The Relative Contribution of Genes and Environment to Alcohol Use in Early Adolescents: Are Similar Factors Related to Initiation of Alcohol Use and Frequency of Drinking?

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Background: The present study assessed the relative contribution of genes and environment to individual differences in initiation of alcohol use and frequency of drinking among early adolescents and examined the extent to which the same genetic and environmental factors influence both individual differences in initiation of alcohol use and frequency of drinking.

Methods: Questionnaire data collected by the Netherlands Twin Register were available for 694 twin pairs aged of 12 to 15 years. Bivariate genetic model fitting analyses were conducted in MX. We modeled the variance of initiation of alcohol use and frequency of drinking as a function of three influences: genetic effects, common environmental effects, and unique environmental effects. Analyses were performed conditional on sex.

Results: Findings indicated that genetic factors were most important for variation in early initiation of alcohol use (83% explained variance in males and 70% in females). There was a small contribution of common environment (2% in males, 19% in females). In contrast, common environmental factors explained most of the variation in frequency of drinking (82% in males and females). In males the association between initiation and frequency was explained by common environmental factors influencing both phenotypes. In females, there was a large contribution of common environmental factors that influenced frequency of drinking only. There was no evidence that different genetic or common environmental factors operated in males and females.

Conclusion: Different factors were involved in individual differences in early initiation of alcohol use and frequency of drinking once adolescents have started to use alcohol.

Key Words: Alcohol Use, Initiation, Adolescence, Genetic Models.

THE INTERNATIONAL REPRESENTATIVE Health Behavior in School-aged Children study of the World Health Organization shows that among those young people who initiated alcohol use before the age of 16 (about 80%), boys reported drinking for the first time at an average age of 12.3 years and girls at an average age of 12 (Currie et al., 2004). As in many other countries, Dutch adolescents start experimenting with alcohol and establish a drinking pattern during this period of early adolescence. However, in the Netherlands, but also in the United Kingdom and Denmark, adolescents start drinking regularly at a younger age than in most other western countries. Among Dutch adolescents 21% of the 13-year olds and 52% of the 15-year olds drink alcohol weekly, while the average in western countries is respectively 12% in 13-year olds and 29% in 15-year olds (Currie et al., 2004). These figures indicate that it is particularly relevant to examine the etiology of initiation and frequency of drinking in early adolescents. In the present study, we assessed the relative contribution of genes and environment to individual differences in initiation of alcohol use and frequency of drinking among early adolescents (12 to 15 years).

Studies on the genetic contribution to the variation in alcohol use in adolescence found that the largest part in the variance of initiation of alcohol use is explained by environmental factors (e.g., Maes et al., 1999; Rose et al., 2001; Stallings et al., 1999), while genetic factors become more important as adolescents grow older and develop more regular drinking patterns (see Hopfer et al., 2003). Most research on alcohol use in adolescents, however, focused on initiation and few studies paid attention to drinking beyond initiation. More importantly, until now little is known about the overlap in etiology of initiation of alcohol use and the adoption of more regular drinking patterns. The present study assessed the relative contribution of genes and environment to individual

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differences in initiation of alcohol use and to frequency of drinking among early adolescents and examined whether the same genetic and environmental factors were related to the two indicators of drinking.

Twin studies are commonly used to examine the relative contribution of genetic and environmental influences on individual differences in behavior. These studies partition the variance of individual differences into (i) the heritability or additive genetic influences (a^2); (ii) common environmental influences which are environmental influences that family members have in common and make them similar to each other (c^2); and (iii) unique environmental influences which are environmental influences that family members experience uniquely and make them different from each other (e^2). Table 1 depicts an overview of univariate and bivariate twins studies on initiation of alcohol use and frequency or quantity of drinking. Twin studies demonstrate that the variation in initiation of alcohol use is moderately heritable, with heritabilities ranging from 0% to 43% (average approximately 30%) (Fowler et al., 2007; Koopmans and Boomsma, 1996; Maes et al., 1999; Pagan et al., 2006; Rhee et al., 2003; Rose et al., 2001; Viken et al., 1999). Common environmental influences explained most of the variation in initiation, with c^2 ranging from 32% to 79% (average approximately 65%). Only Han et al. (1999) reported a relatively high heritability estimate (84%) for males and did not find significant influences of common environment.

In addition to initiation of alcohol use, some twin studies on adolescents' drinking have also focused on other indicators of alcohol consumption, such as frequency of drinking. Viken et al. (1999) showed higher heritabilities (37–47%) and unique environmental influences (27–32%), and smaller common environmental influences (35–22%) for frequency of drinking than for initiation of alcohol use. In contrast, Rhee et al. (2003) found genetic effects for initiation while genes did not contribute to alcohol use. Differences in findings between Viken et al. (1999) and Rhee et al. (2003) might be as a result of differences in the assessment of alcohol use: Viken et al. assessed frequency of drinking with a categorized measure ranging from drinking never to drinking daily, while Rhee et al. defined alcohol use as having six or more drinks during one's lifetime. Moreover, differences in findings might also be explained by age differences between samples and cultural differences between the U.S.A. and Finland.

Viken et al. (1999) and Rhee et al. (2003) examined initiation of alcohol use and frequency of drinking independently. Only two twin studies on adolescents' alcohol use applied a multivariate approach to explore the overlap in factors influencing variation in both initiation and continuation of alcohol use. Pagan et al. (2006) found common environmental factors to play an important role in the variance of initiation (about 60%), and a moderate role in the variance of frequency of use (about 30%), while genes explained a more or less equal part (respectively around 30% and 40%) of the variance in both initiation and frequency of use. Consequently, unique environmental factors explained about 10% of the variance in initiation of alcohol use and around 30% in the variance of frequency of use. Largely the same factors influenced both the variance of initiation and frequency of drinking. Genetic factors influencing the variance of initiation also explained 26% of the variance of frequency of drinking and common environmental factors influencing the variance of initiation also explained 66% of the variance in frequency of drinking. Moreover, Fowler et al. (2007) reported comparable results for the variance of initiation (heritability 26%; common environment 65%). In addition to initiation of alcohol use they measured quantity of drinking during a typical week in the past year. Results indicated that the variance of quantity of drinking was largely predicted by genetic factors (64%) and for a smaller part by unique environmental factors (36%),

Table 1. Overview of Univariate and Bivariate Twin Studies on Initiation of Alcohol Use and Frequency or Quantity of Drinking

					Univariate results		
	Sample	Age	Measure	Sex and age differences	a^2	c^2	Bivariate results
Koopmans and Boomsma (1996)	Dutch	15–17+	Initiation	15–16	0.34	0.58	–
				17+	0.43	0.37	
Han et al. (1999)	US	17–18	Initiation	Male	0.84	–	–
				Female	–	0.76	
Maes et al. (1999)	US	13–16	Initiation	–	–	0.71	–
Viken et al. (1999)	Finnish	16–17	Initiation	16	0.14	0.79	–
				17	0.26	0.67	
			Frequency	16	0.37	0.35	
				17	0.47	0.22	
Rose et al. (2001)	Finnish	14	Initiation	Male	0.18	0.76	–
				Female	–	0.76	
Rhee et al. (2003)	US	12–19	Initiation use[a]	–	0.39	0.32	–
				–	–	0.45	
Pagan et al. (2006)	Finnish	17	Initiation	–	0.30	0.60	*a* 26% overlap
			Frequency	–	0.40	0.30	*c* 66% overlap
Fowler et al. (2007)	UK	11–19	Initiation	–	0.26	0.65	In total 23%
			Quantity	–	0.64	–	Overlap

[a]Alcohol use was defined as having six or more drinks during one's lifetime.

while common environmental factors did not contribute to the variance of quantity of drinking. The variance of quantity of drinking was partly (23%) because of factors also affecting the initiation of alcohol use. In sum, both studies showed comparable results for initiation of alcohol use, but the results for continuation of use diverged.

Previous twin studies which tested for differences in genetic and environmental influences between males and females in adolescent samples revealed contradictory findings. Maes et al. (1999), Rhee et al. (2003) and Viken et al. (1999) did not find sex differences in the magnitude of genetic and environmental effects on the variance of initiation and alcohol use. In contrast, Han et al. (1999) reported higher heritabilities and smaller environmental influences on initiation of alcohol use in males than in females. Rose et al. (2001) found that heritabilities of initiation were higher for females than for males. Common environmental influences were equally important among males and females, while unique environmental factors were more important in males. Results with respect to qualitative sex differences (i.e., do the same or different factors operate in males and females) have also provided mixed results. While some twin studies on adolescent alcohol use reported that mainly the same genetic and common environmental factors operate in males as in females (Maes et al., 1999; Rhee et al., 2003; Rose et al., 2001), others indicated that partially different factors operate (Koopmans and Boomsma, 1996; Viken et al., 1999).

In the current study, we examined the relative contribution of genes and environment to individual differences in initiation of alcohol use and frequency of drinking among early adolescents (12 to 15 years). This relatively young homogeneous group of adolescents was examined because the initiation of alcohol use is typical for early adolescents and should preferably be assessed in this age period. We examined whether the relative contribution of genes and environment differed between males and females and whether the same factors operated in males and females. Further, we tested the overlap in factors related to the variance of initiation of alcohol use and frequency of drinking.

METHODS

Participants

Data reported in this study are part of an ongoing longitudinal survey study of the Netherlands Twin Register. Since 1991, adolescent and young adult twins and their family take part in survey studies on health, lifestyle and personality roughly every 2 years. Twins were asked to participate every 2 years (1991, 1993, 1995, 1997, 2000, 2002, and 2004); parents in 1991, 1993, 1995, 2002, and 2004 and siblings from 1995 onwards. Some individuals participated only once, while others participated several times. Information about sample and data collection is described in detail in Boomsma et al. (2002, 2006).

In the present study, we used data from the 1993, 1995, 1997, and 2000 surveys to create a large cross-sectional dataset. We selected all twins who were between 12 and 15 years of age in 1993, 1995, 1997, or in 2000. At this age, adolescents are experimenting with alcohol and may start to drink regularly, although in the Netherlands young people are legally not allowed to drink alcohol before the age of 16. Initially, we used data on alcohol use of twin pairs from the 1993 wave, but if data of both twins were not available at this wave we used data of the 1995 wave. This was continued until we used data of all five measurement waves as a possible source to construct one cross-sectional dataset. In total, the sample consisted of 694 twin pairs within the age range of 12 to 15 years, of these pairs 125 were monozygotic males (MZM), 89 pairs were dizygotic males (DZM), 183 pairs were monozygotic females (MZF), 106 pairs were dizygotic females (DZF), and 191 pairs were dizygotic opposite sex (DOS). Zygosity was based on DNA polymorphisms, or if not available, on survey questions on the physical similarity of the twins and confusion in identifying twins by family members, friends and strangers. Agreement between zygosity based on DNA results polymorphisms and zygosity based on questionnaires is 97% (Willemsen et al., 2005).

Initiation of Alcohol Use and Frequency of Drinking

To assess initiation of alcohol use the twins were asked at what age they first tried alcohol. Response categories were: (1) "never", (2) "11 or younger", (3) "12" - (8) "17", and (9) "18 or older". When examining the variation in initiation and frequency of use in multivariate, or so-called multiple-stage genetic models that allowed overlap of risk factors for both indicators of drinking, initiation should be defined as a multiple category trait (e.g., never vs. early onset vs. later onset) instead of a binary variable (Heath et al., 2002; Pagan et al., 2006) or else estimates of genetic (or environmental) correlations between initiation and continuation of use might be biased (Heath et al., 2002). We therefore categorized initiation of alcohol use as follows: (1) "never initiated", (2) "at age 13 or after", and (3) "before age 13". This categorization was used because our participants were 12 to 15-year old and initiation before age 12 was less prevalent.

In the surveys, twins were also asked: "How often do you drink alcohol?". Twins could respond to this question on one of eight categories: (1) "I do not drink alcohol", (2) "once a year or less", (3) "a few times a year", (4) "about once a month", (5) "a few times a month", (6) "once a week", (7) "a few times a week", and (8) "daily" (Poelen et al., 2005). The frequency of alcohol use was recoded to : (1) "once a year or less", (2) "a few times a year", (3) "about once a month", (4) "a few times a month", (5) "once a week or more". Categories 6, 7 and 8 of the original measure were summarized into one category (new category 5). If participants did not drink alcohol, in other words, if they scored (1) "never initiated" on the initiation item, they consequently had a missing value on the frequency of drinking scale.

Strategy of Analyses

Genetic model fitting was conducted with the software package MX (Neale et al., 2003). We first calculated the polychoric correlations for twin pairs in all zygosity groups (MZM, DZM, MZF, DZF, and DOS). Because initiation of alcohol use and frequency of drinking are categorical a liability model was used (Falconer and Mackay, 1996). A liability model assumes that a categorical trait reflects an underlying (latent) liability with a normal distribution (with unit variance) and thresholds that divide the sample into for example noninitiators, late initiators, and early initiators. The thresholds are obtained from the prevalences and can be interpreted as a z-value. Polychoric correlations represent the resemblance of twins on the liability distribution. A comparison of MZ and DZ correlations provides insight into the relative contribution of genes and environment to the variation in initiation of alcohol use and frequency of drinking. A higher correlation among MZ twins than among DZ twins indicates genetic influences, but if the correlations among MZ and DZ twins are of similar magnitude, environmental factors, not genetic factors are the main determinants of individual differences in drinking behavior.

In order to examine the relative contribution of genes and environment to individual differences in initiation of alcohol use and

frequency of drinking we used a structural equation modeling approach. We modeled the variance of drinking as a result of three latent factors: (i) additive genetic effects (A); (ii) common environmental effects (C); and (iii) unique environmental effects (E) (Fig. 1). Unique environmental effects also included measurement error. The estimation of the contributions of A, C, and E (i.e., a, c, and e) was based on the differences in genetic relatedness of MZ and DZ twins. In genetic model fitting the correlation between the latent A factors for MZ twins (r_A) was fixed to 1, while the correlation between A factors for DZ twins was fixed to 0.5. The correlation between the common environmental (r_C) latent factors was fixed to 1 and the unique environmental (E) latent factors were not correlated, for both MZ and DZ twins. Figure 1 presents the structural model used in our analyses. We tested whether the genetic and environmental factors related to the variation in initiation of alcohol use were also related to the variation in frequency of drinking and/or whether genetic and environmental factors were specific for the variation in initiation of alcohol use and frequency of drinking. Comparable types of multivariate modeling strategies are described by Heath et al. (2002) and Pagan et al. (2006), our modeling procedure was conform these studies.

The bivariate model tested in this study implies structural missing data for frequency of alcohol use in those twins who never initiated alcohol use. Related to these structural missing data, Heath et al. (2002) indicated that initiation should be defined using multiple categories (e.g., never vs. early onset vs. later onset) instead of two categories, to have enough information to estimate polychoric correlations between initiation of use (3 categories) and frequency of drinking (5 categories) (Heath et al., 2002). The approach outlined by Heath is based on the fact that the frequency of drinking data are Missing at Random (MAR) as the probability of missingness is determined by scores on initiation of alcohol use. Little and Rubin (2002) have shown that if missing data are MAR, Full Information Maximum Likelihood procedures provide unbiased parameter estimates and are recommended (Heath et al., 2002). Therefore, we used the Full Information Maximum Likelihood estimator to estimate the parameters in our models.

Models were fit directly to the raw data using MX. We fitted the complete model as depicted in Fig. 1 and tested whether model parameters for males and females were equal, by comparing the fit of a model in which all parameter estimates are allowed to be different in males and females with the fit of a model in which all parameter estimates are constrained to be equal in males and females. In addition to these tests for quantitative sex differences (i.e., differences in the magnitude of the parameter estimates), we examined qualitative sex differences (i.e., do different factors operate in males and females). Qualitative sex differences were investigated by comparing the fit of a model which freely estimates the genetic correlation in DOS twin pairs with the fit of a model which constrains the genetic correlation at 0.5, as in same-sex DZ twin pairs. A decreased genetic correlation (< 0.5) in DOS twin pairs indicates that different genetic factors are related to initiation of alcohol use and frequency of drinking in males and females. In addition, we subsequently constrained the estimates for a, c, or e parameter specific for the variance of initiation or frequency of drinking at zero or one of the shared a, c, or e parameters from the baseline model. The significance of the constrained parameters are tested by examining the change in -2 log likelihood between the baseline model and the sub model; this difference is evaluated using a chi-square distribution. A significant decrease in chi-square in the constrained model compared to the baseline model indicates a deterioration of the model fit if this particular parameter is not modeled, therefore this parameter should be included in the model.

RESULTS

Descriptives

As can be seen from Table 2 the majority of our participants initiated alcohol use. Females less often initiated than males [χ^2 (2) = 8.25; p = 0.016]. Around half of the participants who initiated alcohol use, started at age 13 or after, and around half started before the age of 13. A relatively small part of the adolescents who had initiated, drank alcohol a few times a month or more. The distribution of frequency of drinking among adolescents who had initiated was not significantly different in males and females [χ^2 (7) = 7.98; p = 0.092].

Table 3 depicts the correlations of initiation of alcohol use and frequency of drinking within twin pairs and between initiation and frequency of drinking of twins 1 and 2 for the five zygosity groups. For initiation of alcohol use, MZ twin correlations were higher than DZ twin correlations, indicating that genes played a role in the variance of initiation of alcohol use. For frequency of use, the correlations indicated common environmental influences on the variation, because the

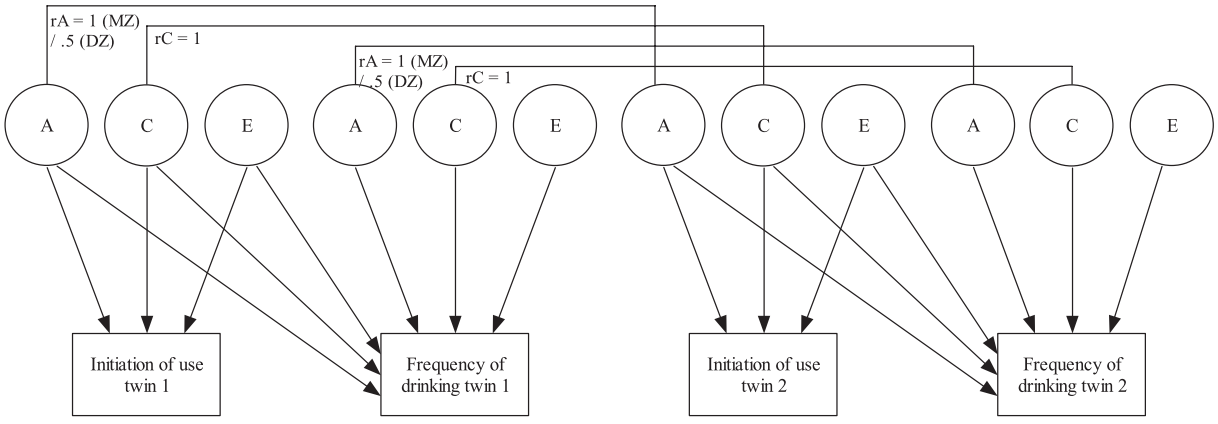

Fig. 1. Bivariate model of alcohol initiation and frequency of drinking. MZ, monozygotic; DZ, dizygotic. The variance of the observed variables initiation of alcohol use and frequency of drinking is caused by three latent factors: (i) the additive genetic factor (A); (ii) the common environmental factor (C); and (iii) unique environmental factor (E). The correlation between the genetic (r_A) latent variable in twins was fixed at 1 for MZ twins and r_A was fixed at 0.5 for DZ twins. The correlation between the common environmental (r_C) latent factors was fixed at 1 for both MZ and DZ twin pairs.

Table 2. Percentage of Initiation of Alcohol Use and Frequency of Drinking Among Twins Who Have Initiated Alcohol Use by Sex

	Males	Females
Initiation of alcohol use	*n* = 561	*n* = 727
Never	32.3	40.0
At age 13 or after	36.7	32.9
Before age 13	31.0	27.1
Frequency of drinking	*n* = 379	*n* = 433
Once a year or less	25.6	31.9
A few times a year	35.9	37.2
About once a month	10.6	9.5
A few times a month	15.3	9.9
Once a week or more	12.7	11.5

Table 3. Number of Twin Pairs in Each Group and Twin Correlations for Initiation of Alcohol Use and Frequency of Drinking

	MZM *n* = 125	DZM *n* = 89	MZF *n* = 183	DZF *n* = 106	DOS *n* = 191
Initiation twin 1–initiation twin 2	0.84	0.60	0.88	0.66	0.56
Frequency twin 1–frequency twin 2	0.85	0.77	0.84	0.79	0.36
Initiation twin 1–frequency twin 1	0.24	0.31	0.29	−0.06	−0.28
Initiation twin 2–frequency twin 2	0.05	0.11	0.31	−0.14	0.14
Initiation twin 1–frequency twin 2	0.22	0.16	0.33	0.08	0.02
Initiation twin 2–frequency twin 1	0.13	0.28	0.24	−0.09	0.15

MZM, monozygotic males; DZM, dizygotic males; MZF, monozygotic females; DZF, dizygotic females; DOS, dizygotic opposite-sex twins.

correlations were relatively high and the MZ and DZ correlations barely differed. The twin correlations for frequency of drinking for DOS twins were relatively low compared to the correlations in same sex twin pairs, suggesting qualitative sex differences in frequency of drinking. The fact that the same picture arises for MZ and DZ twins indicates that the covariance between initiation and frequency of drinking is not likely to be explained by genetic factors. Correlations between initiation of alcohol use and frequency of drinking within one person and between co-twins were relatively low. Previous research also indicated low correlations between initiation of alcohol use and frequency of drinking within one person (Pearson's correlation around 0.08) (Engels et al., 1997).

Genetic Analyses

We examined the bivariate genetic model as shown in Fig. 1. Table 4 shows the model fitting results, with the best fitting model in bold. The saturated model (Table 4, model 1) does not place any constraints on the covariance structure of MZ and DZ twins. The full genetic model (Table 4, model 2) allows for qualitative and quantitative sex differences. This model provides a good fit compared to the saturated model, indicating that a genetic model fits the data well. Next, we examined whether model parameters for males and females were different (quantitative sex differences) and whether different genetic factors were related to the variance of initiation of alcohol use and frequency of drinking in males and females (qualitative sex differences). Dropping qualitative sex differences from the model (Table 4 model 3) did not cause a significant change in the model fit, implying that the same genetic factors operated in males and females for both initiation and frequency of drinking. Dropping quantitative sex differences from the model (Table 4 model 4) did cause n decrease in the model fit, but this decrease was not significant. We also dropped quantitative and qualitative sex differences simultaneously from the model (Table 4 model 5), this did result in a significant decrease of the model fit. Thus, in subsequent models sex-specific parameters were estimated. In addition we tested respectively whether the factor loading of the shared genetic factor, the shared common environmental factor and the shared unique environmental factor on initiation and frequency of drinking could be dropped from the model (Table 4 models 6, 7, and 8). Model fitting results showed that only the elimination of the loading of the shared common environmental factor on frequency of drinking caused a significant decrease in model fit, the other parameters could be eliminated without a significant decrease of the model fit. Thus, in subsequent models, the shared genetic and unique environmental parameters were not included. Finally, as the twin correlations suggested that there is no influence of genes on the variation in frequency of drinking, we dropped the genetic

Table 4. Bivariate Model Fitting Results for Initiation of Alcohol Use and Frequency of Drinking

	−2LL	*n* par	Versus	$\Delta\chi^2$ (df)	*p*-value
1. Saturated model	4630.15	90	–	–	–
2. ACE model with quantitative and qualitative[a] sex diff	4644.48	76	1	14.33 (14)	0.43
3. ACE model, qualitative sex diff dropped	4644.48	74	2	0.00 (2)	>.99
4. ACE model, quantitative sex diff dropped	4654.38	69	2	9.90 (7)	0.19
5. ACE model quantitative and qualitative sex diff dropped	4661.86	67	2	17.38 (9)	0.04
6. ACE model, quantitative sex diff, shared A dropped	4646.67	72	3	2.20 (2)	0.33
7. ACE model, quantitative sex diff, shared C dropped	4652.65	72	3	8.17 (2)	0.02
8. ACE model, quantitative sex diff, shared E dropped	4644.93	72	3	0.45 (2)	0.80
9. ACE model, quantitative sex diff, shared A and shared E dropped	4648.89	70	3	4.41 (4)	0.35
10. ACE model initiation, CE model frequency, shared A and shared E dropped	**4652.94**	**68**	**9**	**3.95 (2)**	**0.14**

A, additive genetic variance component; C, common environmental variance component; E, unique environmental variance component; −2LL, −2 loglikelihood; *n* par, number of parameters; versus indicates to which model the submodel is compared to.

[a]Genetic correlation in dizygotic opposite-sex twins is estimated at the boundary of 0.5. Best fitting model in bold.

factor specific to the variation in frequency of drinking from the model (Table 4 model 10). The resulting model appeared to be the best fitting model.

Table 5 depicts the parameter estimates and 95% confidence intervals of the best fitting model. For males, the common environmental factor loading specific for the variance of initiation of alcohol use and the common environmental factor loading specific for the variance of frequency of drinking were not significant. For females the common environmental factor loading on both initiation and frequency was not significant (confidence intervals of these factor loadings included zero). These insignificant factor loadings were retained in the model. As constraining these factor loadings to zero would result in a cross-twin correlation of zero for frequency of drinking and in a cross-twin cross-trait correlation of zero for initiation and frequency of drinking in DOS twins. Table 5 also presents the percentages of explained variance for initiation of alcohol use and frequency of drinking. Among males 83% of the variance of initiation of alcohol use was explained by additive genetic factors, 15% was explained by unique environmental factors. In females, the largest part of the variance of initiation of alcohol use was explained by additive genetic factors (70%), 19% was explained by common environmental factors and 11% by unique environmental factors.

In males, the same common environmental factor that explained a small part of the variance of initiation of alcohol use also explained the largest part (81%) of the variance of frequency of drinking. Another 18% of the variance in frequency of drinking in males was explained by unique environmental factors specific to frequency of drinking. In females, the overlapping common environmental factor explained a smaller part of the variance (13%) in frequency of drinking than in males. However, the variance of frequency of drinking in females was also for the largest part explained by common environment as specific common environmental factors explained 69% of the variance. The remaining part of the variance in frequency of drinking in females was explained by unique environmental factors (18%).

DISCUSSION

The present study assessed the relative contribution of genes and environment to individual differences in initiation of alcohol use and frequency of drinking among early adolescents (12 to 15-y old). The modeling procedure we used allowed a test of whether and to what degree the same factors were related to individual differences in initiation of alcohol use and frequency of drinking. Results showed that genetic factors were most important in explaining the variance of initiation of alcohol use, as they explained 83% of the variance in males and 70% of the variance in females, and that a much smaller part of the variance was explained by common environmental factors (2% in males and 19% in females). In contrast, common environmental factors explained most of the variance of frequency of drinking (82% of the variance in both males and females), while genetic factors were not involved in the explanation of the variance of frequency of drinking. In males, these factors almost completely overlapped with the factors explaining variation in initiation of alcohol use, while in females variation in frequency of drinking was mainly predicted by common environmental factors specific to frequency of drinking. Our analyses showed that only common environmental factors influencing variation in initiation of alcohol use overlapped with common environmental factors influencing variation in frequency of drinking, while genetic and unique environmental factors did not overlap. Our findings further indicated that parameter estimates were different for males and females, but that the same genetic and common environmental factors operate in males and females.

Our finding that genetic factors are important in explaining the variance of initiation of alcohol use is partly in contrast with previous studies which showed that the variance of initiation of alcohol use was moderately heritable and largely explained by common environmental influences (Fowler et al., 2007; Koopmans and Boomsma, 1996; Maes et al., 1999; Pagan et al., 2006; Rhee et al., 2003; Rose et al., 2001; Viken et al., 1999). The difference between our findings and those of these previous studies may be explained by age

Table 5. Parameter Estimates of the Best-Fitting Model and 95% Confidence Intervals and Percentages of Explained Variance for Specific and Shared A, C, and E Factors Loading on Initiation of Alcohol Use and Frequency of Drinking

	A		C		E	
	a (CI)	%	*c* (CI)	%	*e* (C.I.)	%
Males						
Specific for Initiation	0.91 (0.84–0.95)	83	0.15 (−0.08–0.37)	2	0.38 (0.29–0.48)	15
Specific for Frequency	–	–	0.10 (−0.64–0.55)	1	0.43 (0.34–0.53)	18
Shared factors	–	–	0.90 (0.71–0.94)	81	–	–
Females						
Specific for Initiation	0.84 (0.69–0.93)	70	0.44 (0.18–0.64)	19	0.33 (0.26–0.41)	11
Specific for Frequency	–	–	0.83 (0.21–0.93)	69	0.42 (0.34–0.52)	18
Shared factors	–	–	0.35 (−0.12–0.89)	13	–	–

The percentages of explained variance were obtained by squaring the standardized loadings.
A, additive genetic influences; C, common environmental influences; E, unique environmental influences.

differences between samples. Most previous studies examined mainly older adolescents or examined adolescent samples that were less homogeneous in age (i.e., samples included also older adolescents), while we examined early adolescents as the experimentation with drinking is typical for early adolescents and should preferably be assessed in this age period. We defined early initiation as starting to use alcohol before the age of 13. In contrast to other previous studies, our definition of initiation of alcohol use apparently discriminated between the more problematic (and genetically induced) early adolescent onset and less problematic behaviors (later onset and abstinence until at least 16).

Our finding that common environmental factors mainly affected the variance of frequency of drinking in 12 to 15-year olds is in line with the findings of Rhee et al. (2003), but in contrast with others (e.g., Pagan et al., 2006; Viken et al., 1999). Common environmental factors are those influences from the environment that twins have in common and make them similar to each other. During early adolescence, twins are likely to spend a lot of time with their families and have shared experiences at school and with friends. Therefore, it is likely that shared familial influences and peer influences were incorporated in the common environment. Dutch figures show that about half of the adolescents report to drink with their parents at age 12 to 13 years. This percentage remains rather stable during adolescence (NIGZ, 2006). Many Dutch parents allow adolescents to drink alcohol at home and it is likely that they provide the same rules regarding drinking for their twins (e.g., Van Der Vorst et al., 2005). Moreover, peer influences are also considered to be important factors in adolescents' alcohol use (e.g., Andrews et al., 2002; Petraitis et al., 1995; Urberg et al., 1997). Peer influence will operate as common environmental influence when twins share peers or have similar experiences with peers. Indeed, in our sample 24% indicated to share all friends and 44% of the twins shared at least part of their friends. Our findings indicated that in males the same common environmental factors explained variation in initiation and frequency of drinking. In females, different factors from the common environment mainly explained variation in initiation and frequency of drinking. This finding implies that for a part different common environmental factors explain the variance in frequency of drinking in males and females. We can only speculate about what factors in the common environment are different for males and females. The sex difference might have it's origin in differences in pubertal development in early adolescence (i.e., girls mature faster) (Dick et al., 2000). Adolescents tend to form an identity independent from their parents and foster tighter bonds with their peers during adolescence. Girls do this earlier than boys, because they mature earlier, and girls at that age may therefore be more influenced by their peers than boys. Studies on this topic showed that early matured girls are likely to affiliate with older and deviant peers (Caspi et al., 1993). Previous analyses of our data indeed showed a trend being indicative that friends' drinking was a greater risk factor for drinking in females compared to males (Scholte et al., 2008).

Unique environmental factors explained only a relatively small part in the variance of both initiation of alcohol use and frequency of drinking. This probably reflects tendencies that during early adolescence twins still spend a lot of time with their families and have shared experiences at school and with friends. It also implies a small contribution of measurement error, which shows that we use reliable indicators of initiation and frequency of drinking.

Estimates of explained variances of initiation of alcohol and frequency of drinking considerably differ between different twin studies. Differences in estimates of genetic and environmental influences are likely to be explained by cultural differences between samples and the definition or measurement of alcohol use (see also Table 1). Our study was the first that showed the genes were most relevant in explaining the variance in early initiation of alcohol use. Therefore, we encourage other scholars with twin data also to examine early adolescent onset of alcohol use to determine whether our findings can be confirmed in other samples.

A few limitations of our study need to be mentioned. We obtained the largest possible sample size by using several measurement waves of longitudinal survey data to create a cross-sectional dataset. While constructing this data we assumed that it was not likely that cohort effects in genetic or environmental influences on drinking occurred in this period of 7 years. This cross-sectional approach does not allow us to draw conclusions on predictors of development of individual drinking patterns. Furthermore, while interpreting our results it should be noted that initiation of alcohol use was assessed by asking participants at what age they first tried alcohol. This makes it conceivable that initiation is in fact experimentation (which may lead to initiation). Table 2 shows that 25.6% of the boys and 31.9% of the girls who indicated that they have tried alcohol drink only once a year or less, these adolescents probably only have experimented and have not actually initiated (yet). The other adolescents who indicated that they have tried alcohol drink at least a few times a year and have actually initiated alcohol use. Moreover, it should be noted that we assessed frequency of drinking and not quantity of drinking. Descriptive statistics of quantity of drinking show that the vast majority of 12 to 15-year-old adolescents drink <1 drink a week (this was the lowest category on this measure; see also Poelen et al., 2005). So most of the participants in this age category drink in relative low doses. The frequency of drinking measure shows more variance in this age group (see Table 2) and is therefore more suitable for the analyses we applied in this study. However, frequency of drinking and quantity of drinking are significantly correlated [r (n = 539) = 0.57, $p < 0.001$]. When children have their first drinks within a family context at special occasions like New Year's Eve, or within a deviant peer context where they drink with (older) friends, this might have different meanings and consequences, with the latter indicative of a deviant-prone orientation (Moffitt, 1993). Future research should reveal whether also genetic and environmental effects on alcohol initiation differ for these groups. It is also relevant to pay

attention to the fact that in the Netherlands and also other northern European countries such as the United Kingdom and Denmark adolescents start drinking regularly at a younger age than in most other western countries such as the United States. Young people in the Netherlands may start drinking regularly at a relatively young age, because of permissive attitudes of parents towards drinking (Van Der Vorst et al., 2005) and the cultural embedding of alcohol use in the Netherlands (Engels and Knibbe, 2000). Moreover, twin studies have shown that estimates of genetic and environmental influences depend upon the age of onset of regular drinking (e.g., Hopfer et al., 2003; Rose and Dick, 2005). This implies that the relative high drinking levels in the Netherlands and some other Northern European countries might affect the generalizability of our findings. Therefore, we suggest for future research to test models with A, C, and E influences on initiation of alcohol use and frequency of drinking in countries with similar drinking cultures to the Dutch and in countries with different drinking cultures than the Dutch. In addition, the prevalence data from a study by Currie et al. (2004) presented in the introduction section seem to be higher than our prevalence rates of frequency of drinking (Table 2). However, it should be noted that differences in age between samples and differences in measurements cause this discrepancy in prevalence rates.

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