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Peer Network Drinking Predicts Increased Alcohol Use From Adolescence to Early Adulthood After Controlling for Genetic and Shared Environmental Selection

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Abstract

Research consistently links adolescents' and young adults' drinking with their peers' alcohol intake. In interpreting this correlation, 2 essential questions are often overlooked. First, which peers are more important, best friends or broader social networks? Second, do peers cause increased drinking, or do young people select friends whose drinking habits match their own? The present study combines social network analyses with family (twin and sibling) designs to answer these questions via data from the National Longitudinal Study of Adolescent Health. Analysis of peer nomination data from 134 schools ($n = 82,629$) and 1,846 twin and sibling pairs shows that peer network substance use predicts changes in drinking from adolescence into young adult life even after controlling for genetic and shared environmental selection, as well as best friend substance use. This effect was particularly strong for high-intensity friendships. Although the peer-adolescent drinking correlation is partially explained by selection, the present finding offers powerful evidence that peers also cause increased drinking.

Keywords

alcohol use; adolescent; twins; social network analysis; friends

Peer drinking is widely viewed as a potent influence on adolescent alcohol use (e.g., Bauman & Ennett, 1996; Crosnoe & McNeely, 2008). Despite this popular belief, empirical evidence needs to be interpreted cautiously for two basic reasons. First, adolescents can and do select peers with similar drinking habits (e.g., Urberg, Luo, Pilgrim, & Deglirmencioglu, 2003), raising the basic and essential question of whether the peer-teen drinking correlation is due to selection or causation. Second, studies of peer influences have focused on best friends and perceptions of peer drinking, partly due to ease of assessment. Yet, true peer influences may result from norms set by complex peer networks. In fact, recent reviews highlight both concerns (Brown, Bakken, Ameringer, & Mahon, 2008; Hartup, 2005), which we address in the present study, which combines social network analysis with a genetically informed, longitudinal design to examine the influence of peer group alcohol use on adolescent drinking after controlling for genetic and shared environmental selection.

Similarity between friends likely owes to a combination of selection, where adolescents choose and are chosen by friends who engage, or are likely to engage, in similar behaviors, and causation, where friends actually influence one another (e.g., Hartup, 2005). Thus,

cross-sectional associations between the alcohol use of adolescents and their peers overestimate peer influences both theoretically and empirically (e.g., Arnett, 2007; Bauman & Ennett, 1996; Poelen, Engels, Van Der Vorst, Scholte, & Vermulst, 2007; Urberg et al., 2003). Selection may owe directly to alcohol use, as most adolescents become friends with adolescents who engage in similar behaviors (Allen, Porter, & McFarland, 2006; Simons-Morton & Chen, 2006; Urberg et al., 2003), or be a more general process of association with externalizing youth (e.g., Patterson, DeBaryshe, & Ramsey, 1990). Other possible selection effects influencing similarity in friendship include third variables such as socioeconomic status, neighborhood, and school (Leventhal & Brooks-Gunn, 2000); family characteristics such as attitudes toward risky behavior (Jaccard, Blanton, & Dodge, 2005), parent-youth attachment (Allen, Moore, Kuperminc, & Bell, 1998; Allen, Porter, McFarland, McElhaney, & Marsh, 2007), and parental monitoring (Kiesner, Poulin, & Dishion, 2010; Padilla-Walker, 2006); and personality such as hyperactivity (Young, Heptinstall, Sonuga-Barke, Chadwick, & Taylor, 2005) and temperament (Wills & Cleary, 1999).

Longitudinal studies have documented the importance of selection in the relationship between adolescent and peer behaviors (e.g., Bauman & Ennett, 1996; Urberg et al., 2003). Further, longitudinal designs have found changes in alcohol use paralleling changes in adolescent friendships (Poulin, Kiesner, Pedersen, & Dishion, 2011; Simons-Morton & Chen, 2006) and changes in the influence of friends over time (Poelen, Scholte, Willemsen, Boomsma, & Engels, 2007). Despite these advances, traditional longitudinal studies can only control for selection variables that are measurable and measured. It always remains possible that unmeasured factors contribute to selection in important ways.

Genetically informed designs, in contrast, offer many advantages for parsing selection from causation. For example, when comparing two groups of unrelated individuals, observed differences in alcohol use may due to genetic influences on drinking behavior or environmental experiences such as growing up with an alcoholic parent. However, if we compare groups of monozygotic (MZ) twins, differences alcohol use cannot be attributed either to genetic factors or to shared environmental experiences whether measured or unmeasured. Observed differences between MZ twins must be due to the nonshared environment, such as differences in peer group drinking (to the extent that MZ twins have different peer groups). Thus, twin studies and other genetically informed research designs can parse selection from causation by controlling for genetic and shared environmental selection (whether or not key aspects of the shared environment have been or even can be measured). This leaves observed differences attributable to the nonshared environment, quite likely the targeted nonshared experience, peer drinking in the present context. Because it remains possible that some other nonshared experience accounts for any observed effect, however, we term the conclusions of genetically informed designs “quasicausal” rather than “causal.”

Genetically informed designs have demonstrated both shared environmental and genetic selection in explaining the relationship between adolescent and friend drinking and other problem behaviors (Hill, Emery, Harden, Mendle, & Turkheimer, 2008; Kendler et al., 2007; Poelen, Engels, et al., 2007; Walden, McGue, Iacono, Burt, & Elkins, 2004). Specifically, the relationship between alcohol use and perceptions of *best friends'* alcohol use is accounted for largely by shared environmental influences (Hill et al., 2008; Walden et al., 2004), whereas the relationship between alcohol use and best friend report of substance use is completely accounted for by genetic influences (Hill et al., 2008) for all but high-risk adolescents (Harden, Mendle, Hill, Turkheimer, & Emery, 2008). In short, genetically informed research finds little support for best friend influence when controlling for genetic and shared environmental selection. However, existing genetically informed research, including our own previous work (Harden, Mendel, et al., 2008; Hill et al., 2008), has only

considered dyadic, best friend relationships or perceptions of peer group drinking. It remains possible that peer group behavior or broader social network norms are linked to adolescent drinking causally, not just as a result of selection.

Peer Group Norms and Dynamics

Peer group networks, which subsume relationships within dyads (Bauman, Faris, Ennett, Hussong, & Foshee, 2007), may have considerable influence on adolescent behavior by defining norms for behaviors within the peer culture. In fact, peer group networks are likely to be particularly influential for adolescents and young adults, who are both searching for a social niche and exposed to more and wider groups of peers, and adolescents may be socially rewarded for conforming or socially punished for failing to conform to peer drinking norms (Balsa, Homer, French, & Norton, 2011). In contrast, peer groups may be less influential as close friends and romantic partners likely become increasingly influential with age (Brown, Dolcini, & Leventhal, 2008; Morgan & Grube, 1991; Poelen, Scholte, et al., 2007; Urberg, Shyu, & Liang, 1990). Dynamics, such as friendship quality and exposure, may also be of particular importance within adolescent peer groups. Urberg et al. (2003) found that affiliating with substance-using friends predicted increased substance use, especially for high-quality relationships. In adolescence, drinking becomes more normative. As the normative level of drinking increases, so do the expectations of the individuals in the peer group. Despite negative judgment by parents and authorities, some level of alcohol use may reflect normal adjustment and functioning.

Studying peer groups is not easy; for researchers, peer groups are amorphous and difficult to define. Adolescents, however, can reliably identify members of their peer group (Michell, 1997), recognize social hierarchies (Michell, 1997; Rosenberg, McHenry, & Rosenberg, 1962), and accurately attribute attitudes and behaviors about substance use to specified groups (Michell, 1997). Given the potential importance of behaviors and dynamics within peer groups, it is essential to use methods that take into account the complexities of the peer system (e.g., Crosnoe & McNeely, 2008). Social network analysis (e.g., Wasserman & Faust, 1994) is a method that has demonstrated utility for identifying meaningful structures and patterns in peer networks, even specifically in relation to substance use in adolescence (e.g., Ennett & Bauman, 1993; Ennett et al., 2008; Knecht, Burk, Weesie, & Steglich, 2011; Kobus & Henry, 2010).

Of course, the relationship between adolescent and peer network drinking suffers from the same potential selection effects discussed earlier. To date, no study has combined social network analysis with a genetically informed design to control for shared environmental and genetic selection in an attempt to isolate causal (or quasicausal) influences of peers on adolescents' drinking. Toward this end, the present study

- uses a genetically informed design that includes both twins and other siblings to account for genetic and shared environmental selection;
- employs social network analysis to identify different peer group structures, includes a measure of exposure to friends within identified networks, and relies on peers' own report of their drinking rather than perceptions of their drinking;
- considers the course of alcohol use from early adolescence into early adulthood, a time when drinking typically increases dramatically.

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). Complete details of the Add Health study design are available on the Add Health website (Harris et al., 2009). The current study uses subsamples of the complete Add Health data set, including the social network sample, used for social network analyses; the in-home sample, used to identify the factor structure and longitudinal course of alcohol use behaviors; and the sibling sample, used for behavior genetic modeling. These subsamples are described in detail below. Some analyses were initially conducted using these data and presented in an earlier article examining the effects of best friend substance use on adolescent alcohol by means of a family design (Hill et al., 2008). Brief descriptions of these analyses are presented below, and readers are referred to this article for further description of select methods.

Social network sample—All schools in the United States with at least 30 enrollees ($n = 26,666$) were stratified according to geographic region, urbanicity, school size or type, racial composition, and grade span. From these strata, a random sample of schools was selected, and 79% agreed to participate ($n = 134$ schools). The in-school survey ($n = 90,118$), administered during the 1994–1995 school year, included peer nominations and identification of adolescent siblings. Respondents identified up to five male and five female friends by responding to the items “List your closest male/female friends” and “List your best male/female friend first, then your next best friend, and so on. Girls/boys may include boys/girls who are friends and boyfriends/girlfriends.” Social network data were available for respondents attending schools where over 50% of students participated ($n = 82,629$). There were 75,871 respondents (91.82%) with identifiable nominations. From these 75,871 respondents with available data, there were 509,943 nominations for an average of 6.72 nominations per respondent. In-school data were available for 334,300 (61.83%) of the nominations. Data were unavailable for a given nomination because either the nominee was not on the roster of the respondent's school or sister school (i.e., the student nominated someone outside the school; $n = 124,689$; 24.45% of total nominations) or the nominee was not included in the study ($n = 64,835$; 12.71%). The usable peer nominations reflect only friendships within the school and do not include any friends from outside the school, as no data are available for these individuals. There were minor differences between adolescents nominating peers in the study and adolescents nominating peers not in the study with regard to alcohol use and alcohol problems at Wave I and alcohol problems at Wave II (all $R^2 < 1\%$). Similarly, younger girls were more likely to have a missing nomination, with the differences 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. Although these differences were small, to consider differences in missingness due to covariates and outcomes, we included missing data analysis and considered age, gender, and alcohol use when estimating missing data (missing data analysis is described below).

In-home sample—A subsample of randomly selected students from the in-school survey participated in a follow-up home interview with deliberate oversampling of twin and sibling pairs (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 interviews took place in 1995 and included 20,745 respondents (10,481 female, 10,264 male) between 11 and 21 years of age ($M = 16$; 25th percentile = 14, 75th =

17). The Wave II in-home interviews, completed the following year, included 14,738 adolescents (7,556 female, 7,182 male) between 11 and 23 years of age ($M = 16$; 25th percentile = 15, 75th = 17). In addition to attrition, the decline in sample at Wave II reflects that adolescents exceeding the 12th grade, unless they were part of the sibling sample described below, were not included. This does not affect the sibling sample, as twins and siblings were included regardless of their grade level. Adolescents not included in Wave II due to grade level were included again in Wave III. The Wave III interviews included 15,170 respondents (8,030 female, 7,167 male) and took place between 2001 and 2002 (mean age = 22 years; 25th percentile = 21, 75th = 23).

Sibling sample—Twin zygosity was determined primarily on the basis of self-report and responses to four questionnaire items concerning similarity of appearance. Similar questionnaires have been repeatedly cross-validated with zygosity determinations based on DNA (Loehlin & Nichols, 1976; Spitz et al., 1996). Other sibling relationships (e.g., full, half, and genetically unrelated siblings) were determined by self-report. For the current analyses, only same-sex dyads are used, as using opposite sex pairs may spuriously inflate genetic effects, since MZ twins are always concordant for gender (e.g., Walden et al., 2004). Overall, the sibling sample included 284 MZ pairs, 247 dizygotic (DZ) pairs, 715 full sibling (FS) pairs, 225 half sibling (HS) pairs, 159 cousin (CO) pairs, and 216 genetically unrelated (NR) pairs for a total of 1,846 same-sex pairs.

Measures

Sibling sample alcohol use—Alcohol use for the sibling sample was measured by a series of items from the in-home interviews. Adolescents were asked how often in the past year 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 12 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, or someone they were dating or at school: *never* (0), *once* (1), *twice* (2), *3–4 times* (3), *5 or more times* (4). From these items, two factors, frequency of alcohol use and alcohol problems, were identified using exploratory and confirmatory factor analyses. These factors were used for subsequent latent growth curve analyses (for complete description of these models, see Hill et al., 2008). There were 8,921 (43.003%) adolescents reporting they had never drunk in Wave I, falling to 3,470 (22.874%) indicating that they had never drunk alcohol by Wave III.

Peer substance use—As target report of peer behavior is biased and overestimates the association between targets and their peers (e.g., Hill et al., 2008), direct peer report was used for all analyses. Peer behaviors were assessed in the in-school questionnaire with seven items that asked how often in the past 12 months respondents engaged in a variety of risk behaviors: *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 every day* (6). Previous analyses using these data have indicated two factors, with smoking, drinking, and getting drunk loading onto one substance use factor; these factors were used for subsequent analyses (see Hill et al., 2008, for complete details).

Friendship exposure—Adolescents indicated for each nominated friend whether (1) or not (0) in the past week they went to the friend's house, met after school to hang out, spent time together over the weekend, talked with the friend about a problem, or spoke to the friend on the phone. The means of these items were included as weights on each nomination for subsequent social network analyses to represent friendship exposure.

Statistical Analyses

Statistical analyses included three main parts: (a) social network analysis to identify structure and characteristics of the peer system, (b) longitudinal models of alcohol use, and (c) family designs to consider potential effects of genetic and shared environmental factors in the relationship between peers and alcohol use.

Exploratory social network analysis—Comprehensive sampling in a number of schools permitted social network analysis based on friendship nominations to assess adolescents' relationships within school systems (Bearman, Moody, & Stovel, 1997; de Nooy, Mrvar, & Batagelj, 2005). Peer groups are complex and diverse; social network analysis not only allowed for these differences, but permitted quantification and analysis of characteristics within the groups. A network is a set of objects, or vertices, connected by arcs, which represent relationships between vertex pairs. Figure 1 shows a network of 96 students (represented by vertices) and their friendship nominations (represented by arcs). These arcs were weighted by reciprocity and using the friendship exposure construct such that there is a stronger tie for reciprocal relationships and relationships where the adolescents report more time spent with the nominated peer. The stronger the tie, the smaller the distance between the vertices (see de Nooy et al., 2005, for complete introduction).

Identifying peer groups: Exploratory social network analysis was used to identify subnetworks within schools to identify peer groups via the island algorithm in Pajek (Batagelj, Kejžar, Korenjak-Èerne, & Zaveršnik, 2006). This algorithm was chosen over more common approaches that tend to identify larger and more complicated groups (Gest, Moody, & Rulison, 2007; Moody, 2001). As the goal of this study was to examine the peer group with which an adolescent spends the most time, the islands algorithm was chosen as a technique that identified smaller subgroups.

Islands, or connected parts of a network where the vertices included in the island have greater connections within the island than with vertices outside the island, were defined as having at least three members. The islands algorithm creates subnetworks with a level of relatedness (t) that is greater than the connections to other linked vertices. For the current study, the value of t falls in the range of 1–5, corresponding to the friendship exposure weights described above. Adolescents would have a link of $t = 5$ if they reported the maximum amount of time spent with one another possible. The minimum group size for this analysis was three adolescents; so if there were three adolescents whose nominations reflect a weight of 5, and did not share any other nominations with a weight of 5, then this would represent a complete island. If, however, there were only two adolescents with a weight of 5, the algorithm would extend the island to adolescents linked to these two by a weight of $t = 4$ and continue until at least three adolescents were linked. Once at least three adolescents were linked at a specified level t , all other adolescents sharing a connection of level t were then included in the island. Although this partitioning device has not been used in Add Health, it has shown utility identifying important groups in large networks in physics, history, and biology (e.g., Batagelj et al., 2006; Tzekina, Danthi, & Rockmore, 2007). Subsequent evaluation of the peer groups found that the groups identified subnetworks that were cohesive and meaningful. That is, the islands algorithm identified cohesive peer groups in which it was more likely that adolescents nominated peers within the groups than outside the groups. Further, although peers were not placed into groups based on shared behaviors, characteristics such as academic achievement, involvement in school activities, and risk behaviors were more similar between adolescents within a peer group than for other adolescents in their school, and often were more similar between adolescents within a peer group than between a target and the target's best friend (for more information, contact the first author).

Peer group substance use was calculated for each adolescent by taking the weighted average substance use of the members of his or her group. For example, in a group of four adolescents, A, B, C, and D, the value of peer group substance use for adolescent A would be the average of B, C, and D's substance use weighted by the distance to each of these friends. The distance corresponds to the amount of time friends spend with one another, with friends spending more time being closer together. The weighting of the peers to calculate the means gives more weight to peers closer to the target adolescent in a group.

Friendship exposure: To assess the degree to which adolescents were exposed to their peers, closeness centrality (de Nooy et al., 2005), we calculated a ratio of the distance of reachable peers to the number of reachable peers in a network, for each adolescent. Distances were weighted by the inverse of friendship exposure such that the greater the friendship exposure, the shorter the distance. This measure quantifies the difference between dense groups (i.e., the peers nominate each other, talk on the phone every night, and spend time on the weekends together) and less social groups (i.e., the peers nominate one another but spend little time in social context). Figure 1 shows 96 students in a school with 11 islands. Visual inspection clearly indicates that students closer to their peers are represented by larger vertices (greater closeness centrality) than are students further from their peers. For adolescents with only a few friends who spend lots of time together, closeness centrality would be greater than for an adolescent who can reach many peers, but only through indirect or poor quality connections. Notably, closeness centrality is only a proxy for the construct of salience in peer relationships; many factors are involved in the importance of a friendship beyond the time spent with peers and the density of these relationships. However, this measure does reflect important structural characteristics that may influence the degree to which peers affect behavior.

Latent growth models of alcohol use—Latent growth curve modeling evaluated changes in alcohol use across adolescence. Each data wave included a range of ages, and to facilitate age-based growth modeling and provide sufficient coverage, we collapsed measurements into the following four age groups: early adolescence (A1 = 11–14 years; $n = 12,938$), midadolescence (A2 = 15–17 years; $n = 13,419$), late adolescence (A3 = 18–20 years; $n = 9,523$), and early adulthood (A4 = 21 years and older; $n = 11,615$). When adolescents were measured more than once in a given age group, the mean alcohol use within that age group was used to describe the alcohol use during that period. For example, for adolescents who were measured at ages 11 and 12, their scores at these two times would be averaged to represent their score for early adolescence. There were a maximum of three measurements per person, but the overlapping nature of the data and missing data analysis permitted estimation of growth parameters for all adolescents. Although collapsing data into age categories sacrificed some sensitivity, it was necessary to provide adequate coverage at each age and reliable estimates. This method was preferred to a cohort sequential design to allow for integration with multiple group twin models, and to reduce bias in missing data estimates when siblings fell into separate cohorts. From the alcohol factors at each age category, a linear model was compared with a quadratic model and a dual-slope model permitting different slopes at different developmental periods. For the linear and quadratic models, an intercept, the average level of alcohol use, and a linear slope, reflecting linear change over time, were estimated. For the quadratic model, an additional quadratic term was estimated. For the dual-slope model, an intercept and two slopes connected at a midpoint were estimated. The midpoint occurred between mid- and late adolescence. This time was chosen as a midpoint, as it is developmentally an important shift as adolescents graduate from high school and explore new environments. Alternative parameterizations were tested, but the data were best described by a model with a midpoint between mid- and late

adolescence. The growth factors were permitted to covary to allow changes in alcohol use to be related to the initial alcohol use.

Twin and sibling designs—Univariate twin and sibling models were used to partition variance in growth factors and peer variables. Subsequently, multivariate twin and sibling models were used to gauge whether genetic or environmental factors accounted for the relationships between target and peer behaviors.

Univariate ACE decomposition: Using the sample of same-sex sibling pairs ($n = 1,669$), we separated the variances of the growth factors and peer variables into three parts: additive genetic influences (A), environmental influences shared by siblings (C), and environmental influences unique to siblings (E ; see Figure 2A). The E component also includes residual error variance. Decomposition into ACE components was achieved by considering the different proportions of segregating genes shared by twin and sibling dyads ($MZ = 100\%$, $DZ/FS = 50\%$, $HS = 25\%$, $CO = 12.5\%$, $NR = 0\%$). Twin and sibling models depend on several assumptions, including random mating in the parental generation, similar environments for sibling and twin types, and no Gene $\times$ Environment interaction or correlation. The paths from the latent genetic and environmental variables were fixed to 1, and the variances of the A , C , and E components are estimated. For more information about the logic of behavior genetic modeling, see Neale and Cardon (1992).

Multivariate twin and sibling model: To test whether genetic or shared environmental factors accounted for the relationship between adolescent drinking and peer variables, we divided the variances of peer group substance use, closeness centrality, their interaction, and the individual intercepts and slopes into their ACE variance components, and the intercept and slopes were regressed onto the ACE components of the peer variables. The associations between peer factors and target alcohol use were analyzed as a combination of genetic factors, common environmental factors, and quasicausal pathways (see Figure 2B). Age and gender were used as covariates, and missing data analysis was included. Occasionally, variance estimates for C were negative, which may be the result of sampling error, or may suggest that there are dominance or epistatic processes at work (Turkheimer, D'Onofrio, Maes, & Eaves, 2005). Negative variance estimates are not interpretable and were fixed to 0, and the change in model fit was assessed, resulting in significant changes in fit (see Plomin et al., 1993). The within-family association, the path on E , is considered to be quasicausal and indicates that after controlling for genes and shared environment, the twin with riskier peers drinks more than the twin with less risky peers. The quasicausal pathway remains confounded by factors that vary systematically between siblings. For example, twins in riskier peer groups may have more parental conflict; this conflict may cause greater alcohol use than that of their cotwins. Nevertheless, within-family associations have fewer potential confounds than between-family associations, presenting a stronger case for causation.

Software and missing data analysis: Social networking analyses were conducted with Pajek (Batagelj & Mrvar, 2001), a matrix program freely available that analyzes relationships in large networks. All other analyses were conducted in Mplus (Muthén & Muthén, 2004). Missing data were considered with 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 delinquency. However, MAR assumes that missingness of peer data is unrelated to the level of the peer characteristic of interest after controlling for the level of target delinquency and measured covariates. If measured covariates and the target delinquency explain the relationship between missingness and peer characteristics, missingness is considered a function of the covariates and target alcohol use rather than peer characteristics. MAR cannot be tested; it is

impossible to know the true value of missing data. However, ML is robust to minor violations of this assumption. ML integrates over all possible values of missing peer data and gives more weight to values that are more likely (Allison, 2002; Little & Rubin, 1989). Evaluation of ML under MAR with simpler models suggests that with samples of similar size and much higher rates of missing data (nearly 80%), ML under MAR performs adequately (Schafer & Graham, 2002).

Results

Exploratory Social Network Analysis

Partitioning the school networks into groups of adolescents by means of the island algorithm revealed 5,077 groups ranging in size from three to 90. There were 76,926 adolescents included in peer groups of the 82,629 (93.09%) for whom social network data were gathered. The mean group size was 13.596 ($SD = 10.849$), the median was 10, and the modal group sizes were four ($n = 381$) and five ($n = 381$). Peer group substance use was moderately correlated with target substance use ($r = .459$, $df = 74,701$, $p < .001$), with greater peer group substance use associated with greater target substance use. The relationship between the target and the peer group substance use exceeded the relationship between the target and the best friend alone ($r = .290$).

Substance Use Measurement Models

The dual-slope model fit the data adequately for alcohol use (root-mean-square error of approximation [RMSEA] = .055, comparative fit index [CFI] = .999, Tucker–Lewis index [TLI] = .992) and alcohol problems (RMSEA = .071, CFI = .998, TLI = .987) and fit better than the linear and quadratic model (see Tables 1 and 2; $n = 20,770$). We modeled nonlinear change by estimating the average level of alcohol use, the intercept, and two slopes: the first reflecting earlier change from early adolescence into late adolescence (Slope 1) and the second reflecting later change from midadolescence to early adulthood (Slope 2). Both alcohol use and alcohol problems increased from early to mid- and late adolescence and decreased from mid- and late adolescence to early adulthood (see Table 2). The actual means at each age were not significantly different from those means estimated with the growth model.

Relationship Between Target and Peer Behavior

The intercept and slopes from the measurement model of adolescent alcohol use and problems were regressed on the peer group substance use, closeness centrality, and their interaction via the full in-home sample with peer data available ($n = 13,464$), with age and gender included as covariates. Patterns of relatedness were similar for alcohol use and problems (estimates are available in Table 3), and the models fit well (CFI/TLI > .990 for all models).

Greater substance use in peer groups was related to greater overall alcohol use and problems. Closeness centrality was related to substance use independently, with adolescents who were more central reporting greater substance use ($n = 70,396$, $r = .190$, $p < .001$); however, after including group substance use and the interaction term, closeness centrality was only weakly related to the growth factors ($R^2 < 1\%$). The effect of closeness centrality depended on the group substance use. For adolescents with high closeness centrality, high-substance-using groups predicted a greater increase from early adolescence to late adolescence and a lesser decline into early adulthood, whereas low-substance-using groups predicted less increase in alcohol use into early adulthood. For adolescents with low closeness centrality peers, those in high-substance-using groups steadily decreased over time, whereas those in low-substance-using groups steadily increased over time.

Figure 3 is an illustration of the changes over time for those adolescents initially discordant for substance use. Using the growth structure defined above, we estimated growth parameters for adolescents belonging to three groups: those that initially drank more, less, and about the same as their peer groups. Adolescents from the full in-home sample with peer data available were divided into these groups based on the in-school substance use factor, available for individual adolescents and their peer groups. Adolescents were considered discordant with their peer if their initial substance use score was 1.5 standard deviations greater or lower than their peer groups' substance use score. For both adolescents with initially less and more substance use than their peers, there was an initial increase in alcohol use; however, these groups followed different paths following midadolescence. For adolescents who initially had less substance use than their peers, their alcohol use continued to increase into adulthood. For adolescents who had greater substance use initially, their alcohol use decreased into early adulthood. This may reflect causal processes, normative developmental processes, regression to the mean, or selection and confounding processes. Subsequent analyses examined these alternative explanations of this illustrative example.

Twin and Sibling Designs

Univariate ACE decomposition—Table 4 provides the proportion of variance accounted for by *ACE* components for the growth and peer factors. All models fit well (CFI/TLI > .950). Nearly all the factors reflected variance attributable to genetic, shared environmental, and nonshared environmental factors. Changes in alcohol use and problems reflected little shared environmental variance, although overall shared environmental influences accounted for 19.2% and 13.2% of the variances in alcohol use and alcohol problems, respectively. Nearly three quarters (74.4%) of the variance in peer group substance use owed to genetic factors, whereas shared environmental influences accounted for a majority of the variance in closeness centrality (60.6%). Subsequently, an additional term was included to consider possible unique effects of being twins as compared with being siblings; inclusion of this term did not significantly alter the fit of the models and is not included in future analyses.

Multivariate twin and sibling model—The multivariate twin and sibling models regressing the growth factors onto the *ACE* variance components of the peer factors fit well (alcohol use: RMSEA = .048, CFI = .978, TLI = .977; alcohol problems: RMSEA = .051, CFI = .974, TLI = .973). Full results for alcohol use are shown in Tables 5–7. Alcohol problems showed similar trends unless otherwise noted.

Phenotypic regressions: The regression coefficients for the growth factors on the *ACE* components of the peer factors were constrained to be equal to test the equality of between- and within-family associations. If the model fit did not worsen by fixing these values, the model was consistent with an unmodified model because the coefficients were not accounted for by genetic or environmental factors that make families different. For all but Slope 2 on closeness centrality (which itself was not significant), fixing these paths resulted in a significant decrease in fit, implicating genetic and shared environmental influences on the association between the peer variables and adolescent alcohol use ($p < .001$ for all comparisons). This indicates that the phenotypic relationships were at least partially moderated by genetic and shared environmental factors. In Table 6, the estimate in the phenotypic (or fixed parameter) model reflects the between-family association for alcohol use, and in the *ACE* (or free parameter) model, the within-family association, measuring the extent to which differences in peer substance use account for differences in alcohol use after accounting for genetic and shared environmental factors, is reflected in the coefficient on *E*.

Genetic influences: Regressions on the *A* variance component estimated the extent to which genes account for the relationship between affiliating with alcohol-using peers and drinking alcohol. Genetic factors accounted for significant portions of the covariance between peer variables (group substance use and closeness centrality) and the target alcohol use. These genetic factors accounted for covariation between peer variables and overall level of alcohol use (i.e., the intercept), and for the relationship between peer variables and changes in alcohol use over time (i.e., the slope).

Environmental influences: Regressions on the *C* variance component estimated the extent to which shared environmental factors account for the relationship between target alcohol use and closeness centrality and having substance-using peer groups. The variance of the *C* component of peer group substance use and the interaction term were negative, but constraining the variance of *C* to be 0 and estimating only the variances of *A* and *E* did not result in a significant loss of fit ($\Delta\chi^2 = 5.363$, $\Delta df = 2$, $p = .068$). For closeness centrality, the regressions on *C* for alcohol use and problems were not significantly different from 0, indicating that shared environmental influences do not explain the relationship between closeness centrality and alcohol use.

Quasicausal relationships: The phenotypic regression coefficient of the interception closeness centrality ($b = .147$, $p < .05$) was larger than the path on the *E* component ($e = .035$, ns , $p > .05$), indicating that the relationship was accounted for by genetic and environmental factors and that closeness centrality did not explain additional variance after considering genetic and shared environmental influences (see Table 6).

After controlling for genetic and environmental factors, there remained significant paths on the *E* components of peer group substance use and the interaction term for the growth factors of alcohol use and problems (see Table 6). Post hoc analysis included best friend substance use as a covariate to test whether the effect was driven by a group member. Best friend substance use did not affect the quasicausal path, although it did reduce the estimated role of genetic influences. The group drives the quasicausal path and was consistent with a role of group influence on level and changes in alcohol use and problems over time. A regression on *E* indicates that after controlling for genetic and shared environmental influences, siblings exposed to riskier peers will drink more than their cosiblings. This effect was greatest in the context of high-intensity peer relationships.

Figure 4 shows the difference in change over time for adolescents who have peer groups with substance use 1 standard deviation above and below the mean. Unlike the phenotypic trajectory, which reflected convergence to the mean over time, the path on *E* suggested that after controlling for genetic and shared environmental influences, greater peer substance use predicted a U-shaped trajectory that increased into adulthood. Similarly, despite the increase predicted by low-substance-using peer groups by the phenotypic path, the path on *E* predicted decreases in drinking into adulthood. The *E* path predicted divergence from the mean toward the peer group.

The quasicausal effect was clearest when examining MZ twin pairs discordant for peer group substance use. Figure 5 shows the trajectories for MZ twins initially concordant with their cotwin for alcohol use but discordant for peer group substance use. That is, the twins drink the same initially, but one twin's peer group drinks more and the other twin's peer group drinks less than do the twins. There were only 17 pairs initially concordant with their cotwin with discordant peer groups, and the trajectories illustrated in Figure 5 do not represent significant differences. Examination of the trend suggested by these pairs demonstrates that the twins who initially drank less than their peers increased alcohol use into adulthood, but the twins who initially drank more than their peers did not increase

alcohol use into adulthood. The twins in a riskier peer environment drank more than the twins in the less risky peer environment.

Discussion

This study approaches the complex relationship between peer characteristics and adolescent alcohol use by considering the diversity in peer networks, examining the developmental course of alcohol use from early adolescence to early adulthood, and using family designs to consider the immeasurable and innumerable potential confounding processes between friendships and alcohol use to identify true causal relationships. This is the only study to combine these approaches to consider the complexity of peer groups and to distinguish risk indicators from risk mechanisms in the presence of genetic and environmental influences. The results make a strong case for a combination of selection and influence mechanisms involving peer groups and adolescent alcohol use, and may indicate the primary mechanism through which peer groups affect behavior in socialization.

As expected, before accounting for genetic and shared environmental selection, peer group substance use is related to adolescent drinking. Greater peer group substance use predicts greater overall alcohol use and problems, a greater increase in alcohol use and problems in early and midadolescence, and a less dramatic decline into late adolescence. Within peer groups, the strength of the association with group substance use is greatest when adolescents are close to their friends. Being close to substance-using peers predicts greater and more persistent alcohol use, whereas being close to low-substance-using peers predicts persistently low alcohol use. This is consistent with previous research that has found that for adolescents with higher relationship quality, associations between individuals and friends are stronger (Urberg et al., 2003). This may indicate either that the more time adolescents spend with their peer group, the more typical of the group their behavior becomes, or that adolescents whose behavior is typical of the group spend more time with their peers, or a combination of these processes.

Applying the quasicausal model to the observed relationship between peer group and adolescent drinking supports both genetic-based selection processes and causal peer influence. Although genetic factors attenuated the effect, there remains an additional quasicausal peer group effect whereby greater peer group substance use predicts both greater overall alcohol use and more persistent alcohol use over time. To best understand the quasicausal effect, consider MZ twins who are discordant for levels of risk. MZ twins share the same genes and shared environment, so the obvious question is as follows: Do differences in the level of risk they are exposed to predict these differences in drinking? In this case, the answer is yes. The twin exposed to greater peer group substance use has greater overall substance use and more persistent alcohol use over time than the twin exposed to less peer substance use. This deviates from the findings for best friends where genetic factors completely accounted for the covariance between alcohol use and best friends' substance use in a study using the same alcohol factors and the same data (Hill et al., 2008).

The more robust role of peer groups as compared with best friends may suggest that the process by which friends influence one another is by setting normative expectations within a group rather than an adolescent who drinks promoting drinking in an otherwise "good kid." Rather than one adolescent being the impetus for all others' behavior, reciprocal building of expectations and time spent with peers may drive this interaction. Peer groups may create a culture that rewards certain behaviors and discourages others. Peer groups may be formed based on similarity for a host of behaviors, including substance use behaviors, but to the

extent that adolescents differ from their group in substance use, adolescent alcohol use may change in the direction of the group over time.

Although “peer pressure” is often considered to have a negative connotation, adhering to social expectations is not an inherently negative process. Peer environments are both potentially protective and potentially risky. In groups where normative behaviors are seen as problematic, such adherence may lead to negative outcomes (Dishion & Dodge, 2005). The current study, however, extends socialization processes to include the potential for positive socialization processes in that for adolescents with initially high alcohol use who select into low-substance-using groups, there is a decrease in alcohol use over time.

For the quasicausal path, greater peer group substance use predicts more sustained drinking into early adulthood. Studies have often differentiated between “adolescent-limited” and “life-course persistent” trajectories of alcohol use and other problem behaviors (e.g., Chassin, Flora, & King, 2004; Moffitt, 1993). The adolescent-limited trajectory generally reflects a more normative increase in problem behavior that decreases in adulthood, whereas life-course persistent trajectories are associated with earlier and more sustained problem behavior associated with negative outcomes. Belonging to a high-substance-using peer group may sustain problematic alcohol use by increasing the likelihood of dependency and self-medicating in response to subsequent impairments in functioning, or by influencing the way adolescents conceptualize relationships and normative behavior.

In addition to the quasicausal paths, genetic-based selection processes that likely influence development of a peer group and propensity to drink were implicated. 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, DeFries, & Loehlin, 1977). Active rGE suggests that an individual is influenced by his or her genes to seek out certain environments (i.e., an individual genetically predisposed to drink may seek out other individuals who drink). Evocative rGE suggests that an individual's genes cause others to act in certain ways toward him or her (e.g., an adolescent who is predisposed to drink will attract peers who drink). In this case, as peer selection is a reciprocal process, it is likely both active and evocative rGE processes may be at work. Identification of a role for genetics does not imply that genes determine these characteristics or shed light on specific genes or mechanisms at work. The majority of variance in alcohol use was attributable to environmental factors; genetic factors did not completely explain the covariance between alcohol use and peer factors.

Future Directions and Limitations

Twin models—as used here—assume that there is no Gene $\times$ Environment interaction and may misidentify interaction effects in either genetic or nonshared environmental influences. Previous research has found a role for Gene $\times$ Environment interaction in the relationship between adolescent and best friend alcohol use (Harden, Hill, Turkheimer, & Emery, 2008). Recent research suggests that specific genes may affect the degree to which an individual's drinking is influenced by an unrelated person's drinking behavior (Larsen, van der Zwaluw, Overbeek, Franke, & Engels, 2010). Similarly, future work might examine Gene $\times$ Environment interaction and rGE simultaneously in the relationship between adolescents and peer groups. Another assumption of twin models is random mating in the parental generation, an assumption that is untested in this analysis. Deviations from this assumption may result in an inflated estimate of shared environmental variance (Neale & Cardon, 1992). Although important to consider in future work, it is unlikely that this would substantially change the results, as the estimates for shared environment were quite low and the multivariate paths were not significant. Further, despite the support gleaned from accounting for the numerous potential genetic and environmental factors that vary between families,

potential peer effects are called quasicausal, as they are not free from within-family confounds (e.g., belonging to a peer group with greater substance use may lead to greater parental conflict and in turn lead to greater alcohol use). Similarly, the interrelated relationship between siblings and peer groups would be a valuable avenue for future study. Here twin and sibling pairs occasionally fell within the same island, with this occurring more for twins than siblings and more for MZ twins than DZ twins. When this occurred, the sibling's substance use was included in the calculation of the weighted average, as the pair identifies one another as part of the peer group. Including the siblings was a theoretical decision but may have implications for the estimates of genetic and environmental factors. Siblings may have an effect on both friend selection and substance use, and future research should consider this intricate relationship. Missing peer reports due to failure to nominate peers or nominated peers without data present were treated as missing. Although there were only minor differences between these groups on covariates and outcomes, there may have been important unmeasured differences between these groups. The limits of the missing data analysis used here have not been tested for complex genetic models, but evaluation with simpler models suggests that ML under MAR performs well with similar samples sizes and nearly 80% of data missing (Schafer & Graham, 2002).

Peer group identification, especially in large networks, is a notoriously complex task (Moody, 2001). Finding meaningful groups and evaluating the validity of these groups is still more complicated. However, the island algorithm demonstrated utility in identifying groups of meaningful size within large networks. Due to limited previous use of this algorithm and the difficulty of identifying groups within large systems, further validation efforts are indicated.

Finally, this study focuses on peer group substance use and exposure to the peer group as potential predictors for alcohol use. A myriad of interrelated peer characteristics, including popularity, hierarchy, and friendship quality, are important avenues for future research. Additionally, the current study only has peer measurements at the initial time point, and for most individuals in the current study, there is no information about the peer groups as the individuals enter adulthood. As adolescent peer groups change over time, data and analyses that take these dynamics into account may further elucidate the relationship between peers and alcohol use.

Conclusions

Peer groups may be a particularly important context for promotion and prevention of alcohol use behaviors, especially for relationships with high exposure. These analyses demonstrate the utility in considering diverse characteristics and structures within peer networks and in controlling for genes and shared environment via behavior genetic designs. By simplifying the peer system to dyadic relationships, we may underestimate potential peer effects. Conversely, by failing to consider potential genetic and shared environmental factors, causal effects may be overestimated. Identification of the causal role of peer groups has implications for interventions targeting adolescent alcohol use.

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Figure 1.
Example of social network with vertices weighted by closeness centrality. Sizes of vertices represent closeness centrality, and shading represents group membership.

Figure 2.

Univariate (A) and multivariate (B) twin and sibling models. Multivariate model regressing growth factors on *ACE* components of peer factors: additive genetic influences (*A*), environmental influences shared by siblings (*C*), and environmental influences unique to siblings (*E*). For clarity, one twin is shown for the multivariate model, and a single generic peer factor (*P*) is shown. Actual model includes peer group substance use, closeness centrality, and their interaction. Although not shown here, residual *ACE* components for the growth factors were estimated, and age and gender were included as covariate. MZ = monozygotic; DZ = dizygotic; FS = full sibling; HS = half sibling; NR = genetically unrelated.

Figure 3.

Adolescent alcohol use over time for adolescents concordant and discordant with initial peer substance (Subst) use. Shaded region represents 95% confidence interval.

Figure 4.

Alcohol use trajectories predicted by the regressions on E for high- and low-substance-using peer groups. PG = peer group; SU = substance using.

Figure 5.

Monozygotic twin pairs initially concordant for alcohol use, but discordant for peer group substance use. Data for monozygotic twin pairs initially concordant for drinking but discordant for peer substance use provide a useful illustration of findings indicating peer influence on subsequent drinking. However, differences represent trends and are not significant ($p > .05$; $n = 17$ pairs).

Table 1

Latent Growth Model Fit Indices and Comparative Fit Indices for Dual-Slope Growth Models

Variable	χ^2	df	CFI	TLI	RMSEA	Linear $\Delta\chi^2$	Δdf	p
Alcohol use								
Linear	7062.347	5	.875	.850	.261			
Quadratic	129.489	1	.998	.986	.079	6932.858	4	<.001
Dual slope	101.949	1	.998	.989	.070	6960.398	4	<.001
Alcohol problems								
Linear	6533.154	5	.880	.856	.251			
Quadratic	110.911	1	.998	.988	.073	6422.243	4	<.001
Dual slope	83.202	1	.998	.991	.063	6449.952	4	<.001

Note. CFI = comparative fit index; TLI = Tucker–Lewis index; RMSEA = root-mean-square error of approximation.

Table 2

Estimates of Growth Factors for Dual-Slope Growth Models

Variable	Alcohol use				Alcohol problems			
	<i>M</i>	<i>SE</i>	Correlations		<i>M</i>	<i>SE</i>	Correlations	
			Intercept	Slope 1			Intercept	Slope 1
Intercept	-.017	.005	—		-.018	.005	—	
Slope 1	.074	.003	.237	—	.061	.003	.218	—
Slope 2	-.054	.004	-.593	-.434	-.039	.004	-.522	-.398

Table 3

Standardized Regression Coefficients for Growth Factors on Peer Values for Alcohol Use and Problems (n = 13,464)

Variable	Alcohol use			Alcohol problems		
	Intercept	Slope 1	Slope 2	Intercept	Slope 1	Slope 2
Peer group	.306	-.097	.108	.311	-.144	.106
Closeness centrality	.034	.027	.014	.035	.056	.048
Peer Group × Closeness Centrality	.001 ^a	.204	-.090	-.001 ^a	.173	-.108

^aEstimate not significantly different from 0 ($p > .05$).

Table 4

Proportions of Variance in ACE Components for Peer and Target Factors

Variable	Alcohol use			Alcohol problems			Peer factors	
	Intercept	Slope 1	Slope 2	Intercept	Slope 1	Slope 2	Peer group substance use	Closeness centrality
<i>A</i>	.150	.371	.092	.277	.402	.127	.744	.180
<i>C</i>	.192	.056	.023	.132	.000 ^a	.079	.060	.606
<i>E</i>	.658	.573	.885	.591	.598	.794	.195	.214

Note. *A* = additive genetic influences; *C* = environmental influences shared by siblings; *E* = environmental influences unique to siblings.

^aFor this variable, the estimate for *C* was negative, which may be the result of sampling error or may suggest dominance or epistatic processes at work. A negative estimate is not interpretable and this negative variance estimate was fixed to 0 without loss of fit.

Table 5

Variance and Residual Variance Component Estimates

Variable	<i>A</i>		<i>C</i>		<i>E</i>	
	Estimate	Total (%)	Estimate	Total (%)	Estimate	Total (%)
Variance components						
Group	0.844	80.08			0.210	19.92
Centrality	0.182	17.45	0.605	58.01	0.256	24.54
Group $\times$ Centrality	14.589	81.05			3.411	18.95
Residual variance components						
Intercept	0.151	29.43	0.099	19.30	0.263	51.27
Slope 1	0.058	66.67			0.029	33.33
Slope 2	0.039	15.06	0.053	20.46	0.167	81.07

Note. Negative variance and residual variance estimates were set to 0. *A* = additive genetic influences; *C* = environmental influences shared by siblings; *E* = environmental influences unique to siblings.

Table 6

Regression Coefficients

Variable	Phenotypic^a		A		C		E	
	Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE
Peer group substance								
Intercept	.206	.01	.121	.02			.641	.05
Slope 1	-.025	.01	.037	.01			-.159	.04
Slope 2	.049	.00	.031	.02			.421	.06
Centrality								
Intercept	.147	.01	.730	.26	<i>.056</i>	<i>.03</i>	.036	.07
Slope 1	<i>.006</i>	<i>.01</i>	.223	.11	<i>.004</i>	<i>.02</i>	-.133	.07
Slope2	<i>-.007</i>	<i>.01</i>	-.175	.15	.012	.02	.053	.05
Peer Group × Centrality								
Intercept	.087	.01	.030	.00			.150	.01
Slope 1	.015	.00	.009	.00			-.023	.01
Slope 2	-.020	.00	-.008	.00			.099	.01

Note. Parameters in italics not significantly different from 0 ($p > .05$). *A* = additive genetic influences; *C* = environmental influences shared by siblings; *E* = environmental influences unique to siblings.

^aShared environmental variance (*C*) estimates for peer group substance use and the interaction term were negative and set to 0. For these, the phenotypic models are defined by setting the regression coefficients on *A* and *E* equal ($a = e$).

Table 7

Effect Sizes Owing to Peer Variables

Variable	%	Model fit
Intercept	23.34	
Slope 1	6.12	
Slope 2	12.34	
CFI		.978
TLI		.977
RMSEA		.048

Note. CFI = comparative fit index; TLI = Tucker–Lewis index; RMSEA = root-mean-square error of approximation.