Behavior Genetics, Vol. 15, No. 4, 1985

A Twin Study of Psychomotor and Physiological Responses to an Acute Dose of Alcohol

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Received 6 Nov. 1984—Final 30 Nov. 1984

A battery of psychomotor tasks and physiological measures was administered to 206 pairs of twins before alcohol and then three times at hourly intervals after they ingested a standard dose of ethanol (0.75 g/kg body weight). Repeat measurements were obtained for 41 of these pairs on a second occasion. Performance on motor coordination, standing steadiness, pursuit rotor, arithmetic computation, and reaction-time tasks deteriorated after alcohol, but decrements on the five tasks were generally independent of each other. Measurements of blood pressure, pulse rate, and skin temperature were all elevated following alcohol intake, but these responses were also uncorrelated. The variance in many of these measures increased after alcohol. An analysis of covariance structure revealed that most of this additional variance exposed by alcohol was genetic in origin, particularly for standing steadiness, pursuit rotor, arithmetic computation, and pulse rate. Up to 50% of the variance in body sway after alcohol was estimated to be due to genetic factors expressed only under the influence of alcohol. Although significant correlations were found with blood alcohol concentration, previous drinking experience, and the personality trait Extraversion, little of the genetic variance exposed by

This work was supported by the Australian Associated Brewers.

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0001-8244/85/0700-0305$04.50/0

alcohol could be explained by these predictors. It is concluded that the sources of the considerable genetic variation affecting performance under alcohol must be sought elsewhere.

KEY WORDS: alcohol susceptibility; psychomotor performance; twins; genetics.

INTRODUCTION

There is a large body of literature characterizing "typical" behavioral and physiological reponses to alcohol (e.g., Goldberg, 1966) but less attention has been paid to the variation among individuals in these responses. While the effects of factors such as sex, age, and drinking experience on response have been thoroughly investigated (Powell *et al.*, 1973; Wait *et al.*, 1982), there has been little attempt to assess the extent of genetic variability. Propping (1977a,b) studied EEG patterns and psychomotor performance before and after alcohol ingestion in 52 pairs of male twins. He found some evidence of genetic influences on EEG responses to ethanol but not on psychomotor responses. However, a study with such small numbers of twins has very little power to detect genetic variance (Martin *et al.*, 1978).

We have measured psychomotor performance and physiological variables before and after the ingestion of an acute dose of alcohol (0.75 g/kg) in 206 pairs of 18- to 34-year-old twins. Of these, 41 pairs were retested on a second occasion. In a previous paper we reported on the genetic variation in blood alcohol metabolism in these twins (Martin *et al.*, 1985). In this paper we present our analysis of psychomotor and physiological response data from this experiment. Specifically, we address the following questions:

1. How much variation among individuals is there in the response to alcohol?
2. How repeatable is this variation over occasions?
3. What proportion of the variation in response is due to genetical factors?
4. How much can be explained by the blood alcohol concentration, previous drinking experience, and personality variables?

SUBJECTS AND METHODS

Subjects

Pairs of healthy twins aged between 18 and 34 years (mean, 23.1 years) were recruited from the Sydney and Canberra metropolitan areas. Both members of a twin pair attended on the same day, and between two

and six pairs were tested each day. A total of 206 pairs was tested successfully, and of these, 41 pairs (36 females and 46 males) were also tested on a second occasion between 1 and 17 months after the first (mean, 4.5 months). All subjects were of European (mainly Northern European) extraction. Zygosity determination and other details of the sample are described by Martin *et al.* (1985). Of the 206 twin pairs for whom measurements were available, there were 43 monozygotic (MZ) female, 42 MZ male, 44 dizygotic (DZ) female, 38 DZ male, and 39 DZ opposite-sex (DZOS) pairs. There were no substantial differences in age distribution among the five zygosity groups.

Experimental Protocol

Twins attended a testing session beginning at 9:00 AM, having eaten a light, nonfatty breakfast about an hour earlier. Subjects answered questions on their normal drinking habits, in particular the number of drinks they consumed in a normal week and the age at which they began drinking regularly. They also completed the Eysenck Personality Questionnaire (Eysenck and Eysenck, 1975), which was scored for the four traits Extraversion (E), Psychoticism (P), Neuroticism (N), and the Lie or Social Desirability scale (L).

Subjects were trained to plateau on an apparatus used to test psychomotor performance, and then prealcohol (T_0) measurements were taken of all the physiological and behavioral tests. Subjects were then given an alcohol dose of 0.75 g ethanol/kg body weight diluted to 10% (v/v) in sugarless lemon squash and were asked to drink it in 20 min at a constant rate. After a further 20 min, postalcohol testing began, with repeated measurements of breath alcohol, blood alcohol, blood pressure, pulse, skin temperature, and psychomotor performance including motor coordination, body sway, hand steadiness, simple and complex reaction times, and self-ratings of intoxication. Each subject was measured three times at hourly intervals (T_1, T_2, and T_3), although additional readings of breath and blood alcohol were taken at more frequent intervals to increase information about the ethanol metabolism curve (Martin *et al.*, 1985).

Subjects were started on the testing circuit at 7-min intervals. In an attempt to avoid observer bias toward pair concordance, cotwins were never tested consecutively. Each subject took about 25 min to complete a circuit of the test battery. Further details of the experimental protocol are given by Martin *et al.* (1985).

Test Battery

The test battery used has been described in detail by Belgrave *et al.* (1979). These psychomotor tests have been shown from dose–response curves to be sensitive to ethanol (Franks *et al.*, 1976). Measurements were obtained for psychomotor and physiological variables in the following order.

Motor Coordination

Motor coordination was assessed using the Vienna determination apparatus (VDA; Schufried, Stuttgart, West Germany), which generated a random sequence of visual and auditory stimuli to which the subject had to give specific button or foot-pedal responses. One hundred stimuli were presented at 1.22-sec intervals and three scores were obtained for each test: number of correct responses (VDANC), number of delayed correct (>1-sec) reponses (VDADC), and number of incorrect responses (VDAIC). Since the number of incorrect responses was obtained by difference, these three variables are not independent.

Body Sway

The subject was asked to stand relaxed and as steadily as possible on a platform. A displacement transducer was mounted beneath the platform and any shift in position created an electrical impulse. Oscillations were integrated and the time taken (seconds) to accumulate a given amount of sway was recorded on a Grass polygraph (Quincy, Mass.). The longer the time, the steadier the subject. Measurements were made under two conditions, first with the subject's eyes open (EO) and then with the subject's eyes closed (EC). Duplicate measurements were made under each condition and their average was taken. Subjects wearing high heels removed their shoes for this test.

Pursuit Rotor

This standard apparatus (Schufried, Stuttgart, West Germany) was used to measure hand–eye coordination. Subjects stood and attempted to follow a light target moving in a clockwise circular motion with a photocell stylus which recorded the number of times off the target (PURNO) and accumulated the total time off the target (PURDO). Deviations to the left and right of the target were recorded separately but were added for our purposes. Because subjects stood for this task, it was thought that hand steadiness might be affected by height, but negligible

correlations were found between height and either measure of performance.

Arithmetic Computation

The Arbeit und Konzentration Testgrate (AKTG; Zak, Simbach am Inn, West Germany) is a speeded number task in which simple addition and subtraction computations are presented to the subject, who is asked to do as many computations as possible in 2 min. The number of correct (AKTNC) and incorrect responses (AKTIC) was recorded.

Simple and Complex Rection Times

The apparatus (Schufried, Stuttgart, West Germany) presented visual and auditory stimuli either separately or together at random time intervals. For the visual reaction time (VRT), subjects responded to a white light; for the auditory reaction time (ART), to a tone; and for the complex reaction time (CRT), only to the combination of white light and tone among several other stimuli. Response times (milliseconds) to five stimuli for each condition were recorded.

Physiological Measures

Subjects were asked to lie quietly for 2 min, after which readings of the pulse (PULSE) and systolic and diastolic blood pressure (SYSBP and DIABP) were made. The skin temperature on the cheek (SKTEM) was measured with a probe attached to a digital readout (Medtel LCDT/1) in an attempt to quantify the flushing response to alcohol.

Blood Alcohol Concentrations

For each subject six blood alcohol concentration (BAC) readings were obtained during the experiment. Breath alcohol measurements were also obtained but have not been used in the analysis below. Details are given by Martin *et al.* (1985).

Intoxication Self-Ratings and Willingness to Drive

At the end of each circuit subjects were asked, "How drunk do you feel now, on a scale of 1 = 'completely sober' to 10 = 'the most drunk I have ever been'?" They were also asked, "Would you drive a car now?" and this was scored no = 0 and yes = 1. These variables are designated INTSR and DRIVE.

Statistical Methods

Scaling

It was found that the variance of a body-sway measurement was proportional to its mean value. By transforming raw scores to a log scale we were able to remove this relationship. It was expected that body sway would depend on the height of the center of gravity above the platform on which the subject was standing. Accordingly, we performed a regression analysis of prealcohol log body-sway measurements on height and weight. Initially this was carried out separately for males and females but we found that there was no heterogeneity of the regressions; the same equation could be applied to both sexes. The final regression equations used to correct single body-sway measurements were as follows:

$$\text{BODEO} = \log(\text{EO}) - 3.22589 + 0.00998 \cdot \text{weight (kg)} + 0.00277 \cdot \text{height (cm)},$$

$$\text{BODEC} = \log(\text{EC}) - 3.03177 + 0.00897 \cdot \text{weight (kg)} - 0.00422 \cdot \text{height (cm)}.$$

Almost the same corrected scores were obtained when the height term was omitted. The measurements used below are the means of duplicates, each transformed and corrected as above.

The only other psychomotor measures requiring transformation were reaction times, which were significantly positively skewed but could be centralized by log transformation. All reaction times (milliseconds) were thus transformed and a single observation was taken as the mean of five replicates.

Weekly alcohol consumption and years drinking, as reported by the twins themselves, also had positively skewed distributions and were logarithmically transformed to create the variables LCONW and LYDR, respectively.

A list of all the variables measured in this study and their abbreviations employed in this paper is given in Table 1.

Repeatability

Of the 206 pairs who completed the protocol, 41 pairs (36 women and 46 men) returned on a second occasion between 1 and 17 months later (mean, 4.5 months) and repeated the entire protocol. Paired results of these individuals have been used to estimate repeatabilities. An analysis of variance between and within individuals was performed, with occasions partitioned out of the within-individuals mean square. If the between-occasions term was not significant, then repeatability

Table I. Variables Used in this Study and Their Working Abbreviations

Psychomotor measures	
Motor coordination	
Vienna determination apparatus	
Number correct	VDANC
Delayed correct	VDADC
Incorrect	VDAIC
Standing steadiness	
Body sway (log transformed and corrected for height)	
Eyes-open condition	BODEO
Eyes-closed condition	BODEC
Hand steadiness	
Pursuit rotor	
Number of times off target	PURNO
Dwell time off target	PURDO
Arithmetic computation	
AKTG apparatus	
Number correct	AKTNC
Number incorrect	AKTIC
Reaction times (log transformed)	
Simple visual reaction time	VRT
Simple auditory reaction time	ART
Complex reaction time	CRT
Subjective measures	
Intoxication self-rating	INTSR
"Would you drive a car now?"	DRIVE
Physiological measures	
Systolic blood pressure	SYSBP
Diastolic blood pressure	DIABP
Pulse	PULSE
Skin temperature	SKTEM
Drinking habits	
Normal weekly consumption (log transformed)	LCONW
Years since began drinking (log transformed)	LYDR
Personality measures (Eysenck Personality Questionnaire)	
Extraversion	E
Psychoticism	P
Neuroticism	N
Lie	L
Blood alcohol concentration	BAC

$$r = S_{bi}^2/(S_{bi}^2 + S_{wi}^2),$$

where S_{bi}^2 is the between-individuals component of variance and S_{wi}^2 is the within-individuals component. Performance on many of the psychomotor variables improved with practice so the between-occasions term was significant. In this case we calculated

$$r' = S_{bi}^2/(S_{bi}^2 + S_{I\times O}^2),$$

where $S_{I\times O}^2$ is the individuals × occasions component of variance. Where there is a significant difference between occasions, then r' is tabled.

Genetical Analysis

We require an analysis to detect genes influencing variation and covariation in performance between trials during intoxication that are not acting before alcohol ingestion. One approach is through the genetic analysis of covariance structure, which allows us simultaneously to test hypotheses about both the sources and the structure of covariation (Martin and Eaves, 1977; Martin *et al.*, 1979). Application of this technique to the current problem is also discussed by Martin *et al.* (1981).

Data Reduction

For every character we perform an analysis of variance in each of the five twin groups to calculate the between- and within-pairs matrices of mean products among performance at the four times T_0–T_3. For n pairs of twins the between-pairs matrix has $n - 1$ degrees of freedom and the within-pairs matrix has n degrees of freedom. A large difference in the means of males and females will inflate the mean products within DZ opposite-sex pairs (DZOS), so these are corrected for the vector of mean differences and the corresponding degree of freedom is removed.

Sources of Covariation

The three sources of variation we consider are individual environmental influences (E_1), which also include measurement error, additive gene action (V_A), and environmental influences shared by cotwins but differing among families (E_2). Confounded with estimates of E_2 will be any additive genetic variation accruing from assortative mating, but it seems unlikely that this will be an important factor for the variables we are studying. We do not consider genetic variance due to dominance since the power of our study to detect any variation arising from this source is minimal (Martin *et al.*, 1978). The contributions of E_1, E_2, and V_A to the variation within and between pairs of MZ and DZ twins are derived from standard genetical and statistical theory (Eaves *et al.*, 1978).

Structural Models

We first consider a model in which only two factors contribute to the variance at each trial, one for each of the E_1 and V_A sources. Hence $\mathbf{H}$ is the 4×1 matrix of E_1 factor loadings and $\mathbf{\Delta}$ is the corresponding matrix of V_A loadings. We postulate that each individual has his or her own "genetic level" of performance before alcohol but that alcohol has a uniform effect at subsequent trials in altering this level by a constant factor in every individual. In other words, our null hypothesis is that there are no genetic differences regulating the behavioral response to alcohol.

We also postulate that any specific variation at each trial not explained by the two common factors is solely environmental in origin, so that our model also contains a diagonal matrix **E** of E_1 specific standard deviations (for computational reasons these square roots are more convenient to estimate than the specific variance components themselves). If environmental variation at each trial is all random measurement error, then we expect to find no E_1 factor variance and only specific E_1 variance. If, however, there are environmental differences affecting individuals' performances systematically at each trial, then we expect to find an E_1 general factor.

Hence the model for the four expected mean products matrices may be written as follows:

$$\sum_{BMZ} = \Delta\Delta' + HH' + \mathrm{E}^2,$$

$$\sum_{WMZ} = HH' + E^2,$$

$$\sum_{BDZ} = \tfrac{3}{2}\Delta\Delta' + HH' + E^2,$$

$$\sum_{WDZ} = \tfrac{1}{2}\Delta\Delta' + HH' + E^2.$$

The matrices $\sum_{BMZ}$, $\sum_{WMZ}$, $\sum_{BDZ}$, and $\sum_{WDZ}$ represent the mean products between (B) and within (W) pairs of monozygotic (MZ) and dizygotic (DZ) twins, respectively. There are thus 12 parameters (four each of η_i, δ_i, and e_i) to estimate in our model (which, for subsequent purposes, we designate Model 3).

This model does not tell us whether there are genetic effects specifically involved in the susceptibility to alcohol. If there are such genetic differences, they will affect the variance and covariance among the performances on the three postalcohol trials, but they will not covary with the performance on the prealcohol trial. We can test this hypothesis by fitting a second genetic factor independent of the first and loading on T_1, T_2, and T_3 but not on T_0, i.e., acting in the presence of alcohol but not in its absence. The effects influencing performance at each time are now represented in the path diagram in Fig. 1, and from this, expectations for the variation and covariation between cotwins at different times can be derived. This model (Model 4) will thus estimate an additional three alcohol factor loadings, or 15 parameters in all.

An alternative source of covariation to genetic factors might be shared environmental influences experienced by both members of a twin pair but differing between pairs (E_2). We may thus fit two further models analogous to those already considered but with a matrix of E_2 factor loadings substituted for Δ. This matrix, as in the case of Δ, may contain

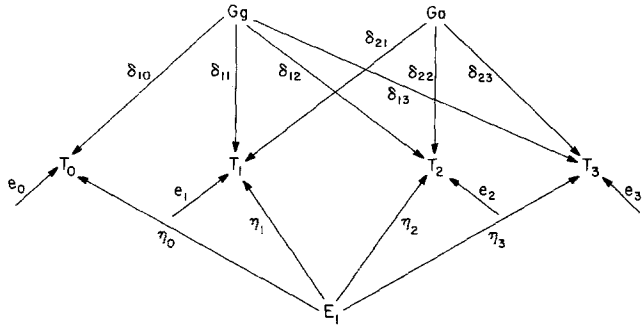


Fig. 1. Path diagram to illustrate the contributions to covariance among measures at four times, T_0 (before alcohol) and T_1, T_2, and T_3 after alcohol ingestion. G_g is a general genetic factor making contributions δ_{1i} at all times; G_a is genetic variance exposed by alcohol, making contributions δ_{2i} at T_1, T_2, and T_3. An individual environmental factor, E_1, makes contributions η_i to covariation at all four times, and specific environmental variance at each time is contributed through the e_i paths. Shared environmental variance may contribute to covariation in the same pattern as the genetic factors but is not shown here for clarity.

only a single general factor, loading on performance at all four times alone (Model 1), or a general factor as well as an alcohol factor which loads only on postalcohol performance at times T_1–T_3 (Model 2).

It is possible that all three sources, E_1, E_2, and V_A, contribute to covariation, and we can thus fit four further models: (i) a general factor for each of E_2 and V_A which estimates 16 parameters (Model 5), (ii) a general and alcohol factor for the E_2 source and a general factor alone for the V_A source (19 parameters; Model 6), (iii) a general factor alone for the E_2 source and a general and alcohol factor for the V_A source (19 parameters; Model 7), and (iv) general and alcohol factors for both the E_2 and the V_A sources (22 parameters; Model 8). In all eight models the structure of E_1 covariation is specified identically, with a single common factor and specific standard deviations estimated at all four times.

Testing of Models

To each set of mean products matrices, we thus fit eight different models. The degrees of freedom against which to judge the chi-square for the goodness of fit of a model are $m[v(v + 1)/2] - p$, where p parameters are estimated. In our case $v = 4$ for the measures of performance at the four sampling times and $m = 10$ if we fit the model to between- and within-mean products matrices for all five twin groups. If the preliminary fitting of univariate models for each sampling time indicates

heterogeneity of fit between males and females, then we fit the multivariate models to MZ and DZ matrices of one sex at a time, in which case $m = 4$.

Central to this paper is the detection of variance expressed only under the influence of alcohol. Its presence is inferred if a significant improvement in fit is observed on the addition of an "alcohol factor" to a model previously containing only a general factor for the same source. Thus, if Model 4 is a significant improvement over Model 3, then alcohol-specific genetic variance is inferred. If Model 2 is an improvement over Model 1, then alcohol-specific variance due to shared environment is inferred. If, as we frequently observe, a significant improvement in fit is obtained in both these cases, then the model giving the best absolute fit is preferred. When discrimination between E_2 and V_A models is difficult, then comparisons must be made among Models 5–8. In general, criteria for the preferred model include not only its absolute fit, but also its parsimony and improvement over simpler or alternative models.

Once a preferred model has been decided, the significance of each parameter estimated in the model is judged against its standard error and its contribution to the total variance in performance at a given time can be calculated.

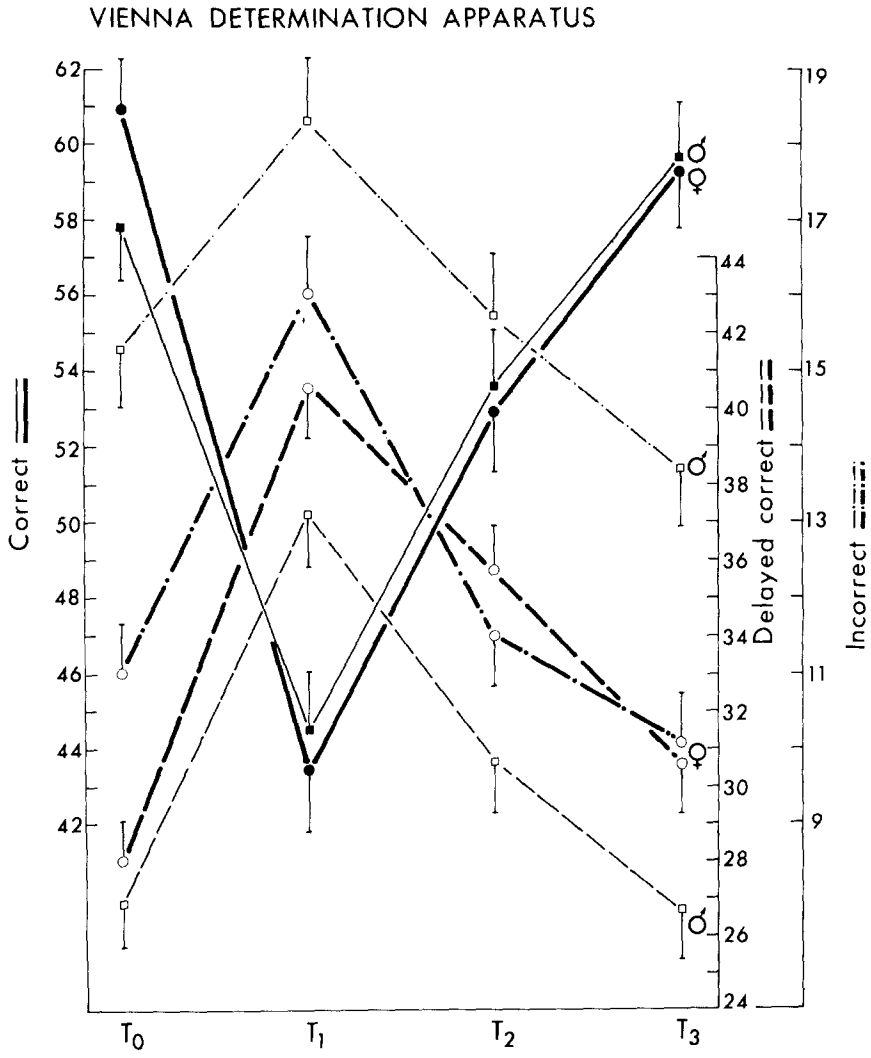
RESULTS

Mean Effects of Alcohol and Sex

It can be seen from Figs. 2–6 that alcohol causes a significant decrement in performance in all psychomotor measures, particularly at T_1. For many of the measures there are sex differences which are consistent at all sampling times, both before and after alcohol. For some measures there are also sex differences in the extent of the decrement following alcohol intake. However, the direction of sex differences varies according to the measure under consideration.

For the motor coordination task (Fig. 2), both males and females achieve similar numbers of correct responses (VDANC), but females make more delayed correct (VDADC) and males more incorrect responses (VDAIC) at all times.

For both body-sway measures (BODEO and BODEC), females and males sway to the same extent before alcohol, but sway increases much more in females after alcohol ingestion (Fig. 3). This difference between the sexes is dramatic and cannot be removed by correction for blood alcohol concentration or habitual alcohol consumption, in both of which there are sex differences (Martin *et al.*, 1985).



Figs. 2–8. Means and standard errors for all measures: T_0 is before alcohol ingestion and T_1, T_2, and T_3 are approximately 40, 100, and 160 min after ingestion.

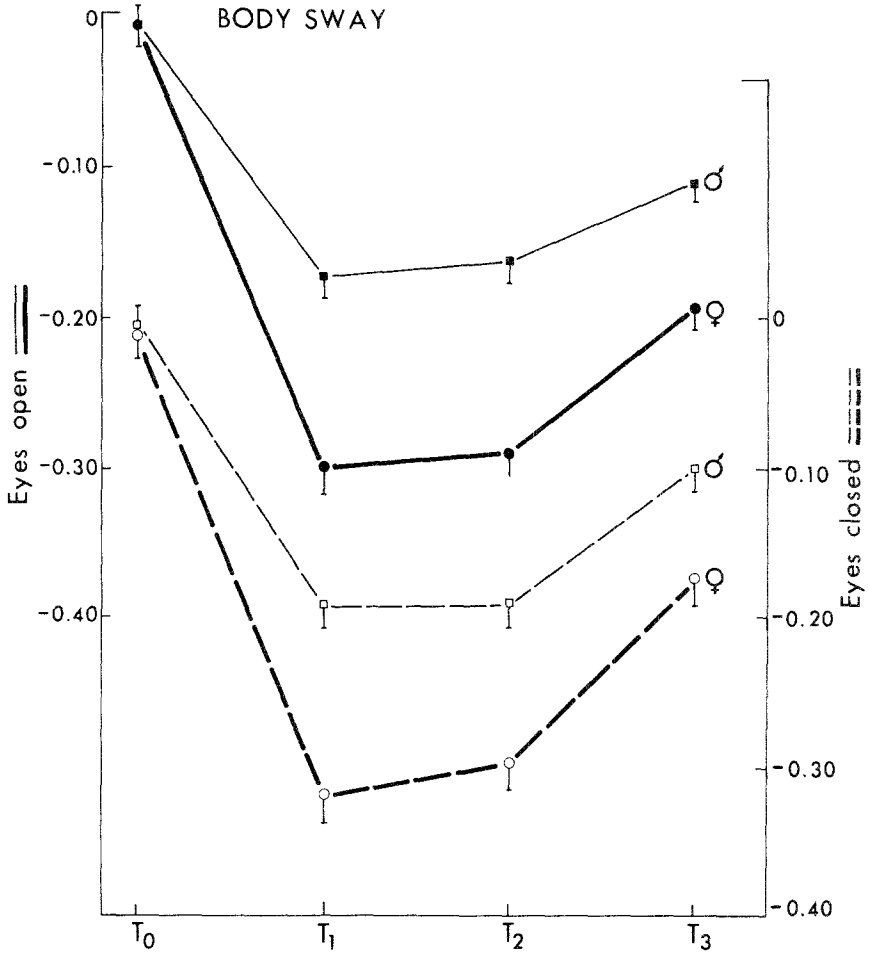

BODY SWAY
0
-0.10
-0.20
-0.30
-0.40
Eyes open
Eyes closed
0
-0.10
-0.20
-0.30
-0.40
♂
♀
♂
♀
T0
T1
T2
T3

Fig. 3.

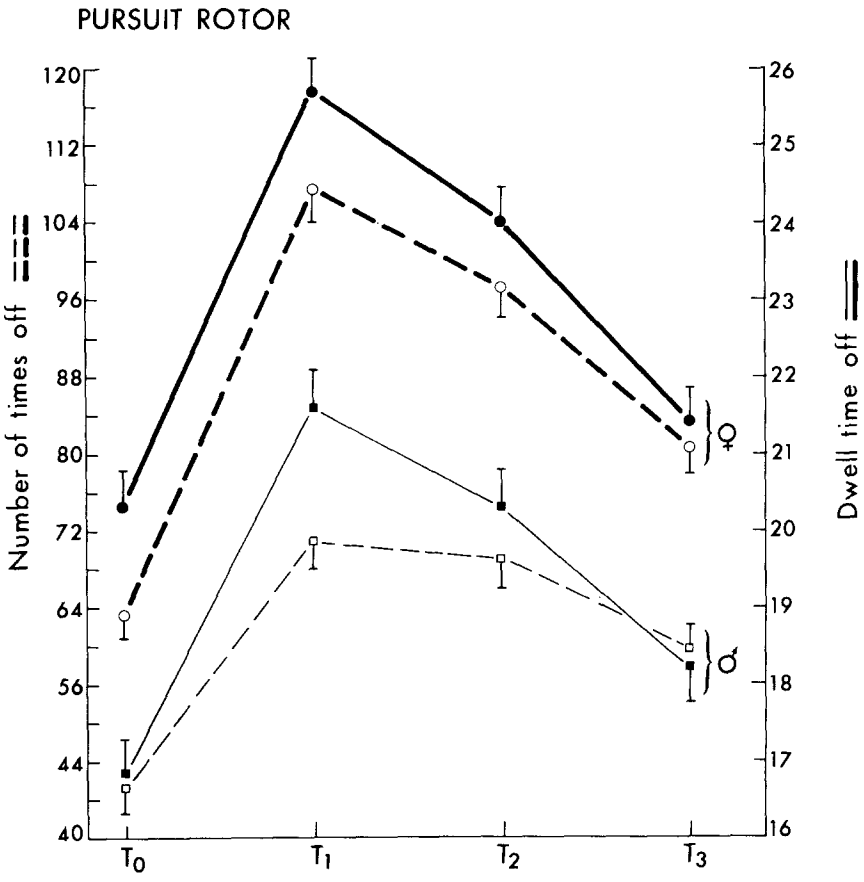

PURSUIT ROTOR
Number of times off
Dwell time off
120
112
104
96
88
80
72
64
56
44
40
26
25
24
23
22
21
20
19
18
17
16
T0
T1
T2
T3
♀
♂

Fig. 4.

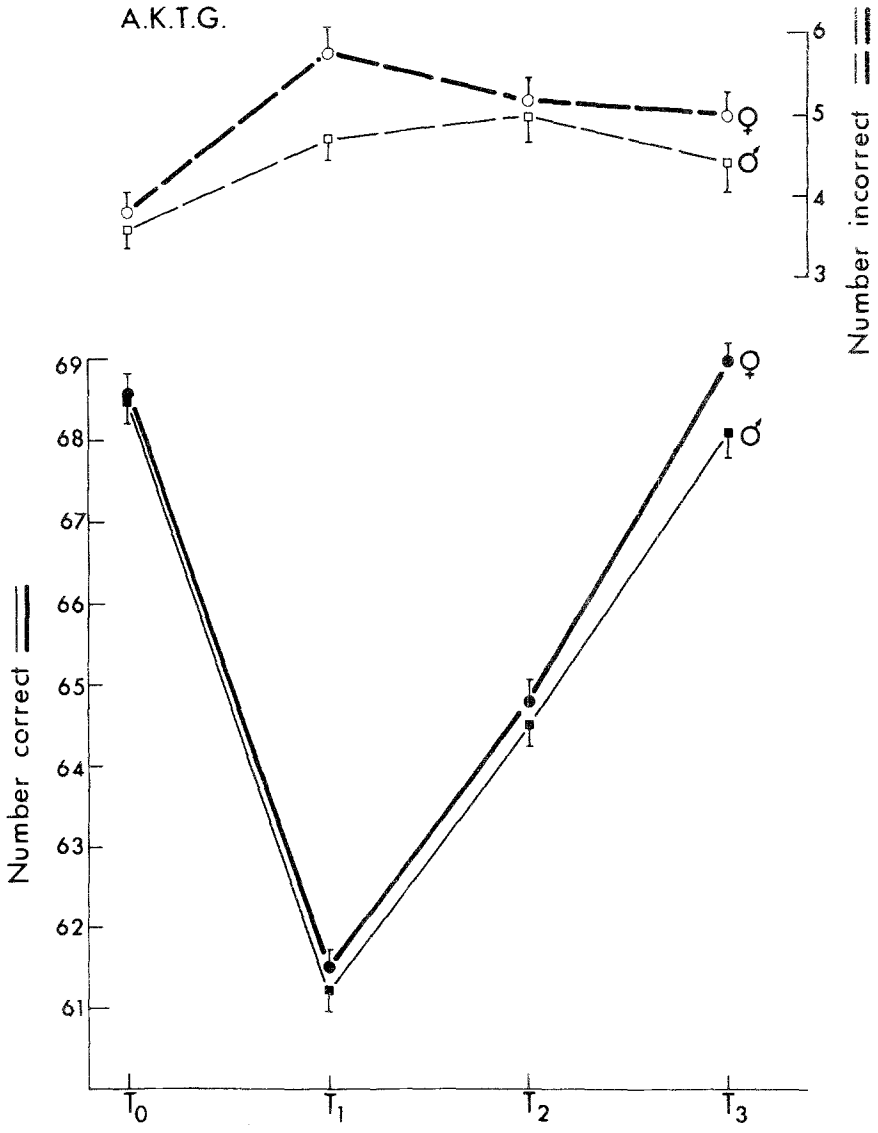

A.K.T.G.
Number incorrect
6
5
4
3
♀
♂
Number correct
69
68
67
66
65
64
63
62
61
T0
T1
T2
T3

Fig. 5.

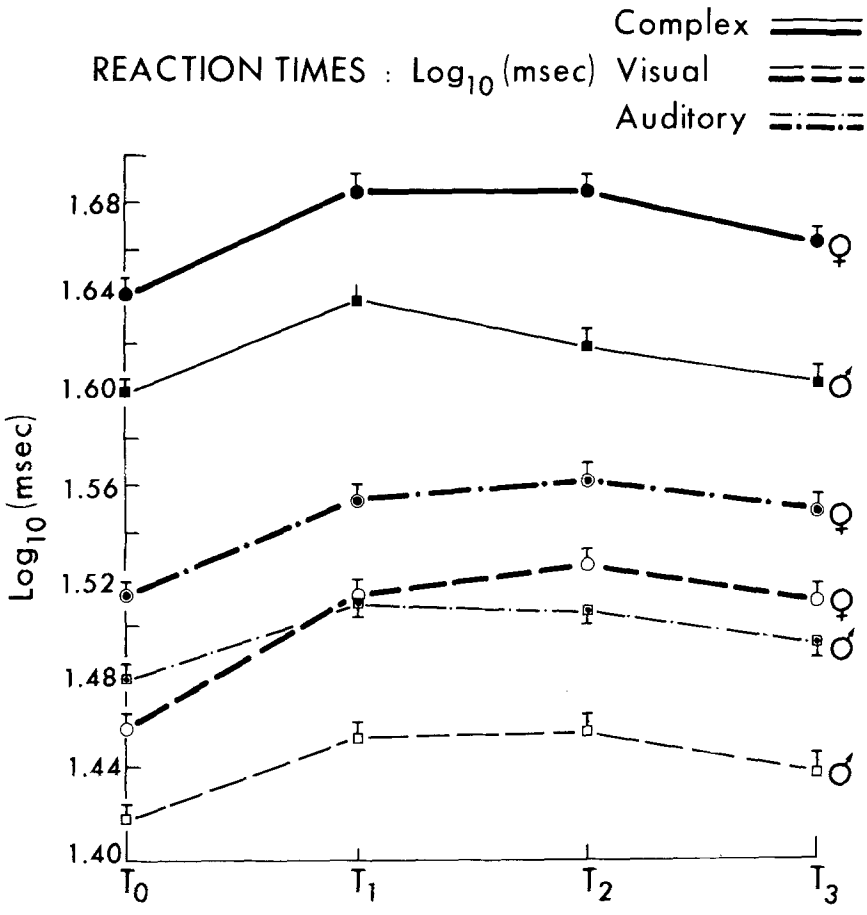

REACTION TIMES : Log10 (msec)
Complex
Visual
Auditory
Log10 (msec)
1.68
1.64
1.60
1.56
1.52
1.48
1.44
1.40
T0
T1
T2
T3

Fig. 6.

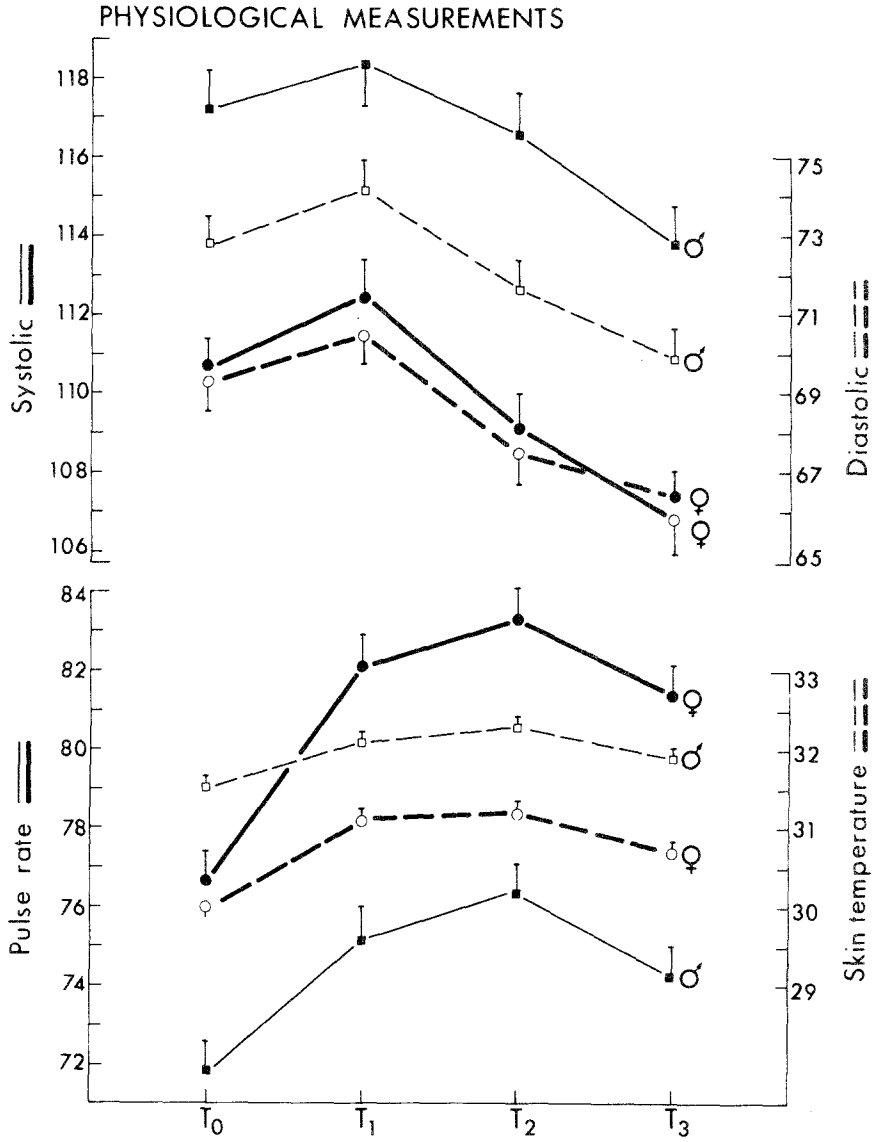

PHYSIOLOGICAL MEASUREMENTS
Systolic
Diastolic
Pulse rate
Skin temperature
118
116
114
112
110
108
106
75
73
71
69
67
65
84
82
80
78
76
74
72
33
32
31
30
29
T0
T1
T2
T3

Fig. 7.

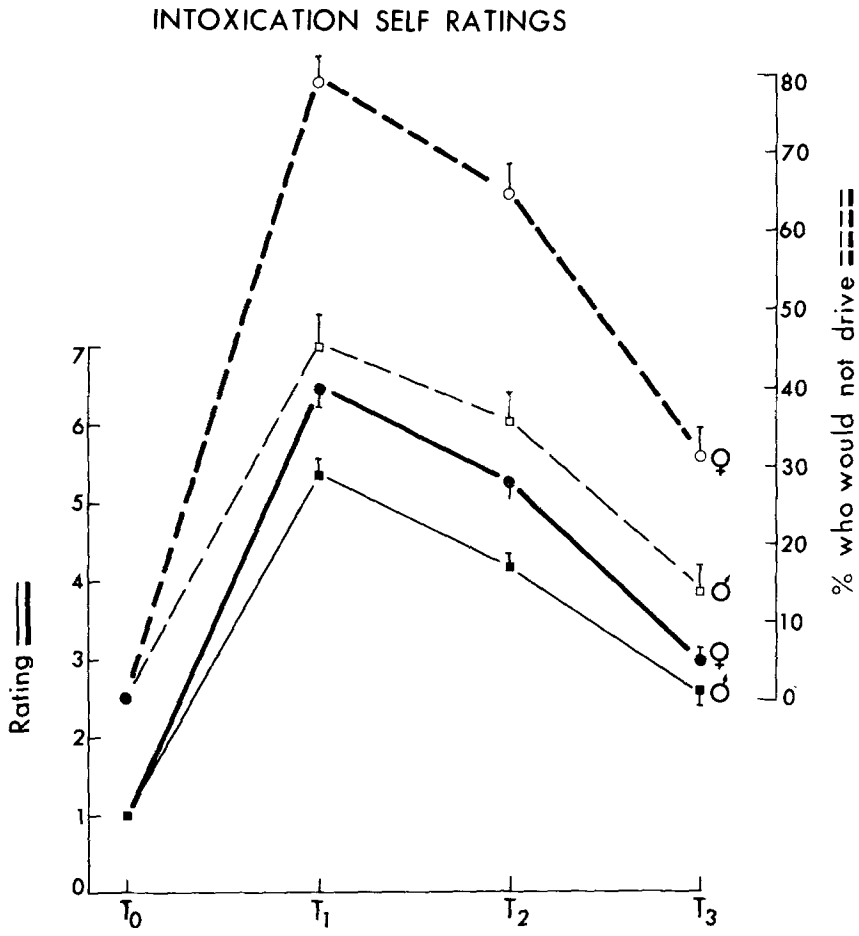

INTOXICATION SELF RATINGS
Rating
% who would not drive
0
1
2
3
4
5
6
7
10
20
30
40
50
60
70
80
T0
T1
T2
T3
♀
♂
♀
♂

Fig. 8.

Males perform better than females on the pursuit rotor (PURNO and PURDO) and reaction-time tasks (VRT, ART, and CRT) (Figs. 4 and 6), but there is no difference in the number of correct arithmetic computations (AKTNC; Fig. 5). Although there are marked effects of alcohol on the performance of all these tasks, there is no evidence that the decrement differs between sexes.

Means and standard errors for systolic and diastolic blood pressure (SYSBP and DIABP) and for pulse (PULSE) and skin temperature (SKTEM) are graphed in Fig. 7. Blood pressure is higher in males than females throughout the experiment. It increases at the first reading after alcohol ingestion (T_1), but by T_2 blood-pressure readings are lower than those taken before alcohol. Since we have not investigated the dose–response relationship between alcohol and these physiological measures, our blood-pressure results are difficult to interpret and may be related as much to the stress of the protocol as to alcohol.

On the other hand, pulse and temperature show responses to alcohol which parallel the psychomotor tests, being elevated at T_1 and T_2 and falling toward their prealcohol values at T_3. Females have higher pulse rates but lower skin temperatures than males throughout the experiment.

All subjects rated themselves as completely sober (rating = 1) and willing to drive a car before alcohol. This was objectively true because the breath alcohol was checked before the start of the experiment and the few subjects who had residual blood alcohol from any drinking on the previous night were excluded. Intoxication self-ratings (INTSR) for T_1–T_3 are graphed in Fig. 8. Because the question "Would you drive a car now?" (DRIVE) was not introduced until after the experiment had been going for several weeks, these responses are missing for some subjects. The percentage who replied "no" at T_1–T_3 is also graphed in Fig. 8. Females rate themselves more drunk than males, and at each time roughly twice as many females as males said that they would not drive. This is consistent with the greater willingness of males to take risks (Eysenck and Eysenck, 1977), as also evidenced by the greater number of incorrect responses (as opposed to delayed correct responses) on the motor coordination task (Fig. 2).

Factor Analysis of Performance Measures and Performance Decrements

Correlations among performance measures at T_0 were subjected to principal-factor analysis, and five initial orthogonal factors with eigenvalues greater than one were extracted. After iteration and rotation to simple structure, the interpretation of these factors is very clear: each factor represents a different psychomotor test, and there is very little

intercorrelation among them. Factor I measures reaction time, Factor II loads on the VDA measures (coordination), Factor III is the pursuit rotor variables (hand steadiness), Factor IV is body sway, and Factor V is the AKTG variables (speeded numbers task). Principal-factor analyses of performance measurements at T_1 and T_2 were similar to those at T_0 except that the fifth factor for AKTG variables was not extracted. At T_3 this factor was extracted again and the results were almost identical to those for T_0.

Principal-factor analysis of the T_1-T_0 performance change scores extracted four initial factors with eigenvalues greater than one, and after rotation to simple structure the loadings on each variable were also clearly interpretable. Factor I measures change in coordination (VDA), Factor II measures change in body sway but also loads on pursuit rotor and can therefore be regarded as a steadiness factor, Factor III loads mainly on reaction time but also to some extent on body sway, and Factor IV loads on change in the speeded arithmetic (AKTG) task. Principal-factor analyses of T_2-T_0 and T_3-T_0 change scores produced factor patterns which were essentially similar.

We conclude that the five sets of psychomotor tests are measuring abilities that are essentially independent, both when individuals are sober and in their response to alcohol. The exception is some degree of correlation in change in the body-sway and pursuit rotor tasks, which appears to reflect a common effect of alcohol on both types of steadiness.

Variability of Response

There was considerable variance in each measure at all times, both before and after alcohol. More importantly, however, many measures, notably VDANC, BODEO, and BODEC, showed a marked increase in standard deviation immediately following alcohol ingestion at T_1. The fact that in many measures the variance appeared to decline toward prealcohol levels after T_1 suggests that the T_1 increase is specifically alcohol related.

Repeatability over Occasions

For the psychomotor tests, there was considerable variation in repeatability between tasks and, in some cases, between measures derived from a single task. For the motor coordination task, repeatabilities were highest for the number of correct responses (VDANC), particularly in males, where the repeatabilty averaged 0.80 over the four sampling times. Repeatabilities were lower in females, particularly for the number incorrect. Mean repeatabilities for the body-sway measures were about 0.6 for

eyes closed (BODEC) in both sexes and eyes open (BODEO) in males and 0.73 for eyes open in females. For the pursuit motor measures, repeatabilities were notably lower, being 0.38 and 0.44 for PURNO in females and males and 0.45 and 0.51 for PURDO. Repeatabilities for AKTNC were remarkably high at all times, the mean being 0.84 in both males and females. However, for AKTIC they were much lower, particularly in females. Among the reaction times, repeatabilities were highest for ART, being 0.74 in females and 0.68 in males, lower for VRT (0.61 and 0.63), and lowest for CRT (0.56 and 0.54). A full analysis of variance showed that for all three reaction times, the occasions × individuals interaction variance was no higher than the variance among the five replicates within occasions.

Repeatabilities for physiological measures were generally low, the mean for systolic blood pressure being 0.41 in females and 0.47 in males, that for diastolic blood pressure 0.34 and 0.37, that for pulse 0.40 in both sexes, and that for skin temperature 0.41 and 0.19. If skin temperature is related to the menstrual cycle, it is surprising that the repeatability of this measure should be lower in males than in females.

Repeatabilities for the intoxication self-rating (INTSR) were high at T_1 (0.78 in females and 0.73 in males) but lower at T_2 and T_3. Repeatabilities of about 0.5 for willingness to drive a car (DRIVE) were calculated as coefficients of association (ϕ) from a 2 × 2 table of yes/no responses on occasion 1 and occasion 2.

Genetical Analysis

In Table II we present the intercorrelations of performance scores at all four times for selected psychomotor and physiological variables. Correlations are generally higher among the three postalcohol measurements than between these and the prealcohol measurement. This, like the increase in variance after alcohol, suggests that alcohol-related influences are systematically affecting performance. However, we need further analysis to see whether these influences are environmental or genetic in origin.

The mean products matrices on which the ensuing analyses are based are available from the authors on request. The eight multivariate models described under Genetical Analysis in Subjects and Methods have been fitted to only one measure from each psychomotor task (in general, the one which has the highest repeatability), except for reaction times, where both CRT and ART are considered. These models have also been fitted to mean products for all four physiological measures. Results of fitting these models to all 10 mean products matrices are shown in Table III,

Table II. Intercorrelations (× 100) of Scores at Four Times for All Measures

	VDANC	VDADC	VDAIC	BODEO	BODEC	PURNO	PURDO	AKTNC	AKTIC	ART	VRT	CRT	SYSBP	DIABP	PULSE	SKTEM
Females (N = 213)																
T_0–T_1	72	69	66	57	55	44	58	82	48	62	58	58	62	58	67	70
T_0–T_2	77	76	68	64	62	45	61	83	40	57	61	59	56	57	66	67
T_0–T_3	75	76	69	72	61	57	63	84	42	67	57	53	59	51	69	64
T_1–T_2	87	82	75	80	74	56	80	87	51	73	66	70	71	67	77	73
T_1–T_3	81	76	69	69	65	54	64	86	59	58	58	62	68	66	74	68
T_2–T_3	90	91	78	83	76	66	75	87	53	63	69	68	78	73	81	80
Males (N = 199)																
T_0–T_1	80	78	83	61	47	53	65	86	52	69	69	51	61	52	71	68
T_0–T_2	84	83	82	59	41	59	65	87	49	69	65	62	62	60	68	59
T_0–T_3	83	82	83	66	53	64	65	87	51	69	66	55	64	59	74	57
T_1–T_2	87	88	84	82	72	68	74	90	52	69	76	64	70	57	81	80
T_1–T_3	83	81	83	76	65	60	66	88	60	65	68	56	62	56	80	71
T_2–T_3	91	90	88	87	72	75	81	91	57	76	74	66	68	61	83	84

Table III. Results of Fitting Multivariate Models to All 10 Mean Products Matrices[a]

Model	E_1		E_2		V_A			VDANC		BODEC		PURNO		AKTNC		CRT		VRT		ART		SYSBP		DIABP		PULSE		SKTEM	
No.	g	s	g	a	g	a	df	χ^2	P	χ^2	P	χ^2	P	χ^2	P	χ^2	P	χ^2	P	χ^2	P	χ^2	P	χ^2	P	χ^2	P	χ^2	P
1	X	X	X				88	110	0.06	166	0.00	93	0.33	128	0.00	97	0.24	132	0.00	158	0.00	133	0.00	128	0.00	122	0.01	244	0.00
2	X	X	X	X			85	98	0.16	133	0.00	82	0.58	119	0.01	95	0.21	125	0.00	142	0.00	121	0.01	127	0.00	108	0.05	207	0.00
3	X	X			X		88	98	0.21	152	0.00	97	0.23	99	0.20	86	0.55	116	0.02	145	0.00	130	0.00	123	0.01	115	0.03	225	0.00
4	X	X			X	X	85	88	0.40	119	0.01	78	0.70	87	0.41	84	0.53	107	0.05	123	0.01	120	0.01	121	0.01	95	0.21	197	0.00
5	X	X	X		X		84	89	0.32	121	0.01	78	0.67	86	0.42	84	0.49	108	0.04	126	0.00	118	0.01	118	0.01	99	0.13	197	0.00
6	X	X	X	X	X		81	78	0.58	109	0.02	73	0.73	82	0.44	82	0.45	104	0.04	125	0.00	111	0.02	118	0.00	92	0.19	189	0.00
7	X	X	X		X	X	81	77	0.60	104	0.05	71	0.78	81	0.47	81	0.47	103	0.05	121	0.00	112	0.01	117	0.01	89	0.26	191	0.00
8	X	X	X	X	X	X	78	74	0.62	103	0.03	71	0.71	81	0.38	81	0.37	103	0.03	120	0.00	110	0.01	117	0.00	89	0.19	189	0.00

[a] Sources of variation are (i) individual environment (E_1), for which general-factor loadings (g) and specific components of variance (s) are estimated; (ii) common environment (E_2), for which general-factor (g) or alcohol-factor (a) loadings may be estimated; and (iii) additive genetic variation (V_A), for which general-factor (g) or alcohol-factor (a) loadings may be estimated. Degrees of freedom (df) remaining to test the goodness of fit for each variable are given. For each model fitted to each variable, the chi-square value (χ^2) and its probability (P) are tabled.

and in the cases where there is heterogeneity over sexes, results are shown separately for males and females in Table IV.

For all measures, models including general genetic factors fit better than corresponding models in which E_2 is substituted for V_A, confirming the importance of genetic variation throughout. We now consider the detection of variance exposed by alcohol ingestion and whether this is environmental or genetic in origin.

Motor Coordination

Models which include an alcohol factor fit significantly better than those which exclude this factor. However, it is not possible to say whether the covariation among the three measures of performance after alcohol derives from shared environmental covariation ($\chi_{81}^2 = 77.86$) or additive genetic covariation ($\chi_{81}^2 = 77.16$). The breakdown of variation for the latter model shows about 10% (or less) of the total variance at T_1 to T_3 due to the alcohol genetic factor, but the former model shows almost exactly the same proportions due to an E_2 alcohol factor (Table V).

The inclusion of both an E_2 and a V_A alcohol factor does not significantly improve on either of the models in which the covariation after alcohol is due to only one source (Table V). We now find that the alcohol covariation is divided between the two sources inconsistently at times T_1, T_2, and T_3: the fact that only one of the six alcohol factor contributions is now significant means that biological interpretation should be applied with caution to the results of this model.

The specific E_1 variance at each time, which is a measure of the true error variance, is 20% or less of the total, which is slightly less than the proportion of nonrepeatable variance calculated from the repeatability data. This suggests that there may be small systematic E_1 influences such as general tiredness which differ between occasions and contribute to E_1 factor variance.

Body Sway

We consider the genetical analysis for the eyes closed condition (BODEC) only, although results for the eyes-open condition are very similar. Poor fits are obtained when models are fitted to all 10 matrices (Table III) but satisfactory fits are obtained when they are fitted to male and female data separately (Table IV), indicating that the breakdown of variance for the two sexes is significantly different.

In males, the preferred model contains genetic general and alcohol factors ($\chi_{25}^2 = 31.94$, $P = 0.16$), and in Table VI we show the breakdown of variance for males under this V_A general- and alcohol-factor model

Table IV. Results of Fitting Multivariate Models to Four Mean Products Matrices for Females and Males Separately in Certain Variables Displaying Heterogeneity Between Sexes

Model No.	E_1		E_2		V_A		df	BODEC				VRT				ART				SYSBP				DIABP				SKTEM			
								Females		Males		Females		Males		Females		Males		Females		Males		Females		Males		Females		Males	
	g	s	g	a	g	a		χ^2	P	χ^2	P	χ^2	P	χ^2	P	χ^2	P	χ^2	P	χ^2	P	χ^2	P	χ^2	P	χ^2	P	χ^2	P	χ^2	P
1	X	X	X				28	62	0.00	62	0.00	33	0.23	46	0.02	52	0.00	36	0.15	48	0.01	37	0.11	43	0.04	39	0.08	76	0.00	67	0.00
2	X	X	X	X			25	47	0.01	43	0.02	29	0.25	41	0.02	45	0.01	27	0.28	33	0.13	30	0.23	38	0.04	37	0.06	66	0.00	44	0.01
3	X	X			X		28	59	0.00	57	0.00	25	0.61	44	0.03	46	0.02	32	0.26	46	0.02	37	0.13	40	0.07	37	0.11	74	0.00	62	0.00
4	X	X			X	X	25	41	0.02	32	0.16	20	0.73	41	0.02	33	0.13	25	0.47	33	0.14	29	0.28	35	0.09	33	0.13	66	0.00	42	0.02
5	X	X	X		X		24	37	0.05	32	0.13	22	0.58	39	0.02	35	0.07	25	0.42	30	0.18	28	0.28	33	0.11	34	0.09	63	0.00	40	0.02
6	X	X	X	X	X		21	22	0.38	30	0.09	21	0.48	34	0.04	35	0.03	19	0.59	26	0.22	26	0.19	33	0.05	34	0.04	59	0.00	32	0.06
7	X	X	X		X	X	21	22	0.38	26	0.21	18	0.65	35	0.02	32	0.05	18	0.64	26	0.19	26	0.22	32	0.05	32	0.06	59	0.00	33	0.04
8	X	X	X	X	X	X	18	22	0.24	25	0.13	18	0.46	34	0.01	32	0.02	17	0.50	25	0.12	25	0.13	32	0.02	32	0.02	58	0.00	31	0.03

Table V. VDA, Number of Correct Responses: Breakdown of Variation (%) at Each Time for a Model in Which Covariation in Postalcohol Performance Is Due to (a) a Genetic Factor or (b) Both a Shared Environment and a Genetic Factor

(a) Genetic alcohol factor

	Individual environment		Shared environment	Genetic	
Trial	General factor	Specific	General factor	General factor	Alcohol factor
T_0	11***	19***	25***	45***	—
T_1	17***	16***	14*	41***	12***
T_2	32***	3***	31***	25***	9**
T_3	22***	14***	28***	30***	6*

$\chi^2_{81} = 77.16, P = 0.58$

(b) Shared environment and genetic alcohol factors

	Individual environment		Shared environment		Genetic	
Trial	General factor	Specific	General factor	Alcohol factor	General factor	Alcohol factor
T_0	10***	20***	27***	—	43***	—
T_1	20***	16***	18**	10***	36***	0
T_2	30***	3***	26***	4	30***	7
T_3	19***	12***	18**	2	43***	6

$\chi^2_{78} = 73.53, P = 0.62$

* $0.01 < P < 0.05$.
** $0.001 < P < 0.01$.
*** $P < 0.001$.

(Model 4). The striking feature of these results is the very large genetic component in postalcohol covariance.

Three sources are required to explain the covariation in females. However, it is not initially clear whether the large amount of alcohol-specific variation is genetic or environmental in origin, since Models 6 and 7 fit equally well. When we consider the results of Model 7 (Table VI), there is no evidence of a substantial alcohol genetic factor but the *general* genetic factor has a very small loading at T_0 and accounts for 40–50% of the variance after alcohol ingestion. It thus assumes the characteristics of an alcohol genetic factor and indeed, in Model 6 (not shown here), retains these characteristics. In both models the common environmental component is large at T_0 but very small at later times, and this is seen to be the cause of the anomaly. We conclude that genetic factors

Table VI. Body Sway—Eyes Closed: Breakdown of Variation (%) at Each Time T_0–T_3 for a Model in Which Covariation in Postalcohol Performance Is Due to a Genetic Factor[a]

Males				
	Individual environment		Genetic	
Trial	General factor	Specific	General factor	Alcohol factor
T_0	4*	26***	70***	—
T_1	7***	26***	23***	44***
T_2	21***	26***	13***	40***
T_3	55***	13***	22***	10**
$\chi^2_{25} = 31.94, P = 0.16$				

Females					
	Individual environment		Common environment	Genetic	
Trial	General factor	Specific	General factor	General factor	Alcohol factor
T_0	17***	25***	47***	11	—
T_1	25***	14***	5	46*	10
T_2	17***	25***	10*	49***	0
T_3	15***	22***	10*	46**	6
$\chi^2_{21} = 22.27, P = 0.38$					

[a] Separate models are fitted for females and males, and a common shared environment factor is fitted for females.

* $0.01 < P < 0.05$.

** $0.001 < P < 0.01$.

*** $P < 0.001$.

are also of great importance in determining the effect of alcohol on this steadiness task in females.

Pursuit Rotor

For PURNO, models including either an E_2 or a V_A alcohol factor give satisfactory fits to the data, but the model containing general genetic and alcohol genetic factors gives the better fit ($\chi_{85}^2 = 77.75, P = 0.70$). The addition of an E_2 general factor or E_2 general and alcohol factors to this model does not significantly improve the fit (Table III). The breakdown of variance at each time for the genetic general and alcohol factors is shown in Table VII. We find that 17–18% of the variance in performance at T_1–T_2 is due to genetic variation expressed in the presence of alcohol,

Table VII. Pursuit Rotor, Number of Times off Target: Breakdown of Variation (%) at Each Time for a Model in Which Covariation in Postalcohol Performance Is Due to a Genetic Factor

	Individual environment		Genetic	
Trial	General factor	Specific	General factor	Alcohol factor
T_0	16*	40*	44*	—
T_1	17*	45*	21*	17*
T_2	32*	29*	21*	18*
T_3	58*	20*	19*	3
		$\chi^2_{85} = 77.75, P = 0.70$		

* $P < 0.001$.

but this drops to a nonsignificant 3% at T_3. The total environmental variance at each time is similar to the nonrepeatable variance.

Arithmetic Computation

For AKTNC the best-fitting, most parsimonious model includes genetic general and alcohol factors ($\chi_{81}{}^2 = 87.28, P = 0.41$). The addition of either an E_2 general factor or an E_2 general and alcohol factor to this model causes no significant improvement in fit. A breakdown of causes of variance (Table VIII) shows that the heritability of performance is high at each sampling time and that a small proportion (5–7%) of the variance in postalcohol performance is due to genetic differences revealed by the alcohol. The E_1-specific components are only slightly smaller than the nonrepeatable variance, so only a small proportion of the E_1-factor var-

Table VIII. AKTG, Number of Correct Responses: Breakdown of Variation (%) at Each Time in Which Covariation in Postalcohol Performance Is Due to a Genetic Factor

	Individual environment		Genetic	
Trial	General factor	Specific	General factor	Alcohol factor
T_0	13*	12*	75*	—
T_1	12*	13*	68*	7*
T_2	13*	10*	70*	7*
T_3	17*	12*	66*	5*
		$\chi^2_{85} = 87.28, P = 0.41$		

* $P < 0.001$.

Table IX. Reaction Times: Breakdown of Variation (%) at Each Time T_0–T_3 for a Model in Which Covariation in Postalcohol Performance Is Due to a Genetic Factor

Trial	Individual Environment		Genetic	
	General factor	Specific	General factor	Alcohol factor
		Complex reaction time		
T_0	12***	41***	47***	—
T_1	19***	39***	33***	9**
T_2	35***	22***	37***	6**
T_3	22***	38***	33***	7*
		$\chi^2_{85} = 83.50$, $P = 0.53$		
		Auditory reaction time		
Females				
T_0	21***	34***	45***	—
T_1	20***	25***	38***	17***
T_2	25***	20***	32***	23***
T_3	13***	32***	55***	0
		$\chi^2_{25} = 32.93$, $P = 0.13$		
Males				
T_0	39***	27***	34***	—
T_1	25***	38***	35***	2
T_2	27***	14***	51***	8
T_3	18***	14***	67***	1
		$\chi^2_{25} = 24.86$, $P = 0.47$		

* $0.01 < P < 0.05$.
** $0.001 < P < 0.01$.
*** $P < 0.001$.

iance can be nonrepeatable, the remainder being genuine individual differences affecting performance at all times.

Reaction Times

For CRT, an improvement of only $\chi_3^2 = 4.25$ is obtained by fitting a genetic alcohol factor in addition to the general genetic factor. However, significant genetic alcohol loadings accounting for 6–9% of the postalcohol variation are estimated, so the breakdown of variance in this model is shown in Table IX.

The picture for auditory reaction time (ART) is different. Poor fits are obtained when models are fitted to all 10 matrices but satisfactory fits are obtained to male and female matrices separately (Table IV). In

females the preferred model is clearly that including genetic general and alcohol factors ($\chi_{25}^2 = 32.93, P = 0.13$). In males, improvement obtained by the inclusion of either an E_2 or a genetic alcohol factor is not as marked as in females and the genetic model is only marginally preferable. If both shared environmental and genetic alcohol factors are included in the model, then the small amount of postalcohol variation is split between them but none of the estimates is significant. Table IX shows the breakdown of variance for the genetic general- and alcohol-factors model separately for each sex. Contributions of the genetic alcohol factor to the variation in females are large and significant at T_1 (17%) and T_2 (23%) but are negligible and nonsignificant at all sampling times in males.

Results for VRT were similar to those obtained for ART, although the fit of models to the male data was less satisfactory.

Physiological Measurements

Apart from a correlation of 0.80 between systolic and diastolic blood pressure, intercorrelations of the four physiological measures are negligible.

For systolic blood pressure, no model gives a satisfactory fit unless the sexes are considered separately. In both sexes there is an improvement in fit when an alcohol factor is included in the model (Table IV), but it makes little difference whether this is due to E_2 or V_A sources. Since the model containing genetic general and alcohol factors is slightly preferred, we table the breakdown of variance for this model in Table X separately for each sex. E_1 variance appears to be more important in males than females, but the genetical alcohol factor accounts for 13% of the variance at T_1 and 8% at T_2. In contrast, in females only a small proportion of variance (3%) at T_1 is accounted for by alcohol-related genetic effects, while 31% is accounted for at T_2: this disparity with the male result suggests anomalous aspects of the female systolic blood pressure data. Breakdowns of variance are similar for the analogous E_2 models and it is not clear which of the two is to be preferred.

For diastolic blood pressure, heterogeneity of fit between sexes is also observed (Table III), so models have been fitted separately to males and females (Table IV). Improvements in fit due to the inclusion of either E_2 or V_A postalcohol factors are small and nonsignificant in either sex. The genetic alcohol-factor model is slightly preferred and results for this model are given separately for females and males in Table X. It can be seen that genetic factors in postalcohol covariance are larger (11–18%) in males than in females (1–5%). Disregarding the anomalous result for SYSBP in females, results for both blood-pressure measurements are

Table X. Systolic and Diastolic Blood Pressures: Breakdown of Variance (%) at Each Time for a Model in Which Postalcohol Covariation Is Due to a Genetic Factor[a]

Trial	Individual environment		Genetic	
	General factor	Specific	General factor	Alcohol factor
		Systolic blood pressure		
Males				
T_0	30***	37***	33***	—
T_1	29***	27***	31***	13**
T_2	19***	30***	43***	8*
T_3	15***	29***	56***	0
		$\chi^2_{25} = 28.70$, $P = 0.28$		
Females				
T_0	13***	47***	40***	—
T_1	11***	32***	54***	3*
T_2	20***	14***	35***	31***
T_3	24***	29***	39***	8*
		$\chi^2_{25} = 32.61$, $P = 0.14$		
		Diastolic blood pressure		
Males				
T_0	19***	25***	56***	—
T_1	10*	48***	29***	13***
T_2	14***	37***	31***	18***
T_3	10**	38***	41***	11**
		$\chi^2_{25} = 32.95$, $P = 0.13$		
Females				
T_0	2	53***	45***	—
T_1	13***	25***	57***	5*
T_2	24***	20***	53***	3
T_3	20***	31***	48***	1
		$\chi^2_{25} = 34.91$, $P = 0.09$		

[a] Separate models are fitted to males and females.
* $0.01 < P < 0.05$.
** $0.001 < P < 0.01$.
*** $P < 0.001$.

consistent with a small genetic factor influencing blood pressure after alcohol ingestion.

Results of model fitting for PULSE indicate no significant heterogeneity between sexes. The preferred model includes no E_2 source of covariation but genetical general and alcohol factors ($\chi_{85}^2 = 95.04$, $P =$

Table XI. Breakdown of Variance for (a) Pulse Rate and (b) Skin Temperature

(a) Pulse rate: breakdown of variance (%) at each time for a model in which postalcohol covariation is due to a genetic factor

	Individual environment		Genetic	
Trial	General factor	Specific	General factor	Alcohol factor
T_0	33***	25***	42***	—
T_1	41***	25***	24***	10***
T_2	40***	15***	22***	23***
T_3	39***	20***	27***	14***

$\chi^2_{85} = 95.04$, $P = 0.21$

(b) Skin temperature: breakdown of variance (%) in males only for a model in which postalcohol covariation is due to a common environmental factor and general variance is due to both common environmental and genetic factors

	Individual environment		Common environment		Genetic
Trial	General factor	Specific	General factor	Alcohol factor	General factor
Males					
T_0	8**	29***	21**	—	42***
T_1	11***	18***	13	19***	39***
T_2	25***	13***	0	19*	43***
T_3	19***	18***	1	9	53***

$\chi^2_{21} = 31.68$, $P = 0.06$

* $0.01 < P < 0.05$.
** $0.001 < P < 0.01$.
*** $P < 0.001$.

0.21). The addition of an E_2 general factor does not improve the fit significantly ($\chi_4^2 = 6.06$) and the estimates of E_2 loadings are trivial and nonsignificant. The breakdown of variance in the preferred model is shown in Table XI, and it can be seen that genetic covariation revealed by alcohol accounts for significant proportions of the variance, being 10% of the total at T_1, 23% at T_2, and 14% at T_3.

Fits to all 10 matrices for skin temperature (SKTEM) are very poor and fits to sexes separately (Table IV) reveal that this is not due just to heterogeneity between sexes but to poor fits for each sex, especially females where no model fits. In males a barely satisfactory fit ($P = 0.06$) is obtained with a V_A general factor and E_2 general and alcohol factors, and the breakdown of variance for this model is given in Table XI. There

are indications of a strong postalcohol common environmental factor, but the contributions of the general E_2 factor are so uneven and the overall fit of the model is so marginal that little confidence can be placed in this result.

Correlates of Performance Decrement

Correlations Between Performance and Blood Alcohol Concentration

Correlations between performance and blood alcohol concentration (BAC) at T_1, T_2, and T_3 are shown in Table XII. Few correlations are significant and none is large. All significant correlations are in the expected direction of decreasing performance with higher alcohol levels, except for intoxication self-ratings and willingness to drive in males at T_1; males with higher BACs rate themselves less drunk and more willing to drive.

Correlations between performance change scores and BACs are also shown in Table XII. We now find more significant correlations, particularly in females, between the change in performance from the prealcohol level and the BAC. Correlations are again in the expected direction; performance deteriorates more in subjects with higher BACs. However, even for the greatest correlation of -0.35 between the change in the number of correct VDA responses and the BAC in females, only 12% of the variance in performance decrement can be explained by variation in BACs.

Correlations Between Performance and Drinking History

Correlations of weekly alcohol consumption (LCONW) and years drinking (LYDR) with performance measurements at T_0 and T_1 and performance change scores (T_1–T_0) are shown in Table XIII. There are several remarkable features of these correlations. Greater weekly alcohol consumption and longer drinking history are associated in males with greater body sway, lower computational speed, and slower reaction times when sober (T_0), implying, perhaps, some long-term detrimental effects of alcohol consumption on psychomotor performance. However, greater consumption is also associated in men with slightly greater steadiness and computational speed after alcohol (T_1), so that the net effect (T_1–T_0) is that males with greater habitual consumption show less decrement in these tasks. Similar results have been reported by Powell *et al.* (1973). The same effect on decrement in body sway is apparent in females, although less markedly, and there is no association with sober performance. While interesting, however, none of these associations accounts

Table XII. Correlations Between Blood Alcohol Concentrations and Performance Measurements at T_1, T_2, and T_3 and Performance Change Scores T_1-T_0, T_2-T_0, and T_3-T_0[a]

	VDANC	VDADC	VDAIC	BODEO	BODEC	PURNO	PURDO	AKTNC	AKTIC	ART	VRT	CRT	INTSR	DRIVE
T_1														
Males	−0.05	0.00	0.10	−0.11	−0.05	0.15*	0.14*	0.00	0.08	0.00	0.08	0.15*	−0.12*	0.15*
Females	−0.22**	0.17*	0.17*	−0.09	−0.03	0.07	0.16*	−0.07	0.09	0.03	0.05	−0.02	0.00	−0.01
T_2														
Males	−0.01	−0.05	0.09	0.03	0.02	0.03	0.06	0.03	0.08	0.01	0.03	0.16*	0.05	−0.01
Females	−0.03	0.05	−0.03	−0.10	−0.11	0.07	0.08	0.07	0.08	0.04	0.09	0.11	0.08	−0.13
T_3														
Males	−0.05	0.00	0.11	0.07	0.02	0.05	0.01	0.03	0.06	−0.01	0.07	0.06	0.12*	−0.04
Females	−0.07	0.07	0.02	−0.23**	−0.16*	0.20**	0.20**	0.11	0.13	0.13	0.12	0.09	0.18**	−0.16**
T_1-T_0														
Males	−0.26***	0.21**	0.18*	−0.16*	−0.08	0.12	0.15*	−0.04	−0.03	−0.05	0.14*	0.13	—	—
Females	−0.35***	0.17	0.30***	−0.19**	−0.09	0.13	0.26***	−0.16*	0.05	0.05	0.14*	0.08	—	—
T_2-T_0														
Males	−0.09	−0.05	−0.06	−0.08	−0.09	0.01	0.11	−0.11	−0.05	0.07	0.11	0.12	—	—
Females	−0.14*	0.05	0.12	−0.05	−0.08	0.12	0.15*	−0.17*	0.05	0.05	0.08	0.09	—	—
T_3-T_0														
Males	−0.11	0.00	−0.09	−0.13	−0.14*	0.07	0.07	−0.10	−0.09	0.07	0.18*	0.03	—	—
Females	−0.20**	0.07	0.16*	−0.20**	−0.10	0.20**	0.28***	−0.18**	0.12	0.13	0.04	0.05	—	—

[a] Pearson correlations are tabled except for those with intoxication self-rating (INTSR) and willingness to drive (DRIVE), which are Kendall's τ. Female $N = 213$ and male $N = 191$ except for DRIVE, where $N = 183$ and 181. Significance tests are two tailed.

* $0.01 < P < 0.05$.

** $0.001 < P < 0.01$.

*** $P < 0.001$.

Table XIII. Correlations of Performance at T_0 and T_1 and Change Scores T_1-T_0 with log Weekly Alcohol Consumption (LCONW) and log Years Drinking (LYDR)

	LCONW												
	BODEO	BODEC	AKTNC	AKTIC	VDANC	VDADC	VDAIC	VRT	ART	CRT	PURNO	PURDO	INTSR
T_0													
Males	−0.22**	−0.31***	−0.22**	0.09	−0.06	0.02	0.08	0.09	0.03	0.03	0.02	0.04	
Females	0.02	0.10	0.02	−0.01	0.01	0.09	−0.17*	−0.03	0.01	0.01	−0.08	−0.09	
T_1													
Males	0.11	0.08	−0.10	0.01	0.02	−0.07	0.06	−0.08	−0.05	0.00	−0.10	−0.04	−0.53***
Females	0.15*	0.16*	0.08	0.01	−0.01	0.11	−0.14*	−0.07	−0.13	−0.12	−0.08	−0.05	−0.33***
T_1-T_0													
Males	0.34***	0.34***	0.26***	−0.08	0.13	−0.14	−0.02	−0.22**	−0.10	−0.03	−0.12	−0.09	
Females	0.16*	0.12	0.10	0.03	−0.03	0.04	−0.01	−0.04	−0.16*	−0.14*	0.01	0.02	
	LYDR												
T_0													
Males	0.05	−0.01	−0.16*	−0.01	−0.09	0.08	0.05	0.23**	0.26***	0.28***	−0.01	0.01	
Females	0.07	0.14*	−0.04	−0.06	−0.03	0.03	0.01	0.09	0.15*	0.08	−0.10	−0.06	
T_1													
Males	0.17*	0.21**	−0.10	0.04	−0.09	0.06	0.09	0.25***	0.19**	0.22**	−0.06	0.06	−0.41***
Females	0.14*	0.16*	−0.02	−0.01	−0.11	0.08	0.09	0.13	0.08	0.10	−0.06	0.02	−0.20**
T_1-T_0													
Males	0.16*	0.25**	0.13	0.05	−0.02	−0.02	0.07	0.02	−0.12	−0.05	−0.05	0.06	
Females	0.10	0.07	0.03	0.04	−0.13	0.08	0.10	0.05	−0.07	0.03	0.05	0.08	

* $0.01 < P < 0.05$.
** $0.001 < P < 0.01$.
*** $P < 0.001$.

for more than 12% of the variance in performance or performance decrement.

Not surprisingly, there are high correlations between previous drinking experience and intoxication self-rating (INTSR), those who drink more or who have been drinking longer rating themselves less drunk at T_1.

Correlations Between Performance and Personality Variables

Correlations of the performance at T_0 and T_1 and the T_1-T_0 change score with Extraversion and Psychoticism are shown in Table XIV. No correlations were found with Neuroticism or the L scale. Few of the tabled correlations are significant, but more extraverted males are less steady (BODEO and BODEC) and obtain fewer correct speeded arithmetic responses (AKTNC) when sober. This effect is not observed at T_1, and consequently these males show a smaller decrement in performance with the alcohol treatment, as also observed by Powell *et al.* (1973). After alcohol more extraverted females have faster auditory reaction times but spend more time off target in the hand steadiness task (PURDO).

Tough-minded ("psychotic") males also tend to sway more (BODEO) and obtain fewer correct responses in the speeded arithmetic task (AKTNC), both before and after alcohol, so there is no net effect on decrement. Females with higher P scores also obtain fewer correct and more incorrect or delayed correct responses on the AKTG task after alcohol and on the VDA both before and after alcohol. They also have faster complex reaction times both before and after alcohol. However, there are no correlations between Psychoticism and performance change scores in either sex.

How Much Variance in Performance Decrement Can We Account for?

We have found some low correlations of performance decrement with blood alcohol concentration, drinking history, and the personality trait Extraversion. However, none of these correlations is large and the predictor variables are themselves intercorrelated; for example, LCONW and LYDR are correlated 0.40 in males and 0.29 in females, Extraversion and LCONW correlate 0.36 and 0.18, and BAC and LYDR are correlated 0.33 and 0.24.

In order to see how much of the variance in performance decrement can be accounted for by BAC, LCONW, LYDR, and Extraversion, we have carried out stepwise regression analyses in which these predictor variables are entered in the order of the greatest variance accounted for at the time of entry. The results of this analysis for the two psychomotor

Table XIV. Correlations of Performance at T_0 and T_1 and Change Scores T_1-T_0 with Extraversion and Psychoticism Scores

	BODEO	BODEC	AKTNC	AKTIC	VDANC	VDADC	VDAIC	VRT	ART	CRT	PURNO	PURDO
						Extraversion						
T_0												
Males	−0.18*	−0.23**	−0.19**	0.01	0.03	−0.03	−0.02	−0.04	−0.05	−0.02	0.04	0.05
Females	−0.01	0.04	0.09	−0.04	0.02	0.02	−0.08	−0.11	−0.10	−0.05	0.09	0.13
T_1												
Males	−0.02	−0.06	−0.11	0.03	0.10	−0.11	−0.01	−0.09	−0.05	0.01	0.00	0.01
Females	−0.08	−0.04	0.07	−0.03	0.08	−0.06	−0.06	−0.06	−0.14*	−0.03	0.02	0.16*
T_1-T_0												
Males	0.15*	0.13	0.16*	0.02	0.11	−0.14	0.01	−0.07	0.01	0.03	−0.04	−0.04
Females	−0.09	−0.08	−0.04	0.00	0.08	−0.10	0.00	0.06	−0.06	0.01	−0.08	0.08
						Psychoticism						
T_0												
Males	−0.15*	−0.05	−0.21**	0.06	−0.14	0.10	0.12	−0.04	−0.09	0.05	0.07	0.09
Females	−0.03	−0.03	−0.09	0.00	−0.15*	0.14*	0.08	−0.05	−0.07	−0.18**	0.02	−0.07
T_1												
Males	−0.12	−0.05	−0.23**	0.08	−0.13	0.10	0.10	−0.01	0.02	−0.05	0.06	0.04
Females	−0.10	−0.05	−0.14*	0.14*	−0.21**	0.19**	0.10	0.02	0.01	−0.17*	0.13	0.02
T_1-T_0												
Males	−0.01	−0.01	−0.02	0.03	−0.00	0.02	−0.02	0.04	0.14	−0.09	−0.01	−0.05
Females	−0.09	−0.03	−0.08	0.15*	−0.12	0.11	0.05	0.07	0.09	0.00	0.09	0.09

* $0.01 < P < 0.05$.
** $0.001 < P < 0.01$.

Table XV. Proportions of Variance Accounted for (r^2) in Performance Change Scores ($T_1 - T_0$) When Predictor Variables Are Entered Stepwise into the Regression Equation

Males		Females	
Variable	r^2	Variable	r^2
	Body sway—eyes closed		
LCONW	0.115***	LCONW	0.013*
BAC (T_1)	0.020**	BAC (T_1)	0.017*
LYDR	0.027*	*E*	0.012
E	0.000	LYDR	0.002
Total	0.162	Total	0.043
	VDA, number correct		
BAC (T_1)	0.069***	BAC (T_1)	0.120***
LCONW	0.030	*E*	0.007
E	0.004	LCONW	0.002
LYDR	0.001	LYDR	0.003
Total	0.103	Total	0.133

* $0.01 < P < 0.05$.
** $0.001 < P < 0.01$.
*** $P < 0.001$.

variables which showed the greatest correlations between performance decrement and predictor variables (viz., BODEC and VDANC) are shown in Table XV.

The greatest proportion of variance accounted for is 16% in BODEC in males, and the bulk of this is by LCONW (12%). BAC accounts for proportionately more variance in females than previous drinking experience, especially for VDANC, where 12% of the variance in performance decrement is due to variation in blood alcohol levels. We speculate that this sex difference may be related to differences between men and women in their drinking experience. Genetical variance accounts for up to half of the total variance in BAC (Martin *et al.*, 1985) and in LCONW (Jardine and Martin, 1984), so a small proportion of the genetic variance in performance decrement probably arises from these sources.

DISCUSSION

There were decrements in psychomotor performance and increases in blood pressure, pulse rate, and skin temperature following alcohol ingestion. This was to be expected given that the mean peak blood alcohol

concentration (BAC) attained in this experiment was 101 mg/100 ml in females (N = 213) and 93 mg/100 ml in males (N = 199) (Martin *et al.*, 1985). Since the maximum impairment for most measures occurred about 40 min after the start of drinking and we estimated the mean time to peak BAC at about 70 min, the absorption phase of ethanol metabolism appears to be the time of the worst performance. Reaction times, pulse rate, and skin temperature, however, were most affected during the elimination phase at about 100 min. Other workers have also found that maximum impairment occurs during the absorption phase for most, but not all, psychomotor and physiological characters (Idestrom and Cadenius, 1968; Vogel-Sprott, 1979).

Although alcohol affected performance on all psychomotor tasks, principal-components analysis revealed that the decrement in performance was essentially independent among the five tests. The only exception was a small correlation between standing steadiness (body sway) and hand steadiness (pursuit rotor). Intercorrelations of the four physiological measures were negligible except for that between systolic and diastolic blood pressures.

There were sex differences for most of the measures regardless of alcohol status, and for some there were sex differences in the effect of alcohol. This was most noticeable in standing steadiness: even after correction for sex differences in height, weight, drinking habits, and BAC, the increase in body sway following alcohol ingestion was much more marked in females than in males. It appears that there are profound differences between men and women in their psychomotor responses to ethanol which cannot be explained away by corresponding differences in these obvious covariates. This result is supported by the intoxication self-ratings. Females reported themselves appreciably more intoxicated than males and approximately twice as many women as men reported that they would not drive a car at each time after the ingestion of alcohol.

There was considerable variation in all measures at each stage of the experiment, both before and after alcohol. For many measures, the variance increased after alcohol, and this was particularly true of motor coordination, body sway, and systolic blood pressure. We also found that correlations among the three measures (T_1, T_2, T_3) of postalcohol performance were higher than between these and the prealcohol performance (T_0). Both these observations indicated extra variation exposed by alcohol but not apparent in the sober state.

The proportion of repeatable variance among the psychomotor tasks ranged from about 0.4 for pursuit rotor to about 0.8 for motor coordination and arithmetic computation measures, with body-sway and reaction-time measures having intermediate values of 0.5–0.7. Repeatabilities for phys-

iological measures were lower, ranging from about 0.4 for most characters down to as low as 0.19 for skin temperature in males. We expect that the repeatability of a measure will be an upper bound to the proportion of heritable variance. Genetic variance in prealcohol performance was detected for most measures, and this is consistent with the results of earlier and smaller twin studies of psychomotor performance (e.g., Williams and Gross, 1980).

Genetical analysis of covariation among the four measurements of performance identifies variance in postalcohol performance which is expressed only under the influence of alcohol. There was a good correspondence between the detection of such alcohol factor variance by this method and the increase in variation following alcohol ingestion already noted. However, our task is to apportion between genetical and environmental causes this extra variance exposed by alcohol.

For motor coordination, there is unequivocal evidence of alcohol-specific variance in performance, but it is unclear whether this is due to genetic or shared environmental factors, and probably both contribute. This finding is consistent over sexes but the alcohol factor variance accounts for a maximum of only 12% of the variance and this occurs at T_1.

There is much more alcohol-factor variance for body sway, particularly in males, where it is clearly due to genetic causes. In men it accounts for 44% of the variance at T_1 and only slightly less at T_2, but it declines to 10% at T_3. In females, the picture is complicated by a large shared environmental effect on prealcohol body sway. However, close scrutiny of the results of model fitting (Table VI) suggests that, as in males, there are genetic factors "switched on" by alcohol which again contribute up to half of the variation in postalcohol body sway.

For the pursuit rotor task there is a small but significant alcohol factor in both sexes which is unequivocally genetic in origin. It accounts for about 18% of the variance at T_1 and T_2 but a negligible 3% at T_3. It is noticeable that the total genetic variance expressed at all four sampling times closely approximates the repeatability, indicating that most of the environmental variance is nonrepeatable for this character.

In the arithmetic computation task (AKTG), the heritability of performance is high at all times. The repeatability of performance in this task was also high (about 0.8) and genetic variance clearly accounts for nearly all the reliable variation. However, while there is significant alcohol-specific variance and this appears to be largely genetic, it accounts for only 5–7% of the total variance after alcohol. These results are consistent in both sexes.

Among the reaction times, significant alcohol-factor variance was detected only for auditory reaction time in females and this seems most

likely to be genetic in origin. It is noteworthy that this is the most repeatable of the reaction times. Reaction time was the only psychomotor task for which the maximum performance decrement was delayed until T_2, and it is interesting that this is also the time when the greatest contribution of genetic variance exposed by alcohol for this character (23%) is observed.

Results for blood-pressure and skin temperature readings are difficult to interpret, and this is hardly surprising in view of their low repeatabilities. It is clear that additional variance for systolic blood pressure in both sexes and skin temperature in males is switched on by the ingestion of alcohol, but it is not clear whether this is genetic or environmental in origin. There is not even significant evidence of alcohol-related variance for diastolic blood pressure in either sex or of skin temperature in females. For pulse rate, on the other hand, considerable new genetic variance was manifest after alcohol ingestion; as with auditory reaction time in females, the time of the maximum alcohol effect and of the greatest alcohol-specific genetic variance (23%) were both observed at T_2.

We have thus demonstrated that there are great differences among individuals in their psychomotor and physiological reponses to alcohol. In many measures, variation among individuals which is not apparent when they are sober is unmasked following alcohol ingestion. In many cases this extra variance is most likely of genetic origin. We now consider whether any proportion of this genetic variance exposed by alcohol might arise from variation in other characters which we have measured.

Correlations of performance decrement have been found with blood alcohol concentration, drinking habits, and the personality trait Extraversion, but none of these is large. Stepwise multiple regression shows that, at most, 16% of the variance in performance decrement on the body-sway task (and less for other variables) can be accounted for by measured covariates. About half the total variance in body sway immediately after ingestion was due to genetic effects unmasked by alcohol, so even if these covariates were completely inherited, they could account for no more than one-third of this new variance in this most favorable case. In fact, we know that only about one-half of the variation in drinking experience and blood alcohol concentration is inherited (Jardine and Martin, 1984; Martin *et al.*, 1985).

We conclude, then, at least for the body-sway task, that more than 80% of the extra genetic variation exposed by alcohol is of unknown origin. Since the variation in blood ethanol levels appears to exert such a minor influence on the variation in psychomotor decrement, future studies should focus on stages of metabolism subsequent to the initial catabolism of ethanol for the source of this genetic variation.

ACKNOWLEDGMENTS

Twin subjects were recruited from the Australian NH&MRC Twin Registry. We thank the twins for their cooperation in a trying protocol, Janet Craig for day-to-day administration, Dr. L. Y. C. Lai for supervision of the blood grouping, Marilyn Olsen for invaluable practical assistance, and David Rowell for expert assistance with computations. Drs. A. C. Heath and K. S. Kendler made valuable comments on the manuscript.

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Edited by C. Robert Cloninger