J Abnorm Child Psychol (2008) 36:81–94
DOI 10.1007/s10802-007-9161-0

Alcohol Use in Adolescent Twins and Affiliation with Substance Using Peers

Jennifer Hill · Robert E. Emery · K. Paige Harden · Jane Mendle · Eric Turkheimer

Received: 4 December 2006 / Accepted: 28 June 2007 / Published online: 31 July 2007

Abstract Affiliation with substance using peers is one of the strongest predictors of adolescent alcohol use. This association is typically interpreted causally: peers who drink incite their friends to drink. This association may be complicated by uncontrolled genetic and environmental confounds because teens with familial predispositions for adolescent substance use may be more likely to select into social networks where drinking is common. We test this alternative hypothesis using a sample of 1,820 twin and sibling pairs, and their same-sex best friends, from three waves of the National Longitudinal Study of Adolescent Health. Across all three waves, peer report of substance use did not influence adolescent alcohol use when genetic and shared environmental predispositions for drinking were considered. The association between alcohol use and peer behavior may be a spurious association attributable to a shared genetic liability to drink alcohol and associate with peers who drink alcohol.

Keywords Peers · Adolescence · Alcohol use · Behavior genetics

As children transition to adolescence, peers emerge as crucial facets of development and socialization (Larson and Richards 1991). Peer selection is a chance for adolescents to choose their extra-familial environment and to supplement childhood backgrounds (Iervolino et al. 2002). Teenagers resemble their friends in a variety of ways, including physical attributes, personality traits, and behaviors (Berkowitz 1969; Fergusson et al. 2002; Guo 2005). In particular, affiliation with peers who use alcohol is one of the strongest correlates of alcohol use in adolescence (e.g. Bauman and Ennett 1996; Dishion and Dodge 2005a; Fergusson et al. 2002). The strength and persistence of the association between individual and peer alcohol use has led to the common assumption that peer behavior is an important *cause* of adolescent alcohol use (Bauman and Ennett 1996).

Evaluating Peer Influence

Nevertheless, the causal influence of peers may have been overestimated in previous studies due to three methodological factors. First, most research uses adolescent report of peer behavior as opposed to self-report obtained from the peers themselves (Bauman and Ennett 1996). However, studies comparing adolescent report of peer risk behaviors to the peer's report of his or her own behaviors have demonstrated that adolescent reports overestimate the similarity between adolescents and their peers by three times (Bauman and Ennett 1996; Kandel 1996). The differences found between peer reports and adolescent reports of peers are likely attributable to a combination of mechanisms; the adolescent may project his or her behavior onto the peer, the peer may present a distorted image to the adolescent to feign closeness or be perceived in a desired manner, and there may be actual differences in knowledge due to changes in the peer's behavior (Bauman and Ennett 1996).

Second, the direction of causation cannot be determined from cross-sectional approaches which do not differentiate peer effects on adolescents from adolescent effects on peer selection (Bauman and Ennett 1996; Kandel 1996). Adolescents choose peers with similar interests, activities, and personalities; therefore, it is unsurprising that adoles-

J. Hill (✉) · R. E. Emery · K. P. Harden · J. Mendle · E. Turkheimer
Department of Psychology, University of Virginia, P.O. Box 400400, Charlottesville, VA 22902, USA
e-mail: jeh8s@virginia.edu

cents who use alcohol are more likely to choose friends who use alcohol (Guo 2005; Jaccard et al. 2005). Attempts to clarify this relationship using longitudinal designs have generally concluded that peer influence is reduced by around 50% when selection effects are considered (e.g. Bauman and Ennett 1996; Kandel 1996).

Third, peers are not assigned randomly to adolescents. A variety of genetic and environmental confounds may influence both selection of alcohol using peers and alcohol use, producing a spurious correlation. For example, consider poverty as a potential confound. Poverty may influence an adolescent's selection of peers who drink alcohol and may also independently increase risk of alcohol consumption. In this case, poverty confounds the causal relationship between peer affiliation and alcohol use. Family factors, socioeconomic status, neighborhoods, and schools are additional examples of the many potential environmental factors that may confound the relationship between peer affiliation and alcohol use (Miles and Carey 1997; Padilla-Walker 2006). Longitudinal studies examining potential confounds, such as adolescent rebelliousness (Curran et al. 1997) or life transitions and stressful life experiences (Fergusson et al. 2002), have noted the importance of examining third variable influences. However, traditional longitudinal studies are limited in that they can only examine measured confounds and cannot examine all of the possible confounds that may create a spurious relationship.

Genetic factors may also create selection effects. Genetic factors are known to influence alcohol use and dependence in adolescence (Rhee et al. 2003; Silberg et al. 2003). In addition, genetic factors are known to influence selection of peers with similar personalities and alcohol use behaviors (Cleveland et al. 2005; Guo 2005). If genotypic factors influence both selection of peers who drink and alcohol use, the association would be an artifact of shared genetic liabilities to select peers and drink alcohol.

Family Designs and Genetic/Shared Environmental Selection

While previous reviews have highlighted the importance of using direct peer report to reduce reporter bias, and longitudinal designs to examine the direction of causation (e.g., Bauman and Ennett 1996; Kandel 1996), potential genetic and environmental confounds have received considerably less attention and warrant further discussion. Identifying genetic and environmental selection effects is essential in determining whether observed associations are spurious consequences of uncontrolled third variables or likely to be true causal processes (Rutter 2003). Family designs are an advantageous way to control for genetic and environmental confounds that may affect the relationship between peer characteristics and adolescent behavior. Because adolescent twin and sibling pairs share genetic and environmental characteristics that are not shared by unrelated adolescents, family designs offer a more accurate estimate of hypothesized causal influences of risk factors on outcomes.

Differences between unrelated individuals are confounded by all the environmental and genetic factors that differ between families, but differences between siblings are only confounded by influences that vary systematically within sibling pairs. Socioeconomic status, for example, varies greatly between families, but relatively little within sibling pairs raised together. Consider a family with low socioeconomic status in which one sibling associates with alcohol using peers and the other does not. If the association between peer behavior and alcohol use is causal, the sibling with alcohol-using peers should engage in more alcohol use than the sibling who does not affiliate with alcohol-using peers. If, on the other hand, the association between peer behavior and alcohol use is an artifact of low socioeconomic status, both siblings should exhibit similar alcohol use, despite the difference in peer affiliation.

By considering monozygotic (MZ) and dizygotic (DZ) twin siblings, not only can the magnitude of potential between family confounds be estimated, but the confound can be identified as genetic or environmental in origin. MZ and DZ twin pairs reared together share a similar home environment but MZ twins are genetically identical whereas DZ twins share only 50% of their genes. To the extent that a confounding variable is an aspect of the family environment, twins within identical and fraternal pairs would be at equal risk for alcohol use. If the confounding variable reflects genetic predispositions, identical twin siblings would have equal risk for alcohol use regardless of peer affiliation, whereas fraternal twin siblings would have less shared risk.

In more formal behavior genetic terms, variability in a risk factor like peer affiliation can be decomposed into genetic risk (A), shared or family environmental risk (C), and nonshared environmental risk (E). Nonshared environment refers to differences in risk among siblings reared together, after the effects of genetic and shared environmental confounds have been controlled. The majority of variation in adolescent behavior is a product of the environment unshared by siblings (Daniels and Plomin 1985). Siblings experience substantial differences in peer environments, and as such, peer relationships may be an important source of unique environmental context for siblings within a family (Daniels and Plomin 1985).

To date, only one study has considered the relationship between adolescent alcohol use and peer affiliation using family designs. Walden et al. (2004) examined early substance use and association with deviant peer groups using twin pairs. Peer group deviance was assessed using target report of his or her peer group's substance use and

delinquency and teacher report of peer group deviance. Environmental confounds completely explained the observed association between peer group deviance and target substance use. Due to limitations of family studies, the environmental confounds cannot be identified specifically, but these results highlight the importance of considering environmental and genetic confounds in understanding adolescent drinking.

Current Study

The present investigation addresses concerns raised regarding previous research of peers and alcohol use in adolescence. (1) To consider potential reporter bias, we measure peer alcohol use using direct peer report. Peers are identified within the sample using target nomination of their same-sex best friend. We also include target report of perceived peer alcohol use so potential differences in these sources of information can be elucidated. (2) To consider potential differences in how peers may influence subtypes of drinking, measurement models are created to examine subtypes of alcohol use. We use exploratory factor analysis on all adolescents in the first wave of data to identify two factors: frequency of alcohol use and problems due to alcohol use. We use confirmatory factor analysis to develop latent constructs specified using strong partial invariance to examine the stability of this structure across the second and third waves of data. (3) To consider genetic and environmental influences on stability and change in alcohol use, we combine twin and sibling methods and longitudinal methods. We use a simplex structure to model target alcohol use across three waves of data for the twin and sibling sample. We decompose the variance in Wave I of alcohol use and the residual variance of Waves II and III into additive genetic, shared environmental, and nonshared environmental components. (4) Finally, to consider unmeasured genetic and environmental confounds, we examine the nature of the relationship between peer affiliation and alcohol use using the twin and sibling sample. We use a multivariate twin and sibling design with peer alcohol use modeled as the risk. We expect that despite genetic and environmental influence, there will be a unique effect of peer affiliation on alcohol use.

Method

Participants

Data were obtained from the National Longitudinal Study of Adolescent Health (Add Health), which was designed to investigate adolescent health and risk behaviors with a special focus on the social contexts in which they occur (Udry 2003). Add Health includes a nationally representative sample of adolescents from 134 representative schools in the United States. The 134 schools, which are 70% of the schools initially recruited, include 80 high schools and their 7th and 8th grade feeder schools (although some high schools contain 7th and 8th grades and serve as their own feeder schools.) Study design included an in-school survey and three waves of in-home interviews. The confidential in-school survey (n=90,118), administered at 129 consenting schools during the 1994–1995 school year, included peer nominations and identification of adolescent siblings who may or may not have been included in the in-school survey. A sub-sample of eligible students, with deliberate oversampling of ethnic minorities, disabled students, and students with adolescent siblings, was selected to participate in a follow-up home interview (78.9% of the selected sample consented to participate). Adolescents who did not participate in the in-school portion were eligible for in-home interviews if they were siblings of respondents who completed the in-school questionnaire.

The Wave I in-home, 90-minute interview took place between April and December of 1995 and included 20,745 respondents (10,480 female, 10,264 male) between 11 and 21 years of age (M=16 years, 25th percentile=14 years, 75th=16 years). The Wave II in-home interview, completed the following year, included 14,738 adolescents (7,556 female, 7,182 male) between 11 and 23 years of age (M=16 years, 25th percentile=15 years, 75th=17 years). The Wave III in-home interview, designed to measure factors involved in the transition from adolescence to young adulthood, included 15,170 respondents (8,030 female, 7,167 male) and took place between August 2001 and April 2002. Participants were between 18 and 28 years of age at Wave III (M=22 years, 25th percentile=21 years, 75th percentile=23 years). Of the 20,745 participants at Wave I, there were 6,007 missing at Wave II and 5,548 missing at Wave III, leaving 14,738 participants (71.04%) with data available across all waves. Each wave includes a range of ages and does not represent distinct cohorts; as such, these analyses cannot identify characteristics of specific developmental periods.

The current study uses the sub-sample composed of all adolescents identified as monozygotic twin pairs (MZ), dyzogotic twin pairs (DZ), full biological siblings (FS), half siblings (HS), and genetically unrelated siblings (NR), subsequently referred to as the target sample (n=3,096 pairs). Twins' zygosity was determined primarily on the basis of self-report and four questionnaire items concerning how often twins were confused with each other and the similarity of their physical appearances. Previous studies have determined that such questions have high validity (greater then 90% accuracy) when compared to zygosity

determinations based on DNA (Spitz et al. 1996). In cases of uncertain zygosity determination, DNA was used to identify twins as MZ or DZ (Harris et al. 2003). For the current analysis, only same-sex dyads are used, as previous research has suggested that using opposite sex pairs may spuriously inflate genetic effects, since monozygotic twins are always concordant for gender (Walden et al. 2004). Overall there were 285 MZ pairs, 248 DZ pairs, 709 FS pairs, 222 HS pairs, and 356 NR pairs included in the analysis, for a total of 1,820 same sex pairs.

Respondents were asked to nominate up to five same-sex and five opposite-sex friends, starting with their closest friend. The same-sex, closest friend was used in the current analysis. Jaccard et al. (2005) have suggested that this friend type best predicts individual behavior. MZ pairs nominated the same friend significantly more often than DZ pairs (χ^2=104.095, df=1, p<0.0001) and twins (MZ and DZ pairs) nominated the same friend significantly more often than non-twins (χ^2=88.825, df=1, p<0.0001). One explanation for the greater likelihood of same-peer nomination by twins as compared to siblings may be differences within sibling pairs in age. To account for differences in ages among siblings that do not exist in twin pairs, age is included as a covariate in all twin and sibling analyses. Age is unlikely the only factor responsible for same-peer nominations as MZ pairs nominated the same friend more often then DZ pairs. This greater likelihood may also be attributable to the greater similarity between MZ twin pairs in personality and appearance than DZ pairs, which may lead to a greater likelihood of selecting the same peers due to these similarities. In the current study, a pair nominating the same peer is treated in the same manner as a pair nominating different peers who have the same level of alcohol use.

Peer nominations were considered valid if the target nominated a same-sex best friend and that peer was included in the in-school sample. Of the 3,640 individual adolescents that were part of a same sex sibling pair, 1,540 (42.31%) had valid peer nominations, 1,210 (33.24%) nominated peers for whom data was not collected, and 800 (21.98%) did not nominate peers. Adolescents without close friends, whether due to failure to nominate a peer or unavailability of the peer data, were modeled as missing. There were minor differences between adolescents who nominated peers not in the study and those who did not nominate peers at Wave I in alcohol use (R^2=0.19%) and at Waves I (R^2=0.24%) and II (R^2=0.38%) in alcohol problems. Participants who nominated a peer not included in the study were more likely to be female and younger than those who did not nominate a peer. However, the differences between these groups were small for both gender (R^2=0.13%) and age (R^2=0.97%). There were no differences in nomination status for zygosity or race. To consider differences in missingness due to covariates and outcomes, missing data analysis was included and considers age, gender, and alcohol use when imputing for missing data.

Measures

Target sample alcohol use measures Alcohol use for the sibling sample was measured by a series of questions from the three waves of in-home interviews. Adolescents were asked at all three waves at what age they first drank alcohol and how often in the past twelve months they drank alcohol, got drunk, and had at least five drinks in a row: every day or almost every day (1), 3 to 5 days a week (2), 1 or 2 days a week (3), 2 or 3 days a month (4), once a month or less (5), 1 or 2 days in the past 12 months (6), or Never (7). They were also asked how often in *the past twelve months*, due to drinking alcohol, they had sex or did something they later regretted, had a fight, were hung over, were sick, or got in trouble with their parents, friends, someone they were dating, or at school: never (0), once (1), twice (2), three to four times (3), five or more times. Research has suggested these measures are internally consistent and have construct validity (Resnick et al. 1997).

Peer sample substance use measures Although siblings were targeted from the in-school sample to be included in the in-home data collection, sibling peers were not. Therefore, few nominated peers were included in the in-home interviews, and the in-school data alone were used to measure direct peer report of risk behavior. Peer report of risk behaviors was assessed in the in-school questionnaire using seven items that ask how often in the past 12 months respondents smoked cigarettes, drank alcohol, got drunk, did something dangerous because they were dared to, raced on a skateboard, roller-blades, or in a car, skipped class, and lied to their parents: Never (0), once or twice (1), once a month or less (2), 2 or 3 days a month (3), once a week (4), 3–5 days a week (5), and nearly everyday (6).

In-home data were used to measure target perception of peer behavior. As research considering the relationship between peer and target behavior generally uses target report of peer behavior, the items "how many of your three closest friends drink," and "how many of your three closest friends smoke" from the in-home Wave I questionnaire were included to compare direct peer report of alcohol use behavior to target report of perceived peer behavior.

Statistical Analyses

Measurement models To develop latent multivariate models of relevant constructs for later use in structural equation models, Exploratory Factor Analysis (EFA), and subsequent Confirmatory Factor Analysis (CFA) were conducted

Table 1 Items and factor loadings used to estimate factor scores for target substance use

	In-home wave		
	I	II	III
Alcohol use			
How many days did you drink alcohol?	1.00	1.00	1.00
How many days did you drink five or more drinks in a row?	1.04	1.04	1.04
How many days have you gotten drunk on alcohol?	1.05	1.05	1.05
Cronbach's alpha	0.90	0.90	0.94
Alcohol problems			
Trouble w/parents because of drinking	1.00	1.00	1.00
Problems at school because of drinking	0.98	0.98	
Problems w/friends because of drinking	1.05	1.05	1.05
Problems w/dating because of drinking	1.02	1.02	1.02
Did something later regretted because of drinking	1.15	1.15	
Were hung-over	1.20	1.20	1.20
Threw up because of drinking	1.12	1.12	1.12
Sexual situation later regret because of drinking	1.02	1.03	1.03
Physical fight because of drinking	1.01	1.01	1.01
Driven while drunk			1.06
Drunk at school or work			0.91
Cronbach's alpha	0.94	0.94	0.93

on the observed indicators of alcohol use for the in-home sample. The items submitted to EFA and CFA at each wave are summarized in Tables 1 and 2. There were 167 (0.81%) participants missing alcohol use data at Wave I, 60 (0.41%) at Wave II, and 71 (0.47%) at Wave III. There were 14,678 (70.75%) participants with complete alcohol use across all waves ($n_{\text{Wave I}}$=20,578, $n_{\text{Wave II}}$=14,678, $n_{\text{Wave III}}$=15,099); missing data analysis was included. An approximation of the EFA solution at Wave I was tested using CFA in the full Add Health data set for the in-home alcohol use items with zeros substituted for estimated loadings less than 0.40. Alcohol use behaviors change from adolescence into adulthood, and although many items were repeated between waves, other behaviors were not assessed at all waves. For example, driving while drunk and being drunk at work were only assessed during adulthood, whereas having problems due to drinking at school was only assessed in adolescence. To evaluate whether items measured the same construct across the three waves, a restricted model in which the factor loadings of items in common between waves were constrained to be equal across waves was compared to a less restricted model in which the factor loadings were allowed to differ across waves.

The most restricted CFA (*strong partial factorial invariance*; Meredith 1993) was specified as follows: items in common across multiple waves were constrained to load onto the same factor for all waves in which they were used; factor loadings for items in common across waves were constrained to be equal for all waves in which they were used; and item thresholds (intercepts) for the categorical items were constrained to be equal for items in common across waves. When strong partial invariance holds across waves, it can be inferred that the same latent construct is being measured at each wave on the basis of common items, even though other items vary from wave to wave. The term *partial* invariance refers to invariance for only those items in common between waves (Byrne et al. 1989). This restricted model is compared to a series of less-restricted models, in which factor loadings, thresholds, or both were allowed to differ across waves.

Longitudinal twin and sibling models of alcohol use The three waves of factor scores for the twin and sibling pairs were used to estimate the genetic and environmental influences on changes in alcohol use and alcohol problems across Waves I–III. Of the 3,640 participants that were a part of a same sex sibling pair, 3,543 (97.34%) had alcohol use data. Missing data analysis was included and age and gender were included as covariates. The simplex twin model, showing only one twin per pair for clarity, is

Table 2 Items and factor loadings used to estimate factor scores for peer substance use

Peer substance use	In-school
Smoke cigarettes	1.00
Drink beer, wine, or liquor	0.99
Get drunk	1.05
Cronbach's alpha	0.84

illustrated in Fig. 1b. The factor scores for Wave III were regressed onto the factor scores for Wave II (*b23*), and the factor score for Wave II was regressed onto the factor score for Wave I (*b12*).

ACE decomposition of simplex model The variance at Wave I and the residual variance of Waves II and III were decomposed into additive genetic influences (A), environmental influences shared by siblings (C), and environmental influences unique to siblings (E). Decomposition into ACE components was achieved by considering the different proportions of segregating genes shared by twin and sibling dyads (MZ=100%, DZ/FS=50%, HS=25%, NR=0%). Twin and sibling models depend on several assumptions,

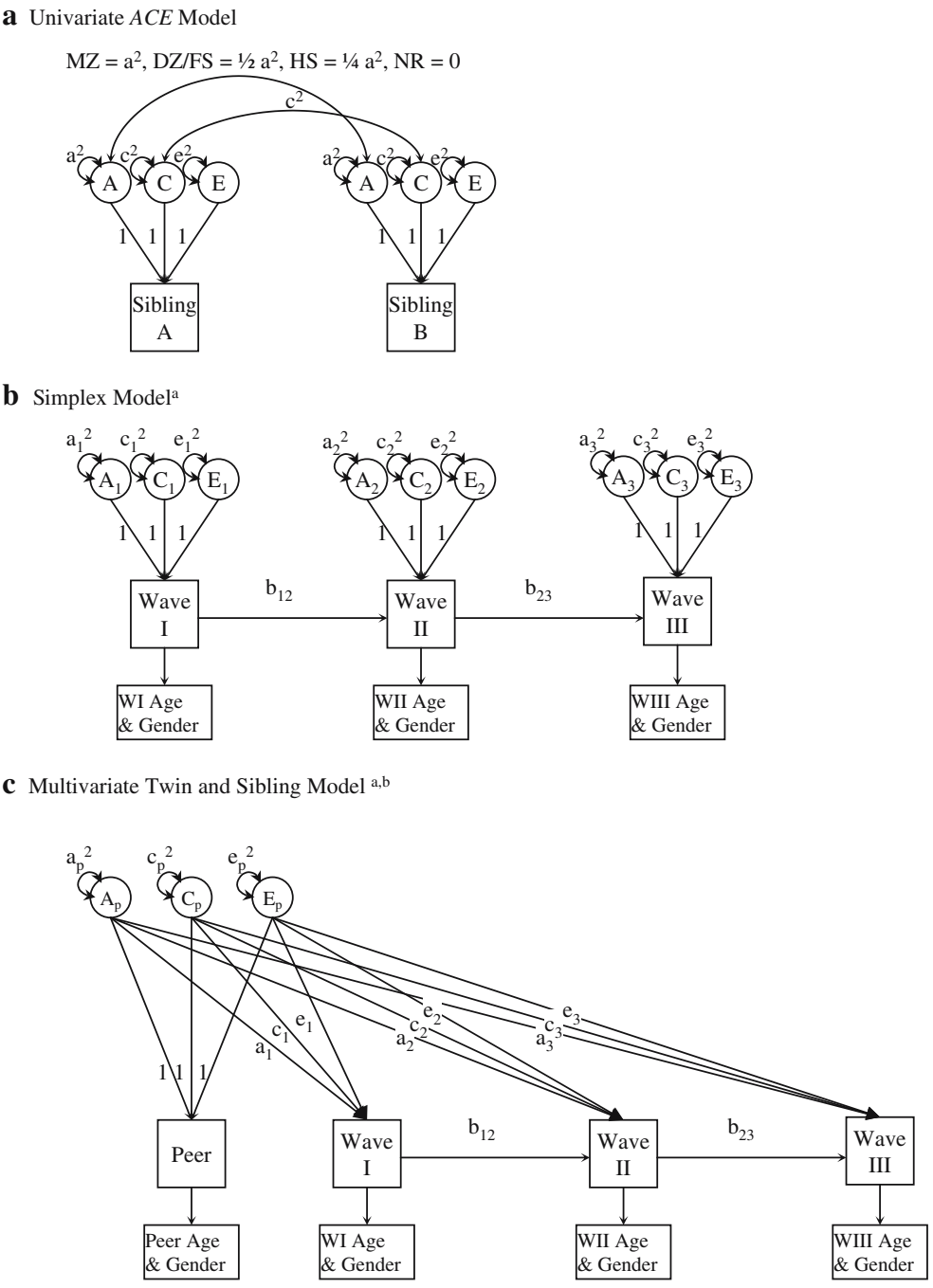

Fig. 1 General univariate (**a**), simplex (**b**), and multivariate twin and sibling (**c**) models

[a] One twin shown for clarity
[b] Waves I-III are decomposed into *ACE* as in Simplex Model above, complete Simplex Model not shown for clarity

including random mating in the parental generation, similar environments for sibling and twin types (MZ environment is assumed to not differ systematically from DZ environment), and no gene-environment interaction. The model we employed to decompose the variances into ACE components is illustrated in Fig. 1a. The paths from the latent genetic and environmental variables are fixed to one and the variances of the ACE components are estimated. For more detailed information about the logic and methodology of behavior genetic modeling, see Neale and Cardon (1992).

Multivariate twin and sibling models The associations between peer and target behavior were analyzed as a combination of genetic confounds, environmental confounds, and quasi-causal pathways using the twin and sibling data. Peer characteristics were modeled as the risks, and the simplex models of the alcohol use factors constituted the outcomes. For each alcohol use factor, the simplex components across the three waves were regressed onto the ACE variance components for the peers (Fig. 1c). All of the 3,543 participants with alcohol use data had available data for target report of peer substance use and 1,540 (43.47%) participants had available peer report of alcohol use. Age and gender were used as covariates and missing data analysis was included.

On occasion, variance estimates for *C* were negative either for the outcome or risk. This pattern may be the result of sampling error, or it may suggest that there are dominance or epistatic processes at work. Negative variance estimates are not interpretable, and in each case, a negative variance estimate was fixed to zero and the change in model fit was assessed.

Missing data analysis Missing data were considered using maximum likelihood (ML) under the assumption that data were missing at random (MAR). The MAR assumption permits missingness in peer data to be a function of measured covariates and target alcohol use. However, MAR assumes that missingness of peer data is unrelated to the level of peer alcohol use after controlling for the level of target alcohol use and measured covariates. If measured covariates and the target alcohol use explain the relationship between missingness and peer alcohol use, missingness is considered to be a function of the covariates and target alcohol use rather then peer alcohol use. MAR cannot be tested as it is impossible to know the true values of missing data, however, ML is fairly robust to minor violations of this assumption (Allison 2002). Maximum likelihood integrates over all possible values of missing peer data, and gives more weight to values that are more likely (Allison 2002; Little and Rubin 1987).

Software and model fitting All analyses were conducted in Mplus (Muthen and Muthen 2004). Mplus uses a probit model to estimate variances and covariances for ordered categorical items. Promax rotation was used for all EFAs. Parameters for the EFA were estimated using weighted least squares estimation. Confirmatory models were estimated using ML, and model fit was assessed using RMSEA, CFI and TLI. RMSEA measures the amount of error per model parameter in approximating the data and values less then 0.05 generally indicate adequate fit (Steiger 1990). The CFI and TLI are normed fit indices that range from zero to one with values greater then 0.95 indicating adequate fit (Bentler 1990; Hu and Bentler 1998). Missing data were estimated using the assumption of MAR, using options available in Mplus.

Results

Measurement Models

Target alcohol use Separate EFAs were conducted for alcohol use items at the three waves using the full in-home sample (items are summarized in Tables 1 and 2). The EFA of alcohol use items for Wave I yielded two eigenvalues greater than one (7.435 and 1.562), and a scree plot suggested two factors. The two-factor solution fit well (RMSEA=0.038), whereas the one-factor solution did not (RMSEA=0.160). The factors retained suggested one factor representing frequency of alcohol use, which included items assessing the number of times the adolescent drank in the past year and how many drinks they had each time, and one factor representing problems due to alcohol use, which included items assessing physical symptoms of drinking and problems in a variety of social contexts due to alcohol use.

The measurement models for alcohol use developed for Wave I were applied to the Wave II and III data, fixing the loadings for the items in common with Wave I to be equal across the three waves (see Tables 1 and 2). The strong partial invariance model fit well for the alcohol use factors (RMSEA=0.039, CFI=0.991, TLI=0.996) and the factors had high internal reliability (Cronbach's alpha >0.80 for all factors, see Tables 1 and 2). Factor scores were estimated for each wave and factor of the in-home data yielding the following six factor scores: frequency of alcohol use (AU-1, AU-2, and AU-3), and problems relating to drinking alcohol (AP-1, AP-2, and AP-3).

Peer substance use Using the full in-school sample, the six externalizing items were submitted to EFA. There were two eigenvalues greater than one (2.707 and 1.188), and the scree plot suggested two factors. The two factor solution fit well (RMSEA=0.019), whereas the one factor solution fit

Table 3 Regression coefficients for simplex models of target alcohol use

Alcohol use	Frequency			Problems		
Regression	*b*	SE	R^2	*b*	SE	R^2
Wave II on I	0.73	0.01	0.54	0.84	0.01	0.70
Wave III on II	0.37	0.02	0.08	0.58	0.02	0.27
RMSEA	0.049			0.048		
CFI	0.956			0.974		
TLI	0.974			0.979		

was poor (RMSEA=0.109). The two-factor solution yielded a substance use factor and a rule-breaking factor which were moderately correlated (r=0.477). Using the factor structure suggested by the EFA, a CFA was fit where items with loadings less than 0.40 were set to zero. The CFA fit well (RMSEA=0.037, CFI=0.959, TLI=0.938), with moderate correlation between the factors (r=0.548). Factor scores were computed, and scores were matched based on best friend nominations. We only used the substance use factor in the present analyses given our focus on that topic. The substance use factor is used for subsequent analyses to represent peer report of substance use (SU-PR). See Tables 1 and 2 for factor loadings.

Target report of peer substance use (SU-TR) was computed by combining the target's report of the number of their friends who drink (0: n=1,563, 44.95%, 1: n=716, 20.60%, 2: n=498, 14.33%, and 3: n=698, 20.09%) and the number who smoke (0: n=1,866, 53.61%, 1: n=749, 21.52%, 2: n=416, 11.95%, and 3: n=450, 12.93%). SU-TR and SU-PR were correlated (r=0.394, n=1,507, p<0.0001).

Longitudinal Twin and Sibling Models

We then fit longitudinal simplex models to the factor scores estimated in the three waves. Complete results for the simplex models are shown in Tables 3 and 4. Wave I alcohol use accounted for 73.41% (AU) and 84.15% (AP) of the variance in Wave II. The variance in Wave II accounted for 37.09% (AU) and 58.01% (AP) of the variance in Wave III. Genetically informative twin and sibling models were used to decompose the variability in the simplex models into genetic and environmental components. The residual variances for Wave II and III generally had a greater non-shared environmental component (which also includes error variance) than the variance in Wave I. However, most of the factors retained portions of genetic, shared environmental, and nonshared environmental residual variance across all three waves.

Associations between Target and Peer Behavior

Correlations, shown in Table 5, between peer factors and target factors were moderate to large for target alcohol use. As expected, the relationship between target behavior and peer self-report of behavior was less than that for target behavior and target report of peer behavior. This difference may be reflective of reporter bias, and is generally consistent with previous findings (Kandel 1996).

The correlations between the target behavior and the peer of the target's twin, known as *cross correlations*, are shown in Table 5. Although not a formal examination of potential genetic and environmental confounds, examination of cross correlations provides an intuitive illustration of effects identified through structural equation modeling. The pattern of cross correlations involving peer self-report were generally suggestive of genetic factors confounding the relationship between peer and target behavior. For example, DZ twins' alcohol use was generally more similar to their own peers' report of substance use (r=0.42) than it was to their twins' peer report of substance use (r=0.21). MZ twins' alcohol use, however, was about as similar to their

Table 4 ACE variance components for simplex models of target alcohol use

	Proportion of variance accounted for by ACE components at each wave					
	I	II[a]	III[a]	I	II[a]	III[a]
A	0.31	0.18	0.21	0.32	0.18	0.25
C	0.14	0.07	0.19	0.20	0.09	0.13
E	0.55	0.75	0.60	0.48	0.73	0.62

[a] Proportion of residual variance not accounted for by previous wave.

Table 5 Correlations between peer substance use and the alcohol use of the target adolescent and the target's twin (cross correlations)

	Peer substance use[b]									
	Peer's report					Target/twin report of peer				
	Full[a] (n=1,540)	MZ (n=234)		DZ (n=205)		Full[a] (n=1,540)	MZ (n=234)		DZ (n=205)	
	Target	Target	Twin	Target	Twin	Target	Twin	Target	Target	Twin
Frequency	0.29	0.38	0.33	0.42	0.21	0.45	0.52	0.38	0.54	0.32
Problems	0.36	0.47	0.42	0.44	0.33	0.53	0.69	0.45	0.60	0.42

[a] Full includes adolescents from MZ, DZ, FS, HS, and NR pairs.
[b] All correlations significant at $p<0.01$.

own peers' report of substance use (r=0.38) as it was to their twins' peer report of substance use (r=0.33). This suggests that genotype, shared completely by MZ twins, influences both peer selection and alcohol use. DZ twin pairs may share less in common with their co-twin's peer because they may not share genetic liabilities for peer selection and alcohol use. Although differences between cross correlations were not significant, the MZ twin cross correlations exceeded the DZ twin cross correlations for frequency of alcohol use and alcohol problems. This pattern may suggest that factors influencing the association between peer selection and alcohol use are genetic in nature.

For target report of peer behavior, the pattern of cross correlations was consistent with shared environmental confounds, and possible additional causal effect of peers. Target alcohol use was moderately correlated with the twin's report of peer substance use suggesting a confounding process. Environment shared by pairs may be responsible for this confound as indicated by the similar patterns of correlations for MZ and DZ pairs. For both MZ and DZ pairs, target alcohol use was more similar to target report of his peer's substance use (r_{MZ}=0.52, r_{DZ}=0.54) than it was to twin report of his peer's substance use (r_{MZ}=0.38, r_{DZ}=0.32). Possible causal peer influence was indicated by generally smaller cross correlations when compared to the correlations between adolescents and their own peers for both MZ and DZ twin pairs. This deflated cross correlation may, alternatively, represent reporter bias. Whereas the correlation between target and peer contains method variance, the target reported on self and peer behaviors, the cross correlation between the target and the twin's peer no longer has method variance as the target reported on self and the twin reported on peer.

Multivariate Twin and Sibling Models

Structural equation modeling was used to test more formally relationships suggested by cross-correlations. The multivariate twin and sibling model regressed the factor scores for each wave onto the ACE components for the peer

Table 6 Fit indices and model comparison for multivariate twin and sibling models

Factor		Coefficients free					Coefficients equal		
Peer	Target	χ^2	df	CFI	TLI	RMSEA	Wave	$\Delta\chi^2$	Δdf[a]
Peer report of substance use	Alcohol use	472.12	300	0.96	0.97	0.040	1	40.10	1
							2	12.67	1
							3	3.56	1
	Alcohol problems	459.31	300	0.97	0.98	0.038	1	63.40	1
							2	7.54	1
							3	9.05	1
Target report of peer substance use	Alcohol use	443.42	305	0.97	0.98	0.035	1	22.19	2
							2	29.94	2
							3	7.69	2
	Alcohol problems	443.23	305	0.98	0.99	0.035	1	46.56	2
							2	24.02	2
							3	6.87	2

[a] The variance of the C component for peer self-report of substance use was not significantly different from zero and this parameter was set equal to zero. Consequently, when the coefficients were set equal, the degrees of freedom changed only one in peer report of substance use (setting $a=e$) and changed two for target report of peer use (setting $a=c=e$).

factors to estimate genetic and environmental confounds in the relationship between having substance using peers and drinking alcohol. The two target factor scores were regressed onto the two peer factor scores for a total of four models. The models fit well with all RMSEAs falling below 0.05 (see Table 6 for complete model fit indices).

Phenotypic regressions The regression coefficients for target factors on the ACE components of the peer factors were constrained to be equal to test the equality of between- and within-family associations. When constrained to be equal, the estimated regression parameter represents the phenotypic relationship between peer and target behavior. Equal ACE regressions are consistent with a causal model, because they indicate that the coefficients are not explained by factors that make families different (i.e., genetic or shared environmental selection). In contrast, unequal ACE regressions indicate selection effects, inconsistent with assumed peer causal influences on substance use. There were several significant relationships indicating phenotypic relationships between peer and target behavior. Constraining the coefficients to be equal resulted in a significant loss of fit across models (see Table 6 for changes in model fit). In Table 7, the estimated parameter in the fixed coefficient model reflects the between-family association, and in the free coefficient model, the within-family association is reflected in the *e* coefficient. In all cases, genetic and environmental confounds were indicated as the between-family associations were larger than the within-family associations between target and peer behavior (see D'Onofrio et al. 2005).

Quasi-Causal Relationships

Non-shared environment regressions Table 7 shows the regression coefficients for target behavior on the ACE components of peer behavior. There were no significant paths on the *E* component of peer report of substance use. The relationship between peer substance use and target alcohol use at Wave I, and the changes in target alcohol use at Waves II and III indicated by the phenotypic regressions were completely explained by shared environmental and genetic confounds at each wave. The phenotypic regression coefficients were larger than the coefficients on the *E* component of peer behavior indicating the influence of genetic and environmental confounds. For example, the phenotypic regression of target problems due to alcohol use at Wave I on peer report of substance use (b=0.21) exceeded the regression on the *E* component (e=0.00), which reflects the relationship between target and peer behavior after considering genetic and environmental confounds.

For target report of peer substance use, the paths on *E* were generally smaller than the phenotypic regression coefficients indicating genetic or environmental confounds. However, there were significant paths on *E* at Wave I for

Table 7 Parameter estimates for multivariate twin and sibling models

Alcohol use		Wave	Coefficients equal[a]		Coefficients free[a]								
					a			*c*[b]			*e*		
Peer	Target		Est.	SE	Est.	SE	R^2	Est.	SE	R^2	Est.	SE	R^2
Peer report	Frequency	1	0.17*	0.02*	0.71*	0.11*	0.33*	0	–	–	0.10	0.05	0.01
		2	0.06*	0.01*	0.32*	0.08*	0.15*	0	–	–	0.06	0.03	0.00
		3	0.09*	0.02*	0.25*	0.09*	0.03*	0	–	–	0.03	0.04	0.00
	Problems	1	0.21*	0.02*	0.72*	0.09*	0.35*	0	–	–	0.00	0.00	0.00
		2	0.03	0.01	0.43*	0.06*	0.39*	0	–	–	0.02	0.06	0.00
		3	0.02	0.02	0.15	0.10	0.02	0	–	–	0.02	0.04	0.00
Target report	Frequency	1	0.12*	0.01*	0.11*	0.03*	0.06*	0.26*	0.05*	0.14*	0.09*	0.02*	0.03*
		2	0.03*	0.00*	0.05	0.02	0.02	0.10*	0.03*	0.09*	0.01	0.01	0.00
		3	0.03	0.01	0.01	0.03	0.00	0.13	0.05	0.05	0.02	0.02	0.00
	Problems	1	0.14*	0.00*	0.14*	0.02*	0.11*	0.30*	0.04*	0.18*	0.08*	0.02*	0.03*
		2	0.01	0.00	0.00	0.01	0.00	0.09*	0.03*	0.16*	0.00	0.01	0.00
		3	-0.01	0.01	0.06	0.02	0.02	0.08	0.04	0.03	0.01	0.02	0.00

* $p<0.05$; parameter estimates that are significantly greater than zero.

[a] "Coefficients equal" indicates that the parameters *a*, *c*, and *e* are fixed to be equal to test if the phenotypic association represents a causal relationship between peer alcohol use and target alcohol use. The coefficient here represents the magnitude of the phenotypic association. "Coefficient free" indicates that these parameters are permitted to vary to differentiate between peer influence and shared liabilities to choose alcohol using peers and to use alcohol.

[b] The variance of the *C* component for peer self-report of substance use was not significantly different from zero. This parameter was not estimated, and subsequently, the target factor was regressed onto only the *A* and *E* components of peer self-report of substance use.

target alcohol use (R^2=3.44%) and target alcohol problems (R^2=3.39%), which is consistent with a potential role of peer influence on adolescent alcohol use. A regression on *E* indicates that after controlling for genetic and shared environmental confounds, the sibling who is exposed to more alcohol using peers will engage in more alcohol use than his or her co-sibling. The within-family association is not free from confounds. Rather, within-family associations are confounded by factors that vary systematically between siblings. For example, the twin with more alcohol using peers may engage in more conflict with his parents, and this conflict may cause the twin to engage in more alcohol use behavior than his co-twin. Nevertheless, it is important to stress that within-family associations have fewer potential confounds than between-family associations and present a stronger case for causal peer influence.

Genetic confounds Regressions on the *A* variance component estimated the extent to which genetics confound the relationship between affiliating with alcohol using peers and drinking alcohol. For the *A* variance component of peer report of substance use, regressions for both Wave I target outcomes were significant and accounted for 33.21% (frequency of alcohol use) and 35.19% (alcohol problems) of the variance in the outcome. Additionally, the *A* component of peer report of substance use accounted for moderate portions of variance in Wave II target alcohol use (14.87%) and alcohol problems (39.27%), and variance in Wave III alcohol use (2.65%). The regressions of Wave I alcohol use and alcohol problems on the *A* variance component of target report of peer substance use were also significant and accounted for 5.59% and 10.74% of the variance in alcohol use and alcohol problems at Wave I respectively.

Environmental confounds Regressions on the *C* variance component estimate the extent to which shared environmental factors confound the relationship between having substance using peers and drinking alcohol. The variance of the *C* component for peer report of substance use was estimated to be negative, but was not significantly different from zero (95%CI=[−0.107,0.051]). Constraining the variance of *C* to be zero and estimating only the variances of *A* and *E* did not result in a significant loss of fit ($\Delta\chi^2$=0.321, Δdf=1). The *C* component of peer report of substance use and subsequent regressions onto the component were not included in the multivariate analysis.

For target report of substance use, the regressions on *C* from all target factors at Wave I were significant and accounted for between 14.66% (frequency of alcohol use) and 17.80% (alcohol problems) of the variance in outcome at Wave I. Additionally, the regressions on *C* for alcohol use and alcohol problems at Wave II were significant and accounted for 8.73% and 15.88% of variance in target outcome respectively.

Discussion

The causal influence of peers on adolescent alcohol use is often overestimated due to reliance on target report of peer behavior, methods that do not differentiate between peer effects on adolescents and adolescent effects on peer selection, and methods that do not consider potential confounds that may influence both peer selection and alcohol use (Bauman and Ennett 1996; Kandel 1996). Accordingly, the current study used a longitudinal family design to examine the relationship between direct report of peer behavior and target alcohol use. This is the only study to apply genetically informed data to the relationship between peer and adolescent alcohol use using direct peer report.

Univariate Decomposition of Affiliation with Substance Using Peers and Alcohol Use

Consistent with previous research, target alcohol use and alcohol problems reflected a combination of genetic and environmental influences (Rhee et al. 2003). Although previous findings suggested that alcohol problems have a greater relative genetic influence than alcohol use (Rhee et al. 2003), there were no discernable differences between genetic and environmental influences on problem alcohol use and frequency of alcohol use. Decomposition of the changes in alcohol use from adolescence into adulthood reflected primarily nonshared environmental influences, but retained genetic and shared environmental portions as well.

Genetic and nonshared environmental variance completely accounted for the variance in peer alcohol use. This is consistent with previous research on direct peer report of substance use (Cleveland et al. 2005). For target report of peer substance use, shared environment accounted for nearly 20% of the variance. Target perception of peer behavior overestimated the association between adolescent and peer behavior, and this overestimation appeared to reflect shared environmental influences.

Association Between Affiliation with Substance Using Peers and Alcohol Use

Target alcohol use and alcohol problems were related to both peer report of substance use and target report of peer substance use. Applying the quasi-causal model to these phenotypic relationships demonstrated little support for causal peer influence. The association between peer and adolescent behavior may reflect other processes, including

selection attributable to genetic and shared environmental factors. Although it was expected that the relationship between peer affiliation and alcohol use would change over time, genetic and shared environmental factors also account for the relationship between peer alcohol use and changes in adolescent alcohol use.

Associations between adolescent outcomes and target report of peer substance use reflected predominantly shared environmental pathways. The confounding role of shared environment in the association between alcohol use and target report of peer substance use was consistent with research that included teacher and target report of peer behavior (Walden et al. 2004). The shared environmental influences did not completely account for the relationship, and nonshared environment and genetic influence accounted for additional covariance in alcohol use. Target perception of peer substance use may influence target alcohol use after considering the roles of genetics and shared environment.

The relationship between direct peer report of substance use and alcohol use in the target adolescent was explained by genetic factors. Genes may influence the correlation between two observed variables in several ways. Two mechanisms through which gene-environment correlations (rGE) may be at work in the current association are active and evocative rGE (Plomin et al. 1977). Active rGE suggests that an individual is influenced by his or her genes to seek out certain environments. For example, an individual genetically predisposed to drink alcohol may seek out other individuals predisposed to drink alcohol. Evocative rGE suggests that an individual's genes cause others to act in certain ways toward him or her. For example, an adolescent who is predisposed to drink alcohol will attract peers who drink alcohol. In this case, as peer selection is a reciprocal process in which adolescents and peers select each other, it is plausible that both active and evocative rGE could be responsible for the observed association. The finding that genes may influence both peer selection and alcohol use behavior does not imply that genes determine characteristics. The majority of variance in alcohol use was attributable to environmental factors; however, the covariance between alcohol use and peer alcohol use is largely attributable to genetic factors. Future research is needed to determine what environmental influences contribute to variation in alcohol use.

Direct Peer Report and Target Report of Peer Behavior

Consistent with previous research, target report of peer behavior overestimated the similarity between target and peer behavior (Bauman and Ennett 1996). The bias appeared to be systematic; the covariance between target report of peer behavior and target alcohol use reflected primarily shared environmental influence, and the covariance between direct peer report and target alcohol use reflected primarily genetic influences. After considering genetic and environmental confounds, peer behavior does not appear to influence target behavior, whereas target perception of peer behavior may influence target behavior.

Differences between peer behavior and target perception of peer behavior may have important treatment and prevention implications for adolescent substance use. Target perception of peer behavior may have a causal influence where actual peer behavior does not after considering shared environmental and genetic confounds. Perhaps mechanisms which cause target perception to differ from peer behavior may affect alcohol use. Treatment and prevention may be most helpful when focusing on adolescents' perceptions and quality of relationships rather then focusing on affiliation with risk-taking peers.

Future Directions and Limitations

Twin models of the kind employed here assume that there is no gene-environment interaction (GxE) and include any GxE effects in the genetic confound. As this paper identifies genetic confounds, future work might use emerging methods that may be able to examine GxE and rGE simultaneously (Eaves et al. 2003).

Missing data are another possible concern. Missing peer reports due to failure to nominate a peer or nominated peers who did not provide data were both treated as missing. Missing data analysis showed only minor differences between these groups on measured covariates and outcomes. However, it is possible that there were important, unmeasured differences between groups. The proportion of missing data may also produce biased estimates as the limitations of the missing data analysis used in the current study have not been tested for complex genetic models. Evaluation of ML under MAR with simpler models suggests that with samples of similar size and high rates of missing data (nearly 80%) ML under MAR performs adequately (Schafer and Graham 2002).

The ages of the youth in this study spanned the entire range of adolescence, yet life experiences within certain developmental periods may alter peer influence on adolescent alcohol use (Fergusson et al. 2002). Adolescents who begin drinking at a young age also report being more susceptible to peer influence (Flory et al. 2004). Thus, another task for future research is to consider the relative influence of peers at different stages of adolescent development.

While we focused on best friends, one peer does not represent the entire peer group. Future research may benefit from examining the influences of the entire peer group, same and opposite sex, on alcohol use in adolescents. Peer dynamics including changes in peer groups, popularity,

quality of friendships, and parental involvement in peer interactions may be important moderators between engagement in substance use and affiliation with substance using peers (Dishion et al. 2004; Lansford et al. 2003; Sullivan et al. 2006; Urberg et al. 2003).

Finally, we should note that, while best-friends may not influence alcohol use, at risk youth still can be adversely affected by associations with deviant peers. Research suggests that congregating deviant youth in treatment groups may increase substance use (Dishion and Dodge 2005a, b). Perhaps adolescents in treatment groups are more vulnerable to peer influence or the peers in such groups have a particularly powerful, negative influence.

Conclusions

The use of family studies can identify true causal mechanisms which can aid in treatment and prevention of substance use in adolescence. Although consideration of genetic and environmental confounds leads to reduced estimates of the magnitude of the causal relation between peers and adolescents, our results do not indicate that peers are inconsequential to adolescent development. Peers may affect development and behavior in a variety of domains not considered in the current study. Nevertheless, our results indicate that peer substance use did not influence adolescent alcohol use when genetic and shared environmental confounds were considered. Despite a phenotypic association between peer and target alcohol use, genetic and shared environmental factors largely explained this relationship. Failure to consider genetic and environmental factors affecting peer selection may overestimate peer influence over adolescent alcohol use.

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