.176^{***}$	-	2	10.74^{**}	
E_1V_A	$.155^{***}$	-	$.201^{***}$	2	6.52^{*}	
$E_1E_2V_A$	$.161^{***}$	$.089^{**}$	$.108^{**}$	1	0.00	.30±.10
Female & Male						
E_1E_2	$.146^{***}$	$.128^{***}$	-	6	175.95^{***}	
E_1V_A	$.122^{***}$	-	$.151^{***}$	6	127.97^{***}	
$E_1E_2V_A$	$.124^{***}$	$.030^{*}$	$.120^{***}$	5	120.57^{***}	
Female & Male & Opposite-sex						
E_1E_2	$.165^{***}$	$.115^{***}$	-	8	240.17^{***}	
E_1V_A	$.124^{***}$	-	$.155^{***}$	8	129.96^{***}	
$E_1E_2V_A$	$.125^{***}$	.004	$.151^{***}$	7	129.44^{***}	

Table 7.27 Parameter estimates (± s.e.) and heritabilities (h^2) from fit of models incorporating different components of variation for males and females.

	$\hat{V}_A$	$\hat{E}_{1_M}$	$\hat{E}_{1_F}$	$\hat{E}_{2_M}$
Normal weekly consumption	0.130 ± 0.006	0.146 ± 0.008	0.102 ± 0.004	0.084 ± 0.013
		$\chi^2_6 = 3.00$		
	$h^2_{female} = 0.56 \pm 0.02$		$h^2_{male} = 0.36 \pm 0.02$	
Consumption last week	0.129 ± 0.006	0.158 ± 0.008	0.106 ± 0.004	0.070 ± 0.013
		$\chi^2_6 = 1.91$		
	$h^2_{female} = 0.55 \pm 0.02$		$h^2_{male} = 0.36 \pm 0.02$	

the values of S_w^2, obtained from the repeatability data, from the estimates of E_1 and so estimate the proportion of total variance due to non-repeatable "error" and that due to repeatable individual environmental differences (Table 7.28).

7.4.5.1 Comparison of older and younger pairs

We decided again to fit models separately for older and younger pairs to see what effect this had on the results of model fitting. We divided the twin pairs into two groups, those 30 and under, and those over 30. The mean squares for the two measures of consumption, and their degrees of freedom, for older and younger pairs, are shown in Table 7.29, separately for males and females.

Just as before models were first fitted to the mean squares for males and females separately and then to all eight statistics together, models were fitted separately to those 30 and under and those over 30, and then to all eight statistics combined. The results of fitting these models to normal weekly consumption and consumption last week are shown in Tables 7.30 and 7.31 respectively.

Although for both measures of consumption in females, the E_1V_A model gives an excellent fit to the data in both younger and older twins, there is significant heterogeneity of fit over age groups for both normal weekly consumption ($\chi_2^2 = 31.49$; $P<.001$) and consumption last week ($\chi_2^2 = 6.79$, $P<.05$). While at least some of this heterogeneity is due to an increase in variance with age affecting $\hat{E}_1$ and $\hat{V}_A$ equally for normal weekly consumption, and $\hat{V}_A$ for consumption last week, it does suggest that the

Table 7.28 Sources of variance (%) for log transformed measures of alcohol consumption.

	Normal weekly consumption		Consumption last week	
	Females	Males	Females	Males
E_1 Error	44	41	45	44
Individual environment	16	37	45	15
	28	4	0	29
E_2	-	23	-	20
V_A	56	36	55	36

Table 7.29 Observed mean squares and their degrees of freedom for log transformed measures of alcohol consumption for older and younger pairs.

	Normal weekly consumption				Consumption last week			
	Females		Males		Females		Males	
	df	mean square	df	mean square	df	mean square	df	mean square
Younger								
MZ Between	570	0.3319	273	0.5946	570	0.3378	273	0.5742
Within	571	0.0867	274	0.1208	571	0.1102	274	0.1415
DZ Between	350	0.2582	205	0.4768	350	0.2724	205	0.4712
Within	351	0.1377	206	0.1941	351	0.1558	206	0.2051
Older								
MZ Between	661	0.4110	292	0.5514	661	0.3999	292	0.5369
Within	662	0.1175	293	0.1792	662	0.1052	293	0.1786
DZ Between	399	0.3237	145	0.5674	399	0.3027	145	0.5386
Within	400	0.1789	146	0.1958	400	0.1764	146	0.2280

Table 7.30 Summary of model fitting for log transformed normal weekly consumption for younger and older twins.

	$\hat{E}_1$	$\hat{E}_2$	$\hat{V}_A$	df	χ^2	h^2
Females						
Younger						
E_1E_2	$.106^{***}$	$.099^{***}$	-	2	35.53^{***}	
E_1V_A	$.085^{***}$	-	$.119^{***}$	2	0.78	.58±.03
$E_1E_2V_A$	$.086^{***}$	.009	$.109^{***}$	1	0.56	
Older						
E_1E_2	$.141^{***}$	$.119^{***}$	-	2	30.38^{***}	
E_1V_A	$.116^{***}$	-	$.143^{***}$	2	0.72	.55±.03
$E_1E_2V_A$	$.117^{***}$	.081	$.131^{***}$	1	0.53	
Younger & older						
E_1E_2	$.125^{***}$	$.110^{***}$	-	6	92.05^{***}	
E_1V_A	$.101^{***}$	-	$.131^{***}$	6	32.99^{***}	
$E_1E_2V_A$	$.102^{***}$	.010	$.121^{***}$	5	32.58^{***}	
Males						
Younger						
E_1E_2	$.152^{***}$	$.196^{***}$	-	2	16.39^{***}	
E_1V_A	$.116^{***}$	-	$.227^{***}$	2	3.13	.66±.03
$E_1E_2V_A$	$.120^{***}$	$.072^{*}$	$.155^{***}$	1	0.38	
Older						
E_1E_2	$.185^{***}$	$.186^{***}$	-	2	0.43	
E_1V_A	$.170^{***}$	-	$.198^{***}$	2	9.29^{**}	
$E_1E_2V_A$	$.180^{***}$	$.164^{***}$	.026	1	0.15	
Younger & older						
E_1E_2	$.168^{***}$	$.191^{***}$	-	6	18.81^{**}	
E_1V_A	$.143^{***}$	-	$.211^{***}$	6	25.41^{***}	
$E_1E_2V_A$	$.151^{***}$	$.118^{***}$	$.090^{**}$	5	12.44^{*}	

Table 7.31 Summary of model fitting for log transformed consumption last week for younger and older twins.

	$\hat{E}_1$	$\hat{E}_2$	$\hat{V}_A$	df	χ^2	h^2
Females						
Younger						
E_1E_2	$.128^{***}$	$.093^{***}$	-	2	18.65^{***}	
E_1V_A	$.108^{***}$	-	$.111^{***}$	2	0.66	.51±.03
$E_1E_2V_A$	$.109^{***}$	.012	$.098^{***}$	1	0.38	
Older						
E_1E_2	$.132^{***}$	$.116^{***}$	-	2	45.21^{***}	
E_1V_A	$.105^{***}$	-	$.142^{***}$	2	0.72	.58±.02
$E_1E_2V_A$	$.104^{***}$	-.009	$.151^{***}$	1	0.59	
Younger & older						
E_1E_2	$.130^{***}$	$.105^{***}$	-	6	70.78^{***}	
E_1V_A	$.107^{***}$	-	$.128^{***}$	6	8.17	
$E_1E_2V_A$	$.107^{***}$	.001	$.127^{***}$	5	8.17	
Males						
Younger						
E_1E_2	$.169^{***}$	$.181^{***}$	-	2	10.56^{**}	
E_1V_A	$.136^{***}$	-	$.210^{***}$	2	2.87	.61±.03
$E_1E_2V_A$	$.140^{***}$	$.072^{*}$	$.136^{**}$	1	0.30	
Older						
E_1E_2	$.195^{***}$	$.171^{***}$	-	2	3.13	
E_1V_A	$.174^{***}$	-	$.192^{***}$	2	4.08	
$E_1E_2V_A$	$.180^{***}$	$.102^{*}$	.085	1	0.40	
Younger & older						
E_1E_2	$.181^{***}$	$.176^{***}$	-	6	15.45^{*}	
E_1V_A	$.155^{***}$	-	$.200^{***}$	6	12.16	
$E_1E_2V_A$	$.161^{***}$	$.088^{**}$	$.108^{**}$	5	5.35	

relative contribution of genetic and environmental factors to total variation is dependent on age. The effect of age is even more striking in males. For both measures of consumption, the E_1V_A model gives a good fit to the data in younger twins, while for older twins the E_1E_2 model is most appropriate. The effect of E_1 also increases with age along with this decrease in V_A and increase in E_2.

7.4.5.2 A model for developmental change

In the previous section we saw that the genetic and environmental causes of variation in alcohol consumption are dependent on age and sex. In this section we shall attempt to model these age changes in the genetic and environmental effects on the phenotype, separately for each sex.

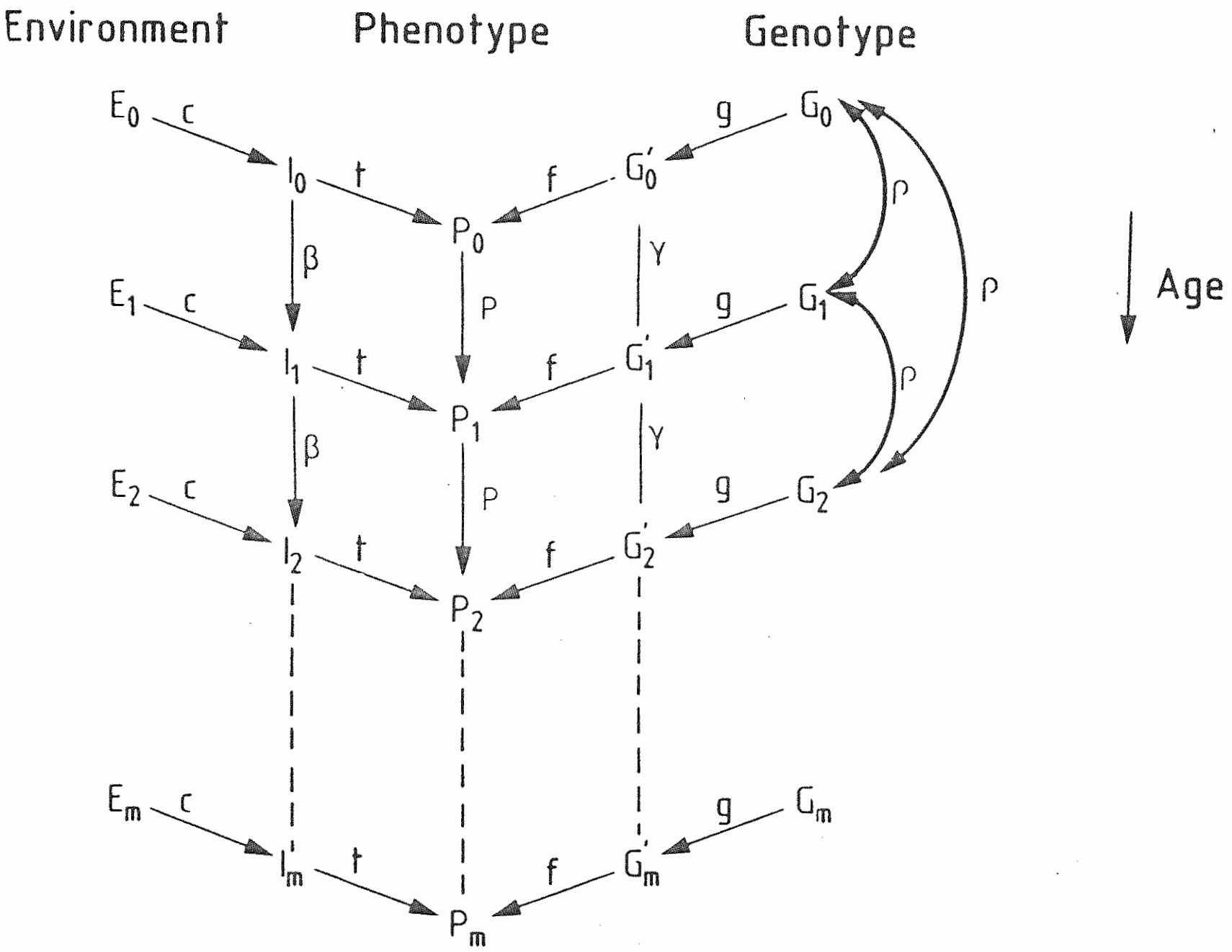
Several approaches have been suggested. As we have seen in the previous section, one can at the simplest level, fit different models of variation to separate age cohorts. Young et al. (1980) developed variance component models which allowed for the expression of entirely different genes at different ages. Eaves (Eaves et al., 1978, Eaves and Eysenck, 1980) used an empirical approach to describe changes in family resemblance with increasing age differences in terms of a negative exponential function. Each method suffers from the weakness that there is no attempt to make predictions for either different age groups and/or familial relationships, or provide a sound theoretical basis for the analysis of developmental change in gene expression.

Recently, Eaves (1985) has offered one theoretical model for development that attempts to overcome these criticisms. Figure 7.1 summarises the basic model which supposes that the phenotype develops from age 0 at m arbitrary intervals and has successive values of $P_0 \ldots . P_m$. At each stage, the phenotype is the product of both genetic (G) and environmental (E) influences. It is assumed that new environmental effects are continually influencing development. Figure 7.1 shows that if environmental effects are "remembered", there will be a path, β, from one occasion (I_{m-1}) to the next (I_m). Similarly, the persistence of genetic effects (G'), which may be seen as a gene product linking genotype to phenotype, may be measured by a path, γ. If at least some, or all, of the genes are responsible for the gene products on each occasion, there will be a correlation, $\rho_{m,n}$, between genetic effects on different occasions. Finally, the phenotype on one occasion (P_m) may be directly influenced by the phenotype on the previous occasion (P_{m-1}), and this may be measured by the path, p. The model assumes that the regression coefficients c, t, f, g, p, β and γ are constant throughout development.*

On this model, Eaves (1985) has derived the expected variances and covariances of family members as a function of age. Where environmental effects are unique to each occasion, the contribution of environmental factors to the phenotypic covariances of individuals measured on occasions m and n is:

$$E_{m,n} = \left(\frac{e}{p-\beta}\right)^2 \left[p^{2+m-n} \frac{1-p^{2(m+1)}}{1-p^2} - \left(p^{1+n-m}\beta + p\beta^{1+n-m}\right) \frac{1-(p\beta)^{m+1}}{1-p\beta} + \beta^{2+n-m} \frac{1-\beta^{2(m+1)}}{1-\beta^2}\right]$$

where e=ct.


Environment
Phenotype
Genotype
E_0
c
I_0
t
P_0
f
G'_0
g
G_0
ρ
β
γ
E_1
c
I_1
t
P_1
f
G'_1
g
G_1
ρ
ρ
Age
β
γ
E_2
c
I_2
t
P_2
f
G'_2
g
G_2
E_m
c
I'_m
t
P_m
f
G'_m
g
G_m

Similarly, where there is age specific gene action, the contribution of genetic factors to the covariances of individuals measured at occasions m and n is:

$$G_{m,n} = \left(\frac{h}{p-\gamma}\right)^2 \left[p^{2+m-n}\,\frac{1-p^{2(m+1)}}{1-p^2} - \left(p^{1+n-m}\beta+p\beta^{1+n-m}\right)\frac{1-(p\beta)^{m+1}}{1-p\beta}\right.$$

$$\left. + \gamma^{2+n-m}\,\frac{1-\gamma^{2(m+1)}}{1-\gamma^2}\right]$$

where $h=fg$.

When the same genes are expressed at every stage of development (i.e. $\rho=1$) we have:

$$G_{m,n} = \left(\frac{h}{p-\gamma}\right)^2 \left[\frac{p(1-p^{m+1})}{1-p} - \frac{\gamma(1-\gamma^{m+1})}{1-\gamma}\right]\left[\frac{p(1-p^{n+1})}{1-p} - \frac{\gamma(1-\gamma^{n+1})}{1-\gamma}\right]$$

An analogous expression holds for constant environmental effects. Estimates of the above parameters may be obtained through a pedigree analysis of the raw scores (Elston and Stewart, 1971; Lange et al., 1976). A description of the technique follows.

Lange et al. (1976) give a standard expression for the log likelihood of an observed pedigree assuming multivariate normality. For a given pedigree of n individuals, we define a vector of observed scores $\underset{\sim}{x}$, and a corresponding vector of expected scores $E\underset{\sim}{x}$. The $E\underset{\sim}{x}$ may be estimated as function of parameters representing effects such as age and sex. The expected covariance matrix of individuals in the pedigree is $\underset{\sim}{\Sigma}$. The $\underset{\sim}{\Sigma}$ may be estimated as a function of (co)variance components

representing genetic and environmental sources of variation. The elements of $\underset{\sim}{\Sigma}$ will depend on the relationship between members of the pedigree and on the causal model for the trait under study. For simplicity, we have given above the paths and the expectations for the covariation of environmental and genetic effects within individuals as a function of age. However, these can easily be adapted to describe changes in covariances between family members that vary with age. In terms of our usual notation (viz E_1, E_2 and V_A), the correlation between MZ and DZ twins for E_2 effects will be analagous in form to $E_{m,n}$. For additive genetic effects, the correlation between MZ twins will be analagous to $G_{m,n}$, for DZ twins $1/2G_{m,n}$. E_1 is not separately estimated but is calculated as a residual category. Thus in the case of the E_1V_A model, E_1 is calculated as

$$\hat{E}_1 = \sigma^2 - \hat{V}_A$$

where σ^2 is the estimated total variance.
A more detailed description of the notation used under our application of Eaves (1985) model will be given later.

To estimate the changes in these covariances as a function of age, we assume an age of onset for the developmental changes. In our case we assume an age of onset of 18 years, which corresponds to the age at occasion m in the model above. The age of the twins at the time of the study corresponds to the age at occasion n in the model. In this way, we are able to examine changes in the covariance between MZ and DZ twins as a function of increasing age.

For a given $\underset{\sim}{Ex}$ and $\underset{\sim}{\Sigma}$, the log likelihood of obtaining the pedigree of individuals with observed scores $\underset{\sim}{x}$ is

$$L = (-1/2)\ln|\Sigma| - 1/2(x-Ex)^{T}\Sigma^{-1}(x-Ex) + \text{constant}$$

The joint log likelihood of obtaining p pedigrees is the sum of the log likelihoods of the individual pedigrees. Estimation involves the selection of parameter estimates which maximise the joint likelihood of observing the given set of pedigrees. We have minimised -L using a subroutine for unconstrained optimisation (E04JAF) from the Numerical Algorithms Group Library (1981). A test of the goodness of fit of the model is cumbersome with this approach, but it is possible to compare alternative hypotheses about the observed variation using the likelihood ratio criterion suggested by Elston and Stewart (1971).

The results of fitting developmental models to data on alcohol consumption are shown separately for females' normal weekly consumption (Table 7.32) and consumption last week (Table 7.33), and males' normal weekly consumption (Table 7.34) and consumption last week (Table 7.35). Models were first fitted estimating means and genetic and environmental components of variance that were assumed to be constant over age (models I-III). By the likelihood ratio criterion, these models were compared to the fit of a full empirical model which estimated a common mean and variance, but separate correlations, for MZ and DZ twins. Having decided on the best fitting model, this model was then elaborated by allowing the mean to vary as a function of

Table 7.32 Summary of fitting models testing for developmental effects on the phenotype for log transformed normal weekly consumption in females.

Model	Mean	Parameter E_1	E_2	V_A	χ^2	df
I	.447	.124	.109	-	58.38***	1
II	.446	.102	-	.131	0.38	1
III	.446	.102	.010	.121	0.00	-
- log likelihood of full model					-1170.67	

Model	Mean: Constant	Mean: Linear age regression	Mean: Quadratic age regression	Parameter σ^2	E_1	$P(E_1)$	$\rho(E_1)$	V_A	$P(V_A)$	$\rho(V_A)$	Improvement[a] χ^2	df
IV	.486	-.001	-	.233	.102	-	-	.131	-	-	3.24	1
V	.340	.007	-.0001	.232	.102	-	-	.130	-	-	9.08*	2
VI	.340†	.007†	-.0001†	.148	.021	.902	.000†	.127	.134	.000†	30.36***	1
VII	.340†	.007†	-.0001†	.151	.022	.902	.000†	.129	.001††	1.00†	30.36***	1

[a] Improvement over model II

† Value of parameter is fixed

†† Parameter went to upper/lower bound

Table 7.33 Summary of fitting models testing for developmental effects on the phenotype for log transformed consumption last week in females.

Model	Mean	Parameter E_1	E_2	V_A	χ^2	df
I	.499	.130	.105	-	59.54^{***}	1
II	.498	.107	-	.128	0.00	1
III	.498	.107	.001	.127	0.00	-
	- log likelihood of full model				-1138.92	

Model	Mean: Constant	Linear age regression	Quadratic age regression	σ^2	Parameter E_1	$P(E_1)$	$\rho(E_1)$	V_A	$P(V_A)$	$\rho(V_A)$	Improvement[a] χ^2	df
IV	.570	-.002	-	.235	.108	-	-	.127	-	-	10.24^{**}	1
V	.394	.008	-.0001	.233	.107	-	-	.126	-	-	18.72^{***}	2
VI	$.394^{\dagger}$	$.008^{\dagger}$	$-.0001^{\dagger}$	.175	.049	.744	$.000^{\dagger}$	.126	$.000^{\dagger\dagger}$	$.000^{\dagger}$	20.55^{***}	1
VII	$.394^{\dagger}$	$.008^{\dagger}$	$-.0001^{\dagger}$	.175	.049	.744	$.000^{\dagger}$	.126	$.001^{\dagger\dagger}$	$1.00^{\dagger}$	20.55^{***}	1

[a] Improvement over model II

† Value of parameter is fixed

†† Parameter went to upper/lower bound

Table 7.34 Summary of fitting models testing for developmental effects on the phenotype for log transformed normal weekly consumption in males.

Model	Parameter Mean	E_1	E_2	V_A	χ^2	df
I	.739	.168	.191	-	8.26**	1
II	.740	.144	-	.211	12.74***	1
III	.740	.151	.117	.090	0.00	-
	- log likelihood of full model				-180.53	

Model	Mean: Constant	Linear age regression	Quadratic age regression	σ^2	E_1	$P(E_1)$	$\rho(E_1)$	E_2	$P(E_2)$	$\rho(E_2)$	V_A	$P(V_A)$	$\rho(V_A)$	Improvement[a] χ^2	df
	Parameter														
IV	.837	-.003	-	.357	.150	-	-	.116	-	-	.091	-	-	6.76*	1
V	.533	.014	-.0002	.355	.151	-	-	.114	-	-	.090	-	-	14.04**	2
VI	.533†	.014†	-.0002†	.324	.024	.924	.000†	.010	.999††	.000†	.287	.945	.000†	b	2
VII	.533†	.014†	-.0002†	.300	.026	.913	.000†	.0003	.995††	1.00†	.271††	.976	.000†	b	2

[a] Improvement over model III

[b] Model was worse than model III

† Value of parameter is fixed

†† Parameter went to upper/lower bound

Table 7.35 Summary of fitting models testing for developmental effects on the phenotype for log transformed consumption last week in males.

Model	Mean	Parameter E_1	E_2	V_A	χ^2	df
I	.824	.181	.176	-	10.32^{**}	1
II	.825	.155	-	.200	6.90^{**}	1
III	.825	.161	.089	.108	0.00	-
	- log likelihood of full model				-159.24	

Model	Mean: Constant	Linear age regression	Quadratic age regression	σ^2	Parameter E_1	$P(E_1)$	$\rho(E_1)$	E_2	$P(E_2)$	$\rho(E_2)$	V_A	$P(V_A)$	$\rho(V_A)$	Improvement[a] χ^2	df
IV	.940	-.003	-	.357	.160	-	-	.087	-	-	.110	-	-	10.78^{**}	1
V	.579	.017	-.0002	.352	.160	-	-	.084	-	-	.108	-	-	21.44^{***}	2
VI	$.579^{\dagger}$	$.017^{\dagger}$	$-.0002^{\dagger}$	.352	.160	$.000^{\dagger\dagger}$	$.000^{\dagger}$	.084	$.001^{\dagger\dagger}$	$.000^{\dagger}$	.108	$1.00^{\dagger\dagger}$	$.000^{\dagger}$	21.44^{***}	2
VII	$.579^{\dagger}$	$.017^{\dagger}$	$-.0002^{\dagger}$	.385	.179	$.000^{\dagger\dagger}$	$.000^{\dagger}$	.001	$.800^{\dagger\dagger}$	$1.00^{\dagger}$	.205	.925	$.000^{\dagger}$	12.22^{**}	2

[a] Improvement over model III

† Value of parameter is fixed

†† Parameter went to upper/lower bound

age (models IV and V), and finally by allowing developmental changes in the genetic and environmental effects on the phenotype (models VI and VII). We shall discuss in detail the results of fitting models to data on normal weekly consumption in females to illustrate the technique.

By the likelihood ratio criterion, the E_1E_2 model fails badly, while the E_1V_A model gives a good fit (Table 7.32). The $E_1E_2V_A$ model has the same number of parameters as the full model, and hence yields an identical minus log likelihood. Only if both simple environmental and genetic models fail will we consider this model as most appropriate. Thus, for normal weekly consumption in females, we regard the E_1V_A model as an adequate description of the data, and the fit of subsequent models was compared against the minus log likelihood of this model. Allowing the mean to vary as a linear function of age (model IV) results in improvement over the E_1V_A model ($\chi^2_1 = 3.24$, $.05<p<.10$), which is further improved by the addition of a quadratic term (model IV). These estimates of the mean as a linear and quadratic function of age were held constant in subsequent models that were fitted. The first developmental model fitted (model VI) assumes that within-family environmental effects are age specific (i.e. $\rho(E_1) = 0$), but that there is "remembering" of these environmental effects over time (i.e. $P(E_1) > 0$). Similarly, additive gene effects are assumed to be age specific ($\rho(V_A) = 0$), with persistence of gene products over time ($P(V_A) > 0$). This model results in significant improvement over the E_1V_A model as well as yielding sensible estimates of all

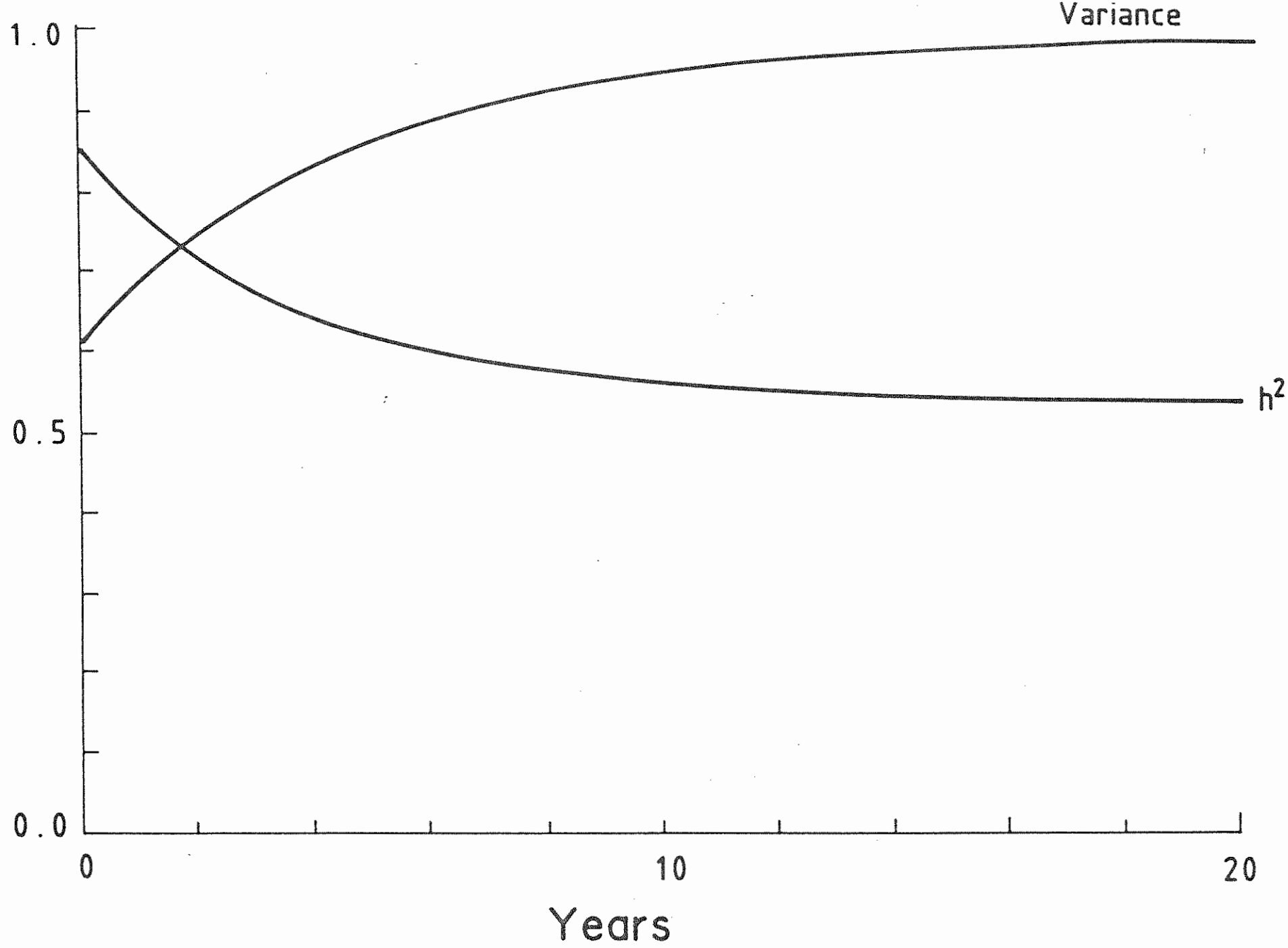
parameters. In contrast, model VII, which assumes that both individual environmental and additive genetic effects persist over time (i.e. $P(E_1)$ and $P(V_A) > 0$), individual environmental effects are age specific ($\rho(E_1) = 0$) and that the same genes act throughout development ($\rho(V_A) = 1.0$), results in $P(V_A)$ going to its lower bound. Thus we accept model VI as the best description of the data.

For consumption last week in females, of the models assuming constant genetic and environmental effects throughout development, the E_1V_A model provides the best description of the data (Table 7.33). Allowing the mean to vary as a function of both linear and quadratic age regressions (model V), results in a significant improvement over the E_1V_A model. Although both developmental models (models VI and VII) result in significant improvement over the E_1V_A model, in both cases $P(V_A)$, the parameter representing the persistence in gene products over time, went to its lower bound.

In males, for both normal weekly consumption (Table 7.34) and consumption last week (Table 7.35), the $E_1E_2V_A$ model provides the best description of the data of the models assuming no developmental changes. For both measures of consumption, improvement over the $E_1E_2V_A$ model was obtained by allowing the mean to vary as a linear and quadratic function of age (model V). The first developmental model fitted to these data (model VI) assumed that within- and between-family environment, and additive genetic effects, were age specific in their action (i.e. $\rho(E_1) = \rho(E_2) = \rho(V_A) = 0$). Furthermore, there was

persistence of these three effects over time (i.e. $P(E_1)$, $P(E_2)$ and $P(V_A) > 0$). For normal weekly consumption, this model resulted in a worsening of fit over the $E_1E_2V_A$ model and furthermore $P(E_2)$ went to its upper bound. For consumption last week, although this developmental model results in significant improvement over the $E_1E_2V_A$ model, the paths representing the persistence of effects over time ($P(E_1)$, $P(E_2)$ and $P(V_A)$) went to their lower bound. The second developmental model (model VII) asumed that within-families environment and additive genetic effects were age specific in their action (i.e. $\rho(E_1) = \rho(V_A) = 0$), but that the same between-families environmental factors were operating throughout development (i.e. $\rho(E_2) = 1$). It was also assumed that there was persistence of these effects over time ($\rho(E_1)$, $\rho(E_2)$ and $\rho(V_A) > 0$). For normal weekly consumption, this developmental model is again worse than the $E_1E_2V_A$ model, and both $P(E_2)$ and V_A go to their upper bounds. For consumption last week, while this model is significantly better than the $E_1E_2V_A$ model, the parameters representing the persistence of within- and between-families environmental effects over time went to their lower and upper bounds respectively. Thus, only for normal weekly consumption in females are we able to find a theoretical model that can adequately describe the changes in genetic and environmental effects as a function of age. This does, however, lead to several predictions that may be tested.

Figure 7.2 shows how the phenotypic variance and heritability (h^2) change when within-family environment and gene effects are assumed to be age specific ($\rho(E_1) = \rho(V_A) = 0$), but


Variance
1.0
0.5
0.0
h^2
0
10
20
Years

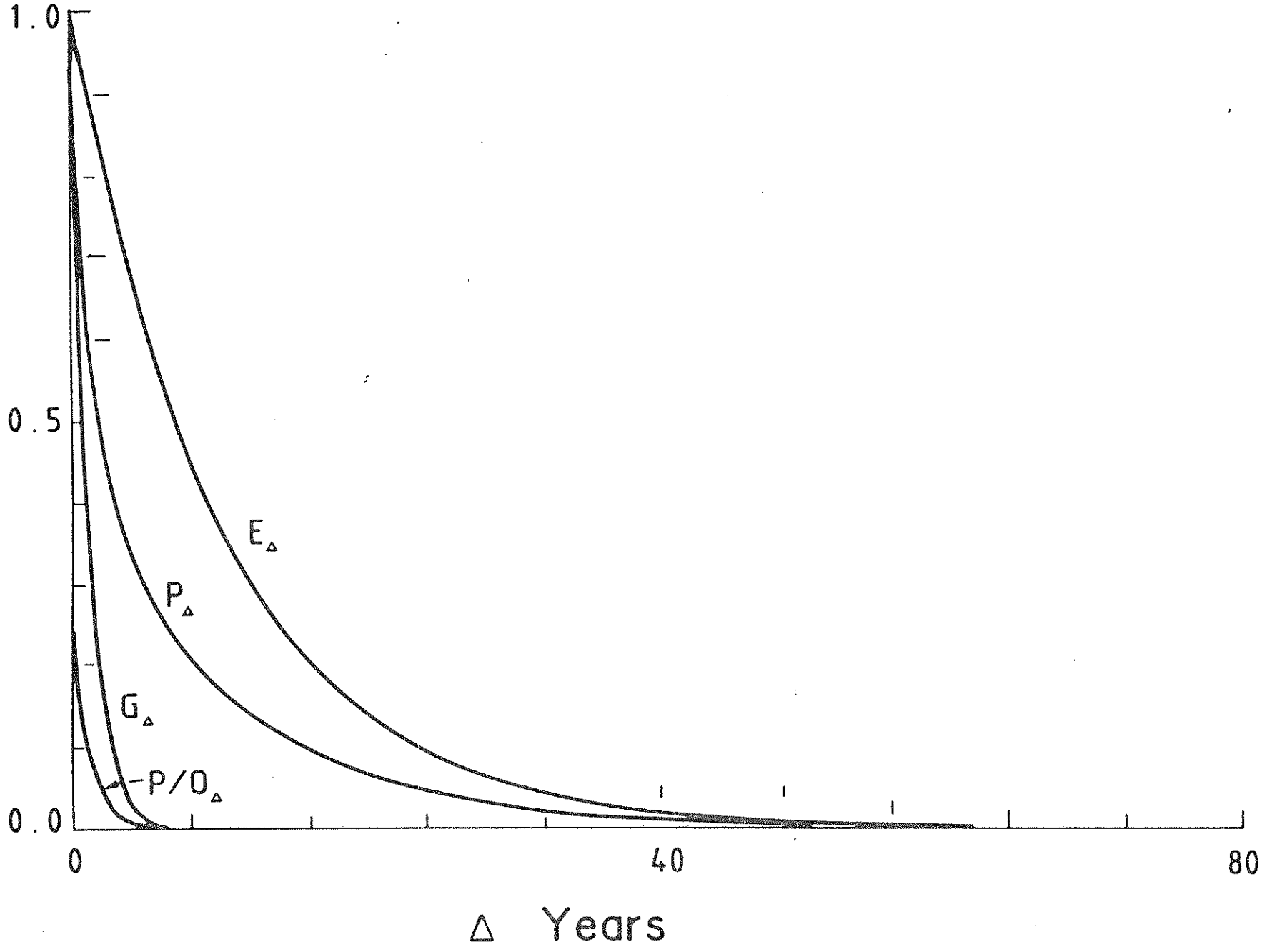
there is persistence of environmental and genetic effects over time ($P(E_1)$ = 0.902, $P(V_A)$ = 0.134, estimates were obtained from the fit of model VI to normal weekly consumption in females). The variance increases, to a limiting adult value, because information received from the environment is continually being incorporated into the phenotype. However, the heritability will decrease with age to a limiting adult value.

Figure 7.3 illustrates predicted changes in correlations in adults as a function of differences in their ages (Δ). The parent offspring correlation (P/O) starts at half the value of the limiting heritability, and then decreases to zero over time. Thus, a naive genetic analysis of parents and their juvenile offspring would mistake these developmental changes as evidence for the expression of dominant genes. Similarly, the genetic (G), environmental (E) and phenotypic (P) correlations within-individuals across occasions start at unity and decline to zero as Δ increases.

Although we have only found one instance where it is possible to give a theoretical explanation of the changes in the contribution of genetic and environmental effects to variation in alcohol consumption over time, this is still superior to empirical approaches that do not lead to testable predictions either for different relationships, or changes in correlations within individuals as a function of increasing time.

7.4.5.3 Comparison of different age cohorts

In section 7.4.5.1 we saw that for both measures of consumption, in females there is an increase in V_A with age,


1.0
0.5
0.0
0
40
80
E△
P△
G△
P/O△
△ Years

while in males E_1 and E_2 increase with age while V_A decreases. However, these conclusions were based on the comparison of only two age cohorts. As we have only been able to find an adequate theoretical explanation of these developmental changes for normal weekly consumption in females, we decided to divide the sample into smaller age cohorts and see if there were perhaps more subtle changes in the contribution of genetic and environmental factors to variation in alcohol consumption with age that our previous analysis had been unable to detect.

The twin sample was divided into cohorts containing at least 100 pairs of twins, separately for males and females. This resulted in 16 cohorts in females, and 8 in males. The mean age, and the mean squares for the two measures of consumption, and their degrees of freedom, for the different age cohorts are shown in Table 7.36, separately for males and females.

Models were fitted to the mean squares for the 16 cohorts in females, and 8 cohorts in males, then to the combined 64 and 32 statistics in females and males respectively. As we were particularly interested in the changes in E_1 and V_A in females, and E_1, E_2 and V_A in males, we compared only the fit of the E_1V_A model in females, and the $E_1E_2V_A$ model in males, over the different age cohorts. The parameter estimates obtained from fitting these models are shown separately for normal weekly consumption (Figure 7.4) and consumption last week (Figure 7.5) in females, and normal weekly consumption (Figure 7.6) and consumption last week (Figure 7.7) in males.

Table 7.36 Observed mean squares and their degrees of freedom for log transformed measures of alcohol consumption for different age cohorts. The mean of age of each cohort is also shown.

Females

Cohort	Mean age	Normal weekly consumption df	Normal weekly consumption mean square	Consumption last week df	Consumption last week mean square
1	18.78				
MZ Between		89	0.3741	89	0.3645
Within		90	0.0781	90	0.0962
DZ Between		56	0.2297	56	0.2845
Within		57	0.0919	57	0.1539
2	20.42				
MZ Between		99	0.3765	99	0.3873
Within		100	0.0874	100	0.0839
DZ Between		69	0.2453	69	0.3189
Within		70	0.1593	70	0.1385
3	22.44				
MZ Between		95	0.2768	95	0.3087
Within		96	0.0930	96	0.1556
DZ Between		47	0.3175	47	0.3557
Within		48	0.1328	48	0.1618
4	24.54				
MZ Between		74	0.2688	74	0.2942
Within		75	0.0786	75	0.0799
DZ Between		45	0.2946	45	0.2472
Within		46	0.0963	46	0.1343
5	26.42				
MZ Between		86	0.3424	86	0.3177
Within		87	0.1218	87	0.1519
DZ Between		56	0.2444	56	0.2063
Within		57	0.2063	57	0.1857
6	28.54				
MZ Between		88	0.3439	88	0.3553
Within		89	0.0606	89	0.0839
DZ Between		47	0.2108	47	0.2617
Within		48	0.1090	48	0.1413
7	30.48				
MZ Between		62	0.3619	62	0.3525
Within		63	0.0841	63	0.1079
DZ Between		46	0.2153	46	0.2153
Within		47	0.1303	47	0.1472
8	32.44				
MZ Between		65	0.2750	65	0.2902
Within		66	0.1158	66	0.1291
DZ Between		38	0.2064	38	0.1812
Within		39	0.1630	39	0.2588
9	34.60				
MZ Between		72	0.4545	72	0.4178
Within		73	0.0993	73	0.1073
DZ Between		46	0.2459	46	0.2695
Within		47	0.1408	47	0.1383

Table 7.36 cont'd

Cohort	Mean age	Normal weekly consumption df	Normal weekly consumption mean square	Consumption last week df	Consumption last week mean square
10	37.01				
MZ Between		59	0.3318	59	0.3477
Within		60	0.1229	60	0.1468
DZ Between		45	0.2858	45	0.2349
Within		46	0.1677	46	0.1933
11	40.00				
MZ Between		66	0.4548	66	0.4067
Within		67	0.0907	67	0.0807
DZ Between		35	0.4797	35	0.4143
Within		36	0.1317	36	0.1232
12	43.46				
MZ Between		79	0.4287	79	0.3800
Within		80	0.1343	80	0.1000
DZ Between		37	0.4154	37	0.3814
Within		38	0.2496	38	0.1862
13	48.09				
MZ Between		59	0.4704	59	0.4281
Within		60	0.1254	60	0.1370
DZ Between		43	0.2468	43	0.2576
Within		44	0.2496	44	0.2468
14	52.94				
MZ Between		73	0.4290	73	0.4594
Within		74	0.1373	74	0.0910
DZ Between		47	0.3249	47	0.3310
Within		48	0.2227	48	0.1679
15	58.42				
MZ Between		72	0.4723	72	0.4668
Within		73	0.1166	73	0.0747
DZ Between		35	0.4385	35	0.3881
Within		36	0.1429	36	0.1521
16	69.21				
MZ Between		79	0.3887	79	0.3820
Within		80	0.1266	80	0.1010
DZ Between		42	0.3540	42	0.2871
Within		43	0.1833	43	0.1664
Males					
1	19.32				
MZ Between		71	0.5979	71	0.5577
Within		72	0.1057	72	0.1602
DZ Between		65	0.4530	65	0.4868
Within		66	0.1458	66	0.1732
2	21.97				
MZ Between		82	0.5527	82	0.5351
Within		83	0.1305	83	0.1394
DZ Between		54	0.5403	54	0.5232
Within		55	0.1970	55	0.2351

Table 7.36 cont'd

Cohort	Mean age	Normal weekly consumption df	Normal weekly consumption mean square	Consumption last week df	Consumption last week mean square
3	25.46				
MZ Between		58	0.6109	58	0.5942
Within		59	0.1177	59	0.1211
DZ Between		50	0.4534	50	0.4298
Within		51	0.1679	51	0.1916
4	29.61				
MZ Between		86	0.4862	86	0.4789
Within		87	0.1560	87	0.1711
DZ Between		45	0.3045	45	0.3329
Within		46	0.2946	46	0.2403
5	33.49				
MZ Between		71	0.5118	71	0.4888
Within		72	0.1843	72	0.1918
DZ Between		33	0.5583	33	0.4905
Within		34	0.1530	34	0.1876
6	39.05				
MZ Between		69	0.5108	69	0.5677
Within		70	0.1666	70	0.1345
DZ Between		33	0.5682	33	0.5828
Within		34	0.2305	34	0.2314
7	49.69				
MZ Between		67	0.6463	67	0.5504
Within		68	0.2080	68	0.1852
DZ Between		34	0.6617	34	0.6664
Within		35	0.1597	35	0.1907
8	65.39				
MZ Between		54	0.6012	54	0.5588
Within		55	0.1361	55	0.1845
DZ Between		30	0.5605	30	0.4764
Within		31	0.2379	31	0.3044

FEMALES NORMAL WEEKLY CONSUMPTION
COHORT
1
2
3
4
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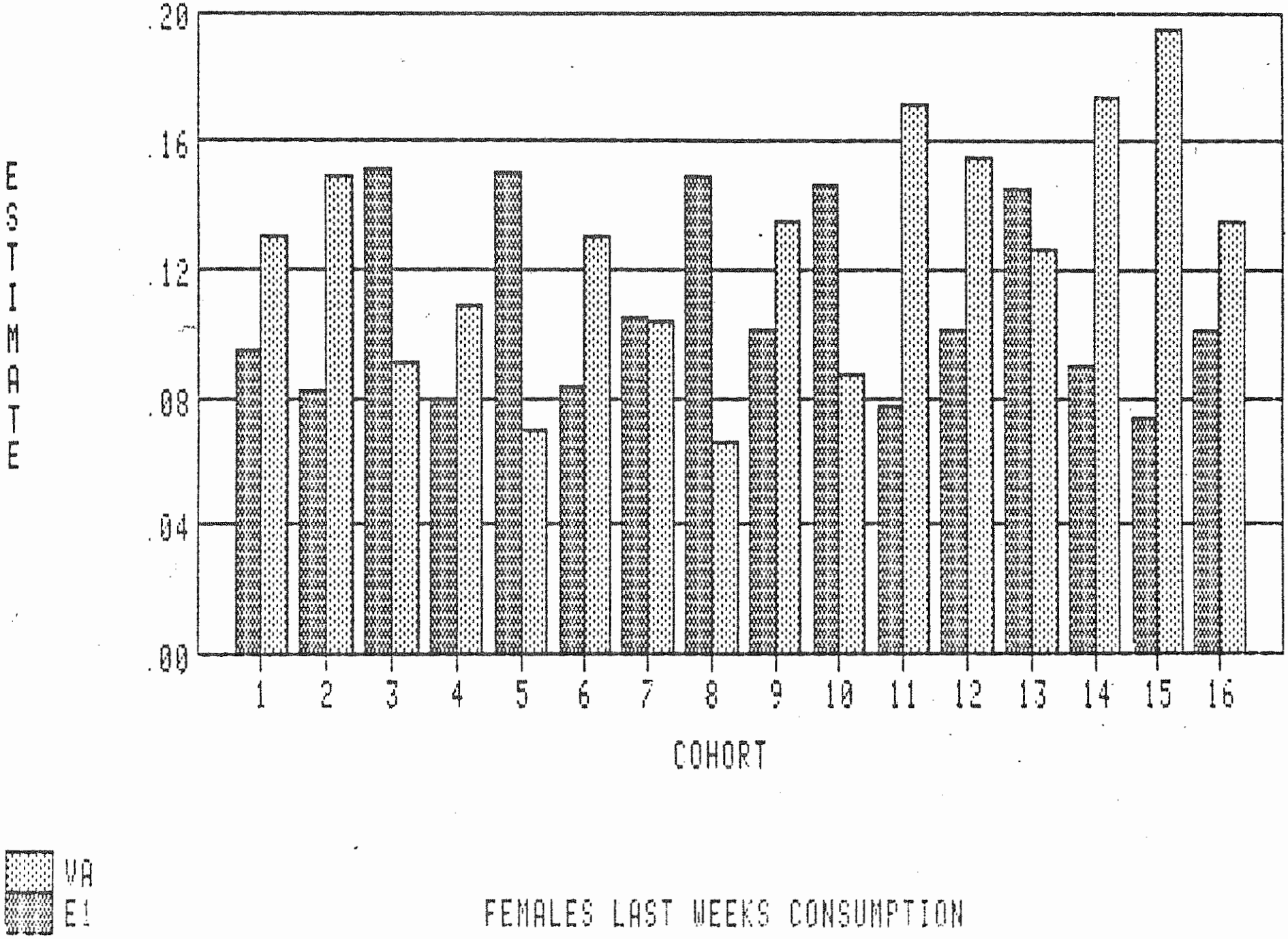
EI
HM


.20
.16
.12
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.00
ESTIMATE
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2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
COHORT
VA
E1
FEMALES LAST WEEKS CONSUMPTION

MALES NORMAL WEEKLY CONSUMPTION

E1 E2 HD

-20 -08 04 16 28 40

BIRTHDATE

COHORT

1 2 3 4 5 6 7 8

MALES LAST WEEKS CONSUMPTION
E1
E3
HH
SERVINGS
10
20
9
14
22
33
COHORT
1
2
3
4
5
6
7
8

In females, there is significant heterogeneity of fit of the E_1V_A model over the different age cohorts for both normal weekly consumption (χ^2_{30} = 79.93, p<.001) and consumption last week (χ^2_{30} = 66.32, p<.001). Furthermore, it is obvious that for both measures of consumption, there is no simple function that could describe the changes in E_1 and V_A with age. This is even more apparent in the male data. Although there is no heterogeneity of fit of the $E_1E_2V_A$ model over the different age cohorts for either normal weekly consumption (χ^2_{21} = 31.11, p>.05) or consumption last week (χ^2_{21} = 16.52, p>.05), the fluctuation in parameter estimates is even more extreme than that found in females. It is not suprising then that our attempts to find a theoretical model to describe developmetal changes in alcohol consumption were unsuccessful, when it is unlikely that we could find an empirical model that could adequately describe the data. Unfortunately, by splitting our sample into small cohorts, our power to discriminate between alternative models of variation is low (Martin et al., 1978), and thus more detailed analysis is unlikely to provide much information. Our analysis does however demonstrate that the changes in the contribution of genetic and environmental factors to variation in alcohol consumption with age are more complicated than has previously been demonstrated (e.g. Partanen et al., 1966; Kaprio et al., 1981).

7.4.5.4 Interaction with marital status

Throughout this chapter we have seen that the causes of variation in drinking behaviour are dependent on age and sex. This leads us to a more general question as to whether we can

identify other factors that might modify the effects of genes and environment on alcohol consumption. In genetic terms, this can be conceived as a search for genotype-environment interaction. Although the present study was not designed to detect genotype-environment interaction, and therefore did not measure possibly relevant environmental variables, it is possible to illustrate one method of detecting genotype-environment interaction by using data on marital status.*

The twin sample was divided into those twin pairs where both twins have a marriage-like relationship (i.e. married or cohabiting), where only one twin has such a relationship, and where neither twin has such a relationship. Table 7.37 shows the variances and covariances for the two measures of consumption for twin 1 and twin 2, divided into these three groups, separately for males and females. For concordant pairs, the order of twin 1 and twin 2 was arbitrary. For discordant pairs, twin 1 was married, twin 2 was not.

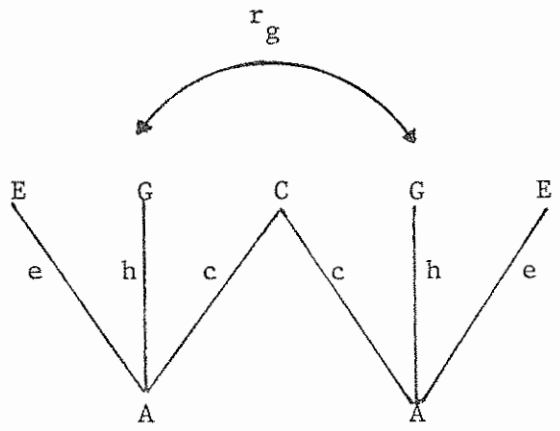
It is possible to fit three basic kinds of model to these data. First, genes and environment could combine additively, in which case gene expression is the same in all twin groups. Second, marital status could govern the degree to which genes are expressed, but the same genes are expressed regardless of whether one is married or not. Third, depending on marital status, different genes could affect alcohol consumption.

In Figure 7.8 we present three path diagrams which summarise our basic model. Figure 7.8(i) applies to concordant pairs who are married, Figure 7.8(ii) to concordant pairs who are single

Table 7.37 Variance-covariance matrices for log transformed measures of alcohol consumption, broken down by sex and marital status.

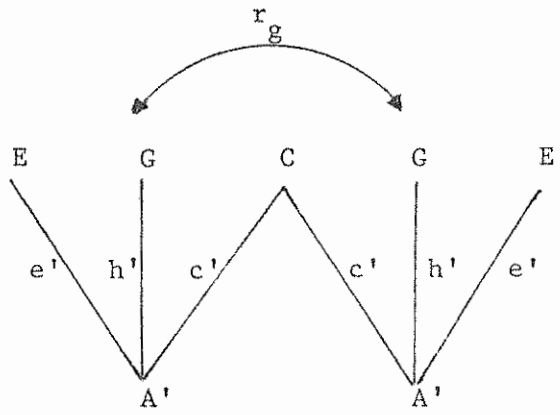
	Concordant married		Concordant single		Discordant	
Females						
Normal weekly consumption						
MZ	(n=650)		(n=307)		(n=276)	
	Twin		Twin		Twin	
	1	2	1	2	1	2
Twin 1	0.2313	0.1290	0.2292	0.1596	0.2295	0.1260
Twin 2	0.1290	0.2339	0.1596	0.2357	0.1260	0.2722
DZ	(n=380)		(n=181)		(n=190)	
Twin 1	0.2433	0.0738	0.2073	0.0808	0.2274	0.0364
Twin 2	0.0738	0.2085	0.0808	0.2073	0.0364	0.2480
Consumption last week						
MZ	1	2	1	2	1	2
Twin 1	0.2316	0.1310	0.2295	0.1547	0.2240	0.1122
Twin 2	0.1310	0.2380	0.1547	0.2440	0.1122	0.2728
DZ						
Twin 1	0.2263	0.0661	0.2775	0.0887	0.2044	0.0271
Twin 2	0.0661	0.2161	0.0887	0.2534	0.0271	0.2420
Males						
Normal weekly consumption						
MZ	(n=268)		(n=187)		(n=112)	
	Twin		Twin		Twin	
	1	2	1	2	1	2
Twin 1	0.3579	0.1861	0.3589	0.2483	0.3795	0.2094
Twin 2	0.1861	0.3377	0.2483	0.3536	0.2094	0.4382
DZ	(n=137)		(n=136)		(n=79)	
Twin 1	0.3433	0.1530	0.3288	0.1648	0.3386	0.1493
Twin 2	0.1530	0.4012	0.1648	0.3217	0.1493	0.3796
Consumption last week						
MZ	1	2	1	2	1	2
Twin 1	0.3270	0.1807	0.3460	0.2063	0.3871	0.2266
Twin 2	0.1807	0.3610	0.2063	0.3396	0.2266	0.4522
DZ						
Twin 1	0.3242	0.1318	0.3353	0.1574	0.3474	0.1257
Twin 2	0.1318	0.4088	0.1574	0.3512	0.1257	0.3659

(i)



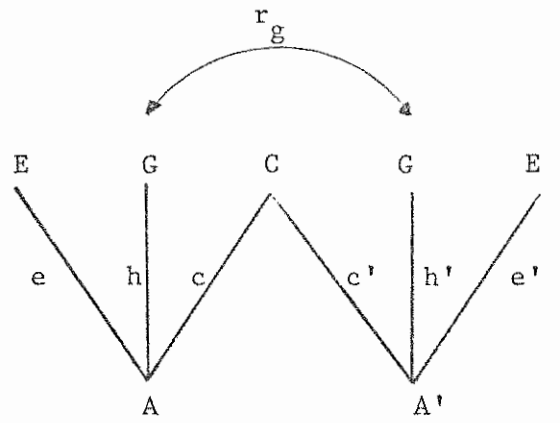
Concordant (married) pairs

(ii)



Concordant (single) pairs

(iii)



Discordant pairs

and Figure 7.8(iii) to discordant pairs where one twin is married, the other is not. In these diagrams, G denotes additive genetic deviations, C between-families environment and E individual environment. The correlations (r_g) between additive genetic deviations will be 1 for MZ twins, 1/2 for DZ twins. For discordant pairs, the correlations between additive gentic effects in married and single pairs will be reduced if the genes affecting alcohol consumption differ according to marital status. The paths to measured alcohol consumption from genotype (h or h'), between-families environment (c or c') or individual environment (e or e') will be the same for MZ and DZ twins, but are allowed to differ according to whether an individual is married or not (denoted by a prime, ').

On this model, the expected total variance, which should be the same in MZ and DZ twins, will be

$$h^2 + c^2 + e^2$$

in those twins who are married, or

$$h'^2 + c'^2 + e'^2$$

in those twins who are single. The expected covariance of married MZ twins will be

$$h^2 + c^2,$$

the expected covariance of MZ twins who are single will be

$$h'^2 + c'^2$$

and the expected covariance of MZ twins who are discordant for marital status will be

$$hh' + cc'.$$

Expected covariances for DZ twins are easily derived by multiplying the genetic terms in the expectations for MZ twins given above by 1/2. Having derived these expected variance-covariance matrices, it is possible to fit alternative models to the full set of observed covariance matrices using the method of maximum likelihood covariance structure analysis described in section 5.3.5.1 of this thesis.

The results of fitting the three basic models described above to both measures of alcohol consumption are shown in Table 7.38, separately for males and females. We have presented the results in terms of our usual notation where

$V_A = h^2$

$V_A' = h'^2$

$E_1 = e^2$

$E_1' = e'^2$

$E_2 = c^2$

$E_2' = c'^2$

and R_{V_A} and R_{E_2} are the correlations between additve genetic and between-families environmental effects in married and single twins respectively. There is an obvious similarity between this approach, looking at scalar and non-scalar differences in genetic and environmental effects in married and single twins, to that used throughout this thesis in the examination of scalar and non-scalar differences in genetic and environmental effects in males and females.

In females, for both measures of consumption, both model I, which assumes that there is no interaction between genotype and marital status, and model II, which assumes that the same genes

Table 7.38 Results of fitting models testing for genetic and environmental interaction with marital status, in log transformed measures of alcohol consumption, separately for males and females.

Females

Normal weekly consumption

Model	V_A	V_A'	R_{V_A}	E_1	E_1'	df	χ^2
I	.130		-	.102		16	40.43***
II	.116	.160	-	.109	.078	14	29.71**
III	.130	.168	.747	.102	.073	13	13.48

Consumption last week

Model	V_A	V_A'	R_{V_A}	E_1	E_1'	df	χ^2
I	.130		-	.102		16	34.60**
II	.116	.152	-	.109	.096	14	27.24*
III	.123	.168	.698	.102	.084	13	7.69

Males

Normal weekly consumption

Model	V_A	V_A'	R_{V_A}	E_2	E_2'	R_{E_2}	E_1	E_1'	df	χ^2
I	.090		-	.116		-	.152		15	22.53
II	.152	.078	-	.040	.176	-	.160	.116	12	8.25
III	.096	.116	.543	.096	.137	1.000†	.160	.109	10	5.39

Consumption last week

Model	V_A	V_A'	R_{V_A}	E_2	E_2'	R_{E_2}	E_1	E_1'	df	χ^2
I	.109		-	.084		-	.160		15	12.52
II	.152	.084	-	.036	.137	-	.168	.144	12	7.63
III	.130	.102	.884	.058	.123	.895	.168	.137	10	7.22

† Parameter went to upper bound

affect alcohol consumption in married and single females, but they have a different effect on the variance, fail badly. Model III, which allows different E_1 effects, and non-scalar differences in V_A effects in married and single females, gives an excellent fit to the data for both normal weekly consumption and consumption last week. We see that there is a consistent tendency for the importance of genetic factors to increase with the lack of a steady partner. Furthermore, the correlations $R_{V_A} = 0.75$ for normal weekly consumption, and $R_{V_A} = 0.70$ for consumption last week, indicate that depending on marital status, there are some differences in the genes affecting alcohol consumption.

In males, for normal weekly consumption, a model allowing no differences in genetic and environmental effects in married and single twin pairs (model I), is able to adequately describe the data. However, there is a significant reduction in chi-square if we allow scalar differences in genetic and environmental effects depending on marital status (model II), ($\chi^2_3 = 14.28$, $p<.01$). No further reductions in chi-square were seen by allowing non-scalar differences in genetic and environmental effects depending on marital status (model III). Thus for normal weekly consumption, the genetic and environmental effects are the same in married and single males, but marital status governs the degree to which genetic and environmental effects are expressed. The importance of genetic factors and individual environment decreases, and between-families environment increases, with the lack of a steady partner. For consumption last week, model I, which assumes that

the same genetic and environmental effects act regardless of marital status, is able to adequately describe the data. No further reductions in chi-square were seen by allowing either scalar or non-scalar differences in genetic and environmental effects depending on marital status (models I and II respectively), thus we regard model I as the best description of the data.

The above results show that the effect of marital status on the causes of variation in alcohol consumption is dependent on sex. We shall now examine whether the effect of marital status on the causes of variation in alcohol consumption is also dependent on age. The twin sample was again divided into younger (30 and under) and older (over 30) twin pairs. The variances and covariances for the two measures of alcohol consumption for older and younger twin pairs are shown in Table 7.39, separately for males and females. The results of fitting models to both measures of consumption are shown separately for younger (Table 7.40) and older (Table 7.41) pairs.

For normal weekly consumption in females, for both younger and older twins, model III which allows different E_1 effects, and non-scalar differences in V_A effects, in married and single females provides the best description of the data. The correlations $R_{V_A} = 0.802$ in younger females, and $R_{V_A} = 0.725$ in older females, indicate that for both age cohorts there are some differences in the genes affecting alcohol consumption depending on marital status. Model III also provides the best description of the data for consumption last week in older female twin pairs,

Table 7.39 Variance-covariance for log transformed measures of alcohol consumption, broken down by age cohort, sex and marital status.

	Concordant married		Concordant single		Discordant	
Younger						
Females						
Normal weekly consumption						
MZ	(n=177)		(n=254)		(n=53)	
	Twin		Twin		Twin	
	1	2	1	2	1	2
Twin 1	0.1787	0.0896	0.2283	0.1579	0.3397	0.2171
Twin 2	0.0896	0.1674	0.1579	0.2273	0.2171	0.4290
DZ	(n=108)		(n=156)		(n=87)	
Twin 1	0.1189	0.0346	0.1978	0.0822	0.1929	0.0427
Twin 2	0.0346	0.1407	0.0822	0.2094	0.0427	0.2222
Consumption last week						
MZ	1	2	1	2	1	2
Twin 1	0.2074	0.0840	0.2334	0.1515	0.1890	0.0878
Twin 2	0.0840	0.2016	0.1515	0.2453	0.0878	0.2302
DZ						
Twin 1	0.1744	0.0332	0.2211	0.0951	0.1898	0.0234
Twin 2	0.0332	0.1664	0.0951	0.2538	0.0234	0.2156
Males						
Normal weekly consumption						
MZ	(n=48)		(n=173)		(n=53)	
	Twin		Twin		Twin	
	1	2	1	2	1	2
Twin 1	0.3529	0.1891	0.3694	0.2567	0.3397	0.2171
Twin 2	0.1891	0.2890	0.2567	0.3515	0.2171	0.4290
DZ	(n=27)		(n=131)		(n=48)	
Twin 1	0.2702	-0.0198	0.3319	0.1642	0.3398	0.1423
Twin 2	-0.0198	0.3413	0.1642	0.3258	0.1423	0.3534
Consumption last week						
MZ	1	2	1	2	1	2
Twin 1	0.3138	0.2384	0.3584	0.2087	0.3374	0.2299
Twin 2	0.2384	0.3916	0.2087	0.3438	0.2299	0.4152
DZ						
Twin 1	0.1554	0.0186	0.2211	0.0951	0.3289	0.1196
Twin 2	0.0186	0.3772	0.0951	0.2538	0.1196	0.3746

Table 7.39 cont'd

	Concordant married		Concordant single		Discordant	
Older Females						
Normal weekly consumption						
MZ	(n=473)		(n=53)		(n=137)	
	Twin		Twin		Twin	
	1	2	1	2	1	2
Twin 1	0.2511	0.1434	0.2318	0.1564	0.2672	0.1532
Twin 2	0.1434	0.2580	0.1564	0.2480	0.1532	0.3329
DZ	(n=272)		(n=25)		(n=103)	
Twin 1	0.2655	0.0894	0.2742	0.0666	0.2565	0.0269
Twin 2	0.0894	0.2357	0.0666	0.1824	0.0269	0.2634
Consumption last week						
MZ	1	2	1	2	1	2
Twin 1	0.2409	0.1485	0.2080	0.1575	0.2605	0.1392
Twin 2	0.1485	0.2517	0.1575	0.2081	0.1392	0.3149
DZ						
Twin 1	0.2473	0.0795	0.2720	0.0351	0.2160	0.0240
Twin 2	0.0795	0.2364	0.0351	0.2219	0.0240	0.2515
Males						
Normal weekly consumption						
MZ	(n=220)		(n=14)		(n=59)	
	Twin		Twin		Twin	
	1	2	1	2	1	2
Twin 1	0.3604	0.1863	0.2393	0.1483	0.4201	0.1987
Twin 2	0.1863	0.3497	0.1483	0.3998	0.1987	0.4172
DZ	(n=110)		(n=5)		(n=31)	
Twin 1	0.3579	0.1899	0.0874	0.1250	0.3451	0.1627
Twin 2	0.1899	0.4137	0.1250	0.2250	0.1627	0.4312
Consumption last week						
MZ	1	2	1	2	1	2
Twin 1	0.3291	0.1699	0.1926	0.1686	0.4372	0.2192
Twin 2	0.1699	0.3559	0.1686	0.2815	0.2192	0.4357
DZ						
Twin 1	0.3576	0.1493	0.1798	0.1379	0.3866	0.1373
Twin 2	0.1493	0.4084	0.1379	0.2955	0.1373	0.3616

Table 7.40 Results of fitting models testing for genetic and environmental interaction with marital status, in log transformed measures of alcohol consumption in younger twin pairs, separately for males and females.

Females

Normal weekly consumption

Model	V_A	V_A'	R_{V_A}	E_1	E_1'	df	χ^2
I	.116		-	.078		16	25.93
II	.084	.144	-	.096	.068	14	11.02
III	.090	.144	.802	.084	.068	13	7.14

Consumption last week

Model	V_A	V_A'	R_{V_A}	E_1	E_1'	df	χ^2
I	.109		-	.102		16	28.41*
II	.068	.144	-	.123	.084	14	7.60
III	.073	.152	.815	.116	.084	13	5.92

Males

Normal weekly consumption

Model	V_A	V_A'	R_{V_A}	E_2	E_2'	R_{E_2}	E_1	E_1'	df	χ^2
I	.160		-	.068		-	.123		15	18.01
II	.168	.130	-	.010	.116	-	.144	.102	12	7.33
III	.168	.130	1.000†	.010	.116	1.000†	.144	.102	10	7.33

Consumption last week

Model	V_A	V_A'	R_{V_A}	E_2	E_2'	R_{E_2}	E_1	E_1'	df	χ^2
I	.144		-	.068		-	.137		15	13.62
II	.109	.221	-	.096	.020	-	.138	.116	12	11.08
III	.109	.221	1.000†	.096	.140	1.000†	.138	.116	10	11.08

† Parameter went to upper bound

Table 7.41 Results of fitting models testing for genetic and environmental interaction with marital status, in log transformed measures of alcohol consumption in older twin pairs, separately for males and females.

Females

Normal weekly consumption

Model	V_A	V_A'	R_{V_A}	E_1	E_1'	df	χ^2
I	.144		-	.116		16	19.96
II	.144	.152	-	.109	.130	14	17.82
III	.144	.194	.725	.109	.084	13	10.49

Consumption last week

Model	V_A	V_A'	R_{V_A}	E_1	E_1'	df	χ^2
I	.144		-	.102		16	30.74*
II	.144	.144	-	.102	.123	14	28.71*
III	.144	.221	.627	.096	.053	13	9.05

Males

Normal weekly consumption

Model	V_A	V_A'	R_{V_A}	E_2	E_2'	R_{E_2}	E_1	E_1'	df	χ^2
I	.029		-	.160		-	.176		15	15.03
II	.020	.109	-	.176	.096	-	.176	.176	12	14.37
III	.044	.058	1.000†	.152	.240	.828	.168	.144	10	12.97

Consumption last week

Model	V_A	V_A'	R_{V_A}	E_2	E_2'	R_{E_2}	E_1	E_1'	df	χ^2
I	.096		-	.090		-	.176		15	12.55
II	.123	.001	-	.068	.281	-	.176	.078	12	7.60
III	.102	.073	.800	.084	.221	.703	.176	.073	10	7.11

† Parameter went to upper bound

the correlation R_{V_A} = 0.627 again indicating that there are some differences in the genes affecting alcohol consumption in married and single females. In contrast, for consumption last week in younger females, model II provides the best description of the data, indicating that in this cohort, the same genetic and environmental effects act in married and single females, but marital status governs the degree to which genetic and environmental effects are expressed. However, for both measures of consumption, in both cohorts, the tendency for the importance of genetic factors to increase with the lack of a steady partner is still apparent. Thus, genetic factors are of least importance in married females under 30, and of greatest importance in single females over 30.

In males, with the exception of normal weekly consumption in younger pairs, model I provides the best description of the data, indicating that marital status and genes and environment combine additively. That is, the same genetic and individual environmental effects act regardless of marital status. For normal weekly consumption in younger males, model II provides the best description of the data, indicating that there are scalar differences in genetic and environmental effects depending on marital status. The importance of genetic and individual environmental effects decreases, and between-families environment increases, with the lack of a steady partner. Thus our results show that the interaction of marital status with the genetic and environmental sources of variation in alcohol consumption is dependent on both age and sex.

7.5 DISCUSSION

The present study demonstrates that the causes of variation in drinking behaviour are more complex than has previously been demonstrated (e.g. Partanen et al., 1966; Kaprio et al., 1981). Our results show that the relative contribution of genetic and environmental factors to variation in drinking behaviour is dependent upon age, sex, and for alcohol consumption in females, marital status.

When studying drinking status we found that for those under 30 years, 89% of the variation in liability to drinking in males is due to family environment and cultural influences, while in females 54% of the variation is due to these effects. Furthermore, there was evidence that the types of environmental influences that predispose people to drink differ between the sexes. In females, genetic factors are also important accounting for 34% of the variance.

In contrast, for those over 30, in both males and females approximately 75% of the variation in liability to drinking is genetic in origin, with the remaining variance due to environmental influences specific to individuals.

Age effects were also apparent when analysing data on the frequency of alcohol consumption. When no correction was made for age, it was not possible to find a model which could adequately describe the data. In younger twin pairs the best fitting model, allowing scalar differences in genetic and environmental effects in males and females, still failed to adequately describe the data. However, in older twin pairs, in

both males and females, 49% of the variation in frequency of alcohol consumption is genetic in origin, and 16% due to the effects of common family environment, the remaining variance due to environmental factors unique to the individual and error.

Our analysis of the frequency of alcohol consumption data assumed that, underlying our observed response categories, there was a continuous and normal distribution of liability to drinking behaviour. We attempted to test this assumption by fitting a model which assumed that there were two dimensions of liability underlying our observed response categories. The first dimension reflected the distinction between non-drinkers and drinkers, the second dimension categorised drinkers on the basis of how frequently they drank. Although this resulted in a highly significant improvement in fit to the data in younger twin pairs, it represented a worsening of fit in older twin pairs. This result suggests that while there may be some differences in the causes of variation in drinking status and, given that one is a drinker, how frequently one drinks, the causes of variation in these two measures are not entirely independent.

An alternative approach to the problem of dimensions in twin similarity has been devised by Eaves (see Eaves and Eysenck, 1980). This approach, based on canonical analysis, does not make any assumptions about the distribution of liability, and simply derives a set of category weights which maximise twin correlations for the weighted phenotype. This approach yields a geometrical representation of the distances between response categories which may be useful in detecting the dimensions in the

twins' responses. For example, when this method was applied to data on the smoking habit (Eaves and Eysenck, 1980), it was shown that non-smokers could not be regarded as one extreme of a single continuum embracing non-smokers and all degrees of smoking. In genetic terms, such models imply interactions between effects responsible for the onset of a habit and factors contributing to intensity once the habit is initiated. Unfortunately, because of time constraints, it was not possible to fit such a model to our data on the frequency of alcohol consumption. However, such an analysis, in the future, may prove useful in elucidating the causes of variation in the frequency of alcohol consumption.

Data relating to alcohol consumption also showed marked age and sex effects. In females, for normal weekly consumption, although there was an increase in E_1 and V_A with age, the heritability decreased from 0.58 to 0.55. For consumption last week, there was an increase in V_A with age, with a corresponding increase in heritability from 0.51 to 0.58.

In males, the effect of age is even more striking. For both measures of consumption, in younger male twins over 60% of the variance is genetic in origin, with the remaining variance due to environmental influences unique to the individual. In older twins, however, genetic differences do not appear to be important, with approximately 50% of the variation due to individual environmental differences, and the remaining 50% due to the effect of common family environment.

Our attempt to find a theoretical explanation for the changes in the contribution of genetic and environmental effects

with age was succesful for normal weekly consumption in females. In this measure, a model which assumed that within-family environmental and additive genetic effects were age specific, but that there was a "remembering" of these environmental effects, and persistence of gene products over time, provided the best description of the data. This leads to several predictions about changes in genetic, environmental and phenotypic correlations within individuals over time, and can also be extended to predict changes in correlations for different family relationships with time. Such analyses would provide further tests of our model. However, for the other measures of consumption, no adequate theoretical explanation for the changes in the sources of variation in alcohol consumption with age could be found. The reason for this was apparent when we analysed the data separately for a large number of different age cohorts. There was considerable fluctuation in our estimates of genetic and environmental sources of variation in the different age cohorts. Furthermore, it was apparent that any attempt to find an empirical model, to guide us in our choice of theoretical model, would be unlikely to be succesful. It should be remembered, however, that we are dealing with cross-sectional data and that perhaps we are seeing cohort rather than developmental effects. A longitudinal study of alcohol consumption would be needed to test this hypothesis.

Another factor found to influence alcohol consumption was marital status. In females, in both younger and older pairs, there is a consistent tendency for the importance of genetic

factors to increase with the lack of a steady partner. The most parsimonious explanation of this finding is that having a steady marriage-like partner attenuates the effects of genes on alcohol consumption, possibly by some social buffering effect. Our results also show that with the exception of consumption last week in younger pairs, there is some difference in the genes affecting alcohol consumption depending on marital status. In contrast, in males, with the exception of normal weekly consumption in younger pairs, marital status and genes and environment combine additively. That is, the same genetic and environmental influences act regardless of marital status.

The significance of the present study lies in three conclusions. First, there are significant genetic and cultural effects on twin resemblance in alcohol consumption. Second, that the effects of genes and environment depend significantly on sex. Third, that the effect of genes and environment depend significantly on age.

Of particular interest is the finding that family environment plays a substantial role in the development of normal drinking behaviour. However, the environmental resemblance between relatives may take a number of different forms. Any attempts at behavioural intervention needs to be based on a better understanding of the major social components of family interaction. Different types of family interaction suggest different therapeutic strategies. For example, children may affect parents, siblings may influence one another and spouses may affect each other. Fortunately, models have been developed

for the contribution of vertical cultural inheritance from parent to child (e.g. Cavalli-Sforza and Feldman, 1973; Rao et al., 1974; Cloninger et al, 1979) and for environmental impact on children of the parental genotype (Nance and Corey, 1976). Models have also been developed for horizontal transmission within a generation (Cavalli-Sforza and Feldman, 1981) including the effects of one sibling on another (Eaves, 1976; Carey, 1983). Unfortunately, the twin design alone is inherently unable to resolve all of the effects we have described.

As we have noted in previous chapters, if there is assortative mating, then part of our estimated contribution of the family environment may reflect assortative mating rather than cultural transmission. Positive marital correlations have indeed been reported for both alcoholism (Hall et al., 1983a, 1983b) and alcohol consumption (Heath, personal communication). Heath studied alcohol consumption in 1553 spouse pairs in America. The spouses were classified as either frequent drinkers, occasional drinkers or non-drinkers, and the polychoric correlation (see section 3.3.7.1) for the spouses was estimated as 0.62 ± 0.02. This correlation could be due to assortative mating or to convergence between spouses following marriage. If it is due to the latter, then Eaves' (1976) model of sibling interaction may be adapted to examine social interaction between spouses. Thus if there is cooperation the phenotypic variance of spouses should be greater than the phenotypic variance of single individuals, if there is competition the reverse will be true. If the marital correlation for alcohol consumption is due to assortative mating,

then this could be due to assortative mating for that variable ('primary phenotypic homogamy': Fisher, 1918) or because of assortative mating for some correlated variable, either some feature of the individuals social background (social homogamy) or some aspect of his phenotype such as educational level or socioeconomic status ('secondary phenotypic homogamy': Fisher, 1918, 1924; Rao et al., 1976, 1979).

Although the twin design alone is unable to resolve these competing models of cultural inheritance, social interaction and assortative mating, Heath et al. (1985) have conducted extensive simulation studies to determine the efficiency of different experimental designs to resolve these effects. They show that extending the classical twin design to include data on the parents of twins allows the detection of sibling interactions and offspring-parent cultural transmission. If we further extend this design to include the spouses and parents-in law of twins, then we are able to determine whether marital correlations arise through assortative mating or convergence, and we are also able to determine the precise basis of assortative mating. Such a study of alcohol consumption would be the single most powerful twin-based design for resolving cultural and biological inheritance. Obviously such a study is necessary if we are to determine more precisely the sources of variation in drinking behaviour.

CHAPTER 8 CONCLUDING REMARKS

In this thesis we have demonstrated the valuable contribution that the classical twin study can make to our understanding of human variation. We have shown the utility of twins for detecting genotype-environment interaction and age- and sex-dependence of genetic and environmental effects. Twin studies are a powerful first step for assessing the broad causes of trait variation. Indeed, they are beset with far fewer problems than other designs (e.g. nuclear-family studies) many of which are rendered almost worthless by the inextricability of genetic and environmental variance (Eaves et al., 1978). The importance of twin studies is further enhanced when it can be shown that their results are generalizable to other relationships. The advantage of the rigorous hypothesis-testing approach that we have adopted in this thesis is that we can use our results as a source of predictions to be tested on other relationships. We shall now consider some of our more striking findings with a view to how they might be more extensively tested in the future, and discuss some of the implications of our results.

in what is certainly one of the largest twin studies undertaken, our results show that without a doubt there are genetical differences leading to human behavioural variation, and that these differences are often dependent on age and sex. Thus for the personality traits we have measured, we found that while the same genes are acting in males and females to produce variation in psychoticism and lie, for psychoticism genetic

differences are more pronounced in males than females, while for lie the reverse is true. For neuroticism there was evidence for the action of different genes in males and females, and for neuroticism and lie genetic differences become more pronounced with age in females but not males. For the attitude of conservatism, while the same genes are acting in females and males, genetic differences become more pronounced with age in males. The basis of these sex and age differences is unknown, and this is an area for future investigation. Prospective longitudinal studies would provide the information needed to examine the changes in the sources of variation with age. However, in view of the problems of sample wastage and the cost and time involved in a longitudinal study, the cross-sequential approach (e.g. Schaie and Strother, 1968) is a more attractive and feasible alternative. In this design, a cross-sectional approach is used to examine variation in a sample of wide age range, and this sample is retested some time later to examine developmental changes.

Such a study of drinking behaviour might help to further elucidate the causes of age dependence in gene expression and environmental influences that we observed in our analysis of data relating to drinking habits. These data were also particularly interesting in that they provided evidence for the importance of family environment in the development of normal drinking habits. We have already noted that this environmental resemblance may take a number of different forms, or may in fact be due to assortative mating (as we found for conservatism). We

reiterate that a study of twins, their parents, spouses and parents-in-law, would provide valuable information on the effects of cultural and biological inheritance on drinking behaviour.

Sibling effects are of great theoretical interest to psychologists and geneticists alike, especially with the emergence of the new discipline of sociobiology (Wilson, 1975; Dawkins, 1976). The basic premise of sociobiology is that individuals influence other individuals, and if such influence has a genetic basis, it may lead to evolutionary change. Although the study of human individual differences has not contributed much to this area as yet, several workers have attempted to extrapolate from animal evidence to explain human social behaviour (e.g. Wilson, 1978; Rushton et al., 1984). Recognising that the genes of one person can effect (through environmental interaction) the phenotype of another, sociobiological phenomena can be viewed in terms of genotype-environment covariation. Furthermore, in the sociobiological literature, interactions between individuals are often discussed in terms of competition (e.g. parent-offspring conflict; Trivers, 1974) or cooperation (e.g. reciprocal altruism; Trivers, 1971). Thus the detection of sibling interaction may provide valuable support for sociobiological interpretations of human social behaviour.

In our analysis of the trait of extraversion, however, we were unable to distinguish between the effects of sibling competition and genetic dominance. Furthermore, our extensive power calculations show that unless the competition effect is

large (greater than 30% of the variance), or dominance accounts for at least 80% of the variance, the discrimination between dominance and sibling interaction is unlikely to prove feasible by model-fitting techniques. However, as Eaves et al. (1978) note, inclusion of family density is the key to the analysis of sibling effects. They point out that singletons and multiple births represent the two extremes of a continuum of density which might be experienced in human families, and that family density will also depend on factors such as total family size and the spacing of siblings. Further work aimed at including these parameters in models of social interaction may prove valuable in the development of further tests of sibling effects.

Our analysis of the causes of variation between the trait of neuroticism and symptoms of anxiety and depression represents an important first step in the bridge between the study of normal personality and psychiatric abnormality. Our results show that genetic variation in anxiety and depression is largely dependent on the same genes which determine variation in neuroticism. Furthermore, there is evidence that additive genetic factors are more important than individual environmental factors in the covariation of anxiety, depression and neuroticism. However, we recognise that many individuals may have personalities vulnerable to disease without developing symptoms. The most important goal for future research in this area is the identification of those factors which account for the fact that some vulnerable individuals develop a given disease while others do not. In particular, a genetic and environmental analysis of the

covariation between neuroticism, life events and their modifiers (see section 5.4) and symptoms of anxiety and depression could help to clarify the relationship between normal personality and disease. Given that genes play substantial role in the aetiology of symptoms of anxiety and depression, it is possible that genes might act via increasing an individual's predisposition to experience life events.

Throughout this thesis we have made the distinction between the effects of within- and between-families environment. This distinction is not merely formal but recognises that there are fundamental differences in the types and effects of different environmental stimuli. Our analysis has shown that environmental differences within-families play a substantial role in the determination of variation in all the traits we have measured. Although part of this variation is due to non-repeatable error, the precise causes of repeatable individual environmental differences are still not known. If we are to determine the nature of these influences a trait-specific approach will be required. Thus in the case of discontinuous variables, studies comparing the environment of "affected" individuals with their "normal" co-twins are needed. In the case of continuous variables, studies of MZ within-pair similarity in environmental experiences and trait similarity are necessary. We should remember, however, that while we have found variables which modify the effects of genes on subsequent behaviour (e.g. the effect of marital status on variation in alcohol consumption in females), one can not necessarily assume that these effects are

environmental in origin. Model-fitting techniques, and the presence of within-pair correlations in MZ twins in the absence of correlations in DZ twins, will be needed to determine the relationship between "environmental" variables and trait similarity.

The results of our analysis of the causes of variation in personality traits and symptoms and the attitude of conservatism have radical implications for our understanding of cultural inheritance in man, and certainly raise serious doubts about naive attempts to explain familial resemblance in purely social terms. Our most striking finding as that for all of these measures, at both the item and trait level, we find virtually no evidence for the importance of family environment. This is neither attributable to an inherent bias in the twin design or lack of power. In fact, Martin et al. (1978) have shown that the twin method is inherently biased against the detection of genetic rather than cultural effects. Furthermore, based on the power calculations of Martin et al. (1978), we know that unless shared family environment is making a trivial contribution to variation in personality and attitudes, our sample size is sufficient to detect its presence.

Our results suggest that geneticists and social scientists have misconceived the role of cultural inheritance in human variation. Workers who emphasise the importance of shared environmental factors must recognise that individuals acquire little from their social environment that is incompatible with their genotype. Our results do not imply that learning and

social environment are unimportant in determining human behaviour. However, it must be recognised that an individual's innate abilities and predispositions help him to select relevant and adaptive opportunities and stimuli from the environment. Any effects of learning serve only to augment rather than eradicate the effects of genotype on behaviour.

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APPENDIX 1

Questionnaire sent to twins

AUSTRALIAN TWIN REGISTRY

TELEPHONE: (062)
49 4486

Department of Population Biology
Research School of Biological Sciences
Australian National University
P.O. Box 475
Canberra City A.C.T 2601

Dear Twin,

Thank you for giving your name to the Australian Twin Registry (which now includes those who have previously given their names to the Victorian Twin Registry).

So far about 10% of all twins in Australia have enrolled on the Registry and with your help we hope to enrol many more pairs. Setting up and financing the Australian Twin Registry has taken a long time, and you and your co-twin may have wondered if you would ever hear from us again. However, we now have financial support from the National Health and Medical Research Council, the University of Melbourne and the Australian National University to carry out this postal survey of all registered twins. This survey will help to explain why some people are more healthy than others. Twins are vital in this research, because by comparing identical with non-identical twins the relative importance of heredity and environment in health differences can be worked out. We hope this work will help in the prevention of disease in Australia.

You will realise that it is an expensive project to send reply-paid mail to several thousand pairs of twins. To obtain full value from the work, we have selected a detailed set of questions relating to your birth and life as a twin, your family, your health, habits, feelings, occupation and beliefs. All of these questions are important for our research and we hope you can spare the time to help us.

This particular survey has been financed by a National Health and Medical Research Council grant to Dr. J.D. Mathews, University of Melbourne; Professor J.B. Gibson and Dr. N.G. Martin, Australian National University.

INSTRUCTIONS:

* *We expect that it will take you about 45 minutes to complete the enclosed questions. You will probably find it easier to complete if you can answer all the questions at one sitting.*
* *It is important that we have your OWN answers to the questions so please DO NOT discuss your answers with your twin.*
* *Before returning the questionnaire in the enclosed reply-paid envelope, please check carefully that you have answered all the questions on every page.*
* *All your answers will be STRICTLY CONFIDENTIAL and the research will be in accordance with National Health and Medical Research Council guidelines.*
* *Mrs. M. Olsen at the above address will be pleased to answer any enquiries.*
* *PLEASE CHECK THE DETAILS ON THE LABELS BELOW AND MARK ANY CORRECTIONS NECESSARY.*

YOURSELF YOUR TWIN

12 20

								1

leave blank

NOW PLEASE TURN OVER

PLEASE DO NOT DETACH ANY PAGES

TWINNING

For each question please tick the box which best describes you.

(1) *Were you the first or second born twin?* → 21

1	First born	
2	Second born	
3	Don't know	

(2) *What was the time between the delivery of the first born and second born twin?* ☐☐ hrs ☐☐ mins ☐ don't know 22 ☐☐☐☐ *leave blank*

(3) *What was your own birthweight?* ☐☐ lbs ☐☐ ozs ☐ don't know 26 ☐☐☐☐ *leave blank*

(4) *What was your twin's birthweight?* ☐☐ lbs ☐☐ ozs ☐ don't know 30 ☐☐☐☐ *leave blank*

(5) *How many placentas (afterbirths) were there at birth?* → 34

1	Single	
2	2 Joined	
3	2 Separate	
4	Don't know	

(6) *Were there any difficulties at your birth? (please specify)*

---

35 ☐ *leave blank*

IF YOU AND YOUR TWIN ARE OF OPPOSITE SEX PLEASE GO TO QUESTION 9

36

(7) *As children were you and your twin mistaken by people who knew you?*

1	Frequently	
2	Sometimes	
3	Rarely	

(8) "Non-identical twins are no more alike than ordinary brothers and sisters. Identical twins on the other hand have such a strong resemblance to each other in stature, colouring, features of the face etc. that people often mistake one for the other".

Having read the above statement, do you think you are? 37

1	An identical twin	
2	A non-identical twin	

Comments: ---

(9) *During childhood were you and your twin ever separated from one another for more than a year?* ☐ No ☐ Yes

If Yes: *How old were you?* 38 ☐☐ age

How long for? ☐☐ years

(10) *How frequently do you and your twin see or contact each other?* 42 ☐ *leave blank*

1	We live together ☐		4	Once or twice a month ☐ see ☐ contact	
2	Almost every day ☐ see ☐ contact		5	A few times a year ☐ see ☐ contact	
3	At least once a week ☐ see ☐ contact		6	Less often ☐ see ☐ contact	

When did you start living apart? (if applicable) 44 ☐☐ age

(11) *Are there any other twins in your family? If so, please state exact relationship and whether twins are identical or non-identical. Attach a separate page if necessary.*

---

46 47 ☐☐ *leave blank*

GENERAL INFORMATION

(12)

	MARITAL STATUS 48	
1	Single	
2	Widowed	
3	Married	
4	Living together but not married	
5	Separated	
6	Divorced	
7	Remarried	

(13) *How many years have you been in your present marital state?* 49 50 ☐☐ yrs

(14) *Do you have any children?* 51 ☐ Yes ☐ No

If YES, please give their sex (male=M and female=F) and their year of birth. Indicate if any are by a previous spouse. Indicate if any are twins.

	Sex	Year of birth	*Leave this column blank*
1			52 ☐☐☐
2			55 ☐☐☐
3			58 ☐☐☐
4			61 ☐☐☐
5			64 ☐☐☐
6			67 ☐☐☐

(15)

	EDUCATIONAL ACHIEVEMENT 71	
1	Less than 7 years schooling	
2	8-10 years schooling	
3	11-12 years schooling	
4	Apprenticeship, diploma, certificate, etc.	
5	Technical or Teachers college	
6	University first degree	
7	University post-graduate training	

Leave blank 70 ☐

(16) *YOUR MAIN OCCUPATION - please describe in detail. e.g. "maintenance fitter in oil refinery".*

	FULL TIME ☐
	PART TIME ☐
	UNEMPLOYED ☐

(17) *YOUR FATHER'S MAIN OCCUPATION (before retirement)*

Leave blank 72 ☐☐☐☐☐ 76

(18)

	RELIGION	*Your religion*	*Father's religion*	*Mother's religion*
		77	78	79
1	No religion			
2	Church of England			
3	Other Protestant			
4	Catholic			
5	Jewish			
6	Greek or Russian Orthodox			
7	Other, please specify			
8	Prefer not to answer			

(19) *How often do you attend church (or other observance)* 80

1	more than once a week	
2	once a week	
3	every month or so	
4	once or twice a year	
5	rarely	

LIFESTYLE & HEALTH

(20) *On average, how many cups of TEA would you drink?* → 81 ☐☐

On average, how many cups of COFFEE would you drink? → 83 ☐☐

Cups each day

(21) *If you were to drink COFFEE in the evening would it stop you from getting to sleep at night?* →

		85
1	always	
2	usually	
3	sometimes	
4	never	

(22) *Which of the following do you take regularly?* leave blank 86 91

1	Asprin, Panadol, Vincents, etc.		6	Laxatives	
2	Sedatives or sleeping tablets		7	Tablets for high blood pressure	
3	Tranquillisers (eg. Valium)		8	Tablets to remove fluid (diuretics)	
4	Antidepressant tablets		9	Tablets for angina (heart pain)	
5	Vitamin tablets		0	Other (specify)	

(23) *Rate your leisure activity using the following as a guide (tick one box)* 92

1	Jogging, cycling to work or vigorous sport (squash, swimming etc.) 3-4 times a week		3	Regular exercise (eg. tennis, golf, etc.) about once a week	
2	Play sport or exercise a couple of times a week		4	Occasional exercise (2-3 times a month) or regular light gardening	
			5	No leisure exercise or sport	

(24) *What is your height?* → ☐ ft ☐☐ ins OR 93 ☐☐☐ Cms

(25) *What is your weight?* → ☐☐ st ☐☐ lbs OR 96 ☐☐☐ Kg

(26) *Do you consider you have good health?* → 99

1	Yes	
2	No	

(27) *In the past 12 months, on how many days would you have stayed home from work because of illness?* → 100 ☐☐ Days

(28)

Do you or any of your blood relatives have any of the following conditions?		Your Self	Your Twin	Blood relatives (specify) 3
Stuttering	102			
Dyslexia, Word blindness, Specific learning disability, Severe reading problems	103			
Autism	104			

(29) *Have you EVER been a smoker? If NO go to next page.* →

1	Yes	105
2	No	

(30) *At what age did you start smoking?* → 106 ☐☐ Years

(31) *If you have stopped smoking, how old were you when you stopped?*

Stopped cigarettes at	→	108 ☐☐ Years
Stopped pipe at	→	☐☐ Years
Stopped cigars at	→	☐☐ Years

(32)

How many CIGARETTES do (or did) you usually smoke in a day?	→	114 ☐☐
How many CIGARS do (or did) you usually smoke in a day?	→	☐☐
How many PIPES of tobacco do (or did) you usually smoke in a day?	→	☐☐
What was the LARGEST number of cigarettes, per day, that you regularly smoked?	→	☐☐

20
2

(33) *Have you EVER taken alcoholic drinks? IF NO go to next page.* → 21

1	Yes
2	No

(34) *At what age did you start drinking alcohol?* → 22 age

(35) *OVER THE LAST YEAR, about how often have you usually taken any alcoholic drinks?* → 24

1	Every day
2	3-4 times each week
3	About twice a week
4	About once a week
5	Once or twice a month
6	Less often

(36) *OVER THE WEEKEND (Saturday & Sunday) would you usually drink?* → 25

1	On two days
2	On one day
3	Not usually

(37) *DURING WEEK DAYS (Monday to Friday) would you usually drink?* → 26

1	Each day
2	On three or four days
3	On two days
4	On one day
5	Not usually

(38) *IF OVER 30, how do your present drinking habits compare with when you were 25-30?* → 27

1	About the same
2	Drink LESS now
3	Drink MORE now

(39) *On average, how many GLASSES would you drink on each day that you take some alcohol?*
Please note:
Beer glass is about 7oz.
Wine glass is about 4oz.
Spirits glass is about 1oz.(1 nip)

28	Weekdays	Weekends
Glasses of beer per day		
Glasses of wine per day		
Glasses of spirits per day		
Glasses of sherry per day		
Other (specify type and amount)		

(40) *Please describe your consumption of alcohol in the LAST WEEK. Write in the chart below the number of glasses you had on each day.*

	Mon	Tues	Wed	Thur	Fri	Sat	Sun
Beer 48							
Wines 62							
Spirits 76							
Sherry 90							
Other 104							

113

(41) *During the LAST WEEK, was your consumption?* →

1	Typical
2	Greater than average
3	Less than average

(42)

How OFTEN have you had any of the following? Tick each condition.	1 Never	2 Only as a child	3 Rarely	4 Quite often	*Tick each condition*	1 Never	2 Only as a child	3 Rarely	4 Quite often
Migraine/sick headaches 21					Warts on skin 32				
Other headaches					Boils or bad pimples				
Sore throats					Psoriasis				
Cold sores (eg. on lip)					Eczema				
Influenza					Other skin trouble				
Glandular fever					Allergy to dust				
Hay fever					Allergy to food				
Sinus trouble					Other allergy				
Bronchitis (chest cold)					Stiff joints in morning				
Asthma or wheezing					Arthritis or rheumatics				
Pneumonia 31					Kidney/bladder infections 42				

(43)

Have you had any of the following conditions? If YES please tick the left hand column. If any of your close relatives has had any of the same conditions (or died from them) BEFORE THE AGE OF 60, please tick in the appropriate columns.	1 Yourself	2 Your Twin	3 Your Mother	4 Your Father	5 Your Spouse	6 Your Sisters	7 Your Brothers	9 Your Children
High blood pressure (hypertension) 43								
Heart attack (infarct or coronary)								
Diabetes								
Stroke (or apoplexy)								
Thyroid trouble								
Ulcer of stomach, duodenum or small bowel								
Jaundice, hepatitis or liver disease								
Piles (Haemorrhoids)								
Diverticulitis								
Cancer or Leukemia (including skin cancer)								
Lump or cyst in breast								
Rheumatoid Arthritis								
Prostate trouble (enlarged prostate) 55								

(44)

OPERATIONS (Don't forget to give age)		No 0	Yes 1	At age
Have you had:	tonsils out? 56			
	appendix out? 59			
	gall bladder removed? 62			
	thyroid gland removed? 65			
	varicose veins done? 68			
MEN ONLY - Have you had:	a vasectomy? 71			
	a prostatectomy? 74			

(45) *Do you have any SERIOUS problem requiring medical treatment? (please specify)*

77

Leave blank

(46)

On WEEKDAYS, what time do you usually go to bed at night?	.....
Does this vary by more than 15 minutes, either way?	☐ No ☐ Yes
On WEEKDAYS after you go to bed, what time do you usually try to get to sleep?	.....
On WEEKDAYS, how long do you think it usually takes you to fall asleep from when you first try to go to sleep?	....hrs.....mins
IF YOU READ TILL YOU FALL ASLEEP, estimate how long you read for:	....hrs.....mins
On WEEKDAYS, how much sleep do you usually get at night?	....hrs.....mins
At WEEKENDS, do you usually get: 1 More than this; 2 About the same; 3 Less	

79 ☐☐☐☐

80 ☐

81 ☐☐☐☐

87 ☐☐☐☐

90 ☐☐☐

93 ☐☐☐☐

97 ☐

(47) *How often does it take you much longer than usual to get off to sleep?* 98

1	Less than once a month	☐
2	1-4 times per month	☐
3	More than once a week	☐
4	Most nights	☐

(48) *How often do you wake up fully during the night?* 99

1	Less than once a month	☐
2	1-4 times per month	☐
3	More than once a week	☐
4	Most nights	☐

(49) *If you wake up during the night, what is the usual reason? GIVE THE THREE MOST COMMON REASONS* Leave blank 100 ☐☐☐☐☐☐

01	Don't know, awake spontaneously	☐	07	Hungry or thirsty	☐
02	Nervous tension, worries	☐	08	Dreams or nightmares	☐
03	Need to pass urine	☐	09	Children or spouse	☐
04	Shortness of breath, coughing	☐	10	Noise	☐
05	Aches or pains	☐	11	Other (specify)	☐
06	Too hot or too cold	☐	12	Never wake during the night	☐

(50) *How would you describe the quality of your usual sleep over the last few months? Would it be:* →

106

1	Very good	☐
2	Good	☐
3	Fair	☐
4	Poor	☐
5	Very poor	☐

(51) *In particular, how would you describe the depth of your sleep? Are you:* →

107

1	Easy to wake	☐
2	About average	☐
3	Hard to wake	☐

(52) *How much would you say the quality of your sleep varies from one night to the other? Would it be:* →

108

1	Very much	☐
2	Moderately	☐
3	Slightly	☐
4	Not at all	☐

(53) *How often do you doze or sleep during the day (including evenings before going to bed and weekends)* →

109

1	Less than once a month	☐
2	1-4 times per month	☐
3	More than once a week	☐
4	Most days	☐

(54) *If you doze, for how long at a time?* →

110 ☐ hrs 111 ☐☐ mins

REPRODUCTIVE HISTORY - WOMEN ONLY - MEN GO TO NEXT PAGE

(55) How old were you when you had your FIRST menstrual period? → [21] Years [23] Months

(56) Have you EVER used a contraceptive pill? If NO go to question 57 → [22]
1 Yes
2 No

How long, in total, have you taken the contraceptive pill (add up all the months) → [26] Years [28] Months

Are you using a contraceptive pill now? → [32]
1 Yes
2 No

IF YES: Did the contraceptive pill cause any upset in you or your health? leave blank [31]

0	No upset	5	Acne (pimples)
1	Weight loss	6	Feeling bloated (swollen)
2	Weight gain	7	Depression (sadness)
3	Nausea (feeling sick)	8	Irritability (easily upset)
4	Irregular cycles (breakthrough bleeding, irregular bleeding)	9	Other (specify)

(57) Have you EVER been pregnant? If NO go to question 58 → [36]
1 Yes
2 No

Are you pregnant now? → [37]
1 Yes
2 No

How many pregnancies have you had? → [38] Number

How old were you with the first pregnancy? → [42] Years

IF YES: Did any pregnancy end in miscarriage or termination? 0 No 1 Yes → at Years; → at Years

With any pregnancy have you had any of the following? leave blank [43]

1	High blood pressure, toxemia	4	Jaundiced baby
2	Difficult birth (forceps, caesarian section)	5	Other birth problems (specify)
3	Stillborn child		

(58) Are your menstrual periods regular? If YES go to question 59 → [50]
1 Yes
2 No

If not regular, have your periods completely stopped? → [51]
1 Yes
2 No

WAS THIS DUE TO:
- Menopause ("natural change of life")? [52] No Yes → at Years
- Hysterectomy (removal of the womb)? [53] No Yes → at Years
- Other (specify) No Yes → at Years

(59) If your periods are more or less regular, what is the average number of days of bleeding? → [61] Days

(60)

Are your periods? 63		Are your periods? 64		Are your periods? 65	
1	Heavy	1	Very painful	1	Very limiting
2	Moderate	2	Moderately painful	2	Moderately limiting
3	Light	3	No trouble	3	Not limiting

(61) What is the average time between the start of one period and the start of the next? → [66] Days

What is the shortest? → Don't know [68] Days

What is the longest? → Don't know [70] Days

		FEELINGS The following statements describe feelings people may have. For each statement please TICK THE BOX which best describes how you are feeling.	1 Not at all	2 A little	3 A lot	4 Unbearably
72	1	Recently I have worried about every little thing.				
	2	Recently I have been so miserable that I have had difficulty with my sleep.				
	3	Recently I have been breathless OR had a pounding of my heart.				
	4	Recently I have been so 'worked up' that I couldn't sit still.				
	5	Recently I have been depressed without knowing why.				
	6	Recently I have gone to bed not caring if I never woke up.				
	7	Recently, for no good reason, I have had feelings of panic.				
	8	Recently I have been so low in spirits that I have sat for ages doing absolutely nothing.				
	9	Recently I have had a pain OR tense feeling in my neck or head.				
	10	Recently the future has seemed hopeless.				
	11	Recently worrying has kept me awake at night.				
	12	Recently I have lost interest in just about EVERYTHING.				
	13	Recently I have been so anxious that I couldn't make up my mind about the simplest thing.				
85	14	Recently I have been so depressed that I have thought of doing away with myself.				

PLEASE CHECK TO SEE THAT YOU HAVE ANSWERED ALL THE QUESTIONS

20

5

		PERSONALITY Please answer each question by PUTTING A CIRCLE AROUND THE "YES" OR THE "NO" following the question. There are no right or wrong answers, and no trick questions. Work quickly and do not think too long about the exact meaning of the questions. PLEASE REMEMBER TO ANSWER EACH QUESTION	1	0
21	1	Do you have many different hobbies?	YES	NO
	2	Do you stop to think things over before doing anything?	YES	NO
	3	Does your mood often go up and down?	YES	NO
	4	Have you ever taken the praise for something you knew someone else had really done?	YES	NO
	5	Are you a talkative person?	YES	NO
	6	Would being in debt worry you?	YES	NO
	7	Do you ever feel "just miserable" for no reason?	YES	NO
	8	Were you ever greedy by helping yourself to more than your share of anything?	YES	NO
	9	Do you lock up your house carefully at night?	YES	NO
30	10	Are you rather lively?	YES	NO

31	11	*Would it upset you a lot to see a child or an animal suffer?*	YES	NO
	12	*Do you often worry about things you should not have done or said?*	YES	NO
	13	*If you say you will do something, do you always keep your promise no matter how inconvenient it might be?*	YES	NO
	14	*Can you usually let yourself go and enjoy yourself at a lively party?*	YES	NO
	15	*Are you an irritable person?*	YES	NO
	16	*Have you ever blamed someone for doing something you knew was really your fault?*	YES	NO
	17	*Do you enjoy meeting new people?*	YES	NO
	18	*Do you believe insurance schemes are a good idea?*	YES	NO
	19	*Are your feelings easily hurt?*	YES	NO
	20	*Are ALL your habits good and desirable ones?*	YES	NO
	21	*Do you tend to keep in the background on social occasions?*	YES	NO
	22	*Would you take drugs which may have strange or dangerous effects?*	YES	NO
	23	*Do you often feel "fed-up"?*	YES	NO
	24	*Have you ever taken anything (even a pin or button) that belonged to someone else?*	YES	NO
	25	*Do you like going out a lot?*	YES	NO
	26	*Do you enjoy hurting people you love?*	YES	NO
	27	*Are you often troubled about feelings of guilt?*	YES	NO
	28	*Do you sometimes talk about things you know nothing about?*	YES	NO
	29	*Do you prefer reading to meeting people?*	YES	NO
	30	*Do you have enemies who want to harm you?*	YES	NO
	31	*Would you call yourself a nervous person?*	YES	NO
	32	*Do you have many friends?*	YES	NO
	33	*Do you enjoy practical jokes that can sometimes really hurt people?*	YES	NO
	34	*Are you a worrier?*	YES	NO
	35	*As a child did you do as you were told immediately and without grumbling?*	YES	NO
	36	*Would you call yourself happy-go-lucky?*	YES	NO
	37	*Do good manners and cleanliness matter much to you?*	YES	NO
	38	*Do you worry about awful things that might happen?*	YES	NO
	39	*Have you ever broken or lost something belonging to someone else?*	YES	NO
	40	*Do you usually take the initiative in making new friends?*	YES	NO
	41	*Would you call yourself tense or "highly-strung"?*	YES	NO
	42	*Are you mostly quiet when you are with other people?*	YES	NO
	43	*Do you think marriage is old-fashioned and should be done away with?*	YES	NO
	44	*Do you sometimes boast a little?*	YES	NO
	45	*Can you easily get some life into a rather dull party?*	YES	NO
	46	*Do people who drive carefully annoy you?*	YES	NO
	47	*Do you worry about your health?*	YES	NO
	48	*Have you ever said anything bad or nasty about anyone?*	YES	NO
	49	*Do you like telling jokes and funny stories to your friends?*	YES	NO
70	50	*Do most things taste the same to you?*	YES	NO

			1	0
71	51	*As a child were you ever cheeky to your parents?*	YES	NO
	52	*Do you like mixing with people?*	YES	NO
	53	*Does it worry you if you know there are mistakes in your work?*	YES	NO
	54	*Do you suffer from sleeplessness?*	YES	NO
	55	*Do you always wash before a meal?*	YES	NO
	56	*Do you nearly always have a "ready answer" when people talk to you?*	YES	NO
	57	*Do you like to arrive at appointments in plenty of time?*	YES	NO
	58	*Have you often felt listless and tired for no reason?*	YES	NO
	59	*Have you ever cheated at a game?*	YES	NO
	60	*Do you like doing things in which you have to act quickly?*	YES	NO
	61	*Is (or was) your mother a good woman?*	YES	NO
	62	*Do you often feel life is very dull?*	YES	NO
	63	*Have you ever taken advantage of someone?*	YES	NO
	64	*Do you often take on more activities than you have time for?*	YES	NO
	65	*Are there several people who keep trying to avoid you?*	YES	NO
	66	*Do you worry a lot about your looks?*	YES	NO
	67	*Do you think people spend too much time safeguarding their future with savings and insurances?*	YES	NO
	68	*Have you ever wished that you were dead?*	YES	NO
	69	*Would you dodge paying taxes if you were sure you could never be found out?*	YES	NO
	70	*Can you get a party going?*	YES	NO
	71	*Do you try not to be rude to people?*	YES	NO
	72	*Do you worry too long after an embarrassing experience?*	YES	NO
	73	*Have you ever insisted on having your own way?*	YES	NO
	74	*When you catch a train do you often arrive at the last minute?*	YES	NO
	75	*Do you suffer from "nerves"?*	YES	NO
	76	*Do your friendships break up easily without it being your fault?*	YES	NO
	77	*Do you often feel lonely?*	YES	NO
	78	*Do you always practice what you preach?*	YES	NO
	79	*Do you sometimes like teasing animals?*	YES	NO
	80	*Are you easily hurt when people find fault with you or the work you do?*	YES	NO
	81	*Have you ever been late for an appointment or work?*	YES	NO
	82	*Do you like plenty of bustle and excitement around you?*	YES	NO
	83	*Would you like other people to be afraid of you?*	YES	NO
	84	*Are you sometimes bubbling over with energy and sometimes very sluggish?*	YES	NO
	85	*Do you sometimes put off until tomorrow what you ought to do today?*	YES	NO
	86	*Do other people think of you as being very lively?*	YES	NO
	87	*Do people tell you a lot of lies?*	YES	NO
	88	*Are you touchy about some things?*	YES	NO
	89	*Are you always willing to admit it when you have made a mistake?*	YES	NO
110	90	*Would you feel very sorry for an animal caught in a trap?*	YES	NO

PLEASE CHECK TO SEE THAT YOU HAVE ANSWERED ALL THE QUESTIONS

ATTITUDES

Here is a list of various topics. For each one please indicate whether or not you agree with it by CIRCLING "YES" or "NO" as appropriate. If you are uncertain please circle "?". It's just your first reaction we want so please do not spend too long on any one topic

21

		2	1	0			2	1	0
1	Death penalty	Yes	?	No	26	Computer music	Yes	?	No
2	Evolution theory	Yes	?	No	27	Chastity	Yes	?	No
3	School uniforms	Yes	?	No	28	Fluoridation	Yes	?	No
4	Striptease shows	Yes	?	No	29	Royalty	Yes	?	No
5	Sabbath observance	Yes	?	No	30	Women judges	Yes	?	No
6	Hippies	Yes	?	No	31	Conventional clothes	Yes	?	No
7	Patriotism	Yes	?	No	32	Teenage drivers	Yes	?	No
8	Modern Art	Yes	?	No	33	Apartheid	Yes	?	No
9	Self-denial	Yes	?	No	34	Nudist camps	Yes	?	No
10	Working mothers	Yes	?	No	35	Church authority	Yes	?	No
11	Horoscopes	Yes	?	No	36	Disarmament	Yes	?	No
12	Birth control	Yes	?	No	37	Censorship	Yes	?	No
13	Military drill	Yes	?	No	38	White lies	Yes	?	No
14	Co-education	Yes	?	No	39	Caning	Yes	?	No
15	Divine law	Yes	?	No	40	Mixed marriage	Yes	?	No
16	Socialism	Yes	?	No	41	Strict rules	Yes	?	No
17	White superiority	Yes	?	No	42	Jazz	Yes	?	No
18	Cousin marriage	Yes	?	No	43	Strait-jackets	Yes	?	No
19	Moral training	Yes	?	No	44	Casual living	Yes	?	No
20	Suicide	Yes	?	No	45	Learning Latin	Yes	?	No
21	Chaperones	Yes	?	No	46	Divorce	Yes	?	No
22	Legalized abortion	Yes	?	No	47	Inborn conscience	Yes	?	No
23	Empire-building	Yes	?	No	48	Coloured immigration	Yes	?	No
24	Student pranks	Yes	?	No	49	Bible truth	Yes	?	No
25	Licensing laws	Yes	?	No	50	Dating parties	Yes	?	No

The information you have given forms a basis for long-term research using the Australian Twin Registry. We hope you will also help in other research projects over the next few years. The results will be published in scientific and medical journals, and summaries of the most important findings will then appear in the daily press for the information of twins and the population at large. In future mailings we hope to include a newsletter which will tell you about progress with the research.

We do thank you for your help in answering so many detailed personal questions, and we believe that the results of this research will justify your time and effort.

One last thing! Do you know any other twins (any age or type) who we could write to, or perhaps we could send you some of our Australian Twin Registry registration pamphlets for you to distribute? (Please attach a page if you need more space).

TWIN NAMES:

ADDRESS:

Please send me ..... Australian Twin Registry pamphlets to distribute.

PLEASE ATTACH ANY COMMENTS YOU WOULD LIKE TO MAKE ON AN ADDITIONAL PAGE.

THANK YOU VERY MUCH FOR YOUR HELP