Familial Resemblances in Alcohol Use: Genetic or Cultural Transmission?*

JUDITH R. KOOPMANS, M.A., AND DORRET I. BOOMSMA, PH.D.

Department of Psychonomics, Vrije Universiteit, De Boelelaan 1111, 1081 HV Amsterdam, The Netherlands

ABSTRACT. *Objective:* Resemblances between parents and children for alcohol use can be due both to cultural transmission and genetic inheritance. We examined the genetic and environmental determinants of the familial resemblances in alcohol use. *Method:* With a parent-twin design a distinction was made between the contribution of genetic effects, the environmental influences shared by siblings and the effects of cultural transmission from parents to offspring. By questionnaire, data on whether subjects had ever used alcohol were obtained from 403 Dutch families with a twin aged 15-16 years old and from 805 families with a twin aged 17 years and older. *Results:* For 15-16 year olds, the resemblance between parents and offspring could be explained either by genetic inheritance or cultural transmission. Shared environment explained between 58% and 88% of the individual differences in adolescent alcohol use. For twins aged 17 years and older, 43% of the individual differences in alcohol use could be attributed to genetic factors and 37% to shared environment. There was no evidence for cultural transmission in this age group. *Conclusions:* For adolescents aged 17 years and older, parental alcohol use did not create an environment that stimulated alcohol use in children. The resemblance for alcohol use between parents and their children aged 17 years and older could be explained by their genetic relatedness. For 15-16 year old adolescents, shared environmental influences were more important than for older adolescents. Only 10% of this shared environmental variance might be influenced by parental alcohol use due to cultural transmission. (*J. Stud. Alcohol* **57:** 19-28, 1996)

THE FAMILIAL aggregation of alcoholism is well established. In a review, Merikangas (1990) showed that first-degree relatives of alcoholics have, on average, a seven-fold increase in the risk of developing alcoholism as compared to controls. Several reviews of family, twin and adoption studies have shown that the familial nature of alcoholism is in part due to genetic factors (Anthenelli and Schuckit, 1990; Devor and Cloninger, 1989; Merikangas, 1990), although the degree of heritability of alcoholism and the extent to which genetic factors play a role in women remain controversial (Heath and Martin, 1994; Kendler et al., 1992). To date, there is not much insight into the mechanisms that are involved in the transmission of alcoholism. Besides the genetic transmission of biochemical traits (Schuckit, 1994) another possible explanation is that familial aggregation of alcoholism is due to cultural transmission, that is the influences of parents' behavior on behavior in their offspring. Of considerable interest is the modeling of parental drinking behavior during adolescence, a transitional period in which adolescents start to use alcohol. There is evidence to suggest that parental alcohol use and parental attitudes toward alcohol use influence adolescents drinking behavior, at least to some extent (Ary et al., 1993; Dielman et al., 1993; Duncan et al., 1994; Weinberg et al., 1994). However, these familial influences may be due both to cultural inheritance and to the genetic relatedness between parents and children.

The separation of genetic effects, environmental influences shared by siblings and cultural transmission from parents to offspring is possible by using a parent-twin design (Boomsma and Molenaar, 1987; Eaves et al., 1978; Fulker, 1982, 1988; Heath et al., 1985). By comparing monozygotic (MZ) twins with dizygotic (DZ) twins, the relative contributions of genetic and shared environmental factors to individual differences can be estimated (Neale and Cardon, 1992). By including the parents of the twins, a distinction is possible between the effects of parental influences (cultural transmission) and the shared environmental effects among the offspring that are not shared with the parents (e.g., peers), while accounting for the genetic relatedness between parents and children.

Several large-scale twin studies have investigated the determinants of alcohol-related behaviors. In these studies self-reported measures of quantity, frequency and density were assessed with questionnaires. Kaprio et al. (1991) reviewed the cross-sectional and longitudinal studies of alcohol use and abuse in the Finnish Twin Cohort, a population-based sample of same-sex adult twin pairs. Results showed modest but significant genetic influences on quantity, density and frequency of alcohol use in men and women, with h^2 (the proportion of the total variance due to genetic factors) ranging from 29% to 45%. Evidence was found for shared environmental influences: twins in more frequent contact showed greater similarity for alcohol use. Carmelli et al. (1993) showed, in a 16-year follow-up of World War II veteran

Received: December 12, 1994. Revision: March 15, 1995.

*This study was supported by the Dutch Heart Foundation and Medical Research NWO (grant NWO 904-61-072).

twins, significant genetic effects for alcohol consumption at baseline (up to 40%) and follow-up (up to 48%) and somewhat lower shared environmental influences (0-32% at baseline, 1-20% at follow-up). Longitudinal stability of drinking behaviors in this cohort was largely due to the continuity of genetic influences.

A study of 572 families of adult twins and their parents in the U.K. found evidence that the amount of alcohol consumption in 1 week was influenced both by genetic factors (37%) and shared environment (42%) (Clifford et al., 1984). The parent-offspring correlations for alcohol consumption were around 0.20. Clifford et al. did not assess the contribution of cultural transmission or genetic inheritance to this parent-offspring resemblance. The shared environmental influences on twins consisted of two parts; one part was manifest when twins lived together, whereas the other part persisted when twins lived apart. The contribution of genetic and shared environmental effects did not differ between men and women. In another study, sex differences in the relative contribution of genetic and environmental factors to variation in alcohol consumption were found in 3,810 pairs of adult Australian twins (Jardine and Martin, 1984). Variation of alcohol consumption in women was explained by genetic factors (56%) without evidence for shared environmental influences. In men, 36% of the total variance was explained by genetic factors and 20% was due to shared environmental effects. The effects of genetic and shared environmental factors were dependent on age. For women, results were similar in younger (30 years and younger) and older (over 30) twins. However, for younger male twins 60% of the individual differences in weekly alcohol consumption were explained by genetic factors and not by shared environment, whereas for older male twins no genetic effects were found and 50% of the total variance was explained by shared environment. Heath et al. (1991a,b) analyzed the same sample of adult Australian twins and showed that abstinence of alcohol use was determined by factors that were largely independent of the factors that determined frequency and quantity of alcohol consumption. Abstinence was strongly influenced by shared environment but not by genetic factors, while frequency and quantity were mainly determined by genetic factors. Consistent with these findings, results from a study of alcohol use in a U.S. volunteer sample of 3,049 female and 1,070 male twins aged 50 to 96, suggested that determinants of whether one drinks differed from those underlying amount of consumption (Prescott et al., 1994). Both genetic (40%) and shared environmental (42%) factors contributed to lifetime abstinence in both men and women, while among drinkers resemblances for alcohol consumption were only explained by genetic influences (43%). The twin studies cited so far all described adult drinking behavior. To our knowledge, there are only two twin studies in which adolescent drinking behavior was assessed. In a study of 1,400 adolescent Australian twin pairs, aged 11 to 18 years, drinking in the previous month was assessed with the question, "Have you had an alcoholic drink in the last four weeks?" (Hopper et al., 1992). Twin associations were represented by log odds ratios. Higher odds ratios in MZ male twins compared to DZ male twins indicated that genetic factors play a role in determining alcohol use in male adolescents. In female twins, MZ and DZ odds ratios were equal, suggesting only shared environmental influences. Retrospective information about abstinence from teenage alcohol use and age of onset of drinking was obtained from 1,589 Australian twin pairs aged 20-30 years (Heath and Martin, 1988). Abstinence from teenage alcohol use was determined by both genetic and shared environmental factors, to differing degrees in males and females. The shared environmental factors that influenced initiation of alcohol use were uncorrelated in males and females. Individual differences in age of onset of teenage drinking in males were not explained by genetic factors but by shared environmental influences (51%). Age of onset of drinking in females was determined by moderate genetic influences (44%) and to a lesser extent by shared environmental factors (14%).

To summarize, twin studies of adult drinking behavior show that genetic factors, and to a lesser extent shared environmental influences, contribute to individual differences in alcohol consumption. For adolescent drinking behavior shared environmental effects seem to be more important. No studies have explicitly addressed the question of cultural transmission for alcohol use. Kendler et al. (1994) used the extended twin-family design to examine the transmission of the vulnerability to alcoholism in 1,030 pairs of adult female twins and their parents. They concluded that alcoholism in parents was genetically transmitted to their children and that there was no evidence for cultural transmission.

In this article we examine to what extent genetic and shared environmental factors contribute to adolescent alcohol use, whether parents and children resemble each other for alcohol use and whether this resemblance is best explained by cultural transmission or by genetic inheritance. Under random mating parents and children have 50% of their genes in common. Genetic inheritance refers to the fact that parents transmit their genetic predisposition for alcohol use to their children. Besides the shared genes, parents and children may share environmental influences. In general, shared environmental factors indicate to what extent family members resemble each other due to the environmental influences that they have in common. In the parent-offspring model that we used the shared environment of the children can be partitioned into environmental influences that the children share with each other but not with their parents and cultural transmission. Cultural transmission refers to the effect of parental behavior (e.g., parental alcohol use) on the shared environment of the children. Several studies have shown that alcohol use in spouses is correlated (Clifford et al., 1984; Gleiberman et al., 1992; Hall et al., 1983; Price and Vandenberg, 1980; Tambs and Vaglum, 1990). This correlation between spouses can be due to assortative mating or to environmental influences shared by spouses. Price and Van-

denberg (1980) found some evidence that spouse similarity for alcohol consumption increased with the length of marriage, but they also showed that there was a significant association for amount of drinking when spouses began dating. This suggests that there is initial assortment for alcohol use. We modeled the correlation between alcohol use in spouses as phenotypic assortative mating. Under this model spouses select each other based on a observed trait or phenotype (e.g., Fulker, 1982). Consequently, the genetic and environmental factors that influence the paternal phenotype become correlated with the latent factors that influence the maternal phenotype. The correlation between the genetic factors of the spouses increases the genetic resemblance between relatives. The increased similarity of DZ twins relative to MZ twins, due to these genetic effects of assortative mating, will inflate the estimates of shared environment in the classical twin model (Fulker, 1982, 1988). In the twin-family model that we use, these effects of phenotypic assortative mating are explicitly taken into account.

We collected questionnaires on health-related behaviors from 1,700 pairs of adolescent twins and their parents. We have previously examined smoking initiation and sports participation in this sample and found that environmental factors that influenced smoking and sports participation were shared between twins but not between parents and children. The resemblance between parents and offspring for smoking and sports participation could entirely be explained by their genetic relatedness and not by cultural transmission (Boomsma et al., 1994; Koopmans et al., 1994).

Method

Subjects and measures

This study is part of the Dutch twin family study on health-related behavior (Boomsma et al., 1994; Koopmans et al., 1994). Questionnaires on health and lifestyle were mailed in 1991 to adolescent twins and their parents. Twin families were recruited by asking all city councils (720) in the Netherlands for addresses of twins aged 12-22 years. A positive response was received from 252 city councils which supplied 3,859 addresses; 177 addresses were available from other sources. After contacting these families by letter, 2,375 twin families indicated that they were willing to participate and 1,700 families returned the questionnaires.

Age of the twins was between 12 and 24 years, less than 4% of the sample were younger than 14 years and 7% were older than 21, the mean ($\pm$SD) age was 17.7 $\pm$ 2.26 years. The average age of the fathers and mothers was 48 $\pm$ 5.6 years and 46 $\pm$ 5.2 years. Zygosity of the twins was determined by questionnaire items about physical similarity and frequency of confusion of the twins by family and strangers (Goldsmith, 1991; Magnus et al., 1983). The classification of zygosity was based on a discriminant analysis, relating the questionnaire items to zygosity based on bloodgroup polymorphisms and DNA fingerprinting in a group of 131 same-sex adolescent twin pairs who participated in a study of cardiovascular risk factors (Boomsma et al., 1993). In that sample zygosity was correctly classified by questionnaire in 95% of the cases. We were able to control the validity of the zygosity questionnaire in a subsample of 88 same-sex twins, aged 16 years, who participated both in our study and in a longitudinal study of brain function (van Beijsterveldt et al., 1994). For these same-sex twins, the agreement between zygosity based on the questionnaire and zygosity based on blood/DNA polymorphisms was 88%. Of the 11 pairs who were misclassified by questionnaire, nine pairs were MZ twins mistakenly assigned as DZ twins.

The questionnaire contained questions about alcohol and tobacco use, sport activities, health, social economical status, religion and a number of personality factors. We asked both parents and twins whether they used or had used alcohol. The question could be answered with "no, seldom or never," "yes, but not any more" and "yes." Less than 2% of the sample of twins and less than 4% of the parents answered "yes, but not any more." Therefore, the last two answers were collapsed into one category, leaving a dichotomous variable for alcohol use. Those who answered "no, seldom or never" but indicated that the quantity of alcohol they consumed in a week was one or more glasses were considered alcohol users. Thus, the variable under study divides the sample into never used alcohol versus ever used alcohol. Of the 1,700 families who returned questionnaires, 1,396 families provided complete data for alcohol use from both father and mother and the twins. The families were divided in five groups by sex and zygosity of the twins; monozygotic males (MZM) and females (MZF), dizygotic males (DZM) and females (DZF) and dizygotic opposite sex twins (DOS).

Statistical analysis

The way we defined alcohol use, a person can be either a drinker or a nondrinker. Quantifying the genetic and environmental factors that contribute to such a dichotomous variable is possible by assuming that the variable has an underlying continuous distribution (Falconer, 1989). This underlying continuous variable has been termed the liability. The liability is due to multiple genetic and environmental influences, giving a normal distribution in liability. A threshold divides the distribution into two classes, affected or not-affected. The correlation in liability, between two family members for example, is called the tetrachoric correlation. We used PRELIS2 (Jöreskog and Sörbom, 1993) to estimate the tetrachoric correlations between twins, spouses and parents and offspring. For each pair of family members (e.g., twin1-twin2; father-mother; father-twin1) a two-by-two contingency table is obtained from which the maximum likelihood estimate of the tetrachoric correlation is computed by PRELIS2, under the assumption that the two variables (e.g.,

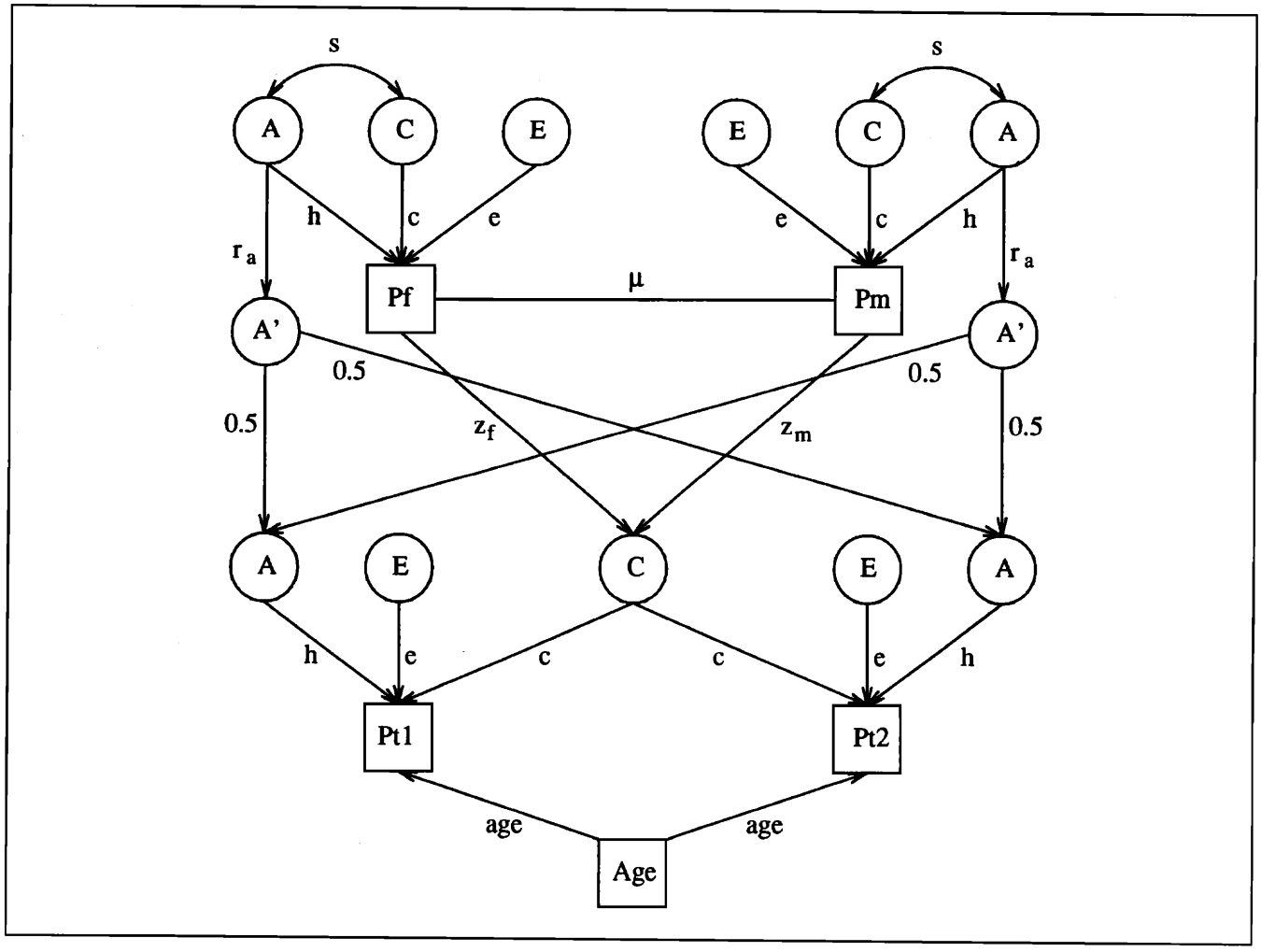

FIGURE 1. Path diagram of the full twin-family model. Circles represent the latent variables and squares the observed variables. Pf and Pm are the observed phenotypes of father and mother, Pt1 and Pt2 the phenotypes of twin1 and twin2. Part of the total variance in twins is explained by the effects of age of the twins. A represents the additive genetic influences, C the shared or common environmental effects and E the unique environmental influences that are specific for each individual. The influence of A, C and E on the phenotype is given by path coefficients h, c and e, respectively. The path coefficients equal the standardized regression coefficients. The model allows for the possibility that genetic factors in the parental generation are different from the genetic factors in the twin generation. r_a estimates the correlation between the parents' genotype and the parents' latent genotype (A') that is transmitted to their children, $r_a = 1$ in the full model. Cultural transmission is represented by z_f and z_m, for father and mother, respectively. The genotype-environment correlation s is induced by cultural transmission and assumed to be at equilibrium. Phenotypic assortative mating is represented by a copath μ (Cloninger, 1980). This path induces correlations among the spouses' latent variables without affecting their within-person covariance structure before assortment. The correlation between the genetic factors in the parents can be expressed as $\gamma = (h+sc)^2 \mu$. The total variance of the twins is computed as $h^2+c^2+age+e^2+2hcs=1$.

drinking in twin1 and drinking in twin2) have a bivariate normal distribution.

A structural equation model was fitted to the twin, spouse and parent-offspring correlations simultaneously to obtain estimates of the genetic and environmental factors that contribute to the familial resemblance of alcohol use. In the full model, the total phenotypic variance in twins is partitioned into an additive genetic part, an individual-specific or unique environmental part, and a shared environmental part (Figure 1). The genetic factors are correlated one in MZ twins, as they are genetically identical. Under random mating, the genetic correlation in DZ twins is a half, as they share on average 50% of their genes. Under phenotypic assortative mating, the genetic correlation in DZ twins is inflated by the correlation between the genotypes of the parents (Falconer, 1989). The genetic correlation in DZ twins thus becomes $0.5(1 + \gamma)$, where γ is the correlation between the genetic factors of the parents (see Figure 1).

By definition, the correlation between the shared environmental factors is unity in MZ and same-sex DZ twins. In opposite-sex twins the correlation between the shared environmental factors in males and females can be less than unity, indicating the extent to which males and females experience different environmental influences. The shared environment of twins is partitioned into a part due to cultural transmission (i.e., the influence of parental drinking behavior on children's environment) and into a part due to environmental influences that are shared by twins only, such as

peers and school. The cultural transmission parameter from the paternal phenotype to the environment in children is allowed to be different from the maternal influences. The genetic inheritance was modeled as a path (with value 0.5) from the parents' genotype to their children's genotype. If there is assortative mating, the genetic resemblance between parents and children is inflated by the correlation between the genotypes of the parents. The model allows for the possibility that genetic factors in the parental generations are different from genetic factors in the twin generation. This genetic correlation between generations is estimated as a path from the parents' genotype to the parents' latent genotype that is transmitted to their offspring. This path is one in the full model. The genetic correlation between generations can only be estimated in a model without cultural transmission, because these two parameters are confounded. The correlation between the spouses was modeled as assortment based on the phenotypes. Phenotypic assortative mating induces correlations between the latent factors that determine the paternal phenotype and the latent factors that determine the maternal phenotype. Due to the effects of cultural transmission the genetic and shared environmental factors within a person become correlated and inflate the phenotypic variance. The assumption of the model is that the effects of assortative mating and cultural transmission have been going on for some generations and have reached a state of equilibrium (Eaves et al., 1978; Fulker, 1982, 1988).

Age of the twins was included in the model as a separate factor, explaining part of the total variance. The effects of age contributes to differences between twin pairs and not to differences within pairs, since twins are always of the same age. If we did not correct for the effects of age, the estimated share environmental influences would be inflated (Neale and Martin, 1989).

For each twin family group, a 5×5 matrix of observed correlations was computed with PRELIS2, giving the correlations for alcohol use between each pair of family members (father, mother, first-born twin, second-born twin) and the correlations between alcohol use and age of the twins. For opposite-sex twins a correlation between male and female twins was computed. Age was recoded to an ordinal variable, resulting in a polychoric correlation between age and alcohol use, because PRELIS still has some problems in estimating the asymptotic weight matrix of the polyserial correlation (a correlation between a continuous and an ordinal variable).

We used Mx, a structural equation modeling package specifically designed for modeling genetically informative data (Neale, 1993), to fit the twin family model to the observed correlation matrices. Parameters were estimated using weighted least squares (WLS). The asymptotic covariance matrix of the observed correlations, which is required as a weight matrix for WLS estimations, was computed with PRELIS2. Different models of familial resemblance were fitted. Under a model where familial resemblance is explained by additive genetic factors, MZ twins, who are genetically identical, are expected to be twice as similar as DZ twins and parents and offspring, who have on average half of their genes in common. Under a shared environmental model, MZ and DZ correlations are predicted to be the same, and parent-offspring correlations depend on the size of the cultural transmission parameters. Under a model where both genetic and shared environmental influences contribute to familial resemblance, DZ correlations are expected to be more than half the MZ correlations. The goodness-of-fit of the models was assessed by likelihood-ratio χ^2 tests and by Akaike's Information Criterion (AIC) (Akäike, 1987). AIC is computed as the χ^2 minus two times the degrees of freedom. This is a measure of the parsimony of the model. The model with the lowest (i.e., largest negative) value of AIC is the most parsimonious and best-fitting model.

TABLE 1. Frequency of alcohol use in males and females for different age groups

	Males		Females	
Age (years)	*n*	%	*n*	%
13<	59	3	68	0
14	145	11	175	6
15	194	26	230	22
16	258	46	270	31
17	231	71	251	48
18	175	73	253	46
19	204	75	264	53
20	165	83	207	52
21>	119	74	131	48
Overall	1,550	55	1,849	37

Results

The number of adolescents who use alcohol is shown in Table 1. Most adolescents start to drink alcohol after the age of 15. At 17 years of age, 71% of the boys and 48% of the girls have used alcohol. The figures in Table 1 show that alcohol use is more common in boys than in girls. This sex difference is also seen in their parents: 88% of the fathers and 65% of the mothers have used alcohol. Because of the prevalence differences in alcohol use between the age cohorts the sample was divided into three age groups: 12 to 14 year old twins, 15 and 16 year old twins, and twins of 17 years and older. For the three different age groups, Table 2 gives the percentages of alcohol users for each zygosity group. In each age group, we tested if alcohol use was independent of zygosity. The alcohol use patterns did not differ significantly between MZ males, DZ males and males from opposite sex twins for the 12 to 14 year old twins ($\chi^2 = 3.42$, 2 df, $p = .18$), for 15-16 year olds ($\chi^2 = 3.41$, 2 df, $p = .18$) and for twins aged 17 years and older ($\chi^2 = 3.95$, 2 df, $p = .14$). For females, alcohol use was also independent of zygosity (12-14: $\chi^2 = 3.26$, 2 df, $p = .20$; 15-16: $\chi^2 = 0.62$, 2 df, $p = .73$; 17 and older: $\chi^2 = 1.80$, 2 df, $p = .41$).

TABLE 2. Concordances and tetrachoric correlations for alcohol use in each zygosity group for different age groups

			Drinking status			Correlation	
	n^a	%Yes	%Both	%One	%Neither	r	SE
12-14 years old[b]							
MZM	40	6	25	75	90		
DZM	32	14	6	16	78		
MZF	46	8	7	2	91		
DZF	49	3	–	6	94		
DOS	51	6m 2f	–	8	92		
15-16 years old							
MZM	68	36	27	19	54	0.80	0.09
DZM	64	34	27	14	59	0.89	0.07
MZF	74	26	22	9	69	0.94	0.05
DZF	73	30	21	19	60	0.78	0.10
DOS	124	44m 30 f	22	31	48	0.58	0.11
17 years and older							
MZM	122	71	61	20	19	0.74	0.09
DZM	129	78	67	22	11	0.60	0.12
MZF	172	47	37	21	42	0.79	0.06
DZF	153	51	35	31	33	0.55	0.10
DOS	229	78m 52f	45	40	15	0.35	0.11

[a] n is the number of complete twin pairs in 12-14 year olds, and n is the number of complete families in the other two age groups.
[b] Due to the small number of subjects in this age group and the empty cells in the DZF and DOS group tetrachoric correlations were not computed.
Note: MZM = monozygotic male twins; DZM = dizygotic male twins; MZF = monozygotic female twins; DZF = dizygotic female twins; DOS = dizygotic opposite-sex twins; m = male; f = female; r = tetrachoric correlations; SE = standard error.

Twin resemblances were expressed as concordances and as tetrachoric correlations (Table 2). Twins in the youngest age group (12-14 years) were highly concordant for not drinking alcohol. For this cohort (n = 218) the tetrachoric correlations could not be computed because of the small numbers of subjects, the low rate of alcohol use and the empty cells in the concordance tables for the DZF and DOS twins. Therefore, genetic analyses were carried out only for the 403 families of 15-16 year old twins and for the 805 families of twins aged 17 years and older. The tetrachoric correlations showed that twins highly resembled each other for drinking behavior. In the group aged 15-16 years, the DZM correlations were as high as the MZM correlations. For females, the DZ correlations seem to be lower than the MZ correlations. In the oldest cohort, the DZ twins were less alike than the MZ twins. The pattern of correlations in the two age groups suggests that shared environment is an important factor in the familial resemblance of alcohol use. With increasing age, the difference between MZ and DZ twins increased, suggesting that genetic factors become more important. In both age groups, the opposite-sex twins were less alike for alcohol use than were the same-sex dizygotic twins. This indicates that drinking in males and females is, to some extent, influenced by different factors.

TABLE 3. Spouse and parent-offspring correlations for alcohol use for two different age groups in the offspring

	15-16 years old			17 years and older		
	n	r	SE	n	r	SE
Spouses	403	0.41	0.06	805	0.55	0.04
Father-son	388	0.12	0.07	731	0.34	0.06
Mother-son	388	0.33	0.06	731	0.31	0.05
Father-daughter	418	0.33	0.08	879	0.37	0.05
Mother-daughter	418	0.22	0.07	879	0.25	0.04

Note: r = tetrachoric correlation; SE = standard error.

TABLE 4. Model fitting results–parent-offspring model for 15-16 year old offspring

Model	χ^2	df	p	AIC
1. ACE, cult.trans. fa ≠ mo	40.80	43	0.57	−45.20
2. ACE, age = 0	57.66	44	0.08	−30.34
3. ACE, cult.trans. fa = mo	40.82	44	0.61	−47.18
4. ACE, cult.trans = 0	41.50	45	0.62	−48.50
5. ACE, cult.trans. = 0,r(G) free	41.54	44	0.58	−46.46
6. CE, cult.trans. fa ≠ mo	43.39	44	0.50	−44.61
7. CE, cult.trans. fa = mo	43.40	45	0.54	−46.60
8. CE, cult.trans. = 0	67.18	46	0.02	−24.82

Note: A = additive genetic factor; C = shared environmental factor; E = unique environment; fa = father; mo = mother; r(G) = the genetic correlation between generations.

Table 3 shows the spouse and parent-offspring correlations in these two age groups. The spouse correlations suggests that the parents of the younger twins resemble each other less than parents of the older twins. However, this difference was not significant, equating the spouse correlations across the 2 age groups by 5 zygosity groups gave χ^2 = 13.33, 9 df, p = 0.15. The overall (± SE) spouse correlation was estimated at 0.51 ± 0.03. The parent-offspring correlations were much lower than would be expected from the twin and spouse correlations. The parent-offspring correlations were not dependent on the sex of the parents nor on the sex of the offspring, both for the 15-16 year old offspring ($\Delta\chi^2$ = 3.57, 3 df, p = 0.31) and for the offspring aged 17 years and older ($\Delta\chi^2$ = 2.63, 3 df, p = 0.45). For the 15-16 year olds the overall parent-offspring correlation was estimated at 0.25 ± 0.04; for the oldest cohort the estimated overall parent-offspring correlation was 0.31 ± 0.03.

With a structural equation model we tested whether the parent-offspring correlations could be explained by cultural transmission or by genetic inheritance or by both. First, the parent-offspring models were fitted separately for twins aged 15-16 years and for the twins aged 17 years and older. The goodness-of-fit parameters for the different models that were considered for the 15-16 year olds are given in Table 4. Under all models, the correlation between the shared environmental factors in boys and girls was estimated as a free parameter; constraining this correlation to one worsened the fit of the model significantly ($\Delta\chi^2$ = 8.00, 1 df, $p < .01$). The first model in Table 4 is the full model with an additive genetic factor (A), a shared environmental factor (C), a unique environmental factor (E) and age of the twins. Under this model, different cultural transmission parameters were esti-

mated for fathers and mothers. The second model shows that the effects of the age of the twins on alcohol use were significant and could not be constrained to zero ($\Delta\chi^2 = 16.86$, 1 df, $p < .001$). The correlation (± SE) between alcohol use and age was 0.26 ± 0.05. Therefore, the factor loading on age of the twins was estimated as a free parameter in the subsequent models. In the third model the cultural transmission parameters were constrained to be equal for fathers and mothers. Model 4 showed that the cultural transmission parameter was not significant. Cultural transmission could be set to zero without a significant increase in the chi-square. Model 5 estimated the correlation between the genetic factors in the parents and genetic factors transmitted to their children. The estimated genetic correlation was equal to one, indicating that the genetic factors influencing alcohol use did not differ between generations. The additive genetic factor could be constrained to zero (Model 6) without worsening the fit of the model, compared to the first model ($\Delta\chi^2 = 2.59$, 1 df, $p = .11$). Under this model, in which only shared environment explained familial resemblance in alcohol use, cultural transmission was significant (Model 8) and equal for fathers and mothers (Model 7).

The model fitting results for the group aged 15-16 years showed that both Model 4 and Model 7 gave a good description of the data. It is not possible to make a distinction between the two models based on likelihood-ratio χ^2 tests. The AIC indicated that Model 4 is a slightly better fitting model. Under Model 4, the resemblances between parents and their 15-16 year old offspring were explained by their genetic relatedness and not by cultural transmission. For this model, additive genetic factors explained 34% of the total variance in alcohol use, 58% was accounted for by shared environmental factors and 7% by age of the twins (Table 6). Under Model 7, familial resemblances in alcohol use were explained by shared environmental influences (88%) and age of the twins (7%) (Table 6). Cultural transmission explained 10% of the shared environmental variance in twins. This means that 79% of the total variance was explained by shared environmental influences in the children that were not shared with their parents, and 9% was explained by cultural transmission. The correlation between shared environmental factors in boys and shared environmental factors in girls was estimated at 0.49 under Model 4 and at 0.44 under Model 7.

The model fitting results for the families of twins aged 17 years and older are given in Table 5. For this age group the additive genetic influences on alcohol use were significant. Constraining the additive genetic component to zero (Model 3) gave a significant reduction of the goodness-of-fit, compared to the first model ($\Delta\chi^2 = 10.92$, 1 df, $p < .001$). The best fitting model for the twins aged 17 years and older was Model 5. Under this model there was no cultural transmission for alcohol use (i.e., the shared environment of twins was not influenced by parental alcohol use). Additive genetic factors accounted for 43% of the individual differences in alcohol use, 37% was accounted for by shared environmental influences and 1% by age of the twins (Table 6). Although the proportion of the total variance that was explained by age of the twins was only 1%, this was significant (Model 2). The correlation (± SE) between alcohol use and age was 0.06 ± 0.03. Under Model 6, the genetic correlation between the generations was estimated at 0.64. However, the fit of this model was not significantly better than Model 5 with a genetic correlation of one. As in the 15-16 year old twins, boys and girls of 17 years and older experienced to some extent different shared environmental influences. The correlation between the shared environmental factors in boys and girls of opposite-sex twins was 0.41.

TABLE 5. Model fitting results–parent-offspring model for offspring aged 17 years and older

Model	χ^2	df	p	AIC
1. ACE, cult.trans. fa ≠ mo	70.67	43	0.005	−15.33
2. ACE, age=0	75.13	44	0.002	−12.87
3. CE, cult.trans. fa ≠ mo	81.60	44	0.000	−6.40
4. ACE, cult.trans. fa=mo	73.10	44	0.004	−14.90
5. ACE, cult.trans.=0	75.01	45	0.003	−14.99
6. ACE, cult.trans.=0,r(G) free	73.10	44	0.004	−14.90

Note: A=additive genetic factor; C=shared environmental factor; E=unique environment; fa=father; mo=mother; r(G)=the genetic correlation between generations.

Next, it was tested whether the same model could explain familial resemblances in alcohol use for both the 15-16 year old twins and the twins of 17 years and older. First, a full parent-offspring model was specified with different parameter estimates for the two age groups, giving $\chi^2 = 111.48$, 86 df, $p = .03$. Then, the parameter estimates were constrained to be equal across the two age groups, giving a significant reduction in the goodness-of-fit ($\Delta\chi^2 = 29.71$, 7 df, $p < .001$). Fitting a model in which the effects of age were allowed to be different for the two age groups still gave a worse fit than the first model ($\Delta\chi^2 = 24.93$, 6 df, $p < .001$). Thus, the magnitude of the genetic and environmental influences on alcohol use in 15-16 year olds was significantly different from the magnitude of the latent factors influencing alcohol use in the older cohort. For 15-16 year old twins the resemblance between parents and offspring could be explained by either genetic inheritance or cultural transmission. For twins aged 17 years and older there was no evidence for cultural transmission.

Discussion

This is the first genetic study in which the transmission of parental alcohol use to their adolescent offspring is examined. The question whether the familial resemblance of alcohol use could be accounted for by cultural transmission was studied in parents and twins aged 15-16 years old and in parents and twins aged 17 years and older. For the adolescents aged 17 years and older we found no evidence for cultural transmission (i.e., parents' alcohol use did not directly influ-

TABLE 6. Standardized parameter estimates for the best fitting twin-family models of alcohol use

Model	h^2	c^2	age	e^2	μ	z_f	z_m	s	r(C)
15-16 year									
Model 4	0.34	0.58	0.07	0.01	0.41	–	–	–	0.49
Model 7	–	0.88	0.07	0.05	0.37	0.19	0.19	–	0.44
17 years and older									
Model 5	0.43	0.37	0.01	0.19	0.57	–	–	–	0.41

Note: h^2 represents the proportion of the total variance that is due to genetic factors; c^2 is the proportion due to shared environment and cultural transmission; age is the proportion of the total variance that can be explained by age of the twins; e^2 is the effect of unique environment. The total variance is computed as $h^2 + c^2 + age + e^2 + 2hcs = 1$, where s = genotype-environment covariance induced by cultural transmission. z_f and z_m represent the cultural transmission parameters for father and mother, respectively. The residual shared environmental variance in twins that is not influenced by cultural transmission is computed as $1 - 2z^2(1 + \mu)$. μ is the assortative mating parameter; r(C) is the correlation between the shared environmental factors in boys and girls.

ence alcohol use in the children). For this age group the resemblance in alcohol use between parents and offspring was explained by their genetic relatedness. The results for the 15-16 year old twins are less conclusive. It was not possible to make a distinction between a model in which the familial resemblances of alcohol use were explained by cultural transmission and a model in which familial resemblances were explained by genetic inheritance. Both models fitted the data equally well. Under a model with cultural transmission, there was no evidence for genetic influences on alcohol use. Resemblances in drinking behavior were explained by shared environmental influences. Due to cultural transmission, part of the shared environment in twins was influenced by parental alcohol use. However, this influence of cultural transmission was rather small. Parental alcohol use contributed only 10% to the shared environmental variance in twins. The residual 90% of the shared environmental variance may consist of the influences of peers, siblings and parental influences that we could not account for in the model, such as parental attitudes towards alcohol use.

Under a model with genetic transmission, the resemblances between parents and offspring for alcohol use were explained by the resemblances in their genetic make-up. Besides the genetic influences in this model, alcohol use in 15-16 year old adolescents was determined by shared environmental influences. These shared environmental factors were not influenced by parental drinking. For older adolescents we found a substantial genetic influence on alcohol use. It is possible that the genetic factors expressed at age 17 and older are correlated with genetic factors expressed at age 15-16. If we can find evidence for such a genetic correlation, a model in which the familial resemblance of alcohol use in 15-16 year olds is explained by genetic transmission will be more likely. This can be resolved by including siblings of the twins to the design. That gives the possibility to compute the correlation in alcohol use between siblings aged 15-16 and siblings aged 17 years and older. Under a model with genetic and shared environmental influences on both 15-16 year old siblings and siblings aged 17 years and older, the expected correlation between the siblings will be higher than under a model with cultural transmission and no genetic effects for 15-16 year old siblings.

Kendler et al. (1994) used the same twin-family design in a study of alcoholism in adult female twins and showed that there was no cultural transmission for alcoholism in women. Tambs and Vaglum (1990) studied alcohol consumption in parents and children aged 18 years and older. The nuclear family design cannot make a distinction between shared genes and shared environment. However, based on the pattern of correlations and estimates of genetic transmission from previous twin studies, Tambs and Vaglum (1990) concluded that there was no evidence for cultural transmission. We have previously examined smoking and sports participation and found that there was no evidence for cultural transmission for these health-related behaviors (Boomsma et al, 1994; Koopmans et al., 1994). The resemblances between parents and offspring for smoking and sports participation were explained by genetic inheritance.

The parent-offspring correlations that we found are consistent with other studies of adolescent alcohol use that found a moderate association between parental drinking and children's alcohol use (Ary et al., 1993; Dielman et al., 1993; Duncan et al., 1994; Hopper et al., 1992; Weinberg et al., 1994). For adolescents aged 17 years and older, we have shown that this association is not due to the shared family environment but due to shared genes. For 15-16 year old adolescents, there is some evidence to suggest that initiation of alcohol use is partly due to the modeling of parental alcohol use. However, the parent-offspring correlations that we found for alcohol use were lower than the spouse and DZ twin correlations. This same pattern of lower correlations between generations than within generations was also found for smoking and sports participation (Boomsma et al., 1994; Koopmans et al., 1994; Pérusse et al., 1988). It suggests that for alcohol use, and for other health-related behaviors, horizontal cultural transmission within generations is more important than vertical cultural transmission between generations.

In all models for parent-offspring resemblances in the families of 15-16 year old twins, shared environmental influences were the most important contributor to the individual differences in adolescent alcohol use. Peers constitute a major part of these shared environmental influences. Several studies have shown that peer use and peer pressure are strongly related to adolescent alcohol use (Ary et al., 1993; Dielman et al., 1993; Duncan et al., 1994; Hopper et al., 1992). Our results showed that shared environmental influences were important for both 15-16 year old twins and twins aged 17 years and older. However, the magnitude of these influences differed between the two age groups. The shared environmental influences on alcohol use become less important as adolescents grow older, whereas the contribution of genetic factors to the individual differences in alcohol use increases. We

found that opposite-sex twins were less alike for alcohol use than were same-sex dizygotic twins. This lower opposite-sex correlation was explained by the reduced correlation between the shared environmental factors in boys and girls of opposite-sex twins. This means that boys and girls experience different types of shared environmental influences, at least to some extent. These sex-specific environmental influences were also found for abstinence from teenage alcohol use (Health and Martin, 1988), smoking (Boomsma et al., 1994) and sports participation (Koopmans et al., 1994). Another explanation for the lower opposite-sex correlation, compared to the same-sex DZ twin correlations, can be a reduced genetic correlation, suggesting that different genes are expressed in males and females. However, sex-specific genetic factors would have resulted in sex-differences in the parent-offspring correlations. We did not observe such sex-differences; parent-offspring correlations were independent from sex of the parent and sex of the children.

With our measure of alcohol use, we assessed whether or not people use alcohol. This may be a broad definition; we did not make a distinction between light and heavy drinkers. However, understanding the factors that influence alcohol use gives us more insight into the development of alcohol-related problems. Only those who initiate alcohol use can develop alcohol problems in the future. Therefore, it is important to investigate the factors that determine why some people drink alcohol and others do not.

Adolescent alcohol use is widely accepted in the Netherlands. The legal age to buy alcohol in a liquor store is 16 years. Even with the availability of alcohol for adolescents there was still a considerable proportion of the sample that stated that they did not use alcohol. For example, in the oldest cohort of twins aged 17 years and older, around 25% of the males and 50% of the females did not use alcohol. This gave us the possibility to assess the factors that contribute to the individual differences in alcohol use. It will be interesting to see if the determinants of adolescent alcohol use are different in countries with a more restricted policy regarding alcohol use.

Our sample is representative of the general population with regard to alcohol use. The number of adolescents who used alcohol in our study was comparable with the percentage of high-school students that spend money on alcohol consumption in a large scale survey of around 11,000 high-school students in the Netherlands (de Zwart et al., 1993), but somewhat lower than the percentage of adolescents aged 10 to 20 that consumed alcohol in the previous month in a national youth health care survey ($N = 8{,}019$) (Plomp et al., 1991).

A problem in our study was that, in a small subsample of same-sex twins, 12% of the twins were misclassified for zygosity by questionnaire. The most common error was that MZ twins were mistakenly classified as DZ twins, which could have resulted in elevated same-sex DZ twin correlations. This would result in overestimated shared environmental influences and underestimated genetic influences. However, we have previously studied sensation seeking in the same sample of 1,700 adolescent twins, using the same classification for zygosity, and found no evidence for elevated DZ twin correlations. As for other aspects of personality, we found for sensation seeking that the DZ correlations were about half the MZ correlations (Koopmans et al., 1995).

In conclusion, the shared environment was the most important determinant of alcohol use in 15-16 year old adolescents. If there is cultural transmission of parents' alcohol use, this explains only 10% of the shared environmental variance in their offspring. This small influence of parental drinking is reduced to zero in adolescents aged 17 years and older. As adolescents grow older, the influence of genetic factors becomes more important and the influence of shared environment decreases.

References

AKAIKE, H. Factor analysis and AIC. Psychometrika **52:** 317-332, 1987.

ANTHENELLI, R.M. AND SCHUCKIT, M.A. Genetic studies of alcoholism. Int. J. Addict. **25:** 81-94, 1990.

ARY, D.V., TILDESLEY, E., HOPS, H. AND ANDREWS, J. The influence of parent, sibling, and peer modeling and attitudes on adolescent use of alcohol. Int. J. Addict. **28:** 853-880, 1993.

BOOMSMA, D.I., KAPTEIN, A., KEMPEN, H.J., GEVERS-LEUVEN, J.A. AND PRINCEN, H.M. Lipoprotein(a): Relation to other risk factors and genetic heritability. Results from a Dutch parent-twin study. Atherosclerosis **99:** 23-33, 1993.

BOOMSMA, D.I., KOOPMANS, J.R., VAN DOORNEN, L.J. AND ORLEBEKE, J.F. Genetic and social influences on starting to smoke: A study of Dutch adolescent twins and their parents. Addiction **89:** 219-226, 1994.

BOOMSMA, D.I. AND MOLENAAR, P.C.M. Constrained maximum likelihood analysis of familial resemblance of twins and their parents. Acta Genet. Med. Gemellol. **36:** 29-39, 1987.

CARMELLI, D., HEATH, A.C. AND ROBINETTE, D. Genetic analysis of drinking behavior in World War II veteran twins. Genet. Epidemiol. **10:** 201-213, 1993.

CLIFFORD, C.A., HOPPER, J.L., FULKER, D.W. AND MURRAY, R.M. A genetic and environmental analysis of a twin family study of alcohol use, anxiety, and depression. Genet. Epidemiol. **1:** 63-79, 1984.

CLONINGER, C.R. Interpretation of intrinsic and extrinsic structural relations by path analysis: Theory and applications to assortative mating. Genet. Res. **36:** 133-145, 1980.

DEVOR, E.J. AND CLONINGER, C.R. Genetics of alcoholism. Ann. Rev. Genet. **23:** 19-36, 1989.

DE ZWART, R., WARNAAR, M.F., VAN PRAAG, B.M.S., DIEKSTRA, R.F.W. AND VAN PRAAG, C.S. Nationaal Scholierenonderzoek, 1992. Den Haag: Nationaal Instituut voor Budgetvoorlichting, 1993.

DIELMAN, T.E., BUTCHART, A.T. AND SHOPE, J.T. Structural equation model tests of patterns of family interaction, peer alcohol use, and intrapersonal predictors of adolescent alcohol use and misuse. J. Drug Educ. **23:** 273-316, 1993.

DUNCAN, T.E., DUNCAN, S.C. AND HOPS, H. The effects of family cohesiveness and peer encouragement on the development of adolescent alcohol use: A cohort-sequential approach to the analysis of longitudinal data. J. Stud. Alcohol **55:** 588-599, 1994.

EAVES, L.J., LAST, K.A., YOUNG, P.A. AND MARTIN, N.G. Model-fitting approaches to the analysis of human behaviour. Heredity **41:** 249-320, 1978.

FALCONER, D.S. Introduction to Quantitative Genetics, 3d Edition, Essex, England: Longman, Scientific & Technical, 1989.

FULKER, D.W. Extensions of the classical twin method. In: BONNÉ-TAMIR, B. (Ed.) Human Genetics, Part A: The Unfolding Genome, New York: Alan R. Liss, Inc., 1982, pp. 395-406.

FULKER, D.W. Genetic and cultural transmission in human behavior. In: WEIR, B.S., EISEN, E.J., GOODMAN, M.M. AND NAMKOONG, G. (Eds.) Proceedings of the Second International Conference on Quantitative Genetics, Sunderland, Mass.: Sinauer Associates, Inc., 1988, pp. 318-340.

GLEIBERMAN, L., HARBURG, E., DIFRANCEISCO, W. AND SCHORK, A. Familial transmission of alcohol use: V. Drinking patterns among spouses, Tecumseh, Michigan. Behav. Genet. **22:** 63-79, 1992.

GOLDSMITH, H.H. A zygosity questionnaire for young twins: A research note. Behav. Genet. **21:** 257-269, 1991.

HALL, R.L., HESSELBROCK, V.M. AND STABENAU, J.R. Familial distribution of alcohol use: I. Assortative mating in the parents of alcoholics. Behav. Genet. **13:** 361-372, 1983.

HEATH, A.C., KENDLER, K.S., EAVES, L.J. AND MARKELL, D. The resolution of cultural and biological inheritance: Informativeness of different relationships. Behav. Genet. **15:** 439-465, 1985.

HEATH, A.C. AND MARTIN, N.G. Teenage alcohol use in the Australian twin register: Genetic and social determinants of starting to drink. Alcsm Clin. Exp. Res. **12:** 735-741, 1988.

HEATH, A.C. AND MARTIN, N.G. Genetic influences on alcohol consumption patterns and problem drinking: Results from the Australian NH&MRC twin panel follow-up survey. Ann. N.Y. Acad. Sci. **708:** 72-85, 1994.

HEATH, A.C., MEYER, J., EAVES, L.J. AND MARTIN, N.G. The inheritance of alcohol consumption patterns in a general population twin sample: I. Multidimensional scaling of quantity/frequency data. J. Stud. Alcohol **52:** 345-352, 1991a.

HEATH, A.C., MEYER, J., JARDINE, R. AND MARTIN, N.G. The inheritance of alcohol consumption patterns in a general population twin sample: II. Determinants of consumption frequency and quantity consumed. J. Stud. Alcohol **52:** 425-433, 1991b.

HOPPER, J.L., WHITE, V.M., MACASKILL, G.T., HILL, D.J. AND CLIFFORD, C.A. Alcohol use, smoking habits and the Junior Eysenck Personality Questionnaire in adolescent Australian twins. Acta Genet. Med. Gemmellol. **41:** 311-324, 1992.

JARDINE, R. AND MARTIN, N.G. Causes of variation in drinking habits in a large twin sample. Acta Genet. Med. Gemellol. **33:** 435-450, 1984.

JÖRESKOG, K.G. AND SÖRBOM, D. New Features in PRELIS 2, Chicago, Ill.: Scientific Software International, Inc., 1993.

KAPRIO, J., ROSE, R.J., ROMANOV, K. AND KOSKENVUO, M. Genetic and environmental determinants of use and abuse of alcohol: The Finnish Twin Cohort studies. In: KALANT, H., KHANNA, J.M. AND ISRAEL, Y. (Eds.) Advances in Biomedical Alcohol Research. Proceedings of the Fifth ISBRA/RSA Congress, Toronto, June 17-22, 1990, New York: Pergamon Press, 1991, pp. 131-136.

KENDLER, K.S., HEATH, A.C., NEALE, M.C., KESSLER, R.C. AND EAVES, L.J. A population-based twin study of alcoholism in women. JAMA **268:** 1877-1882, 1992.

KENDLER, K.S., NEALE, M.C., HEATH, A.C., KESSLER, R.C. AND EAVES, L.J. A twin-family study of alcoholism in women. Amer. J. Psychiat. **151:** 707-715, 1994.

KOOPMANS, J.R., BOOMSMA, D.I., HEATH, A.C. AND VAN DOORNEN, L.J.P. A multivariate genetic anlaysis of sensation seeking. Behav. Genet. **25:** 349-356, 1995.

KOOPMANS, J.R., VAN DOORNEN, L.J.P. AND BOOMSMA, D.I. Smoking and sports participation. In: GOLDBOURT, U., DEFAIRE, U. AND BERG, K. (Eds.) Genetic Factors in Coronary Heart Disease, Dordrecht: Kluwer Academic Publishers, 1994, pp. 217-235.

MAGNUS, P., BERG, K. AND NANCE, W.E. Predicting zygosity in Norwegian twin pairs born 1915-1960. Clin. Genet. **24:** 103-112, 1983.

MERIKANGAS, K.R. The genetic epidemiology of alcoholism. (Editorial) Psychol. Med. **20:** 11-22, 1990.

NEALE, M.C. Statistical Modeling with Mx. Box 710, Medical College of Virginia, Department of Psychiatry, Richmond, Va., 1993.

NEALE, M.C. AND CARDON, L.R. Methodology for Genetic Studies of Twins and Families, NATO ASI Series, Series D: Behavioural and Social Sciences, Vol. 67, Dordrecht: Kluwer Academic Publishers, 1992.

NEALE, M.C. AND MARTIN, N.G. The effects of age, sex, and genotype on self-report drunkenness following a challenge dose of alcohol. Behav. Genet. **19:** 63-78, 1989.

PÉRUSSE, L., LEBLANC, C. AND BOUCHARD, C. Familial resemblance in lifestyle components: Results from the Canada Fitness Survey. Canad. J. Publ. Hlth **79:** 201-205, 1988.

PLOMP, H.N., KUIPERS, H. AND VAN OERS, M.L. Smoking, Alcohol Consumption and the Use of Drugs by Schoolchildren from the Age of 10. Results of the Fourth Dutch National Youth Health Care Survey in 1988/1989, Amsterdam: VU University Press, 1991.

PRESCOTT, C.A., HEWITT, J.K., HEATH, A.C., TRUETT, K.R., NEALE, M.C. AND EAVES, L.J. Environmental and genetic influences on alcohol use in a volunteer sample of older twins. J. Stud. Alcohol **55:** 18-33, 1994.

PRICE, R.A. AND VANDENBERG, S.G. Spouse similarity in American and Swedish couples. Behav. Genet. **10:** 59-71, 1980.

SCHUCKIT, M.A. A clinical model of genetic influences in alcohol dependence. J. Stud. Alcohol **55:** 5-17, 1994.

TAMBS, K. AND VAGLUM, P. Alcohol consumption in parents and offspring: A study of the family correlation structure in a general population. Acta Psychiat. Scand. **82:** 145-151, 1990.

VAN BEIJSTERVELDT, C.E.M., MOLENAAR, P.C.M., DE GEUS, J.C.N. AND BOOMSMA, D.I. Individual differences in P300 amplitude: A genetic study in adolescent twins, submitted for publication.

WEINBERG, N.Z., DIELMAN, T.E., MANDELL, W. AND SHOPE, J.T. Parental drinking and gender factors in the prediction of early adolescent alcohol use. Int. J. Addict. **29:** 89-104, 1994.