

Willingness to Drive When Drunk and Personality: A Twin Study

N. G. Martin¹ and D. I. Boomsma²

In a laboratory study of psychomotor sensitivity to alcohol, twins were asked "Would you drive a car now?" at 1, 2, and 3 h after drinking a standard dose of ethanol (0.75 g/kg). Correlations among these binary items, the Eysenck personality scales, and age were investigated using PRELIS and LISREL. Willingness to drive and Extraversion correlate at all three times in both males and females. In males, willingness to drive also correlates with Psychoticism, and in females it correlates negatively with the Lie (or Social Desirability) scale. Most correlations between cotwins in willingness to drive were significant in both monozygotic (MZ) and dizygotic (DZ) male twins but correlations were lower in female twins. Factor and Markovian models were fitted. In males there seem to be both genetic and cultural influences on willingness to drive when drunk. About half the genetic variance seems to be the pleiotropic effects of genes influencing Extraversion. The correlations with Psychoticism, on the other hand, seem to be largely environmental in origin. The small sample size and lack of proper significance tests mean that these results must be interpreted with caution.

KEY WORDS: alcohol sensitivity; drunk driving; personality; twins; genetics.

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INTRODUCTION

For an alcohol-related road accident to occur, two conditions are necessary. First, the driver's judgment must be impaired so that he is willing to drive even though he knows he has consumed alcohol. Second, his psychomotor performance must be impaired. In a recent laboratory study of 206 pairs of twins we detected genetic variation in several aspects of psychomotor sensitivity to alcohol (Martin *et al.*, 1985b). At the same time we collected data on twins' willingness to drive and in this paper we explore the causes of individual differences in this behavior. Since willingness to drive when drunk might be influenced by personality, we explore its covariation with the Eysenck personality scales and the genetic and environmental causes of this covariation.

METHODS

Twins were trained to plateau on an apparatus measuring reaction time, coordination, and steadiness and measured while sober. They then drank a weight-related dose of alcohol [0.75 g EtOH/kg body weight, diluted to 10% (v/v) in sugarless lemon squash] in 20 min. At 1, 2, and 3 h after the start of drinking they were tested again on the psychomotor tasks. At these times we also asked the question, "Would you drive a car now?" Before they started drinking, twins also completed the Eysenck Personality Questionnaire, which is scored for four dimensions—Extraversion, Psychoticism, Neuroticism, and Lie (or Social Desirability) (Eysenck and Eysenck, 1975). Details of the subjects, measures, and protocol can be found elsewhere (Martin *et al.*, 1985a,b).

ANALYSIS AND RESULTS

Frequencies

Complete data are available for 182 pairs of twins aged 18–34 years (mean, 23 years). When asked "Would you drive a car now?" before they drank the alcohol dose, all subjects responded "yes." The percentage of subjects responding "yes" to this question 1, 2, and 3 h after alcohol is shown in Fig. 1. Many more males than females are willing to drive after drinking.

Reliability

A subsample of 41 pairs (46 male, 36 female individuals) repeated the entire protocol 1–17 months after the first testing (mean, 4.5 months).

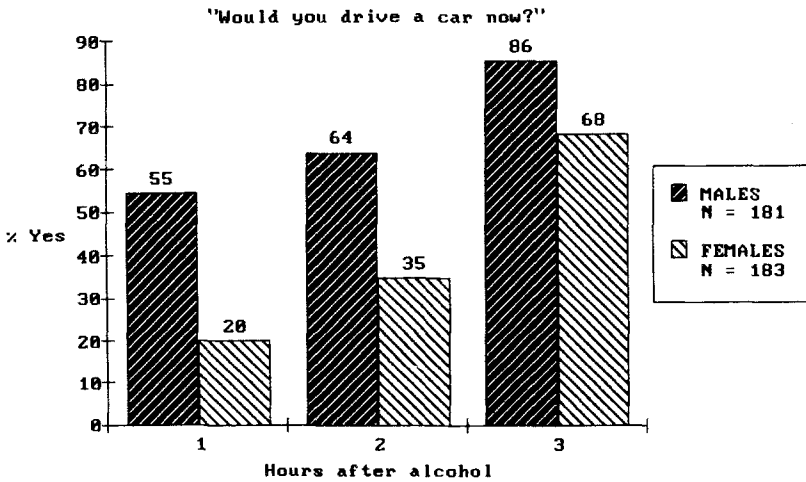


Fig. 1. Percentage of twin subjects responding "yes" to the question "Would you drive a car now?" after a dose of alcohol.

Responses to the willingness to drive questions were obtained from 57 individuals (40 males, 17 females) on both occasions. From their responses on two occasions we can calculate test-retest reliabilities. This is done very simply with PRELIS (Jöreskog and Sörbom, 1986a). For each individual the three yes/no responses and four personality scores are read in for two occasions, so the number of input variables $NI = 14$. Since we know that there are complete data for personality but missing data for the Drive? items we request pairwise treatment of missing values ($TR = PA$). We declare the Drive? items as ordinal since they are dichotomous. PRELIS now calculates the entire 14×14 matrix of correlations, but the only elements of interest to us are the correlations of the same variable measured on two occasions. For the three Drive? items these are calculated as tetrachoric correlations on the assumption that the underlying joint distribution is bivariate normal. They take the values .81, .60, and .81 at 1, 2, and 3 h, respectively, indicating that willingness to drive after alcohol is a highly repeatable response. By relaxing the requirement of PRELIS that there must be at least 200 observations to calculate asymptotic variances of correlations ($MS = 50$ PV on the OU card), we obtain standard errors of these reliabilities as .10, .15 and .11, respectively, although as the manual comments, these should not be taken too seriously for a such a small sample. Test-retest reliabilities of the personality variables are calculated as product-moment correlations and are .89, .69, .86, and .75 for *E*, *P*, *N*, and *L*, respectively, with asymptotic

standard errors of .02, .15, .03, and .05. All test-retest reliabilities are significantly different from zero ($p < .001$.)

Since there are mean differences in response frequencies for the Drive? items between males and females (Fig. 1) and there are also sex differences in means for the personality scales, it is possible that the repeatabilities we report are inflated by these mean differences. However, the repeatabilities for the male subsample alone are .73, .50, and .78 for the Drive? items and .91, .60, .87, and .72 for *E*, *P*, *N*, and *L*, so the high repeatabilities of the variables are not due simply to large sex differences.

Phenotypic Correlations

First, we wish to inspect the phenotypic correlations among the Drive? responses, personality variables, and age. The input data file is thus the entire sample of first visit twins, for this purpose treated as independent individuals. We run two jobs using the select-cases-delete-variable option, (SD SEX = 0 for females, SD SEX = 1 for males). We also use listwise deletion of missing values (TR = LI) and this leaves us with correlation matrices based upon 183 females and 181 males.

Age and the personality measures *E*, *N*, and *L* are approximately normally distributed and are treated as continuous. Psychoticism has a possible range of values from 0 to 25, but the distribution is highly skewed toward low values. Since there were only a few scores >12, these were recoded to 12 (using the RE card) and *P* was declared an ordinal variable, along with the three Drive? responses. Intercorrelations of the continuous variables are product-moment, those of the ordinal variables are polychoric, and those between ordinal and continuous variables are polyserial. These are shown in Table I. PRELIS provides a test of significance for the difference from zero of each correlation and this is indicated in Table I. A test of the model of bivariate normality is also provided for polychoric and polyserial correlations. The test has no degrees of freedom for tetrachoric correlations, but where the test can be applied there do not appear to be more significant departures from bivariate normality than would be expected by chance.

The most consistent correlation between willingness to drive and personality is with Extraversion, which is significant in both sexes at all three times. In males there are also positive correlations with Psychoticism and these are significant at 2 and 3 h. These are consistent with the stereotype of the tough-minded extravert as the drunken driver. The negative correlations with *P* in females (significant at 1 h) are counterintuitive. Since one might have predicted that those with a higher Social Desirability score (i.e., lower Lie score) might answer "no" more often, it is surprising

Table I. Phenotypic Intercorrelations ($\times 100$ —Pearson, Polychoric, and Polyserial) of Age, Willingness to Drive at 1, 2, and 3 h After Alcohol, and EPQ Personality Scores (Females Above Diagonal, Males Below)

	Age	D1	D2	D3	E	P	N	L
Females ($N = 183$)								
Age		3	9	-1	-16*	-13	-8	9
Drive 1 h	28***		75***	54***	22**	-10	12	-24***
Drive 2 h	18*	84***		85***	20**	-15*	-3	-5
Drive 3 h	13	68***	82***		22**	-7	-6	-5
Extraversion	2	29***	23***	33***		1	-20*	-9
Psychoticism	-9	14	25***	16*	6		11	-38***
Neuroticism	-13	-8	-14	-13	-34***	12		-13
Lie	6	-9	-13	-10	-20**	-38***	1	
Males ($N = 181$)								

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

that only the correlation between Drive? 2 h and Lie in females is significant. Similarly, it is somewhat surprising that individuals with higher Neuroticism scores are not less willing to drive, but no correlation with N is seen in either sex. There is a slight tendency for older males in our sample to be more willing to drive after alcohol and this is significant at 1 and 2 h. No significant correlation of age and willingness to drive was observed in females. Although the observations on individuals in Table I are made on different people, since they are pairs of twins they are not strictly independent so the significance of correlations may be somewhat overestimated.

Genetic Analysis: A Factor Model

We confine our analysis of the causes of variation and covariation to males because the correlations of willingness to drive with personality are most consistent in that sex and also because there is greater practical interest in the problem of drunk driving in males.

Our analysis uses the matrices of twin correlations and cross-correlations. For each twin the variables are the three Drive? responses and the E and P scores. We also include the age of the pair, so there are 11 variables in all. The 11×11 matrices of cross-correlations for MZ and DZ males are shown in Table II. These are calculated by PRELIS as

Table II. Intercorrelations ($\times 100$ —Pearson, Polychoric, Polyserial) of Age, Willingness to Drive at 1, 2, and 3 h After Alcohol, and Extraversion and Psychoticism Scores for Twin 1 and Twin 2 (MZ Males Above Diagonal, DZ Pairs Below)

Age	<i>D1T1</i>	<i>D2T1</i>	<i>D3T1</i>	<i>ET1</i>	<i>PT1</i>	<i>D1T2</i>	<i>D2T2</i>	<i>D3T2</i>	<i>ET2</i>	<i>PT2</i>	
MZ males (37 pairs)											
Age		52	60	57	22	-19	39	42	10	5	1
<i>D1T1</i>	35		84	69	52	24	61	65	56	21	8
<i>D2T1</i>	-6	79		78	49	17	58	42	39	19	4
<i>D3T1</i>	5	52	81		86	8	54	38	74	45	-19
<i>ET1</i>	-12	-24	-3	9		-1	48	51	32	54	-20
<i>PT1</i>	4	15	50	22	1		-30	-1	-8	1	44
<i>D1T2</i>	2	49	20	9	21	10		90	39	37	5
<i>D2T2</i>	2	19	46	26	16	10	90		51	50	46
<i>D3T2</i>	-64	-26	-22	5	27	-41	39	51		26	38
<i>ET2</i>	2	-22	1	20	32	-19	37	50	26		-4
<i>PT2</i>	5	-12	17	1	-9	48	5	46	37	-4	
DZ males (34 pairs)											

mixtures of product-moment, polychoric, and polyserial correlations as in the phenotypic matrices above.

The twin correlations (for the same measure—as opposed to cross-correlations for different traits) are highlighted in Table II. It can be seen that the MZ correlations for Drive? at 1, 2, and 3 h are .61, .42, and .74, while the corresponding values for DZ pairs are .49, .46, and .05. Although the variances of these correlations will be large, there is some indication of genetic effects at 1 and 3 h. For comparison, the corresponding correlations for 35 pairs of MZ females are .38, .19, and .19, and those for 38 pairs of DZ females are .32, .34, and .04.

In our structural model we wish to allow for the effects of age. As is shown in the preceding paper (Neale and Martin, 1989) this is simply done in LISREL (Jöreskog and Sörbom, 1986b) by including age in the correlation matrices and specifying a factor which loads on age and also on other variables thought to regress on age. This is seen more clearly in the listing of the LISREL input job, which appears in Appendix I (Fig. A1).

Our first structural model can be seen in Fig. 2. We specify three genetic group factors responsible for variation and covariation of the Drive responses (*D1*, *D2*, and *D3*) and personality scores: (i) G_{ext} will estimate all the genetic variance for extraversion (Ext) and will remove any variance in the Drive? responses caused by the pleiotropic effects of genes influencing extraversion; (ii) G_{psy} estimates pleiotropic effects of

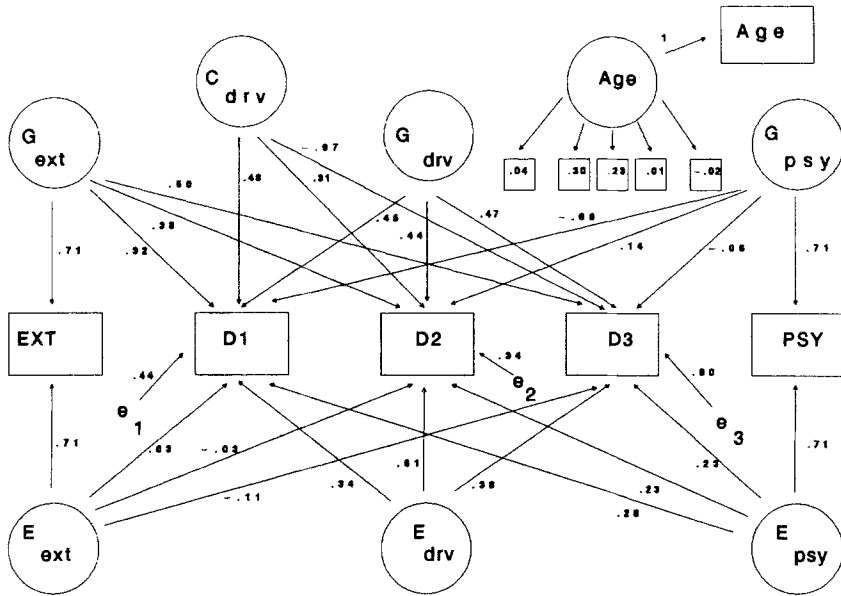


Fig. 2. Factor model for covariation between willingness to drive at 1, 2, and 3 h after alcohol ($D1$, $D2$, $D3$) and the personality variables Extraversion (Ext) and Psychoticism (Psy). LISREL estimates of path coefficients for male twins are shown.

psychoticism (Psy) genes on Drive? responses; and (iii) G_{drv} loads only on the three Drive? responses and estimates genetic effects not covarying with either of the personality dimensions. Analogous environmental group factors E_{ext} , E_{psy} , and E_{drv} are specified to estimate individual environmental covariation. Specific individual environmental variance is allowed at each time (e_1 , e_2 , and e_3 in Fig. 2). Inspection of the correlations also suggests that shared environmental influences may be important in Drive? responses, so we also specify the group factor C_{drv} to estimate covariation between the three Drive? responses due to environmental influences shared by brothers. No shared environmental effects are allowed for personality because no evidence for them has been found in very much larger twin samples (Martin and Jardine, 1986; Plomin and Daniels, 1987).

Specification of this model in LISREL is shown in Appendix I (Fig. A1). Since the input matrices in Table II are not positive definite, we used the approach outlined by Boomsma *et al.* (1989b) to replace the singular matrices by positive definite ones. In this approach an eigenvalue decomposition of the original matrix is carried out and the zero eigenvalue is replaced by a small positive number. In general, this produces only

Table III. Percentages of Total Variance in Males Explained by Latent Factors of First Model (Fig. 2) (Estimated by ML from Data Matrices Adjusted to Be Positive Definite)

	Age	G_{drv}	G_{ext}	G_{psy}	C_{drv}	E_{drv}	E_{ext}	E_{psy}	e_i
Drive 1 h	9	20	10	0	23	11	0	8	19
Drive 2 h	5	19	14	2	9	37	0	5	11
Drive 3 h	0	22	25	0	0	14	1	5	36
Extraversion	0	—	50	—	—	—	50	—	—
Psychoticism	0	—	—	50	—	—	—	50	—

minor changes in the data matrices, as can be seen by comparing the adjusted data matrices in Appendix I (Fig. A1) with the raw matrices in Table II. Consequently, parameters estimated from the adjusted matrices are not generally biased.

Using the adjusted, positive definite matrices has the advantage that it is now possible to use maximum-likelihood (ML) estimation, which is a better-conditioned estimation procedure than unweighted least squares (ULS). We did, however, also analyze the original singular matrices with the ULS option in LISREL and found no large differences in parameter estimates, except for one parameter. Using ULS the unique environmental variance associated with the second Drive? variable could not be estimated, whereas no problems arose with ML estimation.

ML parameter estimates for the factor model are shown in Fig. 2. These are standardized path coefficients and need to be squared to obtain the proportions of variance which are shown in Table III; across a row these variance components should add approximately to one. Inspection of the normalized residuals reveals that where the fit is poor, this is due to heterogeneity between Twin 1 and Twin 2 rather than any systematic inadequacy of the model. Since the designation of Twin 1 and Twin 2 in our study is not a reflection of birth order, this heterogeneity is unlikely to be biologically meaningful.

An Autoregressive Model

Since we are dealing with repeated measures we also fitted a model that specified an autoregressive or Markovian structure for the latent variables. With three observations only, we cannot expect that such a model will fit the data better than a factor model, but it gives a clearer representation of the time-dependent process.

We started by specifying a first-order autoregressive process for both G and E , $\eta_i = \beta\eta_{i-1} + \zeta$, so that for each repeated observation of the Drive? variable there is an underlying genetic and an underlying individual

environmental factor that is correlated with the previous latent factor. The regression of a latent factor on the previous latent factor is estimated by β and the residual part of η , i.e., that part that is uncorrelated with the previous latent factor, is symbolized by ζ .

Cognizant of the results of the first model, extraversion was specified to load on the first genetic factor and psychoticism on the first individual environmental factor. In addition, a unique genetic factor was specified for psychoticism. Further, loadings on a group shared environment factor (C) were specified for only the first two Drive? responses. As in the first model, all observed variables loaded on a latent age factor. Measurement errors for each observed variable were specified in θ . Because we work with input correlation matrices of Twin 1 and Twin 2, measurement scales for the latent factors have to be defined by standardizing Ψ , which means standardizing the residual terms ζ (Boomsma *et al.*, 1989a).

With these specifications LISREL did not converge properly (number of iterations exceeded 250). Inspection of the standardized solution revealed a perfect correlation between the first two latent genetic factors. The reason that LISREL cannot handle this situation is that the residual terms are standardized. This precludes a correlation of one between the latent factors since

$$\begin{aligned} \text{cov}(G1,G2) &= \text{cov}(G1,\beta G1 + \zeta) = \beta \text{var}(G1) = \beta, \\ \text{cor}(G1,G2) &= \text{cov}(G1,G2)/\sqrt{\text{var}(G1)}\sqrt{\text{var}(G2)} \\ &= \beta/\sqrt{[\beta^2 \text{var}(G1) + 1]} = \beta/\sqrt{(\beta^2 + 1)}. \end{aligned}$$

Up until 250 iterations β increases so that the relative weight of the residual variance decreases. No such problems would arise when λ 's are fixed at one and the variances of the ζ 's estimated.

To overcome this problem, only one genetic factor was specified for the first two observations. This model is displayed in Fig. 3, which also shows the standardized parameter estimates. The LISREL input for this model is given as Appendix II (Fig. A2). The latent factors that influence the Drive? responses are the same (G and C) or almost the same (E) for the first two measures, whereas the latent factors that influence the last observation show a much lower correlation with the previous factors (.86 for $G3$ and .75 for $E3$).

The correlations of Psy and Ext with the Drive? variables are mediated through the first factor of the latent series so that, for example, $\text{cor}(\text{Psy}, D1) = .39 * .51 = .2$ and $\text{cor}(\text{Psy}, D3) = .39 * .91 * .75 * .67 = .18$.

Table IV gives the amount of the total variance of the observed variables explained by the latent factors. For the third Drive? variable ($D3$)

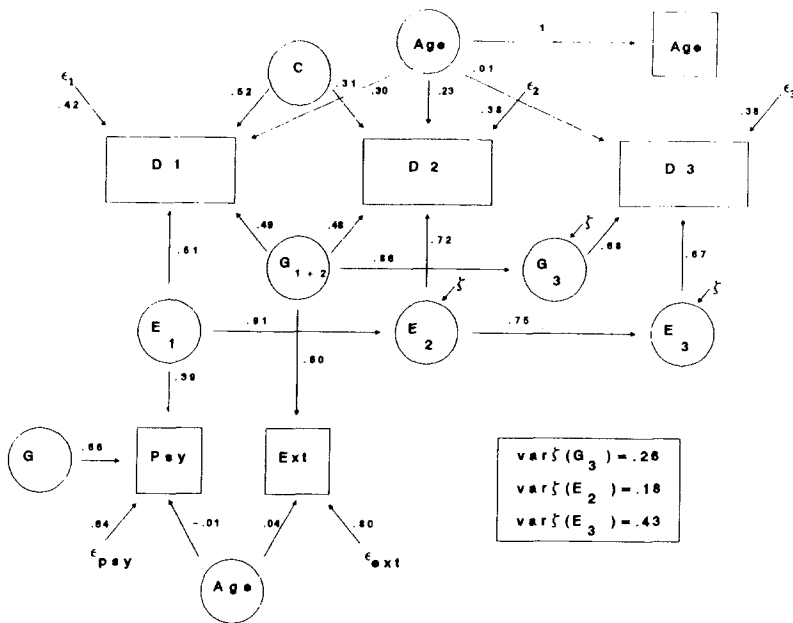


Fig. 3. Autoregressive model and LISREL estimates for male twin data.

about 46% of the observed variance is explained by genetic influences. Of this 46%, part is shared by the previous latent G (i.e., $.86^2 * .68^2 = .34$) and part is “new” variance [i.e., $(1 - .86^2) * .68^2 = .12$].

Judged by the normalized residuals, there is little to choose between this and the factor model. However, the Markovian model is more elegant in that it postulates a much stronger mechanistic hypothesis and is more parsimonious. Since the two models are not nested it is not possible to compare them by likelihood-ratio test. The goodness-of fit chi-square sta-

Table IV. Percentages of Total Variance Explained by Latent Factors of the Second Model (Fig. 3) (Estimates Based on Standardized Solution)

	Age	G1 + 2	G3	G _{psy}	E1	E2	E3	C	ε
Drive 1 h	9	24	—	—	26	—	—	27	17
Drive 2 h	5	23	—	—	—	52	—	9	15
Drive 3 h	0	—	46	—	—	—	45	—	15
Extraversion	0	36	—	—	—	—	—	—	64
Psychoticism	0	—	—	43	15	—	—	—	41

tistics are highly biased because the observed matrices are nearly singular, so we do not consider them.

Both programs shown in the Appendixes ran in less than 6 min using PC-LISREL on an IBM AT clone with an 80286 coprocessor.

DISCUSSION

Willingness to drive after drinking a large dose of alcohol is a repeatable phenomenon, at least in the context of a laboratory study, in that people make the same judgment on two occasions several months apart. This judgment (or lack of it!) appears to be influenced by genetic factors and to be related to the personality traits of extraversion and psychoticism. Age is also important, older males in our sample being more willing to drive soon after drinking. Our interest has been to elucidate the ways in which the associations between personality and drunk driving are modulated by genes and environment. We have fitted two models, the first an exploratory factor model and the second a much stronger mechanistic model which takes account of the time-series nature of the data. A substantial part of genetic variation is willingness to drive when drunk appears to derive from genes also responsible for variation in extraversion. Higher psychoticism scores are also associated with willingness to drive when drunk, but this appears to arise from environmental influences on the individual.

Others have examined the relationship between drunk driving and personality (for a review see Donovan *et al.*, 1983, especially pp. 404–406) but always in drivers arrested for driving under the influence of alcohol or in alcoholics or heavy drinkers at a high risk of drunk driving. In these samples, associations with personality are many and varied but most indicate elevated social deviance or psychopathy, probably reflected in the correlations with psychoticism in our sample. Extraversion scores are elevated in some studies and this is the dominant feature of our results. Interestingly, higher scores for neuroticism and related measures are also found, but this may reflect the state of having been arrested, since we find no such correlation in our volunteer sample.

From the results of the factor model (Table III) we see that the variance of extraversion is half genetic and half due to individual environmental experiences, in line with previous findings (e.g., Martin and Jardine, 1986). But covariation of extraversion with Drive? responses appears to be due almost entirely to pleiotropic gene effects, accounting for 10% of the variance at 1 h, 14% at 2 h, and 25% at 3 h. Individual environmental experiences affecting extraversion have negligible effects on willingness to drive.

The story for covariation of Drive? responses with psychoticism is exactly the reverse. Once again, we see that the variance of psychoticism is equally divided between genetic and environmental causes, also in line with previous findings (e.g., Eaves and Eysenck, 1977). But now any covariation with willingness to drive appears to be environmentally modulated and environmental influences on psychoticism account for 8, 5, and 5% of variance in $D1$, $D2$, and $D3$.

Having removed the genetic variance in Drive? responses due to pleiotropic effects of genes affecting personality, we see from the loadings on G_{drv} that 20, 19, and 22% of the total variance is due to other genetic influences producing covariance between responses at times 1, 2, and 3, respectively. Similarly, a common set of individual environmental influences not related to personality (E_{drv}) but influencing all three responses accounts for 11, 37, and 14% of the variance at 1, 2, and 3 h. Specific individual environmental influences account for 19, 11, and 36% of the variance at 1, 2, and 3 h.

The Age factor removes any variance due to linear regression on age, but as expected from Table I, this is appreciable only for Drive? responses at 1 and 2 h. Finally, the group-shared environmental factor C_{drv} appears to account for 23% of the variance at 1 h and 9% at 2 h. At 3 h the loading is effectively zero.

The second, autoregressive model builds on these findings, but now the persistence and change in genetic and environmental influences on $D1$, $D2$, and $D3$ are more apparent (Fig. 3). A latent genetic factor, which is responsible for all the genetic variance in extraversion, accounts for all of the genetic variance in $D1$ and $D2$. Its influence carries over to $D3$, but new genetic effects come into play 3 h after drinking. This continuity between $D1$ and $D2$ is also seen for environmental effects, and only at $D3$, as subjects start to sober up, is there a suggestion of substantial new environmental factors coming into play. The initial environmental factor influencing willingness to drive also influences the psychoticism score, and its effects carry through to $D2$ and, to a lesser extent, $D3$.

APPENDIXES I AND II

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AGE, "WOULD YOU DRIVE NOW?" T1 - T3, E & P : MZ MALES
DA NG=2 NI=11 NO=37
LA
AGE D1T1 D2T1 D3T1 E1 P1 D1T2 D2T2 D3T2 E2 P2
KM
  1.04  .51  1.00  .61  .83  1.01  .50  .71  .77  1.15  .26
  .50  .50  .76  1.06  -.17  .24  .16  .03  .03  1.02  .42
  .61  .56  .49  .51  -.27  1.04  .37  .66  .43  .47  .45
  -.05  .80  1.07  .15  .55  .39  .64  .38  -.05  .56  .57
  1.06  .05  .21  .19  .46  .54  .00  .42  .08  .09  1.01
  .00  .08  .04  -.18  -.21  .43  .08  -.02  -.19  .00  1.00
MO NY=11 NE=11 NK=20 GA=FU,FI PH=SY,FI LY=ID PS=ZE TE=ZE
PA GA
1 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0
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1 0 0 0 0 1 0 0 0 0 0 0 0 0 0 0 1 0 0 0 1 0 0 0
EQ GA(2,1) GA(7,1) 0. 0. 0. 0. 0. 0. 0. 1.
EQ GA(3,1) GA(8,1) 0. 0. 0. 0. 0. 0. 0. 0. 1.
EQ GA(4,1) GA(9,1) 0. 0. 0. 0. 0. 0. 0. 0. 0. 1.
EQ GA(5,1) GA(10,1) 0. 0. 0. 0. 0. 0. 0. 0. 0. 0. 0. 1.
EQ GA(6,1) GA(11,1) 0. 0. 0. 0. 0. 0. 0. 0. 0. 0. 0. 0. 1.
EQ GA(2,2) GA(7,5) 0. 0. 0. 0. 0. 0. 0. 0. 0. 0. 0. 0. 0. 1.
EQ GA(3,2) GA(8,5) 0. 0. 0. 0. 0. 0. 0. 0. 0. 0. 0. 0. 0. 0. 1.
EQ GA(4,2) GA(9,5) 0. 0. 0. 0. 0. 0. 0. 0. 0. 0. 0. 0. 0. 0. 0. 1.
EQ GA(2,3) GA(7,6) 0. 0. 0. 0. 0. 0. 0. 0. 0. 0. 0. 0. 0. 0. 0. 0. 1.
EQ GA(3,3) GA(8,6) 0. 0. 0. 0. 0. 0. 0. 0. 0. 0. 0. 0. 0. 0. 0. 0. 0. 1.
EQ GA(4,3) GA(9,6) 0. 0. 0. 0. 0. 0. 0. 0. 0. 0. 0. 0. 0. 0. 0. 0. 0. 0. 1.
EQ GA(5,3) GA(10,6) 0. 0. 0. 0. 0. 0. 0. 0. 0. 0. 0. 0. 0. 0. 0. 0. 0. 0. 0. 1.
EQ GA(2,4) GA(7,7) 0. 0. 0. 0. 0. 0. 0. 0. 0. 0. 0. 0. 0. 0. 0. 0. 0. 0. 0. 0. 1./
EQ GA(3,4) GA(8,7) ST 0.5 ALL
EQ GA(4,4) GA(9,7) OU MR
EQ GA(6,4) GA(11,7) AGE, "WOULD YOU DRIVE NOW?" T1 - T3, E & P : DZ MALES
EQ GA(2,8) GA(7,8) DA NO=34
EQ GA(3,8) GA(8,8) LA
EQ GA(4,8) GA(9,8) AGE D1T1 D2T1 D3T1 E1 P1 D1T2 D2T2 D3T2 E2 P2
EQ GA(2,9) GA(7,15) KM
EQ GA(3,9) GA(8,15) 1.03 .32 1.05 -.02 .73 1.07 .03 .53 .80 1.01 -.13
EQ GA(4,9) GA(9,15) -.23 -.04 .09 1.00 .06 .16 .49 .21 .00 1.02 .03
EQ GA(2,10) GA(7,16) .45 .25 .10 .21 .07 1.06 .00 .24 .40 .27 .17
EQ GA(3,10) GA(8,16) .12 .85 1.05 -.60 -.27 -.18 .02 .26 -.38 .38 .50
EQ GA(4,10) GA(9,16) 1.06 .02 -.21 .00 .19 .32 -.19 .37 .51 .26 1.00
EQ GA(5,10) GA(10,16) .03 -.13 .16 .03 -.07 .46 .07 .45 .34 -.04 1.02
EQ GA(2,11) GA(7,17) MO GA=IN PH=SY,FI
EQ GA(3,11) GA(8,17) MA PH
EQ GA(4,11) GA(9,17)
EQ GA(6,11) GA(11,17) same as for MZ twins except off-diagonal 1's replaced by 0.5
EQ GA(2,12) GA(7,18)
EQ GA(3,13) GA(8,19) ST 0.5 ALL
EQ GA(4,14) GA(9,20) OU MR

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Fig. A1. Appendix I. LISREL input for factor model.

REFERENCES

- Boomsma, D. I., Martin, N. G., and Molenaar, P. C. M. (1989a). Factor and simplex models for repeated measures: Application to two psychomotor measures of alcohol sensitivity in twins. *Behav. Genet.* **19**:79–96.
- Boomsma, D. I., van den Bree, M. B. M., Orbeleke, J. K., and Molenaar, P. C. (1989b). Resemblances of parents and twins in sports participation and heart rate. *Behav. Genet.* **19**:123–141.
- Donovan, D. M., Marlatt, G. A., and Salzberg, P. M. (1983). Drinking behavior, personality factors and high-risk driving: A review and theoretical formulation. *J. Stud. Alcohol* **44**:395–428.
- Eaves, L. J., and Eysenck, H. J. (1977). A genotype-environmental model for psychoticism. *Adv. Behav. Res. Ther.* **1**:5–26.
- Eysenck, H. J., and Eysenck, S. G. (1975). *Personality Questionnaire (Junior and Adult)*, Hodder and Stoughton Educational, London.
- Jöreskog, K. G., and Sörbom, D. (1986a). *PRELIS. A Program for Multivariate Data Screening and Data Summarisation. A Preprocessor for LISREL*, Scientific Software Inc., Mooresville, Ind.
- Jöreskog, K. G., and Sörbom, D. (1986b). *LISREL VI. Analysis of Linear Structural Relationships by Maximum Likelihood, Instrumental Variables, and Least Squares Methods*, Scientific Software Inc., Mooresville, Ind.
- Martin, N. G., and Jardine, R. (1986). Eysenck's contributions to behaviour genetics. In Modgil, S., and Modgil, C. (eds.), *Hans Eysenck: Consensus and Controversy*, Falmer Press, London, pp. 13–47.
- Martin, N. G., Perl, J., Oakeshott, J. G., Gibson, J. B., Starmer, G. A., and Wilks, A. V. (1985a). A twin study of ethanol metabolism. *Behav. Genet.* **15**:93–109.
- Martin, N. G., Oakeshott, J. G., Gibson, J. B., Starmer, G. A., Perl, J., and Wilks, A. V. (1985b). A twin study of psychomotor and physiological responses to an acute dose of alcohol. *Behav. Genet.* **15**:305–347.
- Neale, M. C., and Martin, N. G. (1989). The effects of age, sex and genotype on self-report drunkenness following a challenge dose of alcohol. *Behav. Genet.* **19**:63–78.
- Plomin, R., and Daniels, D. (1987). Why are children in the same family so different from one and other? *Behav. Brain Sci.* **10**:1–60.