Co-Twin Dependence Modifies Heritability of Abstinence and Alcohol Use: A Population-Based Study of Finnish Twins

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The role of co-twin dependence (twins' closeness or reliance on the co-twin) was examined as a moderator of genetic and environmental influences on alcohol use in adolescence and early adulthood in a large longitudinal population-based study of Finnish twins (FinnTwin16). The associations between co-twin dependence and alcohol use were studied first at an individual level in adolescence (n = 3362) and early adulthood (n = 2912). Then, maximum likelihood models were fit to the two waves of data from same-sex twin pairs to assess the differences and changes in genetic and environmental influences on alcohol use (abstinence, drinking frequency, intoxication frequency); N = 1342 pairs in adolescence, and N = 1078 pairs in early adulthood. Overall, no significant associations were found between co-twin dependence and individual alcohol use. However, co-twin dependence importantly modulated genetic effects on drinking habits, especially in adolescence, but also in early adulthood. Co-twin–dependent twins reported greater similarity in their alcohol-related behavior across all alcohol-use measures at both time points, and the role of genes and environments varied according to co-twin dependence. Shared environmental factors explained most of the variation in drinking among co-twin–dependent twins in adolescence and contributed to drinking to intoxication during early adulthood. In contrast, among co-twin–independent twin pairs, genetic variance contributed significantly to all alcohol-use measures at both time-points. An interdependent sibling relationship is an important modifier of drinking habits, and it appears to reduce the impact of inherited liabilities on alcohol-related behavior especially in adolescence.

Several studies have revealed that early initiation of alcohol and other substances is a risk factor for escalated later use (Hawkins et al., 1992), as well as a predictive factor for subsequent development of substance-related problems (Anthony & Petronis, 1995; Grant & Dawson, 1997). After initiation, frequency and quantity of drinking typically escalate relatively rapidly from mid to late adolescence (Lintonen et al., 2000). A similar trend of increasing frequency of drinking and increasing frequency of drinking to intoxication was reported in an earlier analysis of the 'FinnTwin16' sample (Viken et al., 1999). Because adolescence is a critical period for the development of behavioral problems, including the abuse of alcohol, the causes and consequences of adolescent alcohol use have been studied intensively.

Twin and adoption studies provide evidence indicating genetic influences on alcohol use and alcohol-related behavior (Hopfer et al., 2003). Similarly, genetic influences on the genesis of alcohol abuse (Walters, 2002) have also been shown. However, some uncertainties remain regarding the relative magnitude of genetic and environmental influences on alcohol use across males and females and across variation in the severity of alcohol use (McGue et al., 1992; Pickens et al., 1991). And there is strong evidence that the magnitude of genetic and shared environmental influences is moderated by age. Twin studies have consistently suggested that during adolescence, shared environmental influences on initiation and alcohol use are more prominent than genetic influences (Han et al., 1999; Rhee et al., 2003; Rose, Dick, Viken, Pulkkinen, et al., 2001; Rose et al., 1999), but the situation is likely to reverse with increasing age, as individuals move into adulthood (Koopmans & Boomsma, 1996; Koopmans et al., 1997; Rose, Dick, Viken, & Kaprio, 2001; Viken et al., 1999). From young adulthood to middle age, genetic and environmental influences are found to be relatively consistent (Kaprio et al., 1987).

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Along with age and gender, specific environmental contexts have been found to moderate the expression of genetic influences. A Dutch study (Koopmans et al., 1999) suggested that, for nonreligious twins, genetic factors explained approximately 40% of the variance of initiation into drinking, but for religious twins, the genetic influences fell to a 0% to 25% range. In a study of Finnish twins (Rose et al., 1999), the magnitude of genetic influences was moderated by regional residency, and the effect was found to be relatively consistent from mid to late adolescence (Rose, Dick, Viken, & Kaprio, 2001). Genetic effects on drinking frequency were more prominent in twins residing in urban areas, while in rural areas, drinking habits were influenced more by shared environmental effects. Further characterization of the relevant features of urban versus rural experience came from a more detailed study of the sociodemographic characteristics of the community of residence such as migration rates, alcohol sales and age structure (Dick et al., 2001).

Heritability estimates of alcohol-related behaviors have been found to vary according to the measure as well. Initiation of alcohol use (ever/never use alcohol) seems to have a small genetic influence (Legrand et al., 1999; Rose et al., 1999), whereas genetic influences on drinking frequency seem to be more substantial (Viken et al., 1999). Maes et al. (1999) reported that drinking with parental permission was not heritable, whereas 72% of the variation in alcohol use without parental permission was explained by genetic influences. Rose et al. (2003) assessed the contribution of familial and nonfamilial environments to children's behavioral experiences on drinking and smoking in early adolescence. Alcohol use in general was negligibly heritable (8% of the variance) with most of the variance attributed to familial (37%) and extrafamilial (26%; school environments, neighborhoods or communities) effects shared by siblings. In contrast, consistent with the report by Maes et al. (1999), alcohol use without parental supervision was found to have a substantial genetic contribution. For unsupervised alcohol use, most of the variance was due to genetic (30%) and shared nonfamilial environmental (32%) influences, whereas the importance of shared familial influences (21%) was less significant.

The influence of parents, siblings and peers on adolescent alcohol use is well established (e.g., Ary et al., 1993). Several studies have suggested that the most important contributor to adolescent alcohol use is the influence of peer groups (Crawford & Novak, 2002; Koopmans & Boomsma, 1996; Zhang et al., 1997), and sibling alcohol use has been found to have a stronger influence than the alcohol use of parents (Duncan et al., 1994; McGue et al., 1996; Windle, 2000). Studies among adult twins have suggested that frequency of co-twin contact is associated with increased similarity in drinking patterns, especially among monozygotic (MZ) twins (Kaprio et al., 1987; Kaprio et al., 1990; Kendler et al., 1992; Prescott et al., 1994; Rose et al., 1990). However, some studies have found the effect to be relatively weak (Lykken et al., 1990), while other studies have failed to find any effect of co-twin contact on twins' similarity in alcohol use (Heath et al., 1989; LaBuda et al., 1997; Reiss, Cederblad, Pedersen, Lichtenstein, Elthammar, et al., 2001).

Some studies have suggested that phenotypic differences among individuals could be associated with the quality of their interpersonal and intimate relationships, that is, genes require certain interactional processes in order to become fully expressed, or in contrast, to not be expressed (e.g., evocative model of Reiss, Cederblad, Pedersen, Lichtenstein, Hansson, et al., 2001); but relatively little is known about these processes and how they change over individual development. In a study of adult female twins, Heath et al. (1989) found that marital status significantly modified genetic effects on drinking habits. In different age groups genetic effects accounted for 31% to 59% of the variance in alcohol use for married twins, whereas for unmarried twins the variance of genetic effects ranged from 60% to 76%, indicating that interpersonal relationships, in this case a relationship to a spouse, reduced the impact of dispositional liability.

In this study, we examine the influence of co-twin dependence on the drinking habits of twins from adolescence to early adulthood. This is the first report, to our knowledge, in which the influence of co-twin relationships, assessed in a population-based sample of cohabiting adolescent twins, was followed up into the twins' early adulthood. Three questions were addressed: (1) Do co-twin–dependent twins differ in their alcohol use from co-twin–independent twins?; (2) Are co-twin–dependent twin pairs more similar in their alcohol-related behavior than co-twin–independent twins?; and (3) Do genetic and shared environmental factors contribute differently to variance in alcohol-related behaviors in co-twin –dependent and co-twin–independent twins? And how does the variance of genetic and environmental effects change from adolescence to early adulthood in these twins differing in their dependence? Our hypothesis is that close and intimate relationships between twin siblings might mediate the genetic propensities of alcohol-related behavior, especially in adolescence. In the present study 'co-twin dependence' might be considered similar to features like co-twin attachment and reliance on a co-twin.

Materials and Methods

Sample

The present report is based on a Finnish Twin Cohort Study, FinnTwin16, a population-based study of five consecutive birth cohorts of Finnish twins, born between the years 1975 and 1979. The birth cohorts were identified from the Central Population Registry of Finland. Baseline assessments were collected through mailed questionnaires sequentially adminis-

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tered during the years 1991 to 1995, within two months of the twins' 16th birthdays. The 5-year cohort yielded replies from 5563 twin individuals, with a male response rate of 81% (n = 2682) and for females 93% (n = 2881). All respondent twins were sent follow-up questionnaires at the ages of 17 years, 18.5 years, and in a third follow-up in early adulthood, at ages 22 to 27 years. The baseline questionnaire included a survey of health habits (including substance use) and attitudes, a symptom checklist and questions of relationships with parents, peers and co-twin. Response rates of 80% to 90% were achieved across all different waves of the study (Kaprio et al., 2002; Rose, Dick, Viken, & Kaprio, 2001).

In the analyses reported here, all twins of known zygosity (N = 5268) were included. Zygosity was determined from validated questionnaire responses of twins' similarity, including questions of genetically influenced characteristics such as eye color, hair color, hair texture as well as questions on how often parents, peers, teachers and acquaintances confused the twins. Questionnaire items concerning zygosity were completed by the twins and by their parents. Previous studies have shown that this kind of method of zygosity determination is highly accurate, giving more than a 95% accuracy rate when compared with blood typing analysis (Rietveld et al., 2000; Sarna et al., 1978).

A further requirement was that individual twins had no missing data on items assessing twin relationship and alcohol use. Twins were excluded for whom we did not have unequivocal data on co-twin dependence at the age of 16 years and those who had omitted questions on drinking, drinking frequency or intoxication frequency at either of the two different time points. Opposite-sex (OS) twin pairs were also excluded from the analyses, because co-twins in OS pairs have quite different interpersonal relationships than do same-sex twin pairs, and they are much less likely to select one another or a common classmate as a best friend in early adolescence (Rose, 2002). At age 16, analyses included 3362 twin individuals, and at ages 22 to 27 years, 2912 twin individuals were included. The model-fitting analyses are based on concordantly co-twin–dependent and concordantly co-twin–independent same-sex (SS) twin pairs. At age 16, the model-fitting analyses consist of 1342 complete twin pairs (2684 twin individuals) and in the follow-up in early adulthood, 1078 complete twin pairs (2156 twin individuals).

Measures

Co-Twin Dependence

The measure of co-twin dependence was based on each twin's self-report of experienced dependence on the co-twin at the age of 16. The question was: 'In your own opinion, are you dependent on your co-twin?', with response alternatives *yes* and *no*. Most twins (99.1%) made an unambiguous response to this simple question. Twins who did not were omitted and only *yes* and *no* answers were accepted. A total of 49 twins (44 pairs) were excluded for answering ambiguously. Twin pairs were classified as co-twin–dependent (both twins of a pair reported co-twin dependence; n = 362 pairs, 14.2%) or as independent (both twins of a pair denied being dependent; n = 1696 pairs, 66.5%). An intermediate group, where one twin of a twin pair reported co-twin dependence and the co-twin reported independence, was classified as intermediate (n = 492 pairs, 19.3%).

Alcohol Use

In both adolescence and early adulthood, twins reported individually on their alcohol use. Alcohol use was measured with a set of structured self-report questionnaire items derived from the Finnish Adolescent Health and Lifestyle Survey (Rimpelä et al., 1988) that are widely used in Finnish epidemiological research. Three alcohol-use measures were included into the analyses at the two time-points: initiation of alcohol use, drinking frequency and intoxication frequency. Frequency of alcohol use was assessed with the question: 'How often do you drink alcohol?' with nine ordered response choices ranging from *daily* to *I don't drink alcohol*. Alcohol use was classified into four categories according to the response alternatives: weekly (including *daily use* to *1 to 2 times per week*), monthly (*1 to 2 times per month*), more rarely (*1 to 6 times per year or less*) and abstinent (*I don't drink alcohol*). Intoxication frequency was assessed with the question: 'How often do you get really drunk?' with four response alternatives, ranging from *once a week or more* to *never*. In the analysis of drinking and intoxication frequency, we included pairs in which twins had reported concordantly their initiation of alcohol use.

Other Covariates

The effects of urban/rural residential status and religiosity were also assessed as possible covariates in relation to co-twin dependence, as these factors have been found to affect the alcohol-use patterns of families and individuals in earlier reports from FinnTwin16 samples (Rose at al., 1999; Winter, Karvonen, Kaprio, et al., 2002; Winter, Karvonen, & Rose, 2002). Urban/rural status was based on the classification of the community of residence at the age of 16 years, and categorized as either urban or rural as defined by Rose et al. (1999). Religiosity was assessed by the Wiggins REL scale of the Minnesota Multiphasic Personality Inventory (MMPI), administered to both twins and parents at the baseline, as described in detail elsewhere (Winter et al., 1999; Winter, Karvonen, & Rose, 2002).

Statistical Analysis

Descriptive distributions of abstinence and drinking patterns are presented in Table 1 for males and females from same-sex twin pairs. Descriptive statistics illustrate the prevalence of alcohol use in co-twin–dependent and –independent twins by twin-type and

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gender. The data were processed using the complex survey data analysis methods with Stata (StataCorp, 2003), to account for the fact that the observations of twin pairs are correlated. To determine differences in alcohol use between co-twin–dependent and –independent twins, the differences in proportions were tested with a design-based chi-square test, namely the Wald *F*-statistics, adjusted for correlated data (Rao & Scott, 1984).

Because the data were ordinal, polychoric correlations were computed from contingency tables using the statistical package Mx (Neale et al., 1999). In the present study, modeling employed contingency tables and maximum-likelihood estimation using the structural equation modeling package Mx (Neale et al., 1999). Significant gender differences were found in the prevalences of co-twin dependence and most outcome variables of alcohol use, so *sex* was added to the SEM twin model to differentiate sex effects from the estimates of variance. Hence, the univariate model-fitting analyses were conducted for concordantly co-twin–dependent and concordantly co-twin–independent twins for the four twin types (male and female monozygotic [MZ] twins and male and female same-sex dizygotic [DZ] twins) to decompose the variance of alcohol-use measures. Intermediate pairs, that is, twin pairs discordant for co-twin dependence, were excluded from the analysis.

First, a full model was fit allowing different additive genetic (A_d), common environmental (C_d) and unique environmental (E_d) influences for co-twin dependent twins and A_i, C_i and E_i influences for independent twins, as well as different ACE effects for males and females to account for the observed variance on alcohol-use measures. The analyses were conducted separately for the two time-points, adolescence and early adulthood. We began with a model that permits the magnitude of genetic and environmental effects to be assessed separately on co-twin–dependent and –independent pairs, but allows the magnitude of paths a^2, c^2 and e^2 to differ across gender. The modeling proceeded by testing whether the paths could be set equal for dependent and independent twins. A more restricted model constraining ACE_d and ACE_i effects to be equal for co-twin dependent and independent twins was compared to the full model by likelihood ratio χ^2. Thresholds were allowed to differ for dependent and independent twins as well as for males and females in both models. After fitting the full model, the most parsimonious model was sought, that is, the model with the fewest parameters, by fitting a series of submodels and testing the significance of each factor by removing the corresponding path from the model. For example, a model in which the genetic influences (path a^2_d and a^2_i) were fixed at 0 was compared with a model containing all three sources of variation (paths a^2_d, c^2_d and e^2_d for dependent and paths a^2_i, c^2_i and e^2_i for independent twins).

The significance of change in model fit, when the full models were constrained equal for co-twin–dependent and –independent twins or parameters were omitted (i.e., removing the corresponding path) from the submodels or the submodels were constrained equal, was tested with estimating the change in χ^2 between the different models. Model fits were also assessed using Akaike's Information Criterion (AIC; Akaike, 1987) and Root Mean Square Error of Approximation values (RMSEA; Neale et al., 1999). The similar model-fitting analyses were conducted for the follow-up data, that is, twins in their early adulthood.

Results

Table 1 gives the distribution of twins' drinking patterns for co-twin–dependent and –independent twins in the two waves of assessment. When twins were analyzed as individuals, twins from co-twin–dependent pairs tended more often to be abstinent and to be drinking less frequently than concordantly independent co-twins, both in adolescence and in early adulthood; however, the difference was significant only for abstinence at age 16 among dependent MZ males, who were significantly more often abstinent than co-twin–independent MZ males (F [1, 349] = 4.78, p = .03). For intoxication frequency, there were no differences between co-twin–dependent and –independent twins.

Twin Correlations

Correlations for drinking patterns are presented for the two waves of assessment in Table 2 with concordantly co-twin–dependent and concordantly co-twin–independent twin pairs separated by zygosity and gender. At the baseline of age 16 years, all correlations differed significantly from zero for all three variables of drinking behavior. Same-sex dizygotic (SSDZ) twin correlations approached, and in some cases numerically exceeded, those found for MZ pairs, offering immediate evidence of the influence of common environmental effects both in co-twin–dependent and –independent twin pairs. Among independent twins, both male and female MZ correlations were significantly higher than the corresponding DZ correlations for both drinking frequency and intoxication frequency, also suggesting genetic influences. In the follow-up, at ages 22 to 27 years, the DZ correlations for abstinence exceeded half the MZ correlation, suggesting shared environmental effects for both co-twin–dependent and –independent twins. For drinking frequency and for intoxication frequency, the difference between MZ and DZ correlations indicates substantial genetic effects in males for both groups. Among females, MZ correlations are higher than DZ correlations, but the difference is smaller, suggesting the influence of common environmental effects, particularly among co-twin–dependent females.

When the correlations were compared between co-twin–dependent and –independent groups at the age of 16 years, most correlations of dependent twins exceeded those of independent twins for both genders,

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Table 1

Distribution of Drinking Habits among Dependent and Independent Twins by Zygosity, Gender and Age at Assessment

	Adolescence (age of 16) *n* = 3362				Early adulthood (ages of 22–27) *n* = 2912			
	Males		Females		Males		Females	
	Dependent %	Independent %	Dependent %	Independent %	Dependent %	Independent %	Dependent %	Independent %
Monozygotic twins								
Abstinent	36	26	26	21	10	6	6	5
	(188)	(488)	(367)	(612)	(146)	(404)	(333)	(552)
Drinking frequency[1]								
weekly	14	19	10	9	57	66	43	46
1–2 times per month	39	36	41	37	28	26	41	37
6 times per year or less	47	45	49	54	15	8	16	17
	(121)	(363)	(273)	(484)	(132)	(379)	(312)	(526)
Intoxication frequency[1]								
weekly	2	2	1	1	20	22	7	10
1–2 times per month	15	17	12	12	39	47	39	37
more rarely	47	43	51	48	34	27	47	44
never	36	38	36	40	7	4	7	9
	(121)	(363)	(273)	(484)	(132)	(379)	(312)	(526)
Dizygotic twins								
Abstinent	24	27	25	21	10	5	10	6
	(123)	(745)	(217)	(622)	(100)	(610)	(193)	(574)
Drinking frequency[1]								
weekly	6	13	9	11	63	62	38	45
1–2 times per month	39	38	38	44	26	29	45	38
6 times per year or less	55	49	53	45	7	9	17	17
	(93)	(524)	(163)	(490)	(90)	(579)	(173)	(539)
Intoxication frequency[1]								
weekly	0	2	1	2	23	23	8	9
1–2 times per month	16	16	14	16	49	47	36	35
more rarely	45	47	49	49	24	28	49	50
never	39	35	36	33	3	3	7	7
	(93)	(524)	(163)	(490)	(90)	(579)	(173)	(539)

Note: Percentages by category and total number of twins in each group in parenthesis (*n*). [1] Includes only the twins who had initiated drinking.

indicating more similar alcohol-related behavior of co-twin–dependent twins in adolescence. One exception is found in intoxication frequency, where the correlation of dependent MZ males is significantly lower than the correlation for independent MZ male pairs. The correlation of co-twin–dependent MZ males might be affected by the fact that no-one of this group had been drinking to intoxication weekly, leaving that category without observations. In early adulthood (ages 22 to 27 years), co-twin–dependent twin correlations for alcohol-use patterns still exceeded the correlations of independent twins, with the exception of DZ males, where the number of dependent DZ male pairs is relatively small. Nevertheless, the results suggest that as young adults, co-twin–dependent twins remain more similar in their alcohol-related behavior than do independent twins.

Model Fitting

Table 3 shows the results of the model-fitting analyses for adolescent twins with the best-fitting full and the best-fitting submodel, that is, the more parsimonious model. Univariate model-fitting suggested that the best-fitting model for all three alcohol-use variables (abstinence, drinking frequency and intoxication frequency) was the model which included separately parameters CE_d for co-twin–dependent twins and ACE_i for independent twins. Constraining the variance components of dependent and independent twins to be equal ($ACE_d = ACE_i$) caused a significant decrease in the fit of the model ($p \leq .05$) in all three alcohol-use measures. In dependent twins, path a^2_d (genetic effect) could be omitted from all alcohol-use variables without significantly compromising the model fit ($p = .90$). In contrast, among twins from concordantly independent pairs, none of the parameters a^2_i, c^2_i or e^2_i could be removed from the models of the three alcohol-use measures without significantly compromising the fit ($p \leq .005$). Constraining the prevalences to equality for dependent and independent twins also caused a significant decrease of fit in abstinence and drinking frequency ($p < .01$), but not in intoxication frequency ($p = .46$).

The model-fitting results suggest that most of the variation in alcohol-related behavior among co-twin–dependent twins in adolescence was accounted for by shared environmental influences. The common environmental effects in alcohol-related behavior were

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Table 2

Polychoric Correlations for Drinking Patterns by Zygosity for Co-Twin Dependent and Independent Same-Sex Male and Female Twin Pairs in Adolescence (n = 1342 pairs) and in Early Adulthood (n = 1078 pairs)

	Males						Females					
	Dependent			Independent			Dependent			Independent		
	(*n*)	*r*	95% CI for *r*	(*n*)	*r*	95% CI for *r*	(*n*)	*r*	95% CI for *r*	(*n*)	*r*	95% CI for *r*
Adolescence												
Abstinence												
Monozygotic twins	(61)	.98	(.88–1.00)	(206)	.86	(.75–.93)	(142)	.98	(.94–1.00)	(261)	.95	(.89–.98)
Dizygotic twins	(23)	.91	(.50–.99)	(327)	.83	(.73–.90)	(61)	.95	(.74–.99)	(261)	.87	(.77–.94)
Drinking frequency[1]												
Monozygotic twins	(37)	.75	(.50–.99)	(140)	.74	(.60–.88)	(101)	.85	(.69–1.00)	(197)	.74	(.62–.86)
Dizygotic twins	(14)	.90	(.65–1.00)	(208)	.59	(.47–.71)	(47)	.80	(.57–1.00)	(196)	.56	(.41–.71)
Intoxication frequency[1]												
Monozygotic twins	(37)	.51	(.13–.76)	(140)	.74	(.63–.82)	(101)	.81	(.69–.89)	(197)	.73	(.61–.81)
Dizygotic twins	(14)	.96	(.70–1.00)	(210)	.54	(.41–.65)	(47)	.91	(.77-.97)	(196)	.60	(.47–.70)
Early adulthood												
Abstinence												
Monozygotic twins	(44)	.83	(.33–.98)	(161)	.77	(.42–.94)	(123)	.95	(.78–1.00)	(227)	.74	(.43–.91)
Dizygotic twins	(14)	.67	(–.29–.99)	(234)	.68	(.32–.89)	(48)	.75	(.26–.96)	(227)	.74	(.43–.91)
Drinking frequency[1]												
Monozygotic twins	(37)	.85	(.64–.95)	(148)	.64	(.46–.77)	(113)	.49	(.28–.65)	(209)	.43	(.27–.56)
Dizygotic twins	(11)	.18	(–.80–.82)	(216)	.21	(.03–.39)	(40)	.48	(.11–.73)	(209)	.32	(.16–.47)
Intoxication frequency[1]												
Monozygotic twins	(37)	.71	(.43–.87)	(148)	.59	(.45–.70)	(113)	.67	(.51–.78)	(209)	.59	(.48–.68)
Dizygotic twins	(11)	.33	(.42–.84)	(216)	.35	(.21–.47)	(40)	.57	(.24–.77)	(209)	.40	(.25–.52)

Note: [1]Correlations for all concordantly drinking female and male twin pairs. Polychoric correlations (r) are computed with Mx.

also important among independent twins, but a significant additive genetic contribution, especially in drinking (a^2_i = 29%) and in intoxication frequency (a^2_i = 38%), was also found in this group. Table 4 presents the partitioning of variance into genetic, common environmental, and unique environmental sources of variance with associated confidence intervals according to the best-fitting full model and the best-fitting submodel in adolescence.

Table 5 presents results of model-fitting analyses in early adulthood for the best-fitting full model and for the best-fitting submodel. The data suggests similar genetic and environmental influences for abstinence and drinking frequency for both co-twin–dependent and –independent twins. For intoxication frequency, the variance components of co-twin–dependent and –independent twins differ, the best-fitting model having components CE_d for dependent twins and AE_i for independent twins. Constraining the prevalences to

Table 3

Model-Fitting Results for Drinking Habits in Adolescence

Model	Equal variance components	Equal prevalences	Fit statistics				
			χ^2	*df*	*p*	AIC	RMSEA
Abstinence							
ACE_d, ACE_i	No	No	11.634	16	.769	–20.366	.000
CE_d, ACE_i	No	No	13.748	17	.685	–20.252	.000
Drinking frequency							
ACE_d, ACE_i	No	No	93.513	52	.000	–10.487	.044
CE_d, ACE_i	No	No	93.529	53	.001	–12.471	.044
Intoxication frequency							
ACE_d, ACE_i	No	Yes	131.670	107	.053	–82.330	.027
CE_d, ACE_i	No	Yes	131.670	108	.061	–84.330	.027

Note: A = additive genetic variance, C = common environmental variance, E = unique environmental variance; ACE_d = ACE model for co-twin dependent twins; ACE_i = ACE model for independent twins.
RMSEA = Root Mean Square Error of Approximation; AIC = Akaike's Information Criterion
More detailed model-fitting results can be obtained by request from corresponding author.

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Table 4

Partitioning of Variance Into Genetic and Environmental Influences on Drinking Habits Under the Best-Fitting Full and Reduced Models in Adolescence

	Variance components for co-twin dependent twins			Variance components for independent twins		
Model	a^2_d	c^2_d	e^2_d	a^2_i	c^2_i	e^2_i
Abstinence[1]						
ACE_d, ACE_i	.11	.87	.02	.14	.78	.09
	(.00–.42)	(.56–.98)	(.01–.06)	(.00–.29)	(.64–.89)	(.05–.14)
CE_d, ACE_i		.97	.03	.14	.78	.09
	—	(.93–99)	(.01–.07)	(.00–.29)	(.64-.89)	(.05–.14)
Drinking frequency[1]						
ACE_d, ACE_i	.02	.79	.19	.29	.44	.27
	(.00–.40)	(.42–.88)	(.12–.28)	(.07–.51)	(.24–.62)	(.20–.34)
CE_d, ACE_i		.81	.19	.29	.44	.27
	—	(.72–.88)	(.12–.28)	(.07–.51)	(.24–.62)	(.20–.34)
Intoxication frequency[2]						
ACE_d, ACE_i	.00	.81	.19	.36	.38	.26
	(.00–.13)	(.67–.87)	(.13–.28)	(.16–.57)	(.19–.54)	(.20–.33)
CE_d, ACE_i		.81	.19	.36	.38	.26
	—	(.72–.87)	(.13–.28)	(.16–.57)	(.19-.54)	(.20–.33)

Note: [1] Separate variance components and prevalences for co-twin–dependent and –independent twins.
[2] Separate variance components, but same prevalences for co-twin–dependent and –independent twins.

equality for dependent and independent twins caused a significant decrease of fit for drinking frequency and intoxication frequency ($p < .001$). In addition, the best-fitting model for abstinence was one that allowed different prevalences for co-twin–dependent and –independent twins.

In early adulthood, most of the variance in abstinence was accounted for by shared environmental influences, so that the influence of genetic effects was negligible. In contrast to the situation in adolescence, the variance components of co-twin–dependent and –independent twins could also be constrained equal. Thus, the best-fitting model for abstinence was the CE model where paths a^2_d and a^2_i are set to 0, and paths c^2 and e^2 are equal for dependent and independent twins. The model suggests that there are possibly similar common environmental effects (78% of the variance in reduced model), which are influencing whether an individual is drinking or abstaining. The influence seems to be highly familial, that is, shared by siblings but not necessarily dependent on the quality of co-twin relationship.

The magnitude of genetic effects on drinking frequency appeared to increase with age, whereas the influence of common environment decreased. The models for drinking frequency among young adults indicated that the genetic effects accounted for most of the variance (47%, full model), with shared environmental effects for drinking frequency barely significant; accordingly, paths c^2_d and c^2_i of common environmental factors could be removed. Thus, the best-fitting model for co-twin–dependent and –inde-

Table 5

Model-Fitting Results for Drinking Habits in Early Adulthood

			Fit statistics				
Model	Equal variance components	Equal prevalences	χ^2	*df*	*p*	AIC	RMSEA
Abstinence							
ACE_d, ACE_i	Yes	No	12.379	18	.827	–23.621	.005
CE_d, CE_i	Yes	No	13.677	19	.802	–24.323	.008
Drinking frequency							
ACE_d, ACE_i	Yes	No	56.963	54	.365	–51.037	.025
AE_d, AE_i	Yes	No	57.173	55	.394	–52.827	.025
Intoxication frequency							
ACE_d, ACE_i	Yes	No	106.039	106	.481	–105.961	.007
CE_d, AE_i	No	No	105.246	106	.502	–106.754	.005

Note: A = additive genetic variance, C = common environmental variance, E = unique environmental variance; ACE_d = ACE model for co-twin–dependent twins; ACEi = ACE model for independent twins; RMSEA = Root Mean Square Error of Approximation; AIC = Akaike's Information Criterion.
More detailed model-fitting results can be obtained by request from corresponding author.

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Table 6

Partitioning of Variance into Genetic and Environmental Influences on Drinking Habits Under the Best-Fitting Full and Reduced Models in Early Adulthood

	Variance components for co-twin dependent twins			Variance components for independent twins		
Model	a^2_d	c^2_d	e^2_d	a^2_i	c^2_i	e^2_i
Abstinence[1]						
ACE_d, ACE_i	.22 (.00–.64)	.61 (.22–.85)	.17 (.09–.31)	*equal*	*equal*	*equal*
CE_d, CE_i	—	.78 (.67–.86)	.22 (.14–.32)	"	"	"
Drinking frequency[1]						
ACE_d, ACE_i	.47 (.19–.61)	.06 (.00–.29)	.47 (.39–.56)	"	"	"
AE_d, AE_i	.53 (.45-.61)	—	.46 (.39–.55)	"	"	"
Intoxication frequency[2]						
ACE_d, ACE_i	.44 (.22–.66)	.17 (.00–.35)	.39 (.33–.46)	"	"	"
CE_d, AE_i	—	.65 (.53–.74)	.35 (.26–.47)	.61 (.53–.68)	—	.39 (.32–.47)

Note: [1] Different prevalences, but same variance components for co-twin dependent and independent twins.
[2] Different prevalences and variance components for co-twin dependent and independent twins.
"equal" signifies that variance components are equal for co-twin dependent and for independent twins.

pendent twins was an AE model and the two twin groups could also be constrained to be equal without significantly compromising the fit. Drinking frequency seemed to be independent from familial or other common environmental effects and the possible influence of the co-twin.

For intoxication frequency, co-twin–dependent and co-twin–independent twins differed. As was true in adolescence, dependent co-twins were showing higher levels of common environmental effects and lower genetic effects than independent twins. For co-twin–dependent twins the model-fitting results indicated that the environmental factors, largely those from common environmental sources, are responsible for phenotypic variation in intoxication frequency, whereas significant genetic factors contributed to intoxication frequency for independent twins. When searching for the most parsimonious model, we found that the best-fitting model was one including the parameters CE_d for co-twin–dependent twins and AE_i for co-twin–independent twins. As the variance components of dependent and independent twins were clearly differing, it seems that the quality of the twin relationship might have an effect on drinking behavior when drinking into intoxication is considered. Table 6 presents the partitioning of variance into genetic, common environmental, and unique environmental sources of variance with associated confidence intervals according to the best-fitting full and best-fitting reduced model in early adulthood.

The analyses of other covariates, that is, urban/rural status and familial religiosity found no associations between these factors and co-twin dependence, neither when twins were analyzed as individuals (all $p > .49$), nor when they were analyzed as concordant dyads of dependent and independent twins (all $p > .31$). Co-twin–dependent twins and co-twin– independent twins were equally likely to be living in urban and rural areas, and they were equally likely to be from religious and nonreligious families.

Discussion

The determinants of drinking behavior develop in the interplay between genetic and environmental influences. The alcohol-related behavior in twins was assessed, focusing on the modulating influence of co-twin dependence. Differences in self-reported drinking behavior were investigated in concordantly co-twin– dependent and concordantly co-twin–independent twin dyads and effects of dependence on the relative influence of genetic and environmental effects on alcohol-related behavior from adolescence into early adulthood.

The primary finding of this study was that a single question on co-twin dependence, scored dichotomously to yield concordantly dependent and independent twin pairs, offers novel insights into twins' similarities for alcohol use in both adolescence and early adulthood. While few differences in prevalence of abstinence were found between dependent and independent twins and no effect of co-twin dependence on drinking or intoxication frequency, significantly higher pair resemblances in co-twin– dependent twins were found for abstaining/drinking of alcohol and drinking behavior compared to independent twins. And the greater similarity in drinking behavior carried over from adolescence into early adulthood.

Another major finding in the study was that the genetic contribution to individual differences in drinking patterns, especially in adolescence, are dependent on the nature of the pair-wise relationship. The model-

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ing results suggest that the drinking behavior of co-twin–dependent twins in adolescence was due more to shared environmental influences, with insignificant genetic factors. In other words, the impact of genetic liability was reduced as a function of the co-twin relationship. Conversely, genetic influences on drinking habits were expressed among independent twins and, especially with drinking frequency and intoxication frequency, genetic influences were as important as shared environmental effects. The importance of non-shared environment in co-twin–dependent twins was less than in independent twins, perhaps reflecting a greater overlap in the shared experiences of co-twin–dependent twin pairs.

In a previous analysis of this same sample, co-twin dependence was found to have a strong association with the patterns of twins' social interactions and leisure time companionship (Penninkilampi-Kerola et al., in press). Differential social contacts may be a relevant contributor as in adolescence, the leisure time spent with the co-twin and having friends in common were strongly associated with co-twin dependence rather than zygosity. In early adulthood, twins who had reported co-twin dependence in adolescence were still in more frequent interaction with each other and were more often living together than were co-twin–independent twins (Penninkilampi-Kerola et al., 2004). Thus, the magnitude of the reciprocal influences that co-twin–dependent twins exert on one another is likely to be greater than for independent twins. However, previous studies on adult twins have provided slightly inconsistent results for the association between contact frequency and twins' similarity in drinking patterns. Some studies have suggested that twins in more frequent contact are more alike in their drinking behavior (Kaprio et al., 1987; Kaprio et al., 1990; Kendler et al. 1992; Rose et al., 1990), while other studies have not found such association (Heath et al., 1989; LaBuda et al., 1997; Reiss, Cederblad, Pedersen, Lichtenstein, Elthammar, et al., 2001).

Compared to earlier studies of older twins, in our analyses both co-twin–dependent and –independent twins were cohabiting in adolescence and were, therefore, in constant contact with each other. Nevertheless, differences were found in the similarity of alcohol use between the two groups. In this context, is it likely that the reciprocal influences can be attributed only to the twins' frequency of contact? It may be more likely that there are embedded processes in sibling interactions, or in interpersonal relationships in general, that influence the choices and mediates behavior both in individual twins and in their dyadic relationship to the co-twin. A study of Heath et al. (1989) found that marital status, that is, being married or having a marriage-like relationship or not modified the level of genetic influences. Co-twin dependence as we measured it, could be one manifestation of these relationship processes in sibling interaction that encompass not only physical contact, but also psychosocial and emotional characteristics that interlace siblings, their behaviors and their environments together. In all likelihood, co-twin –dependent twins, whose relationships are more likely to be closer than the relationship between independent twins, are also more likely to accept and share each other's values and emulate one another's behavior, especially in adolescence where substance use is highly susceptible to modeling and reinforcement. If twins are independent and possibly more apt to strengthen their individuality, modeled behavior will be less likely to be adopted.

Another relevant aspect explaining the differences between co-twin–dependent and –independent twins may be found in differential peer influences on their behavior. Different studies have shown that substance use by close friends and siblings is one of the strongest predictors of adolescent substance use (e.g., Ary et al., 1993; Crawford & Novak, 2002). In a previous analysis of the same sample, it was found that the twin relationship influenced the composition of the peer network and dependent twins were more likely to share their friends (Penninkilampi-Kerola et al., in press); accordingly, it is also plausible that co-twin–dependent twins share their peer influences to a larger extent, and, as a result, have a more limited variation in these influences. In contrast, independent twins may act more self-reliantly in their social interactions and choose from a greater diversity of environmental opportunities and peer networks, which may in part differentiate their patterns of substance use.

Different studies have provided evidence that several factors in family environment, such as religiousness (Koopmans & Boomsma, 1999) and regional residency (Rose, Dick, Viken, & Kaprio, 2001; Rose et al., 1999) may decrease the magnitude of genetic influences so that phenotypic similarity in alcohol use is more influenced by shared environmental influences. Such findings raise the question of possible differences in the family environments of co-twin–dependent and –independent twins. In this study, no differences were found in familial religiosity or in regional residency between dependent and independent twins, findings that suggest that co-twin dependence is more likely to be an independent factor and not mediated by these environmental differences. Additionally, the family dynamics and family cohesion in families of dependent twins may differ from that of independent twins. There may be variation in parental attitudes — upbringing twins as individuals, parental bonding, as well as parental monitoring and discipline — that might influence adolescent alcohol use. There is evidence that family functioning and parenting practices as well as parental alcohol use (Barnes et al., 1994; Koopmans & Boomsma, 1996; McGue et al., 1996) are influencing the alcohol-related behavior of adolescents. In this case, it is plausible that family functioning and parenting practices are influencing directly, but also indirectly by enhancing sibling inter-

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action, and delaying twins from developing independent and autonomous behaviors.

In the follow-up from adolescence to early adulthood, the number of abstinent twins decreased significantly and the rates of drinking and intoxication frequency increased. As young adults, co-twin–dependent and –independent twins seemed to be similar in their alcohol-related behavior at an individual level. However, when twins were analyzed as dyads, we found that, in general, co-twin–dependent co-twins were still more similar in their drinking behavior when compared to independent twins.

For intoxication frequency, a difference in heritability estimates was once again found between the two groups. The model-fitting results indicated that for co-twin–dependent twins, common environment remained the strongest determinant of intoxication frequency, while for independent twins, the influence of common environment was only weak or negligible and the importance of genetic influences was evident. As mentioned earlier, co-twin–dependent twins were in more frequent interaction with each other as young adults as well compared to independent twins. Therefore, it is plausible that they were still sharing their peer networks and attending the same leisure-time social events that influence alcohol-related behavior and drinking habits within and between twin dyads. On the other hand, the relatively small number of dependent twins, especially males, in the follow-up reduced power to test for differences in heritability of intoxication frequency.

The results also have broader implications for singleton populations as the study offers evidence that environmental influences, such as differences in interpersonal relationships, are contributing to the effects of genetic propensities and genetic effects are modulated by shared experiences and imitative modeling between siblings. When the environment exerts a strong moderating effect such as a close relationship to another person, it might diminish the effect of genetic propensity and at the same time increase the influence of shared environmental influences. On the other hand, the genetic factor may be more operative in contexts where social restrictions, such as influence of co-twin, are less distinct. At high levels of family cohesion when there are particularly strong emotional bonds between family members (e.g., between spouses, siblings, parents and children) the modeling effect of family members on each other could be more potent than in less cohesive families, and these modeling effects might be independent from genetic propensities. Similarly, strong emotional attachment and reliance on peers may have the same implications.

Unfortunately, this study doesn't provide data on attachment relationships (or dependence) between family members other than co-twin dependence. However, an earlier study indicates that co-twin dependence is strongly related to twins' social interactions with each other and shared peer networks (Penninkilampi-Kerola et al., in press). Analyzing the associations of contact frequency between different family members may give indications of the influence of cohesive family relationships. It should be noted though that previous studies have suggested that contact frequency as such is not necessarily a sufficient factor to explain this effect or similarity in alcohol-related behavior (e.g., LaBuda et al., 1997; Reiss, Cederblad, Pedersen, Lichtenstein, Elthammar, et al., 2001). This raises the question of the importance of the emotional components in interpersonal relationships, that is, attachment, reliance on others, interpersonal dependence or co-twin dependence. To disentangle these questions, additional research and new data, which provides more detailed and sophisticated measures, is needed, including both structural and emotional components of interpersonal relationships.

It could be considered a limitation that our analyses rely on self-report data of co-twin dependence that is measured only at the age of 16 years. It is possible that dependence measured in adolescence has little relevance at ages 22 to 27 years. However, twins who had reported co-twin dependence in adolescence were found to remain in more frequent interaction in early adulthood, suggesting that the close sibling relationship of dependent twins is likely to continue beyond adolescence (Penninkilampi-Kerola et al., 2004). An earlier study of co-twin dependence (Penninkilampi-Kerola et al., in press) indicated that the construct validity of self-reported dependence is supported by the results patterns of mothers' reports of co-twin dependence, as well as by the co-twins' evaluations of their own dependence and that of their co-twins. Consistent associations were also found between co-twin dependence and twins' social contacts and leisure-time activities, both in adolescence and in early adulthood, indicating that a simple self-report of dependence does measure an important and useful characteristic or quality of the twin relationship that is likely to encompass both structural (e.g., contact frequency, shared peer networks, shared leisure-time activities) and emotional (e.g., reliance on co-twin, attachment, feeling of connectedness) characteristics of the co-twin relationship. Thus, co-twin dependence may assess an important and enduring feeling of being interdependent on another person, and it is expressed in the twin dyad through mutual interaction and interconnectedness in behavior. Moreover, this aspect can also be measured in twins living together, which is the norm for adolescent twins.

Strengths of the study are that the findings are derived from a large population-based sample of twins, with exceptionally good response rates throughout the different waves of the study. Moreover, the study is the first, to our knowledge, to examine longitudinally the contribution of the co-twin relationship to the genetic and environmental variance of a psychosocial health-related trait such as drinking behavior. The fact that twins were cohabiting at the

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baseline extends earlier analyses of older twins, and provides additional information of the role of interpersonal relationships on alcohol use and drinking behavior and on the genetic and environmental variation of these traits.

Our results suggest that attempts to statistically summarize the influences of genes and environment constitute a misleading simplification, because heritability in certain subpopulations will be modulated by differences in psychosocial and environmental characteristics. Therefore, it is increasingly important not only to study the relative proportions of genetic and environmental influences, but to address, as well, how these influences are mediated in different subpopulations and in different psychosocial contexts. As other authors have suggested, however, the comparisons between groups stratified by an environmental covariate such as co-twin contact should be drawn with caution, as the associations found may as well be a reflection of latent genetic, rather than 'purely' environmental, mediation (Eaves et al., 2003). Nevertheless, it is important to recognize and identify the quality of this environmental variation and to understand what its significance to the trait under study is.

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