

A TWIN STUDY OF PERSONALITY, SOCIAL ATTITUDES  
AND DRINKING BEHAVIOUR

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## 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 occurrence 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. <sup>\*</sup> Thus, there is evidence of a substantial genetic component in variation in <sup>symptoms of</sup> anxiety, but much less clear evidence for <sup>symptoms of</sup> 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.

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Anxiety

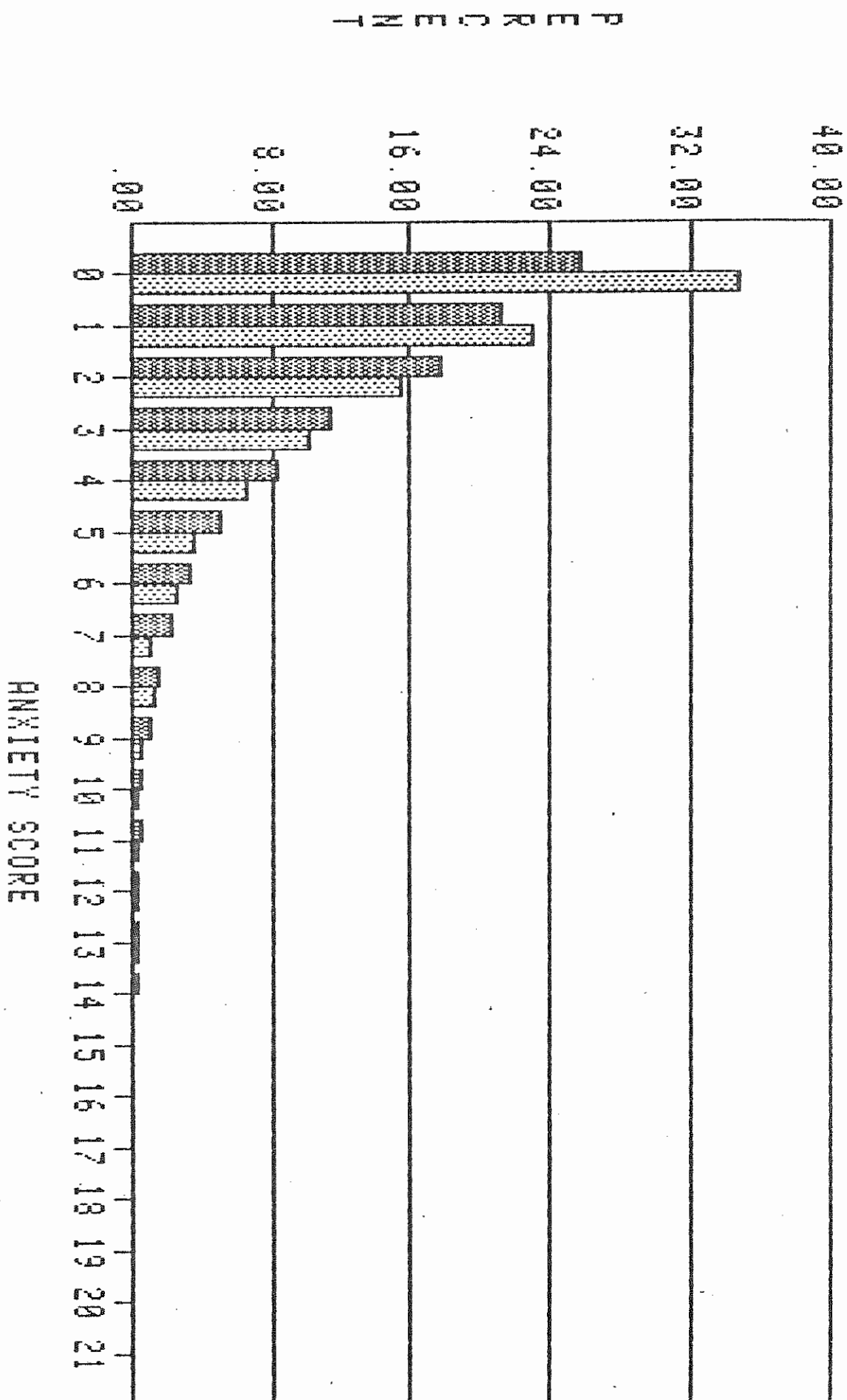
- A1. Recently I have worried about every little thing.
- A2. Recently I have been breathless OR had a pounding of my heart.
- A3. Recently I have been so 'worked up' that I couldn't sit still.
- A4. Recently for no good reason, I have had feelings of panic.
- A5. Recently I have had a pain OR tense 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

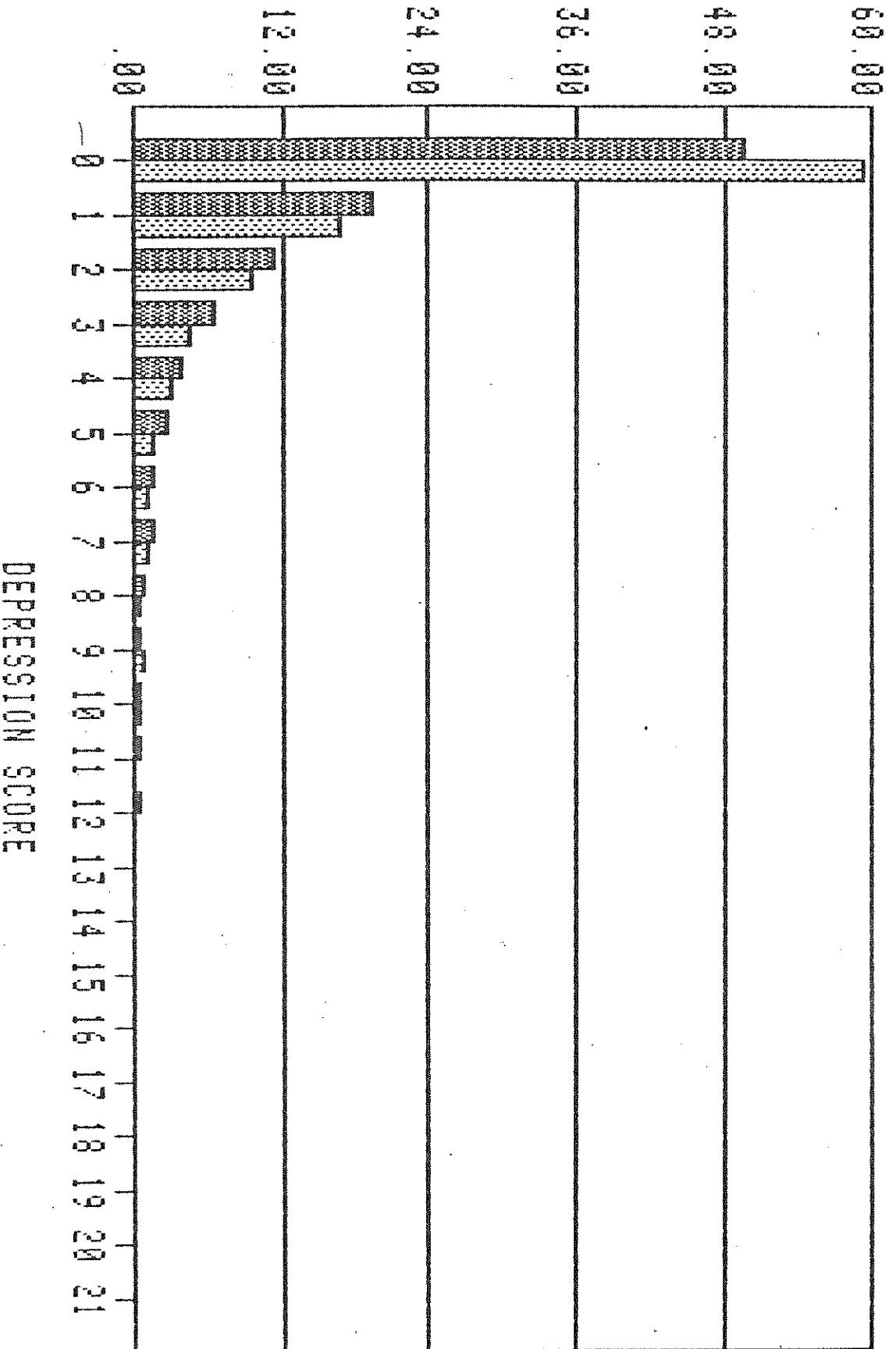
- D1. Recently I have been so miserable that I have had difficulty  
with my sleep.
  - D2. Recently I have been depressed without knowing why.
  - D3. Recently I have gone to bed not caring if I never woke up.
  - D4. Recently I have been so low in spirits that I have sat for ages  
doing absolutely 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.
-

MALES  
FEMALES

DISTRIBUTION OF ANXIETY SCORES



P E R C E N T



MALES  
FEMALES

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.

	MZ F		MZ M	
	L	Q	L	Q
Anxiety				
Raw	.32 <sup>***</sup>	.03 <sup>***</sup>	.44 <sup>***</sup>	.04 <sup>***</sup>
Angle	.14 <sup>***</sup>	.02 <sup>***</sup>	.23 <sup>***</sup>	.04 <sup>***</sup>
$\sqrt{x+1}$	.11 <sup>***</sup>	.05 <sup>***</sup>	.21 <sup>***</sup>	.06 <sup>***</sup>
$\log_{10}(x+1)$	.00 <sup>*</sup>	.11 <sup>***</sup>	.05 <sup>***</sup>	.15 <sup>***</sup>
Depression				
Raw	.47 <sup>***</sup>	.05 <sup>***</sup>	.64 <sup>***</sup>	.03 <sup>***</sup>
Angle	.33 <sup>***</sup>	.05 <sup>***</sup>	.49 <sup>***</sup>	.05 <sup>***</sup>
$\sqrt{x+1}$	.30 <sup>***</sup>	.09 <sup>***</sup>	.46 <sup>***</sup>	.08 <sup>***</sup>
$\log_{10}(x+1)$	.15 <sup>***</sup>	.21 <sup>***</sup>	.30 <sup>***</sup>	.21 <sup>***</sup>

$\sqrt{\log_{10}(x+1)}$ ] 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
<b>Anxiety</b>				
Raw	2.37 <sup>***</sup>	6.92 <sup>***</sup>	1.82	4.88
$\log_{10}(x+1)$	0.42 <sup>***</sup>	0.10 <sup>*</sup>	0.34	0.09
<b>Depression</b>				
Raw	1.50 <sup>***</sup>	6.40 <sup>***</sup>	1.12	4.41
$\log_{10}(x+1)$	0.26 <sup>***</sup>	0.10 <sup>***</sup>	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	$\log_{10}(x+1)$
<u>Females</u>				
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
<u>Males</u>				
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 <sup>a</sup>	901	0.083	901	0.082

<sup>a</sup> 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 <sup>***</sup>	-.01	-.13 <sup>*</sup>	-.14 <sup>**</sup>

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	$\chi^2$	$h^2$
<u>Female</u>							
$E_1E_2$	.068 <sup>***</sup>	.030 <sup>***</sup>	-	-	2	25.47 <sup>***</sup>	
$E_1V_A$	.061 <sup>***</sup>	-	.037 <sup>***</sup>	-	2	0.23	.38 ± .02
$E_1E_2V_A$	.060 <sup>***</sup>	-.002	.039 <sup>***</sup>	-	1	0.16	
$E_1V_AV_D$	.060 <sup>***</sup>	-	.033 <sup>**</sup>	.004	1	0.16	
<u>Male</u>							
$E_1E_2$	.064 <sup>***</sup>	.024 <sup>***</sup>	-	-	2	15.06 <sup>**</sup>	
$E_1V_A$	.058 <sup>***</sup>	-	.031 <sup>***</sup>	-	2	1.29	.35 ± .03
$E_1E_2V_A$	.056 <sup>***</sup>	-.010	.042 <sup>***</sup>	-	1	0.26	
$E_1V_AV_D$	.056 <sup>***</sup>	-	.012	.020	1	0.26	
<u>Female &amp; Male</u>							
$E_1E_2$	.067 <sup>***</sup>	.028 <sup>***</sup>	-	-	6	45.37 <sup>***</sup>	
$E_1V_A$	.060 <sup>***</sup>	-	.035 <sup>***</sup>	-	6	6.70	.37 ± .02
$E_1E_2V_A$	.059 <sup>***</sup>	-.004	.040 <sup>***</sup>	-	5	6.23	
$E_1V_AV_D$	.059 <sup>***</sup>	-	.027 <sup>**</sup>	.009	5	6.23	
<u>Female &amp; Male &amp; Opposite-sex</u>							
$E_1E_2$	.071 <sup>***</sup>	.024 <sup>***</sup>	-	-	8	67.68 <sup>***</sup>	
$E_1V_A$	.060 <sup>***</sup>	-	.034 <sup>***</sup>	-	8	10.36	.36 ± .02
$E_1E_2V_A$	.059 <sup>***</sup>	-.008	.043 <sup>***</sup>	-	7	7.33	
$E_1V_AV_D$	.059 <sup>***</sup>	-	.020 <sup>*</sup>	.016 <sup>*</sup>	7	7.33	

Table 5.9 Estimates ( $\pm$  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 <sup>***</sup>	0.030 <sup>***</sup>	0.038 <sup>***</sup>	0.023 <sup>***</sup>
$\pm$	0.002	0.003	0.002	0.006
$\chi^2_6 = 2.15$ (p = .91)				
	$h^2_{\text{males}} = 0.33 \pm .03$		$h^2_{\text{females}} = 0.39 \pm .02$	

females causes a significant reduction in chi-square ( $\chi_1^2 = 5.18$ ,  $p > .05$ ). The correlation  $r_{V_{AMF}} = 0.67$  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 ( $\chi_2^2 = 27.26$ ,  $p < .001$ ) 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 ( $\chi_3^2 = 24.97$ ,  $p > .001$ ). The correlation  $r_{V_{AMF}} = 0.73$  *(the correlation between  $V_A$  effects in males and females)* 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 ( $\chi_1^2 =$

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

	Females	Males
$E_1$ <ul style="list-style-type: none"> <li>error</li> <li>individual environment</li> </ul>	61 <ul style="list-style-type: none"> <li>38</li> <li>23</li> </ul>	67 <ul style="list-style-type: none"> <li>45</li> <li>22</li> </ul>
$V_A$	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	$\chi^2$	$h^2$
<u>Female</u>							
$E_1E_2$	.069 <sup>***</sup>	.028 <sup>***</sup>	-	-	2	22.04 <sup>***</sup>	
$E_1V_A$	.062 <sup>***</sup>	-	.035 <sup>***</sup>	-	2	1.20	.36 ± .02
$E_1E_2V_A$	.062 <sup>***</sup>	-.000	.035 <sup>***</sup>	-	1	1.20	
$E_1V_AV_D$	.062 <sup>***</sup>	-	.034 <sup>**</sup>	.001	1	1.20	
<u>Male</u>							
$E_1E_2$	.056 <sup>***</sup>	.021 <sup>***</sup>	-	-	2	2.46	
$E_1V_A$	.052 <sup>***</sup>	-	.025 <sup>***</sup>	-	2	1.85	.32 ± .04
$E_1E_2V_A$	.053 <sup>***</sup>	.010	.013	-	1	0.46	
$E_1V_AV_D$	.053 <sup>***</sup>	-	.044 <sup>**</sup>	-.021	1	0.46	
<u>Female &amp; Male</u>							
$E_1E_2$	.065 <sup>***</sup>	.026 <sup>***</sup>	-	-	6	54.72 <sup>***</sup>	
$E_1V_A$	.059 <sup>***</sup>	-	.032 <sup>***</sup>	-	6	30.31 <sup>***</sup>	
$E_1E_2V_A$	.059 <sup>***</sup>	.003	.028 <sup>***</sup>	-	5	30.22 <sup>***</sup>	
$E_1V_AV_D$	.059 <sup>***</sup>	-	.037 <sup>***</sup>	-.006	5	30.22 <sup>***</sup>	
<u>Female &amp; Male &amp; Opposite-sex</u>							
$E_1E_2$	.069 <sup>***</sup>	.023 <sup>***</sup>	-	-	8	76.11 <sup>***</sup>	
$E_1V_A$	.060 <sup>***</sup>	-	.032 <sup>***</sup>	-	8	33.70 <sup>***</sup>	
$E_1E_2V_A$	.059 <sup>***</sup>	-.002	.034 <sup>***</sup>	-	7	33.21 <sup>***</sup>	
$E_1V_AV_D$	.059 <sup>***</sup>	-	.028 <sup>***</sup>	.005	7	33.21 <sup>***</sup>	

Table 5.12 Estimates ( $\pm$  s.e.) obtained after fitting a model allowing different genetic and environmental components of variation in males and females for log transformed depression scores.

	$\hat{E}_{1M}$	$\hat{E}_{1F}$	$\hat{V}_{AM}$	$\hat{V}_{AF}$	$\hat{V}_{AMF}$
	0.053 <sup>***</sup>	0.062 <sup>***</sup>	0.026 <sup>***</sup>	0.036 <sup>***</sup>	0.022 <sup>***</sup>
$\pm$	0.003	0.002	0.003	0.003	0.006
$\chi^2_5 = 8.73$ (p = .12)					
$h^2_{\text{males}} = 0.33 \pm .03$			$h^2_{\text{females}} = 0.37 \pm .02$		

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
$E_1$ <ul style="list-style-type: none"> <li>error</li> <li>individual environment</li> </ul>	63 <ul style="list-style-type: none"> <li>33</li> <li>30</li> </ul>	67 <ul style="list-style-type: none"> <li>29</li> <li>38</li> </ul>
$V_A$	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
<u>Females</u>								
Anxiety	log(x+1)	-	.66***	-.08***	.15***	.61***	-.12***	-.01
Depression	log(x+1)	.66***	-	-.10***	.18***	.57***	-.09***	-.03*
<u>Males</u>								
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  $\Delta_j$  is the matrix of the factor loadings and  $\theta_j^2$  the diagonal matrix of specific variance components. Note, however, that we may complicate the model by introducing correlations between the factors in  $\Phi_j$ , 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 within 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  $S_i$  for the  $i$ -th matrix, having  $N_i$  degrees of freedom. Given some model for the  $S_i$ , we compute the expected values  $\Sigma_i$ , 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  $S_i$  is:

$$\log L = -\frac{1}{2} \sum_{i=1}^{i=k} N_i [\log |\Sigma_i| + \text{tr}(S_i \Sigma_i^{-1})]$$

(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.  $\Sigma_i = S_i$ . In this case we have simply:

$$L_0 = \frac{1}{2} \sum_{i=1}^{i=k} N_i [\log |S_i| + p]$$

When there are  $k$  matrices the  $\chi^2$  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  $-\text{Log } L$  for a given model. We have minimised  $-\text{Log } L$  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
<u>Females</u>					
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)		
<u>Males</u>					
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 unrepeatability 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	Anxiety	Depression
		Angle	log(x+1)	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	Anxiety	Depression
		Angle	log(x+1)	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	$E_1$	$E_{1M}$	$E_{1F}$	$E_{2M}$	$E_{2F}$	$E_{2MF}$	$V_A$	$V_D$
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.

Item	Parameter								
	$E_1$	$E_{1M}$	$E_{1F}$	$E_{2M}$	$E_{2F}$	$E_{2MF}$	$V_A$	$V_{AM}$	$V_{AF}$
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  $E_1M E_1F E_2M E_2F E_2MF$  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  $E_1M E_1F V_{AM} V_{AF}$  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  $E_1M E_1F E_2M E_2F E_2MF$  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 person's 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  $45^\circ$ . 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 heritabilities 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 individual 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.

<u>Item</u>	$\chi^2$	<u>Item</u>	$\chi^2$
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.

<u>Item</u>	$\chi^2$	<u>Item</u>	$\chi^2$
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.

Item	Liberal		Ambiguous		Conservative		$\chi^2$
	Female	Male	Female	Male	Female	Male	
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

Item	Liberal		Ambiguous		Conservative		$\chi^2$
	Female	Male	Female	Male	Female	Male	
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 significance, 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, *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 *more strongly identified in males than females.*

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.

<u>Factor 1: Militarism-punitiveness</u>			
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%
<u>Factor 2: Religion</u>			
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: Racism-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 accounted 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-tendermindedness 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

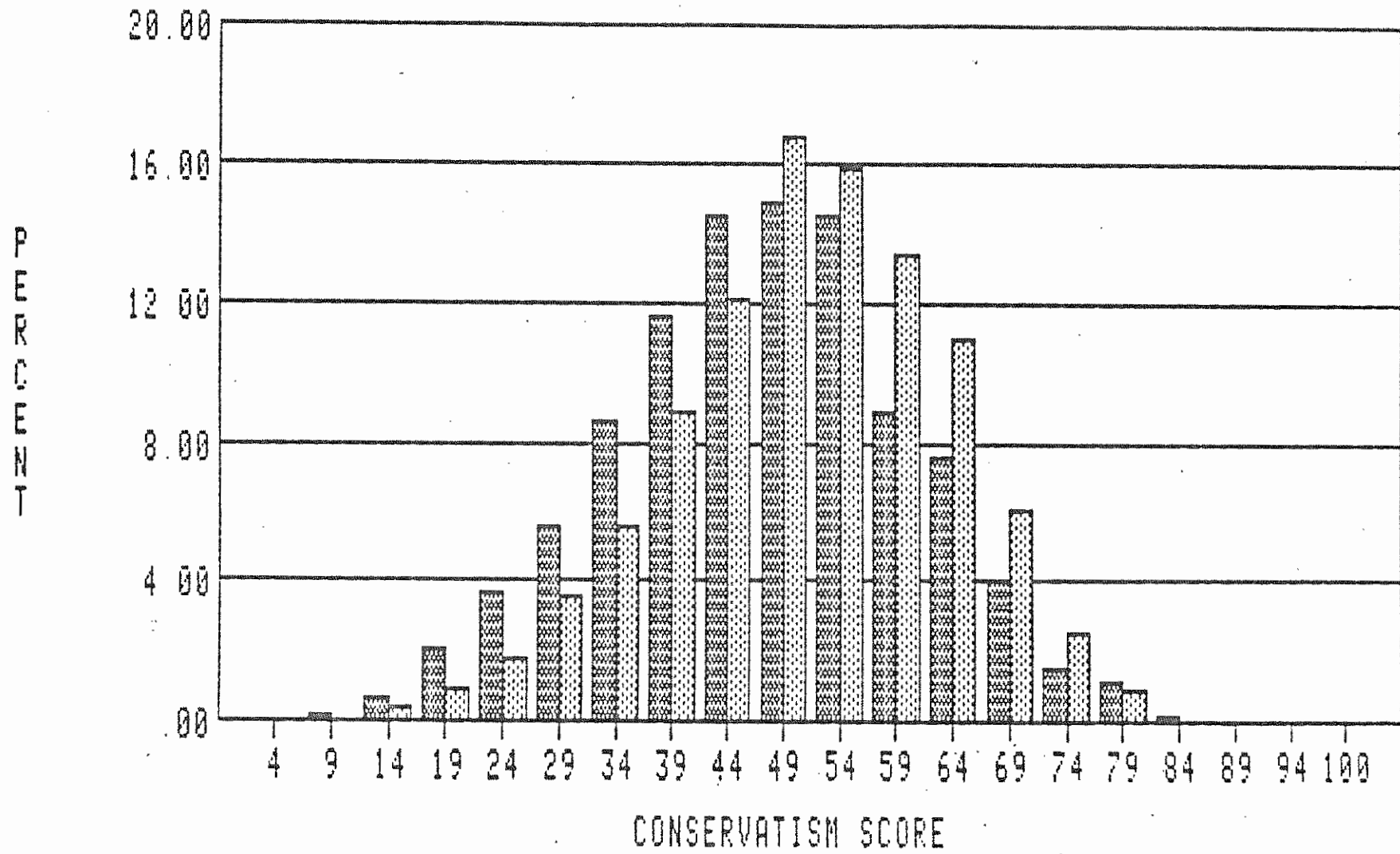
As a test of sampling, two-tailed t-tests were performed between MZ and DZ means and variance ratio tests between the total variances, separately for males and females (Table 6.7). There are no differences in means or variances between MZ and DZ male twins and the MZ and DZ female variances. The MZ female 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



 FEMALES  
 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 questionnaire. 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 <sup>a</sup>	1231	200.39
	Within	1233	43.66
MZ Male	Between <sup>a</sup>	564	250.49
	Within	566	62.44
DZ Female	Between <sup>a</sup>	749	175.62
	Within	751	64.25
DZ Male	Between <sup>a</sup>	350	238.67
	Within	352	85.06
DZ Opposite sex	Between <sup>a</sup>	904	179.92
	Within <sup>b</sup>	905	76.05

<sup>a</sup> Corrected for regression on age

<sup>b</sup> 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 <sup>***</sup>	0.12 <sup>***</sup>

---

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 between  $E_2$  effects in males and females)* The correlation  $R_{E_2MF} = 0.90$  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	$\chi^2$	$h^2$
<u>Female</u>							
$E_1E_2$	51.45 <sup>***</sup>	69.79 <sup>***</sup>	-	-	2	41.31 <sup>***</sup>	
$E_1V_A$	41.82 <sup>***</sup>	-	77.82 <sup>***</sup>	-	2	21.31 <sup>***</sup>	
$E_1E_2V_A$	43.58 <sup>***</sup>	35.67 <sup>***</sup>	41.92 <sup>***</sup>	-	1	0.11	.35 ± .06
$E_1V_AV_D$	43.58 <sup>***</sup>	-	148.92 <sup>***</sup>	-71.33	1	0.11	
<u>Male</u>							
$E_1E_2$	71.11 <sup>***</sup>	87.43 <sup>***</sup>	-	-	2	11.24 <sup>**</sup>	
$E_1V_A$	59.59 <sup>***</sup>	-	97.28 <sup>***</sup>	-	2	13.32 <sup>**</sup>	
$E_1E_2V_A$	62.69 <sup>***</sup>	52.71 <sup>***</sup>	43.28 <sup>**</sup>	-	1	0.20	.27 ± .09
$E_1V_AV_D$	62.69 <sup>***</sup>	-	201.41 <sup>***</sup>	-105.42	1	0.20	
<u>Female &amp; Male</u>							
$E_1E_2$	57.67 <sup>***</sup>	75.35 <sup>***</sup>	-	-	6	108.31 <sup>***</sup>	
$E_1V_A$	47.42 <sup>***</sup>	-	83.99 <sup>***</sup>	-	6	97.73 <sup>***</sup>	
$E_1E_2V_A$	49.58 <sup>***</sup>	40.95 <sup>***</sup>	42.50 <sup>***</sup>	-	5	60.97 <sup>***</sup>	
$E_1V_AV_D$	49.58 <sup>***</sup>	-	165.35 <sup>***</sup>	-81.90	5	60.97 <sup>***</sup>	
<u>Female &amp; Male &amp; Opposite-sex</u>							
$E_1E_2$	62.04 <sup>***</sup>	69.78 <sup>***</sup>	-	-	8	135.90 <sup>***</sup>	
$E_1V_A$	46.43 <sup>***</sup>	-	83.51 <sup>***</sup>	-	8	110.94 <sup>***</sup>	
$E_1E_2V_A$	49.45 <sup>***</sup>	34.33 <sup>***</sup>	47.97 <sup>***</sup>	-	7	64.41 <sup>***</sup>	
$E_1V_AV_D$	49.45 <sup>***</sup>	-	150.95 <sup>***</sup>	-68.66	7	64.41 <sup>***</sup>	

Table 6.12 Estimates ( $\pm$  s.e.) obtained after fitting a model allowing different environmental components of variation in males and females for age-corrected conservatism scores.

$\hat{V}_A$	$\hat{E}_{1M}$	$\hat{E}_{1F}$	$\hat{E}_{2M}$	$\hat{E}_{2F}$	$\hat{E}_{2MF}$
41.54***	62.05***	43.41***	49.41***	34.57***	37.13***
$\pm$ 6.34	3.26	1.69	7.49	6.12	4.96
		$\chi^2_4 = 4.40$ (p = .35)			
$h^2_{\text{males}} = 0.27 \pm .04$			$h^2_{\text{females}} = 0.35 \pm .05$		

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  $\mu$  the observed marital correlation (Eaves, 1977). If an estimate of  $\mu$  is available we can solve the quadratic equation

$$\begin{aligned} A &= h^2_{\mu} \\ &= \mu(V_A(1+(A/1-A)))/V_T \end{aligned}$$

(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  $\mu$  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.

		Females	Males
E <sub>1</sub>	error	18	9
	individual environment	18	32
total genetic		36	41
V <sub>A</sub>	assortative mating	14	11
	family environment	15	21
total genetic		29	32
total genetic		49	38

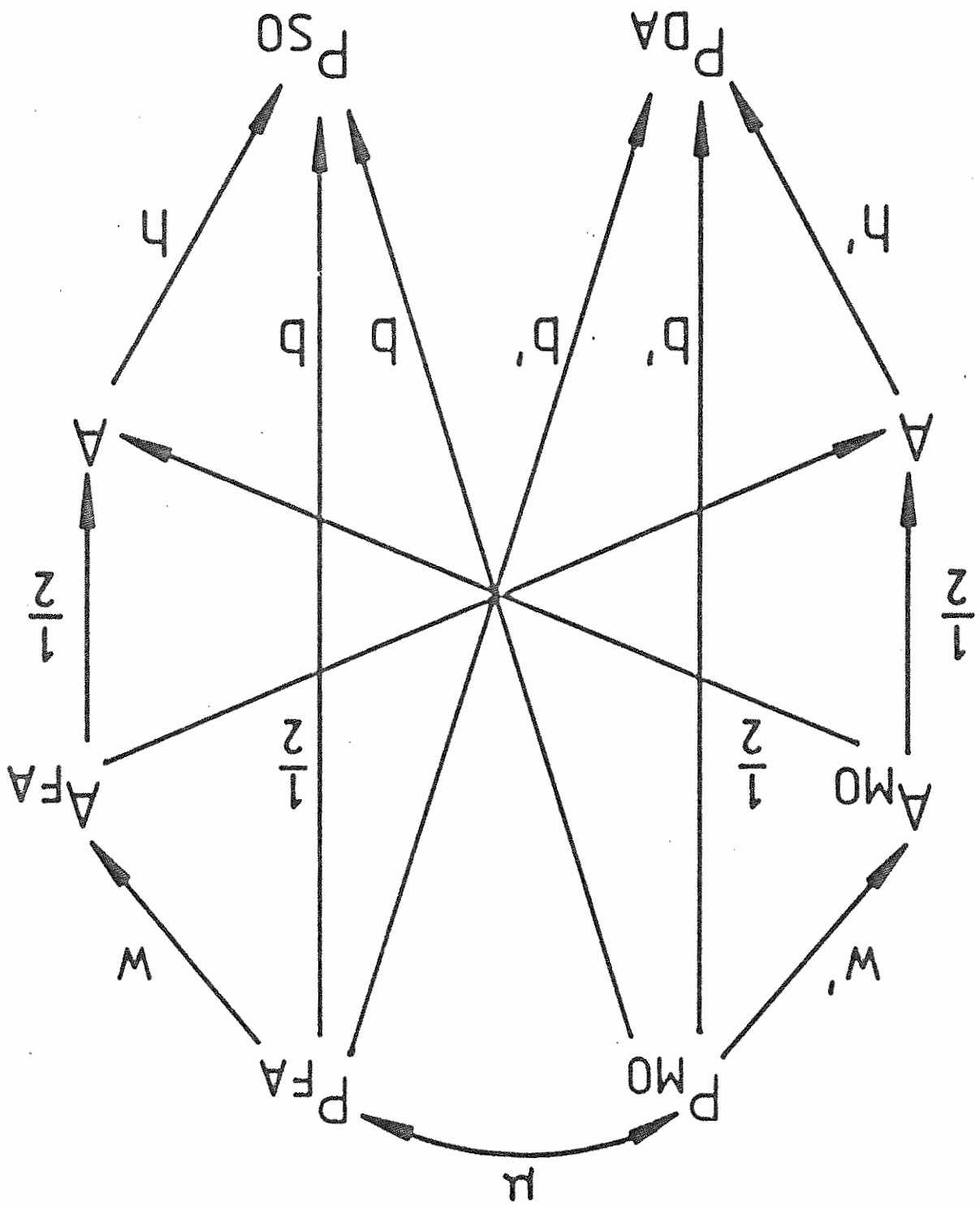
repeatable "error" and that due to repeatable individual environmental differences.

#### 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 phenotype is  $b$  in males and  $b'$  in females. The parameter  $b$ , therefore, represents cultural inheritance in the model. The phenotypic correlation between mates is  $\mu$ . 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  $\mu$  at equilibrium under cultural transmission and assortative mating. For readers unfamiliar with the technique of path analysis, a brief description follows.





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 standardised 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 routes 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  $\mu$  (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,  $h$ ,  $1/2bw$ ,  $1/2\mu bw$ ,  $1/2bw'$  and  $1/2\mu bw'$  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

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

where  $\underline{z}$  is the column vector of  $z$ -transformed observed correlations<sup>2</sup>,  $\bar{\underline{z}}$  the corresponding vector of  $z$ -transformed

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<sup>2</sup> 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.

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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	$\mu$

---

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,  $E$  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 (EO4UAF) from the Numerical Algorithms Group Library (1981). This procedure will yield maximum likelihood estimates of the parameters we choose to estimate. Since the  $z$ '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 ( $\mu$ ) 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'	$\mu$	df	$\chi^2$
1. $b\mu$	-	-	.40	-	.675	4	73.38***
2. $bb'\mu$	-	-	.38	.41	.675	3	68.68***
3. $h\mu$	.79	-	-	-	.675	4	4.61
4. $hh'\mu$	.78	.80	-	-	.675	3	2.84
5. $hb\mu$	.81	-	-.02	-	.675	3	4.46
6. $hh'bb'\mu$	.75	.83	.02	-.02	.675	1	2.70
1. $b\mu$	-	-	.44	-	.400	4	73.38***
2. $bb'\mu$	-	-	.42	.45	.400	3	68.68***
3. $h\mu$	.80	-	-	-	.400	4	7.31
4. $hh'\mu$	.79	.81	-	-	.400	3	6.04
5. $hb\mu$	.71	-	.07	-	.400	3	4.46
6. $hh'bb'\mu$	.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 response 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.<sup>†</sup>

Model	df	Item											
		1	2	3	4	5	6	7	8	9	10	11	12
E <sub>2</sub>	4	48.56 <sup>***</sup>	24.82 <sup>***</sup>	15.72 <sup>**</sup>	26.78 <sup>***</sup>	24.46 <sup>***</sup>	18.62 <sup>***</sup>	19.18 <sup>***</sup>	30.40 <sup>***</sup>	14.36 <sup>**</sup>	23.08 <sup>***</sup>	27.08 <sup>***</sup>	17.12 <sup>**</sup>
V <sub>A</sub>	4	<u>1.52</u>	24.56 <sup>***</sup>	11.22 <sup>*</sup>	18.66 <sup>***</sup>	9.58 <sup>*</sup>	23.16 <sup>***</sup>	7.90	13.60 <sup>**</sup>	<u>3.24</u>	<u>6.36</u>	8.44	19.04 <sup>***</sup>
E <sub>2</sub> V <sub>A</sub>	3	1.26	7.84 <sup>*</sup>	7.36	<u>7.50</u>	<u>0.58</u>	<u>2.94</u>	5.88	10.52 <sup>*</sup>	2.20	4.62	6.68	11.78 <sup>**</sup>
E <sub>2</sub> <sub>M</sub> E <sub>2</sub> <sub>F</sub> E <sub>2</sub> <sub>MF</sub> V <sub>A</sub> <sub>M</sub>	1	-	-	<u>0.18</u>	-	-	-	-	-	-	-	-	-
E <sub>2</sub> <sub>M</sub> E <sub>2</sub> <sub>F</sub> E <sub>2</sub> <sub>MF</sub> V <sub>A</sub> <sub>F</sub>	1	-	-	-	-	-	-	-	-	-	-	-	<u>0.14</u>
E <sub>2</sub> <sub>M</sub> E <sub>2</sub> <sub>F</sub> V <sub>A</sub> <sub>M</sub> V <sub>A</sub> <sub>F</sub>	1	-	-	-	-	-	<u>0.04</u>	<u>0.02</u>	-	-	-	<u>0.06</u>	-
- 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 <sub>2</sub>	4	25.56 <sup>***</sup>	<u>1.32</u>	13.76 <sup>**</sup>	16.62 <sup>**</sup>	16.56 <sup>**</sup>	17.04 <sup>**</sup>	8.76	21.36 <sup>***</sup>	28.82 <sup>***</sup>	23.06 <sup>***</sup>	36.46 <sup>***</sup>	20.48 <sup>***</sup>
V <sub>A</sub>	4	<u>3.76</u>	8.00	24.02 <sup>***</sup>	16.34 <sup>**</sup>	<u>4.54</u>	<u>1.14</u>	6.86	18.72 <sup>***</sup>	<u>7.56</u>	31.66 <sup>***</sup>	7.46	14.20 <sup>**</sup>
E <sub>2</sub> V <sub>A</sub>	3	0.78	1.12	<u>3.74</u>	<u>4.10</u>	3.70	0.74	<u>2.18</u>	13.78 <sup>**</sup>	7.52	<u>3.06</u>	6.04	<u>4.68</u>
V <sub>A</sub> <sub>M</sub> V <sub>A</sub> <sub>F</sub> V <sub>A</sub> <sub>MF</sub>	2	-	-	-	-	-	-	-	-	-	-	<u>0.50</u>	-
- 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 <sub>2</sub>	4	9.54 <sup>*</sup>	25.12 <sup>***</sup>	39.06 <sup>***</sup>	17.74 <sup>**</sup>	31.98 <sup>***</sup>	7.96	28.76 <sup>***</sup>	8.10	27.38 <sup>***</sup>	18.62 <sup>***</sup>	18.56 <sup>***</sup>	25.94 <sup>***</sup>
V <sub>A</sub>	4	9.22	<u>4.58</u>	27.36 <sup>***</sup>	<u>2.52</u>	6.74	<u>6.36</u>	<u>6.64</u>	<u>2.74</u>	<u>2.48</u>	30.32 <sup>***</sup>	13.60 <sup>**</sup>	<u>1.52</u>
E <sub>2</sub> V <sub>A</sub>	3	8.60 <sup>*</sup>	1.46	21.32 <sup>***</sup>	0.48	<u>2.66</u>	5.26	4.44	0.36	2.08	<u>1.84</u>	<u>2.16</u>	1.52
E <sub>2</sub> <sub>M</sub> E <sub>2</sub> <sub>F</sub> E <sub>2</sub> <sub>MF</sub> V <sub>A</sub> <sub>M</sub>	1	<u>0.56</u>	-	-	-	-	-	-	-	-	-	-	-
E <sub>2</sub> <sub>M</sub> E <sub>2</sub> <sub>F</sub> E <sub>2</sub> <sub>MF</sub> V <sub>A</sub> <sub>F</sub>	1	-	-	<u>2.34</u>	-	-	-	-	-	-	-	-	-
- 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.

Model	df	Item											
		37	38	39	40	41	42	43	44	45	46	47	48
$E_2$	4	21.64 <sup>***</sup>	17.62 <sup>***</sup>	12.02 <sup>*</sup>	18.00 <sup>**</sup>	16.14 <sup>**</sup>	27.72 <sup>***</sup>	<u>4.16</u>	13.42 <sup>**</sup>	10.52 <sup>*</sup>	26.16 <sup>***</sup>	26.30 <sup>***</sup>	23.88 <sup>***</sup>
$V_A$	4	<u>0.70</u>	<u>1.08</u>	11.84 <sup>*</sup>	7.76	<u>3.38</u>	<u>2.66</u>	7.12	9.82 <sup>*</sup>	<u>1.98</u>	<u>6.00</u>	<u>8.58</u>	18.76 <sup>***</sup>
$E_2 V_A$	3	0.56	0.42	<u>5.28</u>	<u>5.10</u>	1.78	2.60	3.08	<u>3.68</u>	1.08	2.86	8.50 <sup>*</sup>	12.64 <sup>*</sup>
$E_2 E_2 E_2 V_A$ $E_M E_F E_{MF} E_{AF}$ - log likelihood of full model	1	-	-	-	-	-	-	-	-	-	-	-	<u>0.16</u>
		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 <sup>***</sup>	<u>3.60</u>										
$V_A$	4	42.42 <sup>***</sup>	58.94 <sup>***</sup>										
$E_2 V_A$	3	<u>4.66</u>	2.18										
- log likelihood of full model		7588.14	7657.81										

† Where twice the difference in log likelihood of the models under comparison has  $\chi^2$  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	$T_{1M}$	$T_{1F}$	$T_{2M}$	$T_{2F}$	$E_1$	$E_{1M}$	$E_{1F}$	$E_2$	$E_{2M}$	$E_{2F}$	$E_{2MF}$	$V_A$	$V_{AM}$	$V_{AF}$	$V_{AMF}$
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	$T_{1M}$	$T_{1F}$	$T_{2M}$	$T_{2F}$	$E_1$	$E_{1M}$	$E_{1F}$	$E_2$	$E_{2M}$	$E_{2F}$	$E_{2MF}$	$V_A$	$V_{AM}$	$V_{AF}$	$V_{AMF}$
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	-	-	-	-	-	-	-

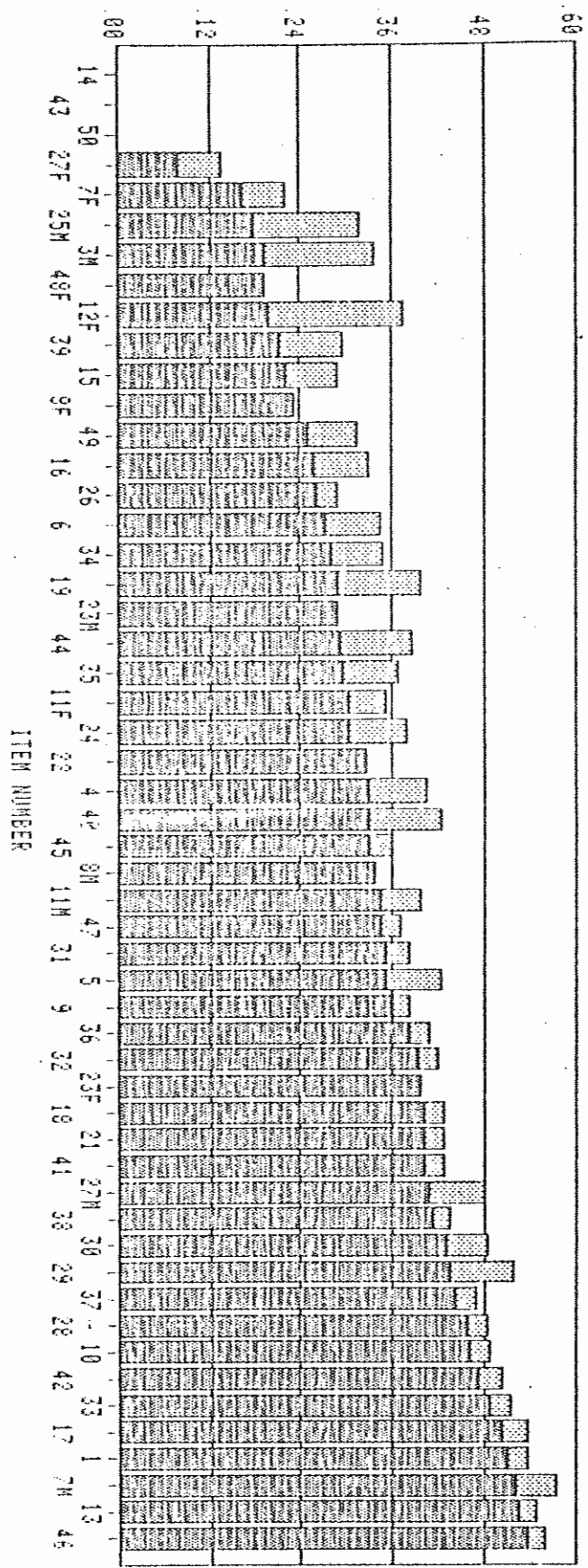
immigration") the  $E_{2M}E_{2F}E_{2MF}V_{AF}$  model is most appropriate, and  $r_{E_{2MF}}$  is 0.57 and 0.59 respectively, indicating that there are some differences in family environmental effects in males and females. For the remaining 4 items the  $E_{2M}E_{2F}V_{AM}V_{AF}$  model is most appropriate. 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  $0.40 \pm 0.005$ , and

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this model resulted in a highly significant heterogeneity chi-square ( $\chi^2_{43} = 226.67, p < .001$ ), 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<sup>†</sup>.

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	Females	Males
$E_2$	0.58 <sup>***</sup>	0.52 <sup>***</sup>
$V_A$	-0.08	-0.06

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<sup>†</sup>  $E_2$  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  $E_1V_A$  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 <sup>also</sup> 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 ( $\chi_1^2 = 0.004$  in males,  $\chi_1^2 = 0.000$  in females) or the frequency of alcohol consumption ( $\chi_6^2 = 9.82$  in males,  $\chi_6^2 = 4.73$  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 response 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
Twin 1	Drinker	978	82
	Non-drinker	69	104

MZM

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

DZF

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

DZM

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

DZO

		Male	
		Drinker	Non-drinker
Female	Drinker	745	39
	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  $E_{1M}E_{1F}E_{2M}E_{2F}V_{AM}V_{AF}$  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  $E_{1M}E_{1F}E_{2M}E_{2F}V_{AM}V_{AF}$  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	$T_M$	$T_F$	$E_2$	$E_{2M}$	$E_{2F}$	$V_A$	$V_{AM}$	$V_{AF}$
$E_2$	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	-	-
$E_{2M}E_{2F}V_{AM}V_{AF}$	1.333	1.061	-	0.603	0.221	-	0.255	0.554

Model	Likelihood ratios		Goodness of fit <sup>†</sup>	
	$\chi^2$	df	$\chi^2$	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
$E_{2M}E_{2F}V_{AM}V_{AF}$	0.56	1	7.72	9

- log likelihood of full model 2607.95

<sup>†</sup> 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  $E_{1M}E_{1F}E_{2M}E_{2F}V_{AM}V_{AF}$  model, while not significantly different from the full model, results in a negative estimate of  $V_{AM}$ . The  $E_{1M}E_{1F}E_{2M}E_{2F}E_{2MF}V_{AF}$  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 ( $r_{E_{2MF}}$ ) 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.

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

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

Model	$T_M$	$T_F$	$E_2$	$E_{2M}$	$E_{2F}$	$E_{2MF}$	$V_A$	$V_{AM}$	$V_{AF}$
$E_2$	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	-	-
$E_{2M}E_{2F}V_{AM}V_{AF}$	1.456	1.303	-	0.889	0.311	-	-	-0.011	0.572
$E_{2M}E_{2F}E_{2MF}V_{AF}$	1.456	1.302	-	0.892	0.539	0.443	-	-	0.337

Model	Likelihood ratios		Goodness of fit	
	$\chi^2$	df	$\chi^2$	df
$E_2$	25.02 <sup>***</sup>	4	28.66 <sup>**</sup>	12
$V_A$	21.04 <sup>***</sup>	4	29.16 <sup>**</sup>	12
$E_2V_A$	10.54 <sup>*</sup>	3	15.32	11
$E_{2M}E_{2F}V_{AM}V_{AF}$	2.06	1	7.76	9
$E_{2M}E_{2F}E_{2MF}V_{AF}$	0.02	1	5.86	9

- log likelihood of full model	993.58
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Table 7.6 Results of fitting models to data on drinking status for older pairs.

Model	$T_M$	$T_F$	$E_2$	$E_{2M}$	$E_{2F}$	$V_A$	$V_{AM}$	$V_{AF}$	$V_{AMF}$
$E_2$	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	-	-	-
$V_{AM}V_{AF}V_{AMF}$	1.215	0.880	-	-	-	-	0.840	0.698	1.000 <sup>†</sup>
$E_{2M}E_{2F}E_{2MF}V_{AF}$	1.215	0.880	-	0.281	0.188	-	0.555	0.501	-

Model	Likelihood ratios		Goodness of fit <sup>†</sup>	
	$\chi^2$	df	$\chi^2$	df
$E_2$	18.28 <sup>**</sup>	4	25.78 <sup>*</sup>	12
$V_A$	7.60	4	14.39	12
$E_2V_A$	4.50	3	11.33	11
$V_{AM}V_{AF}V_{AMF}$	1.16	2	8.20	10
$E_{2M}E_{2F}E_{2MF}V_{AF}$	0.64	1	7.68	9

- log likelihood of full model	1562.62
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<sup>†</sup> 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.

<u>MZF</u>		Twin 2						
		1 <sup>†</sup>	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
<u>MZM</u>		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
<u>DZF</u>		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
<u>DZM</u>		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
<u>DZO</u>		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

<sup>†</sup> 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  $E_{1M}E_{1F}E_{2M}V_{AM}V_{AF}V_{AMF}$  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.

Parameter	Model			
	I	II	III	IV
T <sub>1M</sub>	-1.350	-1.360	-1.358	-1.334
T <sub>2M</sub>	-0.810	-0.816	-0.816	-0.802
T <sub>3M</sub>	-0.403	-0.408	-0.409	-0.401
T <sub>4M</sub>	-0.012	-0.014	-0.016	-0.014
T <sub>5M</sub>	0.426	0.432	0.426	0.421
T <sub>6M</sub>	1.110	1.130	1.120	1.105
T <sub>1F</sub>	-1.046	-1.050	-1.048	-1.055
T <sub>2F</sub>	-0.330	-0.334	-0.334	-0.336
T <sub>3F</sub>	0.103	0.094	0.098	0.100
T <sub>4F</sub>	0.508	0.505	0.501	0.508
T <sub>5F</sub>	0.894	0.891	0.887	0.897
T <sub>6F</sub>	1.394	1.393	1.387	1.401
E <sub>2</sub>	0.523	-	0.143	-
E <sub>2M</sub>	-	-	-	0.383
E <sub>2F</sub>	-	-	-	0.066
V <sub>A</sub>	-	0.660	0.506	-
V <sub>AM</sub>	-	-	-	0.305
V <sub>AF</sub>	-	-	-	0.566
Goodness of fit				
$\chi^2$	652.9 <sup>***</sup>	741.4 <sup>***</sup>	669.7 <sup>***</sup>	777.4 <sup>***</sup>
df	227	227	226	224
Likelihood ratios				
$\chi^2$	135.0 <sup>***</sup>	26.4 <sup>***</sup>	15.6 <sup>**</sup>	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						
		Twin 2							Twin 2						
		1	2	3	4	5	6	7	1	2	3	4	5	6	7
<u>MZF</u>															
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
<u>MZM</u>															
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
<u>DZF</u>															
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
<u>DZM</u>															
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
<u>DZO</u>															
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.

Parameter	Model			
	I	II	III	IV
T <sub>1M</sub>	-1.468	-1.478	-1.476	-1.454
T <sub>2M</sub>	-0.916	-0.926	-0.924	-0.907
T <sub>3M</sub>	-0.447	-0.456	-0.455	-0.446
T <sub>4M</sub>	0.039	0.036	0.036	0.035
T <sub>5M</sub>	0.593	0.597	0.596	0.586
T <sub>6M</sub>	1.515	1.541	1.537	1.518
T <sub>1F</sub>	-1.292	-1.294	-1.292	-1.304
T <sub>2F</sub>	-0.487	-0.489	-0.488	-0.495
T <sub>3F</sub>	0.053	0.049	0.049	0.047
T <sub>4F</sub>	0.614	0.608	0.608	0.611
T <sub>5F</sub>	1.224	1.219	1.218	1.227
T <sub>6F</sub>	1.971	1.967	1.964	1.979
E <sub>2</sub>	0.513	-	0.054	-
E <sub>2M</sub>	-	-	-	0.294
E <sub>2F</sub>	-	-	-	0.013
V <sub>A</sub>	-	0.685	0.626	-
V <sub>AM</sub>	-	-	-	0.429
V <sub>AF</sub>	-	-	-	0.647
Goodness of fit				
$\chi^2$	699.3 <sup>***</sup>	2047.4 <sup>***</sup>	1843.2 <sup>***</sup>	2456.8 <sup>***</sup>
df	227	227	226	224
Likelihood ratios				
$\chi^2$	103.2 <sup>***</sup>	10.0 <sup>*</sup>	9.2 <sup>*</sup>	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.

Parameter	Model			
	I	II	III	IV
T <sub>1M</sub>	-1.241	-1.243	-1.242	-1.224
T <sub>2M</sub>	-0.703	-0.703	-0.703	-0.693
T <sub>3M</sub>	-0.357	-0.357	-0.357	-0.352
T <sub>4M</sub>	-0.074	-0.073	-0.074	-0.074
T <sub>5M</sub>	0.248	0.252	0.250	0.244
T <sub>6M</sub>	0.805	0.817	0.812	0.796
T <sub>1F</sub>	-0.867	-0.869	-0.865	-0.874
T <sub>2F</sub>	-0.192	-0.196	-0.194	-0.197
T <sub>3F</sub>	0.153	0.149	0.150	0.150
T <sub>4F</sub>	0.418	0.414	0.414	0.417
T <sub>5F</sub>	0.659	0.654	0.654	0.659
T <sub>6F</sub>	1.105	1.102	1.100	1.109
E <sub>2</sub>	0.529	-	0.155	-
E <sub>2M</sub>	-	-	-	0.337
E <sub>2F</sub>	-	-	-	0.082
V <sub>A</sub>	-	0.660	0.494	-
V <sub>AM</sub>	-	-	-	0.358
V <sub>AF</sub>	-	-	-	0.547
Goodness of fit				
$\chi^2$	293.1**	265.4*	253.2	272.4*
df	227	227	226	224
Likelihood ratios				
$\chi^2$	59.5***	12.1*	6.1	0.8
df	4	4	3	1
- log likelihood of full model		6936.58		

model, while the  $E_{1M}E_{1F}E_{2M}E_{2F}V_{AM}V_{AF}$  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.

Parameter	Model			
	I	II	III	IV
T <sub>1M</sub>	-1.259	-1.265	-1.266	-1.252
T <sub>2M</sub>	-0.656	-0.662	-0.663	-0.658
T <sub>3M</sub>	-0.102	-0.104	-0.104	-0.106
T <sub>4M</sub>	0.507	0.511	0.511	0.502
T <sub>5M</sub>	1.489	1.509	1.510	1.500
T <sub>1F</sub>	-0.743	-0.741	-0.741	-0.748
T <sub>2F</sub>	-0.099	-0.102	-0.102	-0.104
T <sub>3F</sub>	0.517	0.512	0.512	0.514
T <sub>4F</sub>	1.159	1.152	1.153	1.158
T <sub>5F</sub>	1.950	1.944	1.945	1.955
E <sub>2</sub>	0.452	-	-0.018	-
E <sub>2M</sub>	-	-	-	-0.000
E <sub>2F</sub>	-	-	-	0.144
V <sub>A</sub>	-	0.625	0.645	-
V <sub>AM</sub>	-	-	-	0.685
V <sub>AF</sub>	-	-	-	0.443
Goodness of fit				
$\chi^2$	304.3 <sup>***</sup>	225.4 <sup>**</sup>	225.9 <sup>***</sup>	218.1 <sup>**</sup>
df	164	164	163	161
Likelihood ratios				
$\chi^2$	74.3 <sup>***</sup>	8.1	8.1 <sup>*</sup>	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.

Parameter	Model			
	I	II	III	IV
T <sub>1M</sub>	-1.170	-1.171	-1.171	-1.161
T <sub>2M</sub>	-0.682	-0.686	-0.687	-0.680
T <sub>3M</sub>	-0.311	-0.316	-0.317	-0.312
T <sub>4M</sub>	0.061	0.056	0.056	0.057
T <sub>5M</sub>	0.683	0.684	0.685	0.681
T <sub>1F</sub>	-0.628	-0.631	-0.632	-0.636
T <sub>2F</sub>	-0.178	-0.182	-0.183	-0.185
T <sub>3F</sub>	0.141	0.135	0.134	0.134
T <sub>4F</sub>	0.416	0.411	0.410	0.411
T <sub>5F</sub>	0.911	0.907	0.907	0.910
E <sub>2</sub>	0.419	-	-0.076	-
E <sub>2M</sub>	-	-	-	0.321
E <sub>2F</sub>	-	-	-	-0.083
V <sub>A</sub>	-	0.557	0.640	-
V <sub>AM</sub>	-	-	-	0.261
V <sub>AF</sub>	-	-	-	0.633
Goodness of fit				
$\chi^2$	200.7*	157.1	156.9	151.9
df	164	164	163	161
Likelihood ratios				
$\chi^2$	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  $\underline{A}$ . The elements of  $\underline{A}$  may be calculated from the expected proportions of twins in the 2x2 contingency table of non-drinker vs drinker ( $\underline{B}$ ), and the 6x6 contingency table of frequency of alcohol consumption given that both twins are drinkers ( $\underline{C}$ ).

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  $j=2,3,\dots,7$  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  $i=2,3,\dots,7$  and  $m=i-1$ .

Where both twins are drinkers

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

where  $i=2,3,\dots,7$ ,  $j=2,3,\dots,7$ ,  $m=i-1$  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  $E_1 E_1 M E_1 F E_2 E_2 M E_2 F E_2 M F V_A F$  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  $E_1 V_A$  model was most appropriate. For older twin pairs, the  $E_1 V_A$  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
$E_{2M}$	0.819	-
$E_{2F}$	0.885	-
$E_{2MF}$	0.510	-
$V_A$	-	0.748
$V_{AF}$	0.018	-
Polychotomous		
$T_{1M}$	-0.870	-1.036
$T_{2M}$	-0.278	-0.577
$T_{3M}$	0.297	-0.239
$T_{4M}$	0.906	0.129
$T_{5M}$	1.720	0.744
$T_{1F}$	-0.900	-0.544
$T_{2F}$	-0.282	-0.092
$T_{3F}$	0.269	0.224
$T_{4F}$	0.843	0.498
$T_{5F}$	1.757	0.990
$V_A$	0.642	0.565
Goodness of fit		
$\chi^2$	568.07 <sup>***</sup>	323.41 <sup>***</sup>
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  $E_{1M}E_{1F}E_{2M}E_{2F}V_{AM}V_{AF}$  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 <sup>***</sup>	0.239	73.91 <sup>**</sup>	0.239
MZ Males				
Mean	12.21	0.725	13.54	0.818
Variance	391.06 <sup>*</sup>	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 <sup>***</sup>	115.75 <sup>***</sup>	13.02	407.36
$\log_{10}(x+1)$	0.444 <sup>***</sup>	0.232 <sup>***</sup>	0.764	0.362
Consumption last week				
Raw	5.10 <sup>***</sup>	68.94 <sup>***</sup>	14.35	359.72
$\log_{10}(x+1)$	0.501 <sup>***</sup>	0.234 <sup>***</sup>	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-	20-	30-	40-	50-	60-	70-	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-	20-	30-	40-	50-	60-	70-	80-	90-	100-	110-	120-	150-	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-	20-	30-	40-	50-	60-	70-	80-	90-	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-	20-	30-	40-	50-	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-	20-	30-	40-	50-	60-	70-	80-	90-	100-	110-	120-	150-	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				Total
		18-24	25-44	45-64	65 and over	
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 unrepeatability, 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.

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	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	$\chi^2$	$h^2$
<u>Female</u>						
$E_1E_2$	.125 <sup>***</sup>	.110 <sup>***</sup>	-	2	60.76 <sup>***</sup>	
$E_1V_A$	.102 <sup>***</sup>	-	.131 <sup>***</sup>	2	1.50	.56±.02
$E_1E_2V_A$	.103 <sup>***</sup>	.010	.121 <sup>***</sup>	1	1.09	
<u>Male</u>						
$E_1E_2$	.168 <sup>***</sup>	.191 <sup>***</sup>	-	2	8.62 <sup>*</sup>	
$E_1V_A$	.143 <sup>***</sup>	-	.211 <sup>***</sup>	2	11.56 <sup>**</sup>	
$E_1E_2V_A$	.151 <sup>***</sup>	.118 <sup>***</sup>	.091 <sup>**</sup>	1	0.07	.25±.09
<u>Female &amp; Male</u>						
$E_1E_2$	.138 <sup>***</sup>	.135 <sup>***</sup>	-	6	171.51 <sup>***</sup>	
$E_1V_A$	.115 <sup>***</sup>	-	.157 <sup>***</sup>	6	135.65 <sup>***</sup>	
$E_1E_2V_A$	.118 <sup>***</sup>	.045 <sup>**</sup>	.111 <sup>***</sup>	5	120.80 <sup>***</sup>	
<u>Female &amp; Male &amp; Opposite-sex</u>						
$E_1E_2$	.161 <sup>***</sup>	.118 <sup>***</sup>	-	8	279.41 <sup>***</sup>	
$E_1V_A$	.118 <sup>***</sup>	-	.160 <sup>***</sup>	8	143.60 <sup>***</sup>	
$E_1E_2V_A$	.119 <sup>***</sup>	.001	.159 <sup>***</sup>	7	143.50 <sup>***</sup>	

Table 7.26 Summary of model fitting for log transformed consumption last week.

	$\hat{E}_1$	$\hat{E}_2$	$\hat{V}_A$	df	$\chi^2$	$h^2$
<u>Female</u>						
$E_1E_2$	.130 <sup>***</sup>	.105 <sup>***</sup>	-	2	61.63 <sup>***</sup>	
$E_1V_A$	.107 <sup>***</sup>	-	.128 <sup>***</sup>	2	0.84	.55±.02
$E_1E_2V_A$	.107 <sup>***</sup>	.002	.126 <sup>***</sup>	1	0.82	
<u>Male</u>						
$E_1E_2$	.181 <sup>***</sup>	.176 <sup>***</sup>	-	2	10.74 <sup>**</sup>	
$E_1V_A$	.155 <sup>***</sup>	-	.201 <sup>***</sup>	2	6.52 <sup>*</sup>	
$E_1E_2V_A$	.161 <sup>***</sup>	.089 <sup>**</sup>	.108 <sup>**</sup>	1	0.00	.30±.10
<u>Female &amp; Male</u>						
$E_1E_2$	.146 <sup>***</sup>	.128 <sup>***</sup>	-	6	175.95 <sup>***</sup>	
$E_1V_A$	.122 <sup>***</sup>	-	.151 <sup>***</sup>	6	127.97 <sup>***</sup>	
$E_1E_2V_A$	.124 <sup>***</sup>	.030 <sup>*</sup>	.120 <sup>***</sup>	5	120.57 <sup>***</sup>	
<u>Female &amp; Male &amp; Opposite-sex</u>						
$E_1E_2$	.165 <sup>***</sup>	.115 <sup>***</sup>	-	8	240.17 <sup>***</sup>	
$E_1V_A$	.124 <sup>***</sup>	-	.155 <sup>***</sup>	8	129.96 <sup>***</sup>	
$E_1E_2V_A$	.125 <sup>***</sup>	.004	.151 <sup>***</sup>	7	129.44 <sup>***</sup>	

Table 7.27 Parameter estimates ( $\pm$  s.e.) and heritabilities ( $h^2$ ) from fit of models incorporating different components of variation for males and females.

	$\hat{V}_A$	$\hat{E}_{1M}$	$\hat{E}_{1F}$	$\hat{E}_{2M}$
Normal weekly consumption	$0.130 \pm 0.006$	$0.146 \pm 0.008$	$0.102 \pm 0.004$	$0.084 \pm 0.013$
		$\chi^2_6 = 3.00$		
	$h^2_{\text{female}} = 0.56 \pm 0.02$		$h^2_{\text{male}} = 0.36 \pm 0.02$	
Consumption last week	$0.129 \pm 0.006$	$0.158 \pm 0.008$	$0.106 \pm 0.004$	$0.070 \pm 0.013$
		$\chi^2_6 = 1.91$		
	$h^2_{\text{female}} = 0.55 \pm 0.02$		$h^2_{\text{male}} = 0.36 \pm 0.02$	

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 <sub>1</sub> <ul style="list-style-type: none"> <li>— Error</li> <li>— Individual environment</li> </ul>	44 <ul style="list-style-type: none"> <li>— 16</li> <li>— 28</li> </ul>	41 <ul style="list-style-type: none"> <li>— 37</li> <li>— 4</li> </ul>	45 <ul style="list-style-type: none"> <li>— 45</li> <li>— 0</li> </ul>	44 <ul style="list-style-type: none"> <li>— 15</li> <li>— 29</li> </ul>
E <sub>2</sub>	-	23	-	20
V <sub>A</sub>	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	$\chi^2$	$h^2$
<u>Females</u>						
Younger						
$E_1E_2$	.106 <sup>***</sup>	.099 <sup>***</sup>	-	2	35.53 <sup>***</sup>	
$E_1V_A$	.085 <sup>***</sup>	-	.119 <sup>***</sup>	2	0.78	.58±.03
$E_1E_2V_A$	.086 <sup>***</sup>	.009	.109 <sup>***</sup>	1	0.56	
Older						
$E_1E_2$	.141 <sup>***</sup>	.119 <sup>***</sup>	-	2	30.38 <sup>***</sup>	
$E_1V_A$	.116 <sup>***</sup>	-	.143 <sup>***</sup>	2	0.72	.55±.03
$E_1E_2V_A$	.117 <sup>***</sup>	.081	.131 <sup>***</sup>	1	0.53	
Younger & older						
$E_1E_2$	.125 <sup>***</sup>	.110 <sup>***</sup>	-	6	92.05 <sup>***</sup>	
$E_1V_A$	.101 <sup>***</sup>	-	.131 <sup>***</sup>	6	32.99 <sup>***</sup>	
$E_1E_2V_A$	.102 <sup>***</sup>	.010	.121 <sup>***</sup>	5	32.58 <sup>***</sup>	
<u>Males</u>						
Younger						
$E_1E_2$	.152 <sup>***</sup>	.196 <sup>***</sup>	-	2	16.39 <sup>***</sup>	
$E_1V_A$	.116 <sup>***</sup>	-	.227 <sup>***</sup>	2	3.13	.66±.03
$E_1E_2V_A$	.120 <sup>***</sup>	.072 <sup>*</sup>	.155 <sup>***</sup>	1	0.38	
Older						
$E_1E_2$	.185 <sup>***</sup>	.186 <sup>***</sup>	-	2	0.43	
$E_1V_A$	.170 <sup>***</sup>	-	.198 <sup>***</sup>	2	9.29 <sup>**</sup>	
$E_1E_2V_A$	.180 <sup>***</sup>	.164 <sup>***</sup>	.026	1	0.15	
Younger & older						
$E_1E_2$	.168 <sup>***</sup>	.191 <sup>***</sup>	-	6	18.81 <sup>**</sup>	
$E_1V_A$	.143 <sup>***</sup>	- <sup>***</sup>	.211 <sup>***</sup>	6	25.41 <sup>***</sup>	
$E_1E_2V_A$	.151 <sup>***</sup>	.118	.090 <sup>**</sup>	5	12.44 <sup>*</sup>	

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	$\chi^2$	$h^2$
<u>Females</u>						
Younger						
$E_1E_2$	.128 <sup>***</sup>	.093 <sup>***</sup>	-	2	18.65 <sup>***</sup>	
$E_1V_A$	.108 <sup>***</sup>	-	.111 <sup>***</sup>	2	0.66	.51±.03
$E_1E_2V_A$	.109 <sup>***</sup>	.012	.098 <sup>***</sup>	1	0.38	
Older						
$E_1E_2$	.132 <sup>***</sup>	.116 <sup>***</sup>	-	2	45.21 <sup>***</sup>	
$E_1V_A$	.105 <sup>***</sup>	-	.142 <sup>***</sup>	2	0.72	.58±.02
$E_1E_2V_A$	.104 <sup>***</sup>	-.009	.151 <sup>***</sup>	1	0.59	
Younger & older						
$E_1E_2$	.130 <sup>***</sup>	.105 <sup>***</sup>	-	6	70.78 <sup>***</sup>	
$E_1V_A$	.107 <sup>***</sup>	-	.128 <sup>***</sup>	6	8.17	
$E_1E_2V_A$	.107 <sup>***</sup>	.001	.127 <sup>***</sup>	5	8.17	
<u>Males</u>						
Younger						
$E_1E_2$	.169 <sup>***</sup>	.181 <sup>***</sup>	-	2	10.56 <sup>**</sup>	
$E_1V_A$	.136 <sup>***</sup>	-	.210 <sup>***</sup>	2	2.87	.61±.03
$E_1E_2V_A$	.140 <sup>***</sup>	.072 <sup>*</sup>	.136 <sup>**</sup>	1	0.30	
Older						
$E_1E_2$	.195 <sup>***</sup>	.171 <sup>***</sup>	-	2	3.13	
$E_1V_A$	.174 <sup>***</sup>	-	.192 <sup>***</sup>	2	4.08	
$E_1E_2V_A$	.180 <sup>***</sup>	.102 <sup>*</sup>	.085	1	0.40	
Younger & older						
$E_1E_2$	.181 <sup>***</sup>	.176 <sup>***</sup>	-	6	15.45 <sup>*</sup>	
$E_1V_A$	.155 <sup>***</sup>	-	.200 <sup>***</sup>	6	12.16	
$E_1E_2V_A$	.161 <sup>***</sup>	.088 <sup>**</sup>	.108 <sup>**</sup>	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  $P_0 \dots P_m$ . 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,  $\beta$ , 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,  $\gamma$ . 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  $c$ ,  $t$ ,  $f$ ,  $g$ ,  $p$ ,  $\beta$  and  $\gamma$  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:

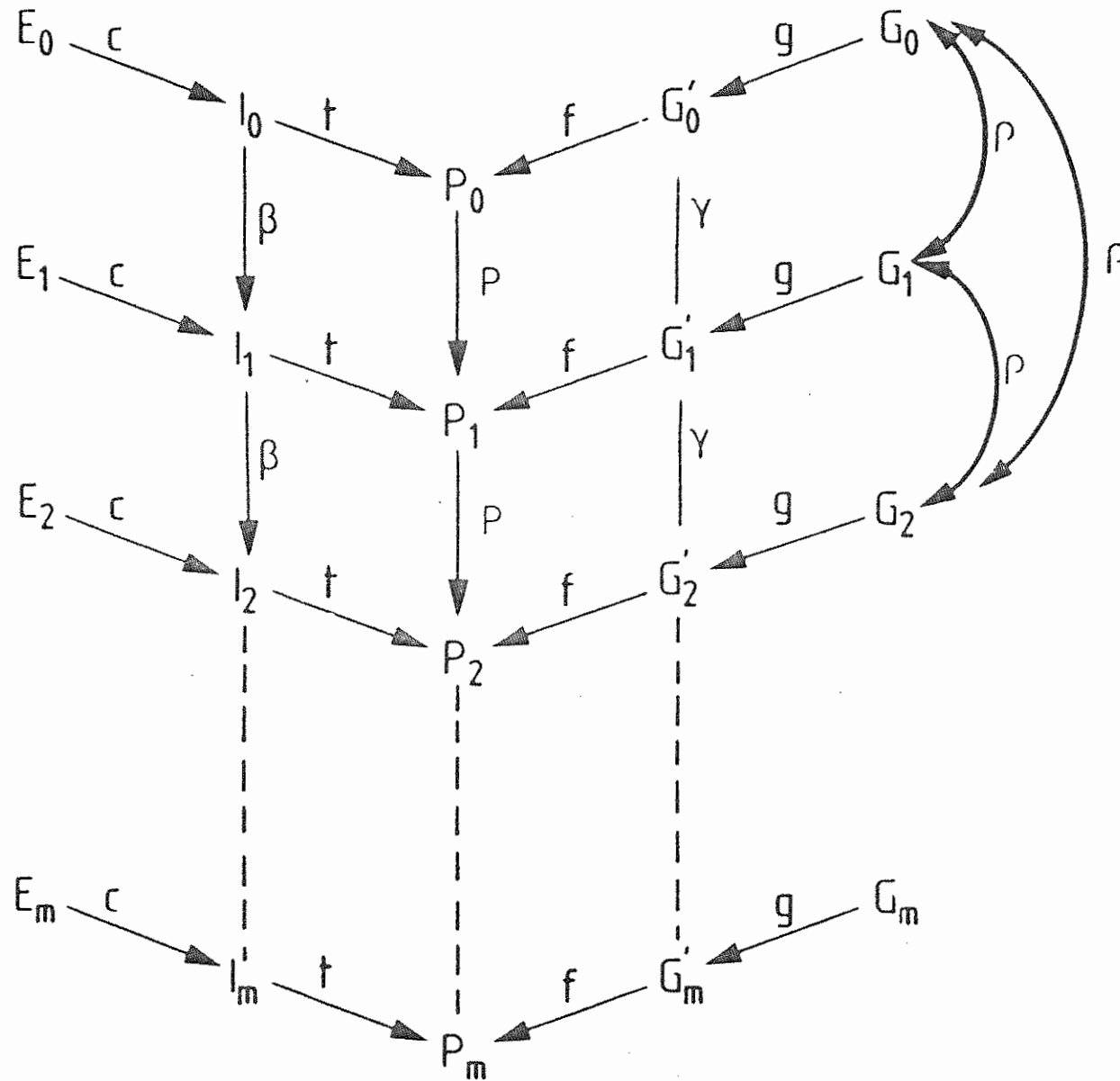
$$E_{m,n} = \left(\frac{e}{p-\beta}\right)^2 \left[ p^{2+m-n} \frac{1-p^{2(m+1)}}{1-p^2} - (p^{1+n-m} \beta + p \beta^{1+n-m}) \frac{1-(p\beta)^{m+1}}{1-p\beta} \right. \\ \left. + \beta^{2+n-m} \frac{1-\beta^{2(m+1)}}{1-\beta^2} \right]$$

where  $e=ct$ .

Environment

Phenotype

Genotype



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:

$$G_{m,n} = \left(\frac{h}{p-\gamma}\right)^2 \left[ p^{2+m-n} \frac{1-p^{2(m+1)}}{1-p^2} - (p^{1+n-m} \beta + p \beta^{1+n-m}) \frac{1-(p\beta)^{m+1}}{1-p\beta} \right. \\ \left. + \gamma^{2+n-m} \frac{1-\gamma^{2(m+1)}}{1-\gamma^2} \right]$$

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  $\underline{x}$ , and a corresponding vector of expected scores  $\underline{Ex}$ . The  $\underline{Ex}$  may be estimated as function of parameters representing effects such as age and sex. The expected covariance matrix of individuals in the pedigree is  $\underline{\Sigma}$ . The  $\underline{\Sigma}$  may be estimated as a function of (co)variance components

representing genetic and environmental sources of variation. The elements of  $\Sigma$  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  $\sigma^2$  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  $\underline{Ex}$  and  $\underline{\Sigma}$ , the log likelihood of obtaining the pedigree of individuals with observed scores  $\underline{x}$  is

$$L = (-1/2)\ln|\underline{\Sigma}| - 1/2(\underline{x} - \underline{Ex})^T \underline{\Sigma}^{-1} (\underline{x} - \underline{Ex}) + \text{constant}$$

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	Parameter				$\chi^2$	df
	Mean	$E_1$	$E_2$	$V_A$		
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	Parameter											Improvement <sup>a</sup>	df
	Mean			$\sigma^2$	Parameter						$\chi^2$		
	Constant	Linear age regression	Quadratic age regression		$E_1$	$P(E_1)$	$\rho(E_1)$	$V_A$	$P(V_A)$	$\rho(V_A)$			
IV	.486	-.001	-	.233	.102	-	-	.131	-	-	3.24	1	
V	.340	.007	-.0001	.232	.102	-	-	.130	-	-	9.08*	2	
VI	.340 <sup>†</sup>	.007 <sup>†</sup>	-.0001 <sup>†</sup>	.148	.021	.902	.000 <sup>†</sup>	.127	.134	.000 <sup>†</sup>	30.36***	1	
VII	.340 <sup>†</sup>	.007 <sup>†</sup>	-.0001 <sup>†</sup>	.151	.022	.902	.000 <sup>†</sup>	.129	.001 <sup>††</sup>	1.00 <sup>†</sup>	30.36***	1	

<sup>a</sup> Improvement over model II

<sup>†</sup> Value of parameter is fixed

<sup>††</sup> 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	Parameter					
	Mean	$E_1$	$E_2$	$V_A$	$\chi^2$	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	Parameter											Improvement <sup>a</sup>	df
	Constant	Mean		$\sigma^2$	$E_1$	$P(E_1)$	$\rho(E_1)$	$V_A$	$P(V_A)$	$\rho(V_A)$	$\chi^2$		
		Linear age regression	Quadratic age regression										
IV	.570	-.002	-	.235	.108	-	-	.127	-	-	10.24**	1	
V	.394	.008	-.0001	.233	.107	-	-	.126	-	-	18.72***	2	
VI	.394 <sup>†</sup>	.008 <sup>†</sup>	-.0001 <sup>†</sup>	.175	.049	.744	.000 <sup>†</sup>	.126	.000 <sup>††</sup>	.000 <sup>†</sup>	20.55***	1	
VII	.394 <sup>†</sup>	.008 <sup>†</sup>	-.0001 <sup>†</sup>	.175	.049	.744	.000 <sup>†</sup>	.126	.001 <sup>††</sup>	1.00 <sup>†</sup>	20.55***	1	

<sup>a</sup> Improvement over model II

<sup>†</sup> Value of parameter is fixed

<sup>††</sup> 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				$\chi^2$	df
	Mean	$E_1$	$E_2$	$V_A$		
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	Parameter													Improvement <sup>a</sup>	
	Mean			$\sigma^2$	Parameter						Improvement <sup>a</sup>			$\chi^2$	df
	Constant	Linear age regression	Quadratic age regression		$E_1$	$P(E_1)$	$\rho(E_1)$	$E_2$	$P(E_2)$	$\rho(E_2)$	$V_A$	$P(V_A)$	$\rho(V_A)$		
IV	.837	-.003	-	.357	.150	-	-	.116	-	-	.091	-	-	6.76*	1
V	.533	.014	-.0002	.355	.151	-	-	.114	-	-	.090	-	-	14.04**	2
VI	.533 <sup>†</sup>	.014 <sup>†</sup>	-.0002 <sup>†</sup>	.324	.024	.924	.000 <sup>†</sup>	.010	.999 <sup>††</sup>	.000 <sup>†</sup>	.287	.945	.000 <sup>†</sup>	b	2
VII	.533 <sup>†</sup>	.014 <sup>†</sup>	-.0002 <sup>†</sup>	.300	.026	.913	.000 <sup>†</sup>	.0003	.995 <sup>††</sup>	1.00 <sup>†</sup>	.271 <sup>††</sup>	.976	.000 <sup>†</sup>	b	2

<sup>a</sup> Improvement over model III

<sup>b</sup> Model was worse than model III

<sup>†</sup> Value of parameter is fixed

<sup>††</sup> 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	Parameter				$\chi^2$	df
	Mean	$E_1$	$E_2$	$V_A$		
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	Parameter													Improvement <sup>a</sup>	
	Mean			$\sigma^2$	Linear age			Quadratic age			$V_A$	$P(V_A)$	$\rho(V_A)$	$\chi^2$	df
	Constant	Linear regression	Quadratic regression		$E_1$	$P(E_1)$	$\rho(E_1)$	$E_2$	$P(E_2)$	$\rho(E_2)$					
IV	.940	-.003	-	.357	.160	-	-	.087	-	-	.110	-	-	10.78**	1
V	.579	.017	-.0002	.352	.160	-	-	.084	-	-	.108	-	-	21.44***	2
VI	.579 <sup>†</sup>	.017 <sup>†</sup>	-.0002 <sup>†</sup>	.352	.160	.000 <sup>††</sup>	.000 <sup>†</sup>	.084	.001 <sup>††</sup>	.000 <sup>†</sup>	.108	1.00 <sup>††</sup>	.000 <sup>†</sup>	21.44***	2
VII	.579 <sup>†</sup>	.017 <sup>†</sup>	-.0002 <sup>†</sup>	.385	.179	.000 <sup>††</sup>	.000 <sup>†</sup>	.001	.800 <sup>††</sup>	1.00 <sup>†</sup>	.205	.925	.000 <sup>†</sup>	12.22**	2

<sup>a</sup> Improvement over model III

<sup>†</sup> Value of parameter is fixed

<sup>††</sup> 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.

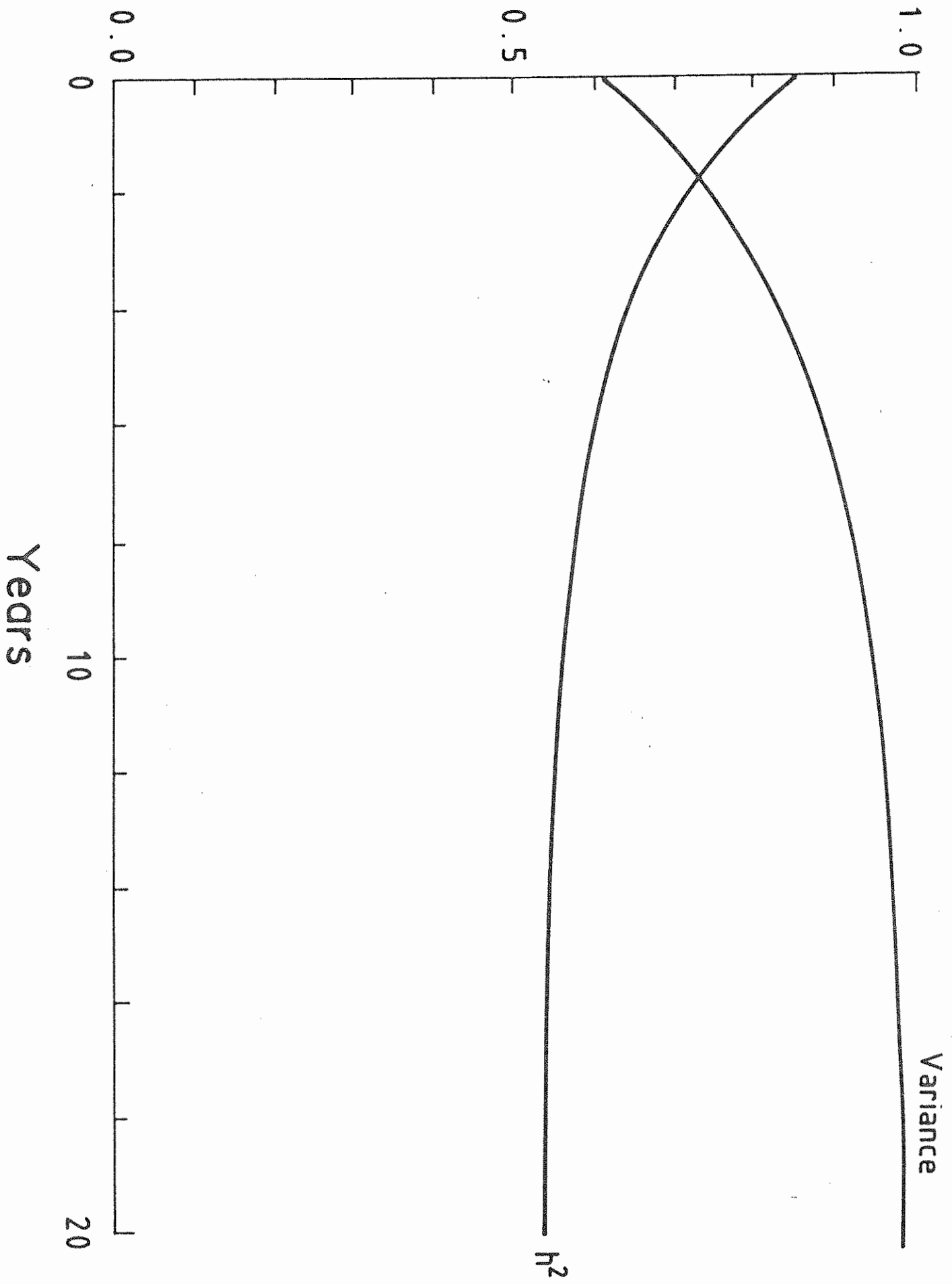
For consumption last week in females, of the models assuming constant genetic and environmental effects throughout development, the  $E_1V_A$  model provides the best description of the data (Table 7.33). Allowing the mean to vary as a function of both linear and quadratic age regressions (model V), 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) assumed 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





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.

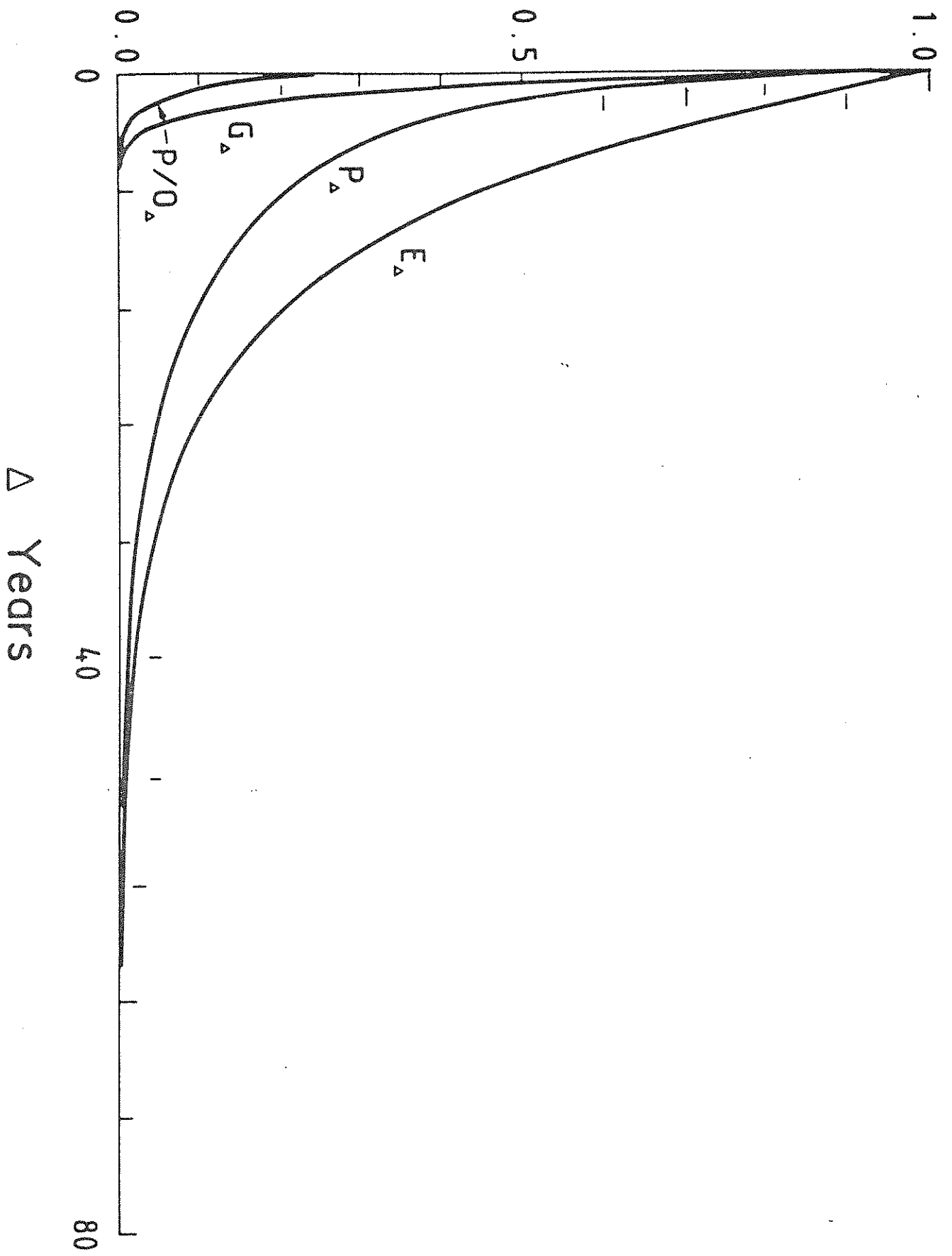
Figure 7.3 illustrates predicted changes in correlations in adults as a function of differences in their ages ( $\Delta$ ). The parent offspring correlation ( $P/O$ ) starts at half the value of the limiting heritability, and then decreases to zero over time. Thus, a naive genetic analysis of parents 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  $\Delta$  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,





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.

<u>Females</u>		Normal weekly consumption		Consumption last week	
Cohort	Mean age	df	mean square	df	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		Consumption last week	
		df	mean square	df	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
<u>Males</u>					
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

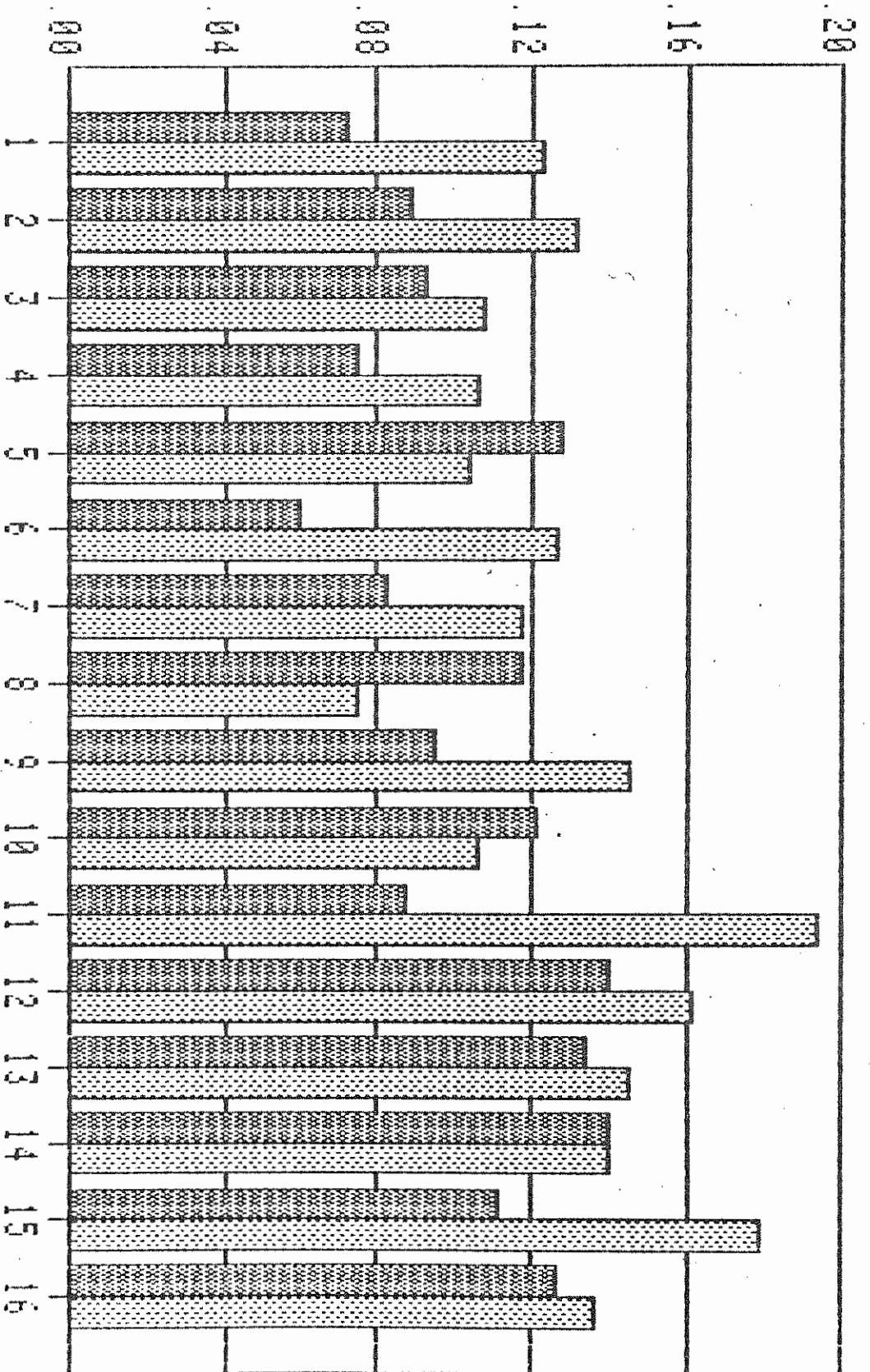
Cohort	Mean age	df	Normal weekly consumption mean square	df	Consumption last week mean square
3	25.46				
MZ Between		58	0.6109	58	0.5942
Within		59	0.1177	59	0.1211
DZ Between		50	0.4534	50	0.4298
Within		51	0.1679	51	0.1916
4	29.61				
MZ Between		86	0.4862	86	0.4789
Within		87	0.1560	87	0.1711
DZ 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

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ESTIMATE



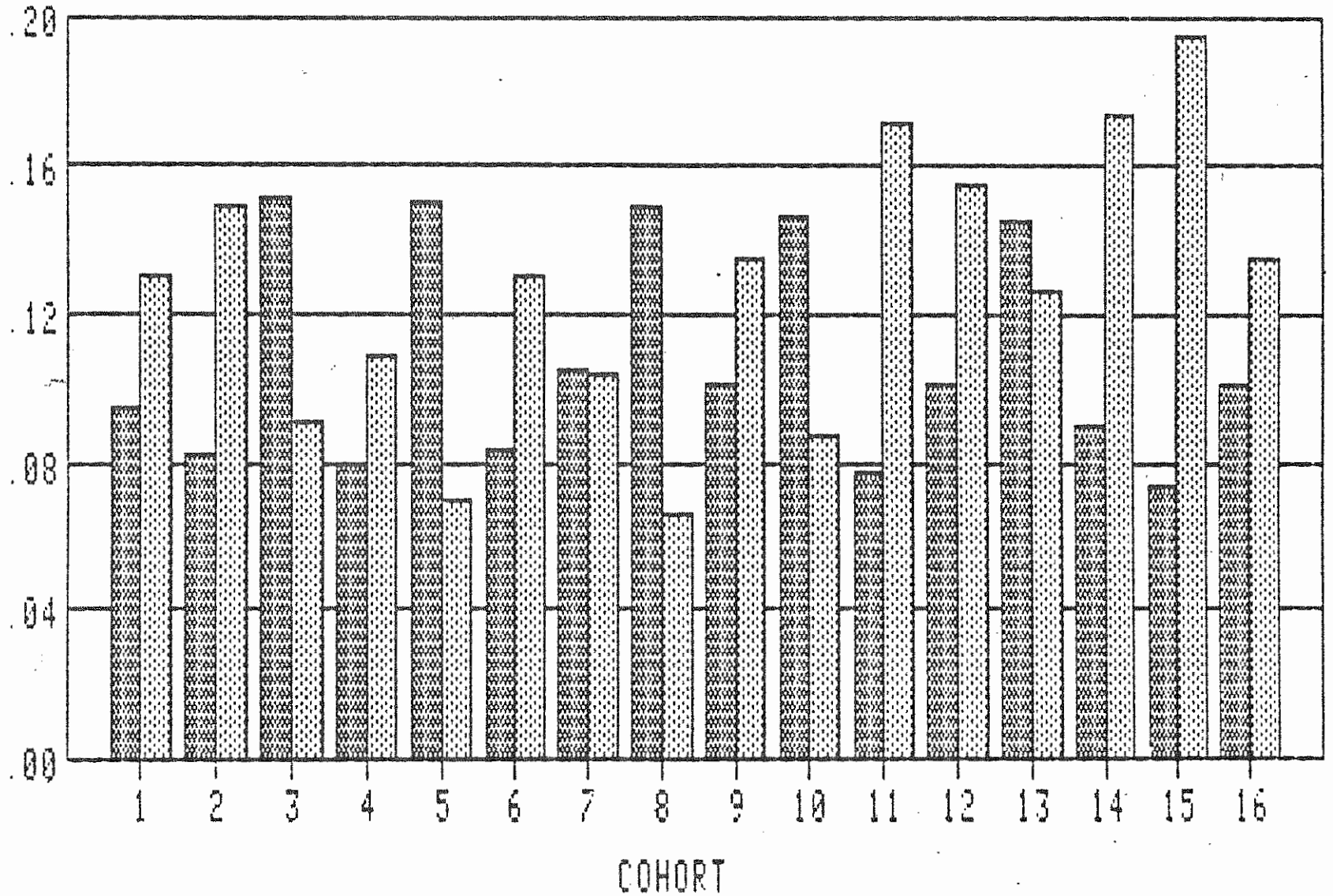
WA  
EI



COHORT

FEMALES NORMAL WEEKLY CONSUMPTION

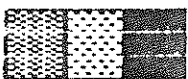
E  
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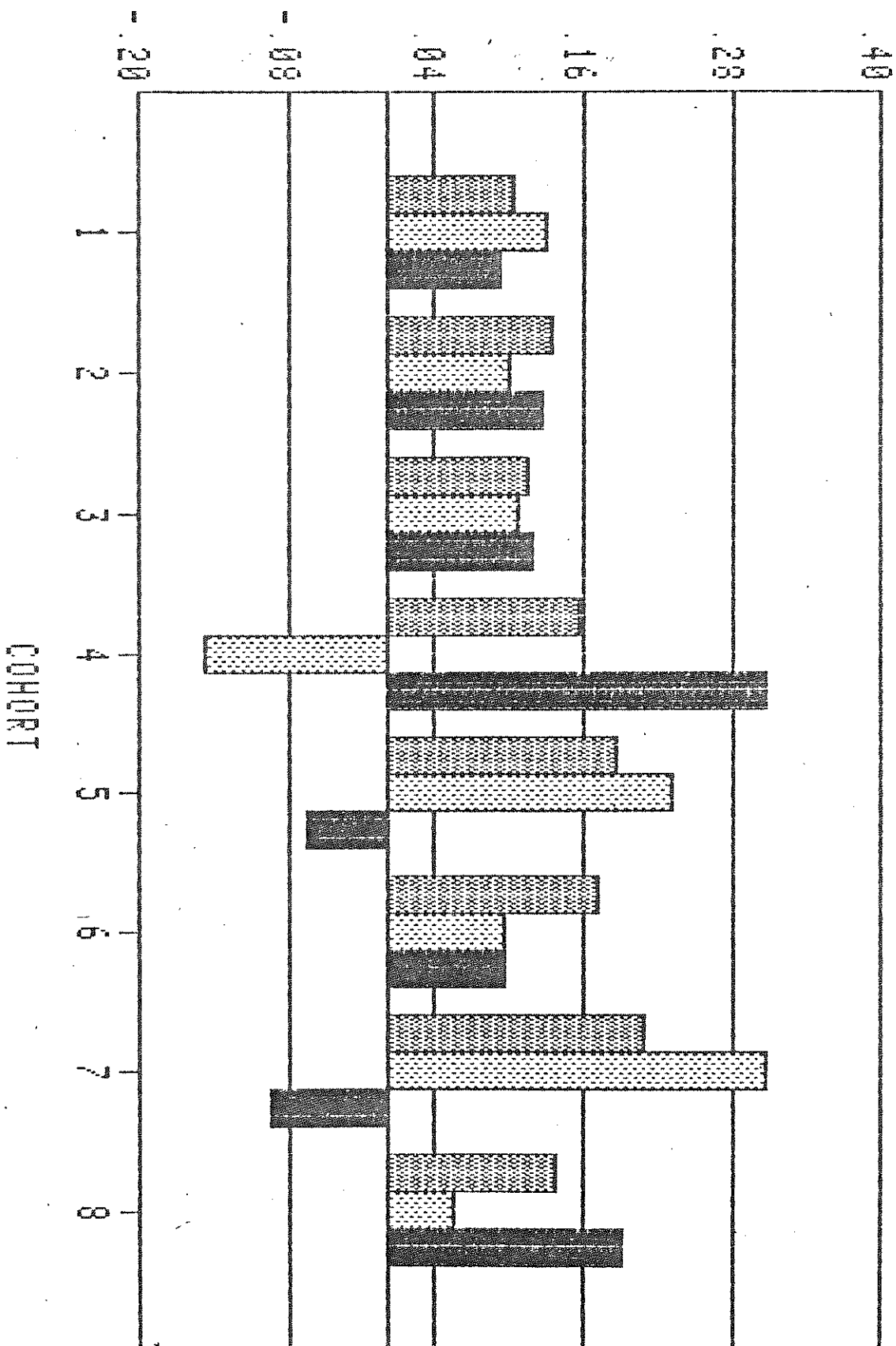
VA  
E1

FEMALES LAST WEEKS CONSUMPTION

WEST INDIAN

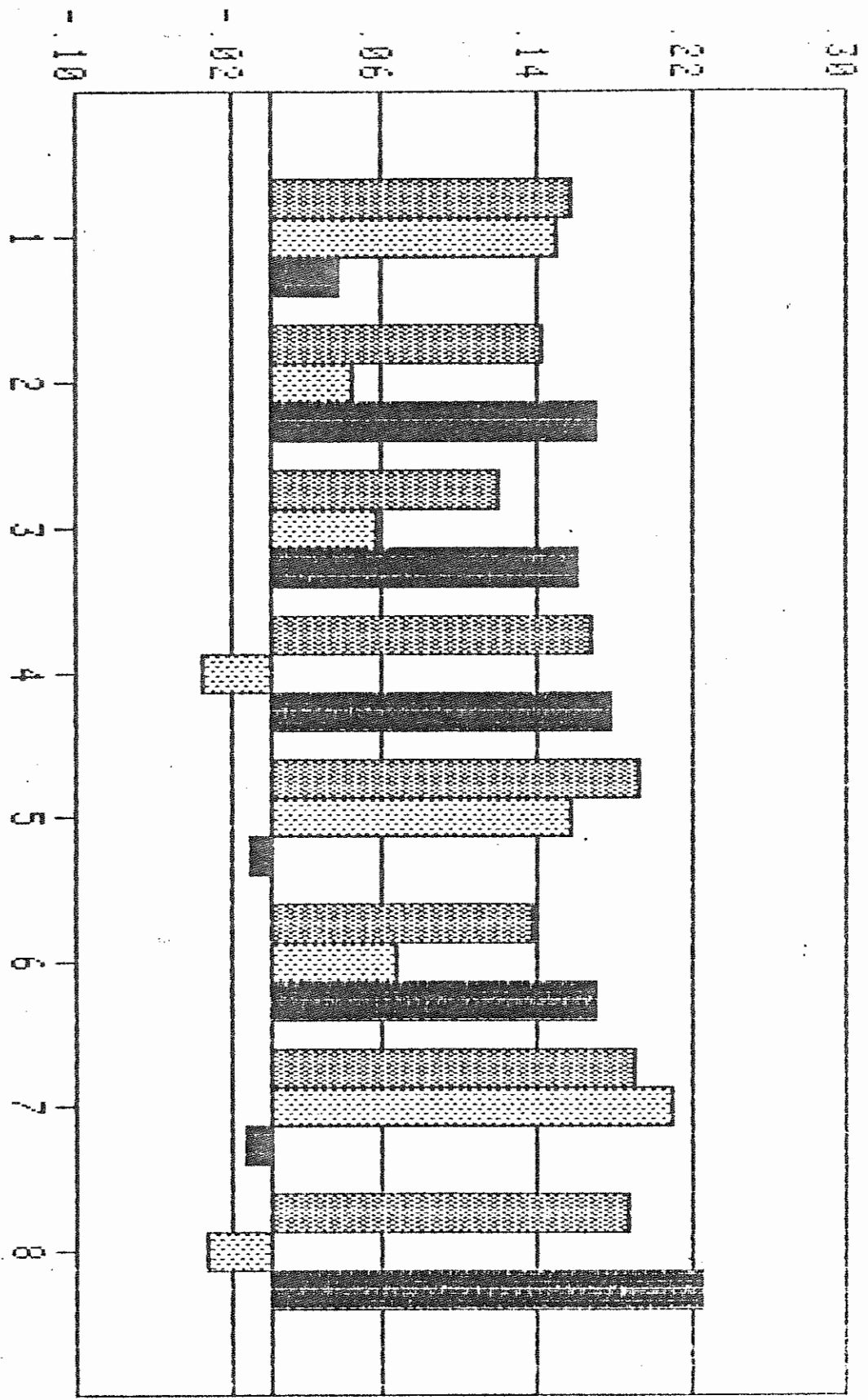
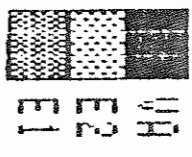


WH  
E2  
E1



MALES NORMAL WEEKLY CONSUMPTION

ESTIMATE



MALES LAST WEEKS CONSUMPTION

In females, there is significant heterogeneity of fit of the  $E_1V_A$  model over the different age cohorts for both normal weekly consumption ( $\chi^2_{30} = 79.93, p < .001$ ) and consumption last week ( $\chi^2_{30} = 66.32, p < .001$ ). 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 ( $\chi^2_{21} = 31.11, p > .05$ ) or consumption last week ( $\chi^2_{21} = 16.52, p > .05$ ), the fluctuation in parameter estimates is even more extreme than that found in females. It is not surprising then that our attempts to find a theoretical model to describe developmental 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. \*

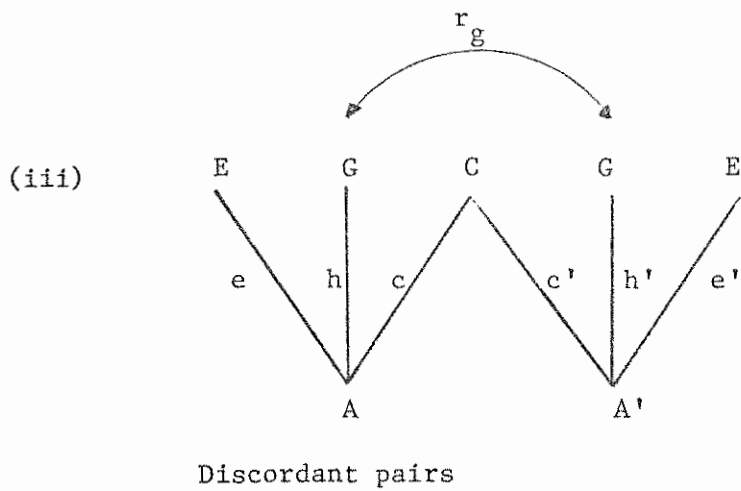
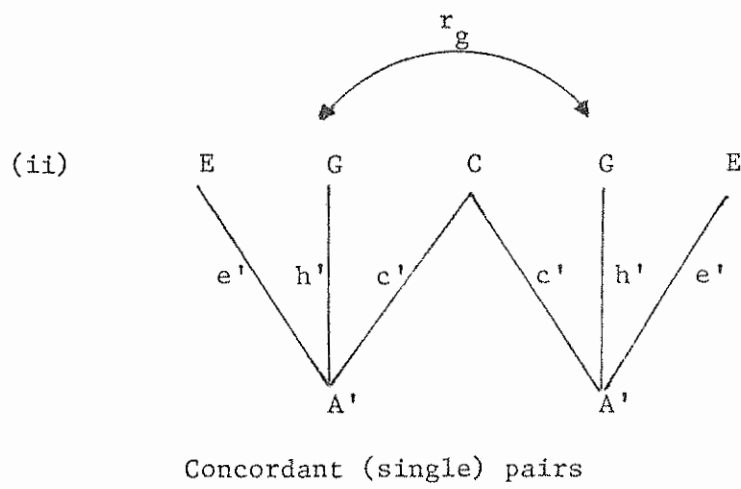
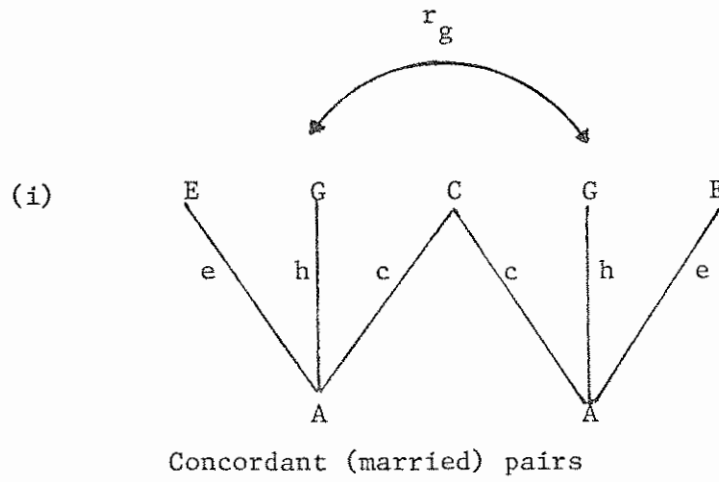
The twin sample was divided into those twin pairs where both twins have a marriage-like relationship (i.e. married or cohabiting), where only one twin has such a relationship, and where neither twin has such a relationship. Table 7.37 shows the variances and covariances for the two measures of consumption 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	
<u>Females</u>							
Normal weekly consumption							
MZ		(n=650) Twin		(n=307) Twin		(n=276) 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
<u>Males</u>							
Normal weekly consumption							
MZ		(n=268) Twin		(n=187) Twin		(n=112) 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



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 genetic 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

$$\begin{aligned} V_A &= h^2 \\ V_{A'} &= h'^2 \\ E_1 &= e^2 \\ E_{1'} &= e'^2 \\ E_2 &= c^2 \\ E_{2'} &= c'^2 \end{aligned}$$

and  $R_{V_A}$  and  $R_{E_2}$  are the correlations between additive 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

<u>Model</u>	$V_A$	$V_{A'}$	$R_{V_A}$	$E_1$	$E_1'$	df	$\chi^2$
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

<u>Model</u>	$V_A$	$V_{A'}$	$R_{V_A}$	$E_1$	$E_1'$	df	$\chi^2$
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

<u>Model</u>	$V_A$	$V_{A'}$	$R_{V_A}$	$E_2$	$E_2'$	$R_{E_2}$	$E_1$	$E_1'$	df	$\chi^2$
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 <sup>†</sup>	.160	.109	10	5.39

Consumption last week

<u>Model</u>	$V_A$	$V_{A'}$	$R_{V_A}$	$E_2$	$E_2'$	$R_{E_2}$	$E_1$	$E_1'$	df	$\chi^2$
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

<sup>†</sup> 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		
<u>Younger</u>							
<u>Females</u>							
Normal weekly consumption							
MZ	(n=177) Twin		(n=254) Twin		(n=53) 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
<u>Males</u>							
Normal weekly consumption							
MZ	(n=48) Twin		(n=173) Twin		(n=53) 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	
<u>Older</u>							
<u>Females</u>							
Normal weekly consumption							
MZ		(n=473) Twin		(n=53) Twin		(n=137) 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
<u>Males</u>							
Normal weekly consumption							
MZ		(n=220) Twin		(n=14) Twin		(n=59) 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.

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Females

Normal weekly  
consumption

<u>Model</u>	$V_A$	$V_{A'}$	$R_{V_A}$	$E_1$	$E_1'$	df	$\chi^2$
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

<u>Model</u>	$V_A$	$V_{A'}$	$R_{V_A}$	$E_1$	$E_1'$	df	$\chi^2$
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

<u>Model</u>	$V_A$	$V_{A'}$	$R_{V_A}$	$E_2$	$E_2'$	$R_{E_2}$	$E_1$	$E_1'$	df	$\chi^2$
I	.160		-	.068		-	.123		15	18.01
II	.168	.130	-	.010	.116	-	.144	.102	12	7.33
III	.168	.130	1.000 <sup>†</sup>	.010	.116	1.000 <sup>†</sup>	.144	.102	10	7.33

Consumption  
last week

<u>Model</u>	$V_A$	$V_{A'}$	$R_{V_A}$	$E_2$	$E_2'$	$R_{E_2}$	$E_1$	$E_1'$	df	$\chi^2$
I	.144		-	.068		-	.137		15	13.62
II	.109	.221	-	.096	.020	-	.138	.116	12	11.08
III	.109	.221	1.000 <sup>†</sup>	.096	.140	1.000 <sup>†</sup>	.138	.116	10	11.08

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<sup>†</sup> 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

<u>Model</u>	$V_A$	$V_{A'}$	$R_{V_A}$	$E_1$	$E_1'$	df	$\chi^2$
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

<u>Model</u>	$V_A$	$V_{A'}$	$R_{V_A}$	$E_1$	$E_1'$	df	$\chi^2$
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

<u>Model</u>	$V_A$	$V_{A'}$	$R_{V_A}$	$E_2$	$E_2'$	$R_{E_2}$	$E_1$	$E_1'$	df	$\chi^2$
I	.029		-	.160		-	.176		15	15.03
II	.020	.109	-	.176	.096	-	.176	.176	12	14.37
III	.044	.058	1.000 <sup>†</sup>	.152	.240	.828	.168	.144	10	12.97

Consumption  
last week

<u>Model</u>	$V_A$	$V_{A'}$	$R_{V_A}$	$E_2$	$E_2'$	$R_{E_2}$	$E_1$	$E_1'$	df	$\chi^2$
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

<sup>†</sup> 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 successful 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 successful. 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  $0.62 \pm 0.02$ . 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 is 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.

21

(1) Were you the first or second born twin? →

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    <sup>22</sup>       *leave blank*

(3) What was your own birthweight?     lbs     ozs     don't know    <sup>25</sup>       *leave blank*

(4) What was your twin's birthweight?     lbs     ozs     don't know    <sup>30</sup>       *leave blank*

(5) How many placentas (afterbirths) were there at birth? →

1	Single	
2	2 Joined	
3	2 Separate	
4	Don't know	

(6) Were there any difficulties at your birth? (please specify)

-----  <sup>35</sup> *leave blank*

IF YOU AND YOUR TWIN ARE OF OPPOSITE SEX PLEASE GO TO QUESTION 9

(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?

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?    <sup>38</sup>   age

How long for?      years

(10) How frequently do you and your twin see or contact each other?    <sup>42</sup>   *leave blank*

1 We live together <input type="checkbox"/>	4 Once or twice a month <input type="checkbox"/> see <input type="checkbox"/> contact
2 Almost every day <input type="checkbox"/> see <input type="checkbox"/> contact	5 A few times a year <input type="checkbox"/> see <input type="checkbox"/> contact
3 At least once a week <input type="checkbox"/> see <input type="checkbox"/> contact	6 Less often <input type="checkbox"/> see <input type="checkbox"/> contact

When did you start living apart? (if applicable)    <sup>44</sup>   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.    <sup>46</sup>  <sup>47</sup>  *leave blank*

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GENERAL INFORMATION

(12) **MARITAL STATUS** 48

1	Single	
2	Widowed	
3	Married	
4	Living together but not married	
5	Separated	
6	Divorced	
7	Remarried	

(14) Do you have any children?  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
1		
2		
3		
4		
5		
6		

leave this column blank

51		
52		
53		
54		
55		
56		
57		

(13) How many years have you been in your present marital state? 49 50  

--	--

 yrs

(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	

(16) **YOUR MAIN OCCUPATION** - please describe in detail. e.g. "maintenance fitter in oil refinery".

	FULL TIME	
	PART TIME	
	UNEMPLOYED	

leave blank 70

(17) **YOUR FATHER'S MAIN OCCUPATION** (before retirement)

--

(18) **RELIGION**

	Your religion	Father's religion	Mother's religion	
1	No religion	77	78	79
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			

leave blank 72 75

(19) How often do you attend church for other observances. 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? →

1	always	
2	usually	
3	sometimes	
4	never	

85

(22) Which of the following do you take regularly? *leave blank*

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    Cms <sup>93</sup>

(25) What is your weight? →  st  lbs OR    Kg <sup>95</sup>

(26) Do you consider you have good health? → 

1	Yes	<input type="text"/>
2	No	<input type="text"/>

<sup>99</sup>

(27) In the past 12 months, on how many days would you have stayed home from work because of illness? →  Days <sup>100</sup>

(28) Do you or any of your blood relatives have any of the following conditions?

	Your		Blood relatives (specify) 3
	Self; 1	Twin; 2	
Stuttering 102	<input type="text"/>	<input type="text"/>	
Dyslexia, Word blindness, Specific learning disability, Severe reading problems 103	<input type="text"/>	<input type="text"/>	
Autism 104	<input type="text"/>	<input type="text"/>	

(29) Have you EVER been a smoker? If NO go to next page. → 

1	Yes	<input type="text"/>
2	No	<input type="text"/>

<sup>105</sup>

(30) At what age did you start smoking? →  Years <sup>106</sup>

(31) If you have stopped smoking, how old were you when you stopped?

Stopped cigarettes at	→	<input type="text"/>	Years
Stopped pipe at	→	<input type="text"/>	Years
Stopped cigars at	→	<input type="text"/>	Years

<sup>108</sup>

(32) How many CIGARETTES do (or did) you usually smoke in a day? →

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? →

<sup>114</sup>

(33) Have you EVER taken alcoholic drinks? If NO go to next page. → 

1	Yes	
2	No	

(34) At what age did you start drinking alcohol? → 

--

 age

(35) OVER THE LAST YEAR, about how often have you usually taken any alcoholic drinks? → 

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? → 

1	On two days	
2	On one day	
3	Not usually	

(37) DURING WEEK DAYS (Monday to Friday) would you usually drink? → 

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? → 

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)

		Weekdays	Weekends
Glasses of beer per day	28		
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							

(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 <sup>2</sup>					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 <sup>31</sup>					Kidney/bladder infections <sup>42</sup>				

(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	8 Your Children
High blood pressure (hypertension) <sup>43</sup>								
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) <sup>55</sup>								

(44) OPERATIONS (Don't forget to give age)

	No <sup>0</sup>	Yes <sup>1</sup>	At age
Have you had:	tonsils out?	56	
	appendix out?	59	
	gall bladder removed?	62	
	thyroid gland removed?	65	
	varicose veins done?	68	
<u>MEN ONLY</u> - Have you had:	a vasectomy?	71	
	a prostatectomy?	74	

(45) Do you have any *SERIOUS* problem requiring medical treatment? (please specify)

(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

78

83

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:

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:

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:

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)

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? hrs mins

REPRODUCTIVE HISTORY - WOMEN OVER 40 - MIN 90 TO NEXT PAGE

(55) How old were you when you had your FIRST menstrual period? →  Years  Months

(56) Have you EVER used a contraceptive pill? If NO go to question 57 →  Yes  No

Altogether, for how long have you taken the contraceptive pill (fill in all the months) →  Years  Months

Are you taking a contraceptive pill now? →  Yes  No

IF YES: Did the contraceptive pill cause any upset to you or your health? *leave blank*

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 →  Yes  No

Are you pregnant now? →  Yes  No

You may pregnancies have you had? →  Number

How old were you with the first pregnancy? →  Years

IF YES: Did any pregnancy end in miscarriage or termination?  No  Yes → at  Years

With my pregnancy have you had any of the following? *leave blank*

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 →  Yes  No

If not regular, have your periods completely stopped? →  Yes  No

WAS THIS TRUE TO: Menopause ("natural change of life")?  No  Yes → at  Years

Hysterectomy (removal of the womb)?  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? →  Days

(60) Are your periods? 63

1	Heavy	<input type="text"/>
2	Moderate	<input type="text"/>
3	Light	<input type="text"/>

Are your periods? 64

1	Very painful	<input type="text"/>
2	Moderately painful	<input type="text"/>
3	No trouble	<input type="text"/>

Are your periods? 65

1	Very limiting	<input type="text"/>
2	Moderately limiting	<input type="text"/>
3	Not limiting	<input type="text"/>

(61) What is the average time between the start of one period and the start of the next? →  Days

What is the shortest? →  Days

What is the longest? →  Days

Don't know 68

Don't know 70

<u>FEELINGS</u>		1	2	3	4
The following statements describe feelings people may have. For each statement please TICK THE BOX which best describes how you are feeling.		Not at all	A little	A lot	Unbearably
72	1				
	2				
	3				
	4				
	5				
	6				
	7				
	8				
	9				
	10				
	11				
	12				
	13				
85	14				

PLEASE CHECK TO SEE THAT YOU HAVE ANSWERED ALL THE QUESTIONS

20

5

<u>PERSONALITY</u>		1	0
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.		YES	NO
PLEASE REMEMBER TO ANSWER EACH QUESTION			
21	1		
	2		
	3		
	4		
	5		
	6		
	7		
	8		
	9		
30	10		

		1	0
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

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

	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	Pyjama 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