HHS Public Access

Author manuscript

J Abnorm Child Psychol. Author manuscript; available in PMC 2016 August 01.

Published in final edited form as:

J Abnorm Child Psychol. 2015 August ; 43(6): 1131–1143. doi:10.1007/s10802-014-9969-3.

Selection and Socialization Effects in Early Adolescent Alcohol Use: A Propensity Score Analysis

Matthew D. Scalco[a], **Elisa M. Trucco**[b], **Donna L. Coffman**[c], and **Craig R. Colder**[a]

[a]The State University of New York at Buffalo, Department of Psychology, 204 Park Hall, Buffalo, NY 14260-4110

[b]University of Michigan, Department of Psychology, 1012 East Hall, 530 Church Street, Ann Arbor, MI 48109-1043

[c]Penn State University, The Methodology Center, 204 E. Calder Way, Suite 400, State College, PA 16801

Abstract

The robust correlation between peer and adolescent alcohol use (AU) has been taken as evidence for both socialization and selection processes in the etiology of adolescent AU. Accumulating evidence from studies using a diverse range of methodological and statistical approaches suggests that both processes are involved. A major challenge in testing whether peer AU predicts an adolescent's drinking (socialization) or whether an adolescent's drinking predicts peer AU (selection) is the myriad of potentially confounding factors that might lead to an overestimation of socialization and selection effects. After creating AU transition groups based on peer and adolescent AU across two waves (N = 765; age = 10-15; 53% female), we test whether transitions into AU by adolescents and peers predict later peer and adolescent AU respectively, using (1) propensity score analysis to balance transition groups on 26 potential confounds, (2) a longitudinal design with three waves to establish temporal precedence, and (3) both adolescent (target) and peer self-report of peer AU to disentangle effects attributable to shared reporter bias. Both selection and socialization were supported using both peer self-report of AU and adolescent-report of peer AU. Although cross-sectional analyses suggested peer self-reported models were associated with smaller effects than perceived peer AU, longitudinal analyses suggest a similar sized effect across reporter of peer AU for both selection and socialization. The implications of these findings for the etiology and treatment of adolescent AU are discussed.

Keywords

adolescence; alcohol use; selection; socialization; influence; propensity scores

Early adolescent substance use (SU) continues to be a major public health concern in the United States (Kessler et al., 2005). Initiation of alcohol use (AU) before age 15 is associated with higher risk for later substance abuse disorders (Grant & Dawson, 1997;

Correspondence regarding this article should be directed to Matthew D. Scalco, Department of Psychology, 204 Park Hall, SUNY at Buffalo, Buffalo, NY 14260-4110. mscalco@buffalo.edu Phone: 716-645-0216 Fax: 716-645-3801.

Sung, Erkanli, Angold, & Costello, 2004) and many other adverse outcomes including decreased school attendance, car accidents, binge drinking, other drug use, and later depression (Grueber, DiClemente, Anderson, & Lodico, 1996; Marmorstein, 2009). Although the literature documents many correlates of adolescent SU (Chassin, Colder, Hussong, & Sher, in press; Ennett et al., 2008; Zucker, 2006), researchers often emphasize the importance of social context, especially peer SU, as one of the most robust and proximal influences of early adolescent SU (Ennett et al., 2006; Hawkins, Catalano, & Miller, 1992). Given that the risk associated with early initiation and subsequent heavy use has been linked to long-term negative outcomes (Chassin, King, & Flora, 2004) understanding whether or not peer SU is a causal factor in the early initiation and escalation of SU will aid in the development of preventative interventions.

Although the relationship between peer and adolescent AU is robust (Hawkins et al., 1992), there has been considerable debate concerning the direction of the relationship. The core dispute centers on whether peers socialize/influence adolescents to drink (i.e., peer AU predicts adolescent AU) or whether adolescent drinkers select into drinking peer groups (i.e., adolescent AU predicts later affiliation with alcohol using peers; Bauman & Ennett, 1996). There is evidence for both selection and socialization of early adolescent AU across longitudinal studies that utilized various methodologies including ecological momentary assessment, social network analysis, and cross-lagged panel designs (Curran, Stice, & Chassin, 1997; Dishion & Skaggs, 2000; Knecht, Burk, Weesie, & Steglich, 2010; Poelen et al., 2007; for a review see Leung, Toumbourou, & Hemphill, 2011). These general conclusions must be tempered in light of the fact that previous work on peer selection and socialization has generally not accounted for the wide range of known correlates of adolescent and peer SU, which include temperament and personality (Chassin, Flora, & King, 2004), psychopathology (King, Iacono, & McGue, 2004; Colder et al., 2013), peer delinquency (Scalco et al., 2014), parental drinking and alcoholism (Alati et al., 2014; Hussong, Curran, & Chassin, 1998), pubertal timing (Costello, Sung, Worthman, & Angold, 2007), amongst others (Chassin, Colder, Hussong, & Sher, in press; Zucker, 2006). These variables are potential confounders for the association between peer and adolescent AU. The current study attempts to address confounding by testing both selection and socialization effects for AU in a large sample (N = 765) of early adolescents (age 10-15) using propensity score analysis (PSA) to adjust for 26 potential confounds and interactions among confounders. We also utilized a longitudinal design to establish temporal precedence and both adolescent (target) report and peer self-report of peer AU to disentangle shared reporter bias.

Socialization and Selection

Theoretical formulations of peer socialization emphasize the importance of positive reinforcement for deviancy, including alcohol and drug use (Dishion, Spracklen, Andrews, & Patterson, 1996). Such positive reinforcement is believed to play a role in the development of a deviant peer culture, exposure to which predicts later SU and further delinquent behavior (Dishion & Skaggs, 2000; Osgood, Wilson, Bachman, O'Malley, & Johnston, 1996). A large number of studies utilizing panel designs and regression methods find that peer AU prospectively predicts adolescent AU even after controlling for prior

levels of adolescent AU (Leung et al., 2011), which is generally taken as support for socialization. Theoretical formulations of peer selection (homophily) emphasize an adolescent's ability to shape their own social environment by choosing friends and environments based on delinquency, AU, and other individual differences (Haynie & Osgood, 2005; Jaccard, Blanton, & Dodge, 2005; Shalizi & Thomas, 2011). Evidence suggests that children and adolescents select friends based on proximity (Epstein, 1989) and similarity on a wide range of variables including age, gender, race, academic/athletic achievement, behavior, and attitudes (Hartup, 1983; Haynie & Osgood, 2005). Similar to the literature on socialization of AU, many studies using panel designs have found that adolescent AU predicts peer AU even after controlling for prior levels of peer use (Leung et al., 2011), which is generally taken as support for selection.

Despite the consistency of findings from panel studies supporting socialization and selection, proponents of social network modeling, especially stochastic actor-based models, have questioned these results on a number of methodological grounds (for a review of the assumptions and parameters estimated by these models see Snijders, Bunt, & Steglich, 2010; Steglich, Snijders, & Pearson, 2010). The most central issue involves the adequacy of cross-lagged panel designs to account for the co-evolution of adolescent social network ties over time (Burke, Vorst, Kerr & Stattin, 2012; Mudnt, Mercken, & Zakletskaia, 2012; Snijders et al., 2010). Cross-lagged panel designs assume stability in network ties and independence of observations, giving rise to concerns about potential confounding of socialization and selection processes. Despite these critiques of panel studies in which peer and adolescent AU predict each other over time, a number of social network studies employing stochastic actor-based methods, which arguably can better disentangle socialization and selection, have also found support for selection and socialization effects for AU in early adolescence (Burk et al., 2012; Knecht et al., 2010; Mercken, Steglich, Knibbe, & Vries, 2010; Osgood et al., 2013). An exception is Mudnt et al. (2012) who found support for selection, but not socialization. Some of these network studies indicate that selection is associated with a larger effect size than socialization in early adolescence (Burk et al., 2012; Knecht et al., 2010), and others indicate either the opposite pattern (Mercken et al., 2012) or that socialization and selection have similar sized effects (Osgood et al., 2013). In addition, one study demonstrated that socialization effects are stronger when considering close friend peers as opposed to the larger peer network even after controlling for genetic and shared environmental effects using a nationally representative twin sample (Cruz, Emery, & Turkheimer, 2012).

Selection/Socialization Effects within a Broader Theoretical Context

Developmental ecological models conceptualize risk and protective factors from multiple levels including the individual (e.g., personality and temperament), social context (e.g., families and peers), and the broader community (e.g., neighborhood characteristics; Bronfenbrenner, 1979). Moreover, factors from multiple levels are thought to interact and reciprocally influence each other to increase or decrease liability for risky behavior. As such, it is likely that selection and socialization effects operate within a complex and dynamic developmental context in which peers are but one important factor. Although only a few studies have tested variables from multiple levels simultaneously (Ennett et al., 2008;

Trucco, Colder, Wieczorek, Lengua, & Hawk, 2014), there is evidence in the literature that temperament (Giancola & Parker, 2001; Windle, 2000), adolescent problem behavior (Scalco, et al., 2014), parental drinking (Engels et al., 1999), and early pubertal timing (Wichstrom, 2001), as well as demographic variables such as gender, age, and ethnicity (Haynie & Osgood, 2005) predict peer delinquency and peer SU, which are generally interpreted as contributing to selection effects (homophily). Once selection has occurred parenting is likely involved in maintaining or exacerbating adolescent and peer SU over time through inadequate parental monitoring which provides increased opportunity to spend unstructured time with peers (Osgood et al., 1996). In addition to effects on peer selection, each of these variables has also been linked to an adolescent's own SU (Alati et al., 2014; Costello et al., 2007; Hitner & Swickert, 2006; Hussong, et al., 1998; King, et al., 2004; Kohl & Mermelstein, 2004; Trucco et al., 2014; for reviews see Chassin et al., in press; Zucker, 2006). Therefore, not only do these factors represent potential confounds regarding the causal mechanism of selection, they also represent potential confounds for socialization effects given that observed effects from peer to adolescent SU may be artifacts of earlier or concurrent selection *or* earlier socialization. Moreover, peer delinquency has been consistently linked to early adolescent SU even when some of the variables listed above served as statistical controls (Ferguson & Meehan, 2011; Scalco et al., 2014), suggesting that peer delinquency may be a particularly important confounder in evidence for socialization of SU.

While a few studies have attempted to consider the wide range of influences on adolescent and peer SU and delinquency (e.g., Haynie & Osgood, 2005; Ennett et al., 2008) many studies have not (e.g., Burke et al., 2012) despite using sophisticated analytic methods. Moreover, even studies that included statistical control variables were unable to account for many known correlates of adolescent and peer SU due to the limitations in the number of statistical control variables that can be incorporated into modeling techniques such as hierarchical linear models (HLM), structural equation models (SEM), OLS regression, ANOVA, and social network models (Rubin, 2005; Kao, Toulis, Airoldi, & Rubin, 2013). Systematically ruling out alternative hypotheses is critical to testing causal models in science (Holland, 1986; Meehl, 1978; Popper, 1963; Rubin, 2005) and the fact that peer and adolescent AU cannot be manipulated experimentally necessitates the use of more sophisticated methodologies to rule out alternative causal explanations. It is worth noting that stochastic actor-based models attempt to adjust for confounding *between* assessments using simulation techniques that model small unobserved changes in network structure and behavior. A limitation of these models is that the “microstep” changes that occur between assessments are assumed to be explained entirely by information in the prior microstep, leaving variables exogenous to the model as potential confounds (e.g., temperament, demographics, family functioning, pubertal development, and peer delinquency). Shalizi & Thomas (2011) described this confounding as latent homophily and social influence and argued that observational social network studies have not ruled out these alternative causal explanations.

The current study attempts to meet the methodological challenge of confounds by using PSA in a longitudinal panel design (Rosenbaum & Rubin, 1983; Rubin, 2005). A benefit of the

PSA design over the social network approach is that a wide range of confounds that precede initiation and escalation of AU can be used to simulate random assignment to “treatment” groups. In the case of socialization, this involves simulating random assignment of targets to a peer who used alcohol vs. a peer who did not use alcohol and then assessing the effect of having a peer who uses alcohol on later target AU. To the extent that all confounders are used to model the propensity scores, an unbiased estimate of a causal effect can be obtained because individuals with similar propensity scores have similar distributions (i.e., balance) on the confounders and can therefore be treated as though they were randomly assigned.

In addition to considering confounders, we also used both adolescent-report and peer self-report of peer AU in separate models to address concerns about potential bias associated with perception of peer AU. The association between peer and adolescent AU is smaller when peers report on their own SU (Bauman & Ennett, 1996). Evidence suggests that adolescents’ reports of their peers’ use tends to be biased in the direction of their own use (Henrey, Kobus, & Schoeny, 2011). However, such errors in perception of peer behavior may be critical to the development of adolescent SU and may still influence an adolescent's drinking behavior (D'Amico & McCarthy, 2006; Trucco et al., 2011).

Summary and the Present Study

In the current study, we test associations between adolescent AU and peer AU using PSA to control for a variety of potential confounding variables and interactions between confounders (see Table 1 for a full list of confounders) as well as a longitudinal design to establish temporal precedence from early- (age = 11) to mid-adolescence (age = 15). To test socialization, *peer* AU at baseline (Wave 1; W1) and one year later (Wave 2; W2) was used to create transition groups. To test selection, *target* adolescent AU at W1 and W2 was used to create transition groups. Transition groups were then compared on target AU at W2 and Wave 3 (W3) for the socialization models and on peer AU at W2 and W3 for the selection models. We used propensity scores to adjust for confounding when estimating whether our independent variables (IVs) were related to our dependent variables (DVs).

Regarding socialization, we hypothesized *a priori* that targets whose peer group shifted from no AU to AU between W1 to W2 would have higher levels of later target AU than targets whose peers remained abstinent at both W1 and W2. Regarding selection, we predicted that target adolescents who initiated AU between W1 and W2 would have higher subsequent peer AU than target adolescents who did not transition into AU. If, after balancing transition groups on the confounders, adolescent drinking does not predict later peer drinking or peer drinking does not predict later adolescent drinking, it would suggest that what researchers call “selection” and “socialization” effects of AU are explained by other factors. We also predicted that effect sizes would vary systematically by reporter of peer AU. That is, for socialization and selection, we expected perception of close friend AU to have the largest effects followed by peer self report of AU (Cruz et al., 2012; Bauman & Ennett, 1996). Given the mixed literature on whether selection or socialization has a larger effect size in this age range, we did not make specific predictions concerning the effect sizes across tests of socialization and selection.

Method

Participants

Recruitment—Participants (target adolescents) were recruited from the community using random-digit-dialing and were drawn from two longitudinal studies examining risk and protective factors for adolescent SU. Children were required to be between the ages of 10 and 12 and 11 and 12 years old at recruitment for each study, respectively. At each assessment, target adolescents provided the names of four close friends and one was recruited into the study (peers) to provide a collateral report of the target adolescent's peer environment. Peers were required to be within two years of age of the target adolescent and could not be a sibling. Moreover, targets were allowed to nominate different peers at each wave to capture the fluid nature of adolescent peer relationships in this age range (Knecht et al., 2010). Hence, peer self-reported AU is best viewed as a collateral report of the peer context of the target adolescent. A more detailed description of recruitment, descriptive statistics, length of interviews, compensation to participants and other procedural details for target adolescents can be found in Colder et al., (2011) and Colder et al., (2013). Procedure and descriptive statistics are reported below for peer adolescents given that this information has not been previously reported for both samples.

Description—The first sample included 378 families while the second sample included 387 families for a total of 765 families. Averaging across samples, adolescents were 10-13 years old (M_{age} = 11.3, *SD* = 0.76; 53% female) while peers were 8-15 (M_{age} = 11.5, *SD* = 1.15; 55% female) at the first assessment. Mean ages of targets at W1, W2, and W3 were 11.8 (*SD* = 0.79), 12.9 (*SD* = 0.79), and 13.9 (*SD* = 0.79), and the mean ages of peers at each assessment were 11.5 (*SD* = 1.15), 12.4 (*SD* = 1.27), and 13.4 (*SD* = 1.18), respectively. From W1 to W3, between 80.2% - 82.4% of the peer participants were White/non-Hispanic, between 10.4 - 13.5% were Black/African-American, between 1.4 - 3.5% were Hispanic, between .4 - .9% were Asian/Pacific Islander, and between 2.2 – 5.1% were of another (usually mixed) race/ethnicity. Most of the peer participants (73.2 to 77.5%) reported being from two-parent homes across all three assessments, and the median income of peer families ranged from $69,750 to $73,500, with 7 - 10.6% of the peer families receiving public assistance

Procedure

Procedures were similar for both samples. Three annual assessments were completed in a laboratory setting. For target and peer families, consent (caregiver) and assent (adolescent) procedures were completed and then the adolescent and caregiver were taken to separate rooms to enhance confidentiality and privacy. The interviewer read questionnaires aloud and responses were entered directly into a computer by the interviewer. Questions deemed “sensitive” (e.g., questions assessing SU and delinquency) were entered into the computer by the adolescent to further increase confidentiality.

Measures

Confounders—A set of 26 covariates were used in the PSA to account for bias in assessing socialization and selection effects related to early adolescent AU (see Table 1 for

details about measures). Prior work has established a variety of correlates of peer and adolescent AU that could confound the relationship between peer and adolescent SU (Chassin et al., in press; Zucker, 2006). In the current study, confounders included demographics (index of socioeconomic status, age, parental marital status, minority status, and gender), pubertal status, temperament (effortful control, negative affect, surgency, Behavioral Inhibition System [BIS] fear, BIS anxiety, Behavioral Activation System [BAS] impulsivity/fun seeking, BAS reward responsiveness, and BAS drive), psychopathology (depressed mood, oppositional defiant disorder [ODD], conduct disorder [CD], ADHD-inattention, and ADHD-hyperactivity), family environment (alcohol specific parenting, parental AU [average daily drinking averaged across mother and father], parental problematic AU [alcohol related negative consequences averaged across mother and father], and adolescent AU with parents permission), W1 peer delinquency, W1 AU for the socialization models, and W1 peer AU for the selection models. Given that our outcomes were adolescent and peer AU, we excluded SU items from our W1 peer delinquency measure to prevent overlap among confounders and our outcomes.

Target Alcohol Use at Waves 1-3—Items from the National Youth Survey (NYS) assessed lifetime AU at W1 and past year AU at W2 and W3 (Elliott & Huizinga, 1983). Lifetime AU was assessed with one dichotomous item (no/yes), while past year AU was assessed with open-ended frequency and quantity items, multiplied to form a quantity × frequency index (Q×F). Lifetime variables were used at W1 to control for all prior AU exogenous to our model. Fifty-three (6.9%) targets used alcohol at W1, while there were 712 abstainers (93.1%). Past year AU was used at W2 and W3 to model yearly changes in adolescent AU thereafter. As expected given the age of our sample, rates of AU were low and Q×F indices were skewed and kurtotic. Accordingly, ordinal variables were created from Q×F indices at W2 and W3 based on visual inspection of the distributions. Although creating categories from continuous variables is generally not recommended, this approach is useful for highly skewed variables (MacCallum, et al., 2002). The ordinal variables represented no use (W2 n = 652, 88.3%; W3 n = 536, 74.8%), a few sips to one drink (W2 n = 56, 7.6%; W3 n = 94, 13.1%), > 1 and < 4 drinks (W2 n = 22, 3.0%; W3 n = 50, 7.0%) and > 4 drinks (W2 n = 8, 1.1%; W3 n = 37, 5.1%).

Peer Alcohol Use—Peer AU was assessed with both target-report (TR) of peer AU (or perceived peer use) and peer self-report (PR) of peer AU. TR of peer AU was assessed using one item taken from Fergusson, Woodward, and Howard (1999), “Tell whether or not any of your three close friends have ever used alcohol without parent's permission.” Responses were keyed as yes (1) or no (0). As expected given the age of the sample, rates of perceived peer AU were low at W1 (no use n = 712, 93.1%; use n = 53, 6.9%), W2 (no use n = 614, 83.5%; use n = 121, 16.5%), and W3 (no use n = 715, 73.8%; use n = 188, 26.2%).

PR of peer AU was assessed with the same items that were used for target AU from the NYS. Lifetime variables were used at W1 to control for all prior peer AU exogenous to our model. Past year peer AU was used at W2 and W3 to model yearly change in peer AU thereafter. As with target AU, rates of peer AU were low at W1 (no use n = 635, 90.1%; use n = 70, 9.9%), W2 (no use n = 566, 86.2%; use n = 91, 13.8%), and W3 (no use n = 484,

76.7%; use *n* = 139, 23.3%). As described above, ordinal variables were created from the Q×F indices at W2 and W3. The ordinal variable represented no use (W2 *n* = 566, 86.2%; W3 *n* = 484, 76.7%), a few sips to one drink (W2 *n* = 52, 7.9%; W3 *n* = 66, 10.6%), > 1 and < 4 drinks (W2 *n* = 16, 2.4%; W3 *n* = 52, 5.1%) and > 4 drinks (W2 *n* = 23, 3.5%; W3 *n* = 41, 6.6%).

Observed Transition Groups—For PR models, peer W1 lifetime AU (no use vs. use) and a dichotomized peer W2 past year AU item (no use vs. use) was used to create the transition groups to test whether changes in peer use across W1 and W2 predicted target use at W2 and W3 (socialization). The ordinal W2 alcohol variables were not used to create transition groups due to small cell sizes. Accordingly, there were four possible transition groups based on different patterns of PR of peer AU across W1 and W2. Likewise, for TR of peer AU, W1 and W2 dichotomous variables were used to create the same four transition groups. Peers who did not use alcohol at either wave were put into the no peer AU group, which served as the control group (referred to as peer control; PR *n* = 601; TR *n* = 594). Peers who did not drink at W1, but did use alcohol at W2 were put into the peer initiation group (PR *n* = 87; TR *n* = 90). Peers who used alcohol at both waves were placed in the peer stable use group (PR *n* = 28; TR *n* = 31). Finally, peers who used alcohol at W1, but not at W2 were placed in the peer desistance group (PR *n* = 49; TR *n* = 19). As can be seen from the *n* in each group, small cell sizes precluded comparing peer stable use and peer desistance to any other groups in our socialization models.

For models assessing whether target AU predicted peer AU (selection), four possible transition groups were also created (target control, target initiation, target stable use, and target desistance). For the target control, target initiation, target stable use, and target desistance groups, the *n* in each group were 635, 62, 24, and 17, respectively. Again, only the target initiation and target control groups had large enough cell sizes for comparison. It is important to note that given the age of our sample, transition from no AU to AU for either peers or targets between W1 and W2 represents early initiation of AU. As such, small groups are to be expected.

Data Analysis

Separate propensity score models were run: (1) to test socialization and selection effects and (2) for each reporter of peer AU (TR or PR). Given that separate models were run for each comparison, each has a slightly different sample size. PSA involves two statistical models: (1) the model relating the confounders to the IV from which the propensity scores are estimated and (2) the model relating the IV to the DV in which the propensity scores adjust the data. Taking the example of the peer initiation vs. peer control group comparison, the propensity scores used to balance the socialization effect are estimated by regressing the IV (peer initiation vs. peer control) on the set of confounders in a generalized boosted regression model. The predicted probability of each participant being in their observed group is then saved. These predicted probabilities are the propensity scores (i.e., the probability of observed group membership conditioned on the confounders) and are used in the final outcome model (as weights; see below) to balance the groups when testing hypotheses. For groups to be reasonably balanced on the confounders using the weights, the

propensity scores within each group must have some degree of overlap. No overlap in propensity scores means that the groups are so different that there would be no way of achieving balance on the confounders so that groups can be treated as randomly assigned. Balance is formally assessed by comparing the IV (e.g., the transition groups in our case, peer initiation vs. peer control) on the confounders before and after weighting the data using the standardized mean difference (SMD). Good balance is achieved in the weighted model when the difference between the groups on the confounders is small or below |.2|.

Propensity Score Estimation—Propensity scores (π_i) were estimated in R using generalized boosted modeling (GBM) in the Twang package (McCaffery et al., 2004). GBM has been shown to improve propensity score estimation when there are a large number of confounders and can automatically include interactions among confounders (Buhlman & Yu, 2003; McCaffery et al., 2004). Once π_i is estimated, the inverse is taken ($1/\pi_i$ for alcohol initiators and $1/[1 - \pi_i]$ for control participants) to create the weights. In all models, interaction depth was set between 3 and 5-way interactions; shrinkage was set between 0.005 and 0.0005; and iterations were set between 10,000 and 25,000 (see McCaffery et al., 2004). Simulation work has shown that confounders having a stronger relationship with the IV than the DV can inflate the variance of the causal effect estimate (Brookhart et al., 2006). Peer delinquency satisfied these conditions in our TR socialization model and all selection models. Accordingly, in these models peer delinquency was included in the final outcome analyses as a covariate.

Assessing Overlap and Balance—Overlap was checked for each propensity score model using overlapping histograms and all models had good overlap (histograms available upon request from the first author). Although there is no gold standard for measuring balance, simulation and theoretical work has suggested that the SMD is a better measure of balance than others measures (e.g., *t* test statistic and Kolmogorov-Smirnov [KS]; Ho, Imai, King, & Stuart, 2007; Stuart et al., 2013). As such, we used the SMD's to assess balance.[1] SMDs were compared before and after weighting for each confounder for each propensity score model. In the case of models in which multiple imputation was used, the mean SMD across imputations was presented. SMDs at or below .2 (in absolute value) after weighting are considered indicative of adequate balance given that .2 represents a small effect (Cohen, 1988). All confounders in which SMDs were above .2 after weighting were included as covariates in the final outcome analysis to rigorously test hypotheses.

Estimation of the Final Model—Final outcome models were run as path models in Mplus 7 using all three waves to establish temporal precedence. Robust maximum likelihood estimation (MLR) was used to handle both the ordinal outcomes and the inverse propensity weights which are treated like survey weights (Muthén & Muthén, 1998-2007). These models assume proportional odds amongst the logits for the ordinal outcomes. Nested log-likelihood tests, the Akaike Information Criteria (AIC), and Bayesian Information Criteria (BIC) were used to evaluate the proportional odds assumption, and results supported

[1]Although only SMDs are presented, *t* test statistics and KS test statistics were also examined. The three measures of balance were generally consistent before weighting (between 8% and 12% of tests disagreed before weighting) and were always consistent after weighting. Hence inferences were very similar across the different measures of balance before weighting and identical after weighting.

the assumption (nested log-likelihood tests *ps* > .10 and lower AIC and BIC for the constrained models). Accordingly, cumulative logits were used to model the ordinal outcomes.

For the socialization effects, target AU at W2 was regressed on the peer initiation vs. peer control group comparison. Target AU at W3 was regressed on target AU at W2 and the peer initiation vs. peer control group comparison. For the selection effect, peer AU at W2 was regressed on target initiation vs. target control. Moreover, peer AU at W3 was regressed on peer AU at W2 and the target initiation vs. target control group comparison. Confounders on which adequate balance was not achieved were included as statistical controls for W2 and W3 outcomes in all models. To provide estimates of effect size, odds ratios (OR) were computed given that standardized βs and R^2s were not available for our imputation models.

Assumptions—PSA makes several assumptions. First, PSA assumes unconfoundedness. To the extent that there are additional confounds not included in the present analysis reported effects may be biased. Second, PSA assumes no interference. That is, the treatment applied to one subject cannot affect the potential outcomes of another subject. Given lack of a nested structure in our data, the no interference assumption was likely met in the present analysis. Third, PSA assumes non-zero probability of treatment in that each person in the population could have been exposed to either condition (treatment or control) and this is also tenable with our data.

Missing Data—Retention for target families was strong in the combined sample with an attrition rate of 3.8% and 6.5% at W2 and W3, respectively. We tested for differences between participants with data at all three waves and participants with missing data at either W2 or W3 on demographic variables in Table 1, W1 target AU, and W1 peer AU using chi-square and ANOVA. There were no significant differences between those with or without missing data suggesting that missing data did not have a substantial impact on study findings related to target families. Given the low rate of missing data at W2 (3.79%) and lack of significant differences, missing data at W2 was excluded from analyses resulting in a sample of 736 for the TR models. Missing data from W2 to W3 was handled using full information maximum likelihood (FIML).

Missing data also occurred for peer data because for some targets we were unable to recruit one of the four close friend peers. Proportion of missing peer data was 8.3% at W1, 13.9% at W2, 18.6% at W3. We tested for differences between participants with missing peer data at any wave and without missing peer data on the same set of variables listed above for target adolescents. Targets with missing peer data tended to report lower socioeconomic status ($F[1, 764] = 34.49$, $p < 0.01$; $R^2 = 0.04$), minority status (Δx^2 [1] = 15.02, $p < 0.01$; phi = 0.14) and to be from families in which the parents were not married (Δx^2 [4] = 25.37, $p < 0.01$; phi = 0.18), but the effects were small. Groups were not different on target or peer AU at W1. These few small differences suggest that missing peer data likely did not have a strong influence on findings. Nonetheless, given the higher rate of missing data for peers, multiple imputation (20 imputations) was used. Our imputation model included all 26 variables in Table 1, and peer age and gender. Imputed data sets were created using the package Mix in R (Schafer, 1997). Multiple imputation involved running the propensity

score model within each imputed data set, creating weights within each data set, saving each data set separately, and then using the imputation function in Mplus 7 to estimate the final model across the imputed data sets.

Results

Socialization Effects: Peer Alcohol Use Predicting Target Alcohol Use

TR of Peer Use—The SMDs before and after weighting for all models can be found in the Technical Appendix (see Online Resource 1). For the peer initiation vs. peer control group comparison, the groups were unbalanced on age, gender, parental marital status, negative affect, surgency, depressed mood, fear (BIS), anxiety (BIS), CD, pubertal status, W1 target AU, and W1 target AU with parent's permission before weighting. After weighting, variables with SMDs above .2 were age, gender, and fear (BIS). As such, the final outcome analysis assessed the effect of peer initiation vs. peer control group status on W2 and W3 target AU above and beyond peer delinquency and the variables on which adequate balance was not achieved. In support of socialization, there was a significant positive effect of group (peer initiation vs. control) on W2 AU ($b = 1.69$, $p < .001$) and W3 AU ($b = 1.35$, $p < .001$) indicating that perceived peer initiation of AU between W1 and W2 was associated with higher probability of target use at W2 and W3 after controlling for peer delinquency and equating the transition groups on the 25 confounders and interactions among confounders. Table 2 contains odds ratios for all outcome models.

PR of Peer Alcohol Use—Before weighting the peer initiation vs. peer control group comparison was unbalanced on socioeconomic status, age, parental marital status, effortful control, negative affect, surgency, depressed mood, fear (BIS), ODD, alcohol parenting, pubertal status, and W1 target AU (age, marital status, negative affect, fear [BIS], pubertal status, and W1 target AU overlapped with the TR model). The only SMD above .2 after weighting was age. Consistent with hypotheses and the TR models, the effect of group was significant on W2 AU ($b = 0.89$, $p = .01$) and W3 AU ($b = 0.91$, $p = .002$). This suggests that peer initiation between W1 and W2 was associated with increased probability of target use at W2 and W3 after balancing on confounders.

Selection Effects: Target Alcohol Use Predicting Peer Alcohol Use

TR of Peer Alcohol Use—For the target initiation vs. target control group comparison, before weighting the groups were unbalanced on age, minority status, drive (BAS), parental AU, pubertal status,W1 target AU with parent's permission, and W1 peer AU. After weighting, SMDs suggested that groups were still unbalanced on age, minority status, W1 target AU with parent's permission, and W1 peer AU. Consistent with a selection effect, there was a significant effect of group on W2 ($b = 2.89$, $p < .001$) and W3 peer AU ($b = 1.04$, $p = .007$) indicating that target initiation of use between W1 and W2 was associated with higher probability of peer AU at W2 and W3 after balancing on the 25 confounders and controlling for peer delinquency.

PR of Peer Alcohol Use—For the target initiation vs. target control group comparison in which peer self-reported use was the outcome, the groups were unbalanced on age, minority

status, drive (BAS), parental AU, pubertal status, and W1 target AU with parent's permission before weighting. SMDs above .2 after weighting were age and W1 target AU with parent's permission. Consistent with a selection effect, there was a significant effect of group on W2 (b = 1.10, p = .006) and W3 peer AU (b = .76, p = .01) indicating that target initiation of AU between W1 and W2 is associated with higher probability of PR of peer AU at W2 and W3 after controlling for peer delinquency and balancing on the 25 other confounders.

Discussion

Although previous research provides evidence that peer AU predicts target AU and vice versa, garnering support for both selection and socialization processes in the etiology of early adolescent AU (Leung et al., 2011; Burk et al., 2012), no research to our knowledge has attempted to adjust for the wide range of potential confounders and interactions among confounders that might account for the association between adolescent and peer AU. A major contribution of this study was to test associations between early peer and adolescent AU above and beyond 26 potential confounding variables using propensity scores. We utilized a longitudinal design to establish temporal precedence and PR of peer AU as well as TR of peer AU to disentangle effects due to shared reporter bias. Initiation groups were generally unbalanced on a range of potential confounders when compared to the control groups consistent with prior literature on AU, including pubertal timing, temperament, externalizing pathology, depressed mood, parenting, and parental AU. Our results, theoretical formulations, and prior empirical work suggest that early initiation of AU is nested within multiple spheres of ecological influence (Ennett et al., 2006; Bronfenbrenner, 1979), supporting the importance of considering potential confounding variables and utilizing propensity scores when testing whether early peer and adolescent AU predict each other over time. In general, even after controlling for confounders, results supported our hypotheses and the larger literature. That is, target adolescent AU predicted peer drinking, supporting selection, and peer AU predicted target adolescent drinking, supporting socialization. Although cross-sectional analyses suggested peer self-reported models were associated with smaller effects than perceived peer AU as has been found in previous literature (Bauman & Ennett, 1996), longitudinal analyses suggest a similar sized effect across reporter of peer AU.

Selection and Socialization of Alcohol use in Early Adolescence

This rigorous test of the prospective associations between peer and target AU provided evidence that both selection and socialization are involved in the early stages of adolescent AU. This supports a growing literature that has utilized a range of methodological innovations and theoretical models to disaggregate selection and socialization processes and found evidence for both (Leung et al., 2011; Osgood et al., 2013). Some reports in the literature indicate that selection has a larger effect than socialization in early adolescence (Burk et al., 2012; Knecht et al., 2010), while others indicate the opposite pattern (Mercken et al., 2012) or comparable effect sizes (Osgood et al., 2013). At W2, our results suggested comparable effect sizes for both peer AU predicting target drinking (selection) and target AU predicting peer drinking (socialization). At one year after our transitions (W3), effect

sizes were similar across reporter of peer AU and across selection and socialization. Averaging across reporter, the OR for socialization was 2.43 and the OR for selection was 2.49. Results from 11 longitudinal studies during early- to mid-adolescence (age: 10-15) that utilized perceived peer AU and/or peer self-reported AU and controlled for prior levels of adolescent and peer AU when testing effects (Burke et al., 2012; Cruz et al., 2012; Curran et al., 1997; D'Amico & McCarthy, 2006; Engels et al., 1999; Merken et al., 2012; Mudnt et al., 2012; Knecht et al., 2010; Osgood et al., 2013; Poelen et al., 2007; Reifman et al., 1998) suggest small to medium effect sizes (Cohen, 1988) for both selection (ORs range: 1.06 - 3.60; βs range: 0.12 – 0.27) and socialization (ORs range: 0.80 - 3.04; βs range: 0.14 – 0.23). Our results fall in the middle of this range and also suggest small to medium effects for both selection and socialization.

Although previous research has controlled for factors such as age, ethnicity, gender, and prior levels of adolescent and peer AU (Burk et al., 2012; Knecht et al., 2010; Mercken et al., 2012), no studies to our knowledge have controlled for other factors that may be particularly important confounders of both tests of selection and socialization. For instance, onset of problem behavior (CD and ODD) and peer delinquency have been found to precede initiation of SU, including AU (Colder et al., 2013; King et al., 2004; Scalco et al., 2014) and effects of these variables have been shown to be exacerbated by early pubertal timing (Costello et al., 2007; Wichstrøm, 2001). To the extent that this sequencing of risk factors represents risk for selection into an early alcohol using peer group, problem behavior, peer delinquency, and pubertal development are an important set of confounders to consider when testing selection and socialization effects. Given that we controlled for these factors, our estimates of effect size for the selection and socialization effects may be more precise than the previous literature. Moreover, mixed findings regarding the relative importance of socialization and selection may be a function of inconsistencies across studies in accounting for confounders and interactions among confounders. Initiation groups based on peer self-report (socialization effects) were unbalanced on a large range of confounders in our study (6). Thus, previous literature using peer self-report and not controlling for other correlates of AU, may be overestimating the effect size of selection and socialization (Mouw, 2006; Shalizi & Thomas, 2011).

Recent critiques of observational studies, including social network modeling (Mouw, 2006; Shalizi & Thomas, 2011), have argued that unobserved differences between adolescents and peers (latent homophily and socialization) make selection and socialization very difficult to separate; however, it is possible that when a large number of factors related to socialization and selection are included in a PSA that these critiques become less impactful. It is important for future research to control for a wide range of confounders when attempting to disentangle selection from socialization, at least for early adolescent AU. We propose that future work controlling for confounders are likely to replicate our findings that selection and socialization have similar sized effects on early adolescent AU.

Consistent with prior literature, the selection and socialization effects at W2 were smaller when peer self-reported AU was used as opposed to perceived peer use (Bauman & Ennett, 1996; Osgood et al., 1996). Empirical evidence suggests that adolescent reports of their peers AU behavior tend to be biased in the direction of their own use inflating effect sizes of

the relationship between peer and adolescent AU (Henry et al., 2011). As previous researchers have pointed out, while larger effects for perceived peer AU may suggest shared method variance or biased reporting of peer use (Henry et al., 2011), it may also indicate that perception of peer use (arguably a cognitive and not a behavioral factor), regardless of accuracy, influences subsequent adolescent AU (Trucco et al., 2011). At W2, perceived peer AU still had a larger effect size after all the confounders were used to balance the groups (even confounders which are strongly related to inaccuracies in TR of peer AU, such as target AU; see Henry et al., 2011) providing limited support for the premise that larger effects sizes in models utilizing perception of peer use may be due to over and under reporting of peer SU by targets who do or do not use, respectively. However, this pattern was limited to our W2 outcomes, which were partially cross-sectional. At W3, effect sizes did not vary by reporter providing some evidence for the validity of our measurement (Cronbach & Meehl, 1955) and suggesting that controlling for prior levels of AU and balancing on the 26 confounders reduced discrepancy between reporters. It is possible that inaccuracies in adolescent report of peer SU behavior may be reduced by including certain confounders and covariates and/or using advanced modeling techniques (e.g., PSA, SEM). This may be a useful direction for future research given the financial and logistic difficulties of collecting social network data.

Limitations

Results from this study should be understood within the context of certain limitations. First, yearly assessment waves provide limited temporal resolution. Given that our AU items asked about use in the past year, it is not possible to distinguish precisely whether peer use at W2 preceded target use at W2 and vice versa. Although balancing on W1 target and peer AU for socialization and selection, respectively helps establish temporal precedence, it cannot definitively rule out the possibility that either selection or socialization processes may systematically precede the other in time, as would be required to adequately establish a causal effect (Holland, 1986; Meehl, 1978). However, we note that even studies that have obtained measurements over two month intervals have found that network structure changes significantly and that selection and socialization effects were both significant (Knecht et al., 2010). As such, we contend that this is a methodological challenge for much of the literature on selection and socialization of SU given that reports of adolescent and peer SU are based on a fixed interval (e.g., past two months or past year) and do not precisely establish when that use happened within the interval in question. Perhaps combining ecological momentary assessment, social network modeling, and PSA would provide the additional rigor required to establish bi-directional causal effects between peer and adolescent SU and test the mechanisms underlying the effects.

Given that the co-evolution of network ties were not considered in the present analysis due to sampling only one peer, we were unable to control for structural network confounders on early adolescent AU. As such, effects on adolescent AU due to popularity, reciprocation of friendship, and density of users in the network could not be ruled out in the current analysis as confounders. In addition, given that peers were allowed to be different across the waves, the model cannot determine whether specific close friend peers are moving into or out of specific peer drinking groups; however, the study can rule out several confounders that

studies using social network analysis have not ruled out, such as temperament, psychopathology, peer delinquency, and pubertal timing. Extending social network models to adjust for confounding variables using propensity scores may provide even more precise estimates of the robust relationship between peer and adolescent AU (Kao et al., 2013).

Statisticians and philosophers in the propensity score and causal inference literatures have argued that propensity scores can provide an unbiased estimate of a causal effect within correlational data when *every possible* confounder of the relationship being examined is used to estimate propensity scores (Rubin, 2005). To the extent that certain confounders are not included in the current analysis (Holland, 1986) or that unobserved differences exist between respondents (latent homophily and socialization; Mouw, 2006; Shalizi & Thomas, 2011), effects of peer and adolescent AU on each other may still be confounded by other factors, and hence not causally related to each other. We view the current study as a first step and it will be important for future studies to build on what we have done, replicating our findings using different measures of our confounders, other potential confounders, and other modeling approaches, including social network analysis. Recent claims that social network studies are confounded by latent homophily and social influence (Mouw, 2006; Shilizi & Thomas, 2011) may be directly tested by combining social network analysis with PSA and SEM.

Clinical Implications and Conclusions

Previous literature has supported reciprocal effects between peer and adolescent AU providing support for selection and socialization of early AU despite using different methodological approaches to disentangle estimated effects (Leung et al., 2011; Burk et al., 2012). Our analyses add to this broader literature in two ways. First, we provide evidence that prior reported socialization and selection effects are likely biased, but are robust even after adjusting for a large number of potential confounders. Second, our results support ecological theories of development that suggest that adolescent risky behavior is nested within multiple spheres of ecological influence (Bronfenbrenner, 1979). Multidimensional Family Therapy (MDFT), one treatment modality amongst many for adolescent SU problems (Waldron & Turner, 2008), emphasizes Bronfenbrenner's ecological model and the importance of intervening on multiple levels including schools, parents, peers, and the juvenile justice system (Liddle, 2010). Perhaps due to its underlying ecological philosophy, MDFT has been shown to effect a wider range of developmental outcomes compared to other treatments such as cognitive behavioral therapy and has also been shown to decrease affiliation with delinquent and substance-using peers as long as one year post treatment (Liddle, 2010). Our results suggest that interventions aimed at altering the peer group should not only attempt to reduce unsupervised time with substance using peers (and thus prevent further socialization and maintenance of SU) but also address the adolescent's motivation to affiliate with and select substance-using friends.

Our results suggest a comparable effect size for socialization and selection. Although comparable effect sizes could be interpreted in a variety of ways, one possibility is that selection and socialization processes are mutually interdependent during the early stages of SU, as suggested by other researchers (Cruz et al., 2012; Mudnt et al., 2012). To the extent

that adolescents initiate and continue to use substances together in the face of a dynamic social network, maintaining a strict dichotomy between selection and socialization may not capture the true complexities of the relationships between peer and adolescent SU. Understanding the mechanisms of the complex relationship between adolescent and peer SU may further aid in improving interventions that attempt to intervene on adolescent peer groups. Furthermore, adjusting for confounders may be particularly important for studying adolescent SU given that experimental designs are not plausible.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

This research was funded by two grants from the National Institute on Drug Abuse (R01 DA020171 and R01 DA019631) awarded to Dr. Craig R. Colder.

References

Alati R, Baker P, Betts KS, Connor JP, Little K, Sanson A, Olsson CA. The role of parental alcohol use, parental discipline and antisocial behaviour on adolescent drinking trajectories. Drug and Alcohol Dependence. 2014; 134:178–184. [PubMed: 24479151]

Bauman KE, Ennett ST. On the importance of peer influence for adolescent drug use: Commonly neglected considerations. Addiction. 1996; 91:185–198. [PubMed: 8835276]

Brookhart MA, Schneeweiss S, Rothman KJ, Glynn RJ, Avorn J, Sturmer T. Variable selection for propensity score models. American Journal of Epidemiology. 2006; 163:1149–1156. [PubMed: 16624967]

Bronfenbrenner, U. The Ecology of Human Development: Experiments by Nature and Design. Harvard University Press; Cambridge, MA: 1979.

Buhlmann P, Yu B. Boosting with the L_2 loss: Regression and classification. Journal of the American Statistical Association. 2003; 98:324–339.

Burk WJ, Van der Vorst H, Kerr M, Stattin H. Alcohol use and friendship dynamics: Selection and socialization in early-, middle-, and late-adolescent peer networks. Journal of Studies on Alcohol and Drugs. 2012; 73(1):89–98. [PubMed: 22152666]

Chassin, L.; Colder, CR.; Hussong, A.; Sher, K. Substance use and substance use disorders.. In: Sameroff, AJ., editor. Developmental psychopathology. 3 ed.. in press

Chassin L, Flora DB, King KM. Trajectories of alcohol and drug use and dependence from adolescence to adulthood: The effects of familial alcoholism and personality. Journal of Abnormal Psychology. 2004; 113(4):483–498. [PubMed: 15535782]

Cohen, J. Statistical power analysis for the behavioral sciences. 2nd ed.. Erlbaum; Hillsdale, NJ: 1988.

Colder CR, Hawk LW, Lengua LJ, Wieczorek WF, Eiden RD, Read JP. Trajectories of reinforcement sensitivity during adolescence and risk for substance use. Journal of Research on Adolescence. 2013; 23:345–356. [PubMed: 23772169]

Colder CR, Trucco EM, Lopez HI, Hawk LW, Read JP, et al. Reinforcement Sensitivity theory and laboratory assessment of BIS and BAS in children. Journal of Research in Personality. 2011; 45:198–207. [PubMed: 21603055]

Colder CR, Scalco MD, Trucco EM, Read JP, Lengua LJ, Wieczorek WF. Prospective associations of internalizing and externalizing problems and their cooccurrence with early adolescent substance use. Journal of Abnormal Child Psychology. 2013; 41(4):667–677. [PubMed: 23242624]

Costello E, Sung M, Worthman C, Angold A. Pubertal maturation and the development of alcohol use and abuse. Drug and Alcohol Dependence. 2007; 88S:S50–S59. [PubMed: 17275214]

Cronbach LJ, Meehl PE. Construct validity in psychological tests. Psychological Bulletin. 1955; 52(4): 281–302. [PubMed: 13245896]

Cruz JE, Emery RE, Turkheimer E. Peer network drinking predicts increased alcohol use from adolescence to early adulthood after controlling for genetic and shared environmental selection. Developmental Psychology. 2012; 48(5):1390–1402. [PubMed: 22390657]

Curran PJ, Stice E, Chassin L. The relation between adolescent alcohol use and peer alcohol use: A longitudinal random coefficients model. Journal of Consulting and Clinical Psychology. 1997; 65(1):130–140. [PubMed: 9103742]

D'Amico EJ, McCarthy DM. Escalation and initiation of younger adolescents' substance use: The impact of perceived peer use. Journal of Adolescent Health. 2006; 39(4):481–487. [PubMed: 16982381]

Dishion TJ, Spracklen KM, Andrews DW, Patterson GR. Deviancy training in male adolescents friendships. Behavior Therapy. 1996; 27(3):373–390.

Dishion TJ, Medici Skaggs N. An ecological analysis of monthly 'bursts' in early adolescent SU. Applied Developmental Science. 2000; 4(2):89–97.

Elliott DS, Huizinga D. Social group and delinquent behavior in a national youth panel. Criminology: An Interdisciplinary Journal. 1983; 21:149–177.

Ellis, LK.; Rothbart, MK. Revision of the Early Adolescent Temperament Questionnaire.. Poster presented at the Biennial Meeting of the Society for Research in Child Development; Minneapolis, MN. 2001.

Engels RCME, Knibbe RA, De Vries H, Drop MJ, Van Breukelen GJP. Influences of parental and best friends' smoking and drinking on adolescent use: A longitudinal study. Journal of Applied Social Psychology. 1999; 29(2):337–361.

Ennett ST, Bauman KE, Hussong A, Faris R, Foshee VA, Cai L, DuRant RH. The peer context of adolescent substance use: Findings from social network analysis. Journal of Research on Adolescence. 2006; 16(2):159–186.

Ennett ST, Foshee VA, Bauman KE, Hussong A, Cai L, Luz H, et al. The social ecology of adolescent alcohol misuse. Child Development. 2008; 79(6):1777–1791. [PubMed: 19037949]

Epstein, JL. The selection of friends: Changes across the grades and in different school environments.. In: Berndt, TJ.; Ladd, GW., editors. Peer relationships in child development. 1989. p. 158-187.

Ferguson CJ, Meehan DC. With friends like these..: Peer delinquency influences across age cohorts on smoking, alcohol and illegal substance use. European Psychiatry. 2011; 26(1):6–12. [PubMed: 21067903]

Ferguson DM, Woodward LJ, Horwood LJ. Childhood peer relationship problems and young people's involvement with deviant peers in adolescence. Journal of Abnormal Child Psychology. 1999; 27:357–370. [PubMed: 10582837]

Giancola PR, Parker AM. A six-year prospective study of pathways toward drug use in adolescent boys with and without a family history of a substance use disorder. Journal of Studies on Alcohol. 2001; 62:166–178. [PubMed: 11327183]

Grant BF, Dawson DA. Age at onset of alcohol use and its association with DSM-IV alcohol abuse and dependence: Results from the National Longitudinal Alcohol Epidemiologic Survey. Journal of Substance Abuse. 1997; 9:103–110. [PubMed: 9494942]

Gruber E, DiClemente RJ, Anderson MM, Lodico M. Early drinking onset and its association with AU and problem behavior in late adolescents. Preventive Medicine. 1996; 25:293–300. [PubMed: 8781007]

Hartup, WW. Peer relations.. In: Mussen, P., editor. Handbook of child psychology. Vol. 4. Wiley; New York: 1983. p. 104-196.

Hawkins JD, Catalano RF, Miller JY. Risk and protective factors for alcohol and other drug problems in adolescence and early adulthood: Implications for substance abuse prevention. Psychological Bulletin. 1992; 112:164–105.

Haynie DL, Osgood D. Reconsidering peers and delinquency: How do peers matter? Social Forces. 2005; 84(2):1109–1130.

Henry DB, Kobus K, Schoeny ME. Accuracy and bias in adolescents' perceptions of friends' substance use. Psychology of Addictive Behaviors. 2011; 25:80–89. [PubMed: 21244119]

Hitner JB, Swickert R. Sensation seeking and alcohol use: A meta-analytic review. Addictive Behaviors. 2006; 31:1383–1401. [PubMed: 16343793]

Ho DE, Imai K, King G, Stuart EA. Matching as non-parametric processing for reducing model dependence in parametric causal inference. Political Analysis. 2007; 15:199–236.

Holland PW. Statistics and causal inference. Journal of the American Statistical Association. 1986; 81:945–960.

Hussong AM, Curran PJ, Chassin L. Pathways of risk for accelerated heavy AU among adolescent children of alcoholic parents. Journal of Abnormal Child Psychology. 1998; 26(6):453–466. [PubMed: 9915652]

Jaccard J, Blanton H, Dodge T. Peer influences on risk behavior: An analysis of the effects of a close friend. Developmental Psychology. 2005; 41(1):135–147. [PubMed: 15656744]

Kao, E.; Toulis, P.; Airoldi, E.; Rubin, D. Causal estimation of peer influence effects.. Poster presented at the International Conference for Machine Learning; Atlanta, GA. 2013, June;

Kessler RC, Berglund P, Demler O, Jin R, Merikanglas KR, Walters EE. Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the national comorbidity survey replication. Archive of General Psychiatry. 2005; 62:593–602.

King KM, Iacono WG, McGue M. Childhood externalizing and internalizing psychopathology in the prediction of early substance use. Addiction. 2004; 99:1548–1559. [PubMed: 15585046]

Knecht AB, Burk WJ, Weesie J, Steglich C. Friendship and alcohol use in early adolescence: A multilevel social network approach. Journal of Research on Adolescence. 2011; 21(2):475–487.

Kodl M, Mermelstein R. Beyond modeling: Parenting practices, parental smoking history, and adolescent cigarette smoking. Addictive Behaviors. 2004; 29:17–32. [PubMed: 14667418]

Leung RK, Toumbourou JW, Hemphill SA. The effect of peer influence and selection processes on adolescent AU: A systematic review of longitudinal studies. Health Psychology Review. 2011; 5:1–32.

Liddle HA. Multidimensional family therapy: A science-based treatment system. The Australian and New Zealand Journal of Family Therapy. 2010; 31:138–148.

MacCallum RC, Zhang S, Preacher KJ, Rucker DD. On the practice of dichotomization of quantitative variables. Psychological Methods. 2002; 7(1):19–40. [PubMed: 11928888]

Marmorstein NR. Longitudinal associations between alcohol problems and depressive symptoms: Early adolescence through early adulthood. Alcoholism: Clinical and Experimental Research. 2009; 33:49–59.

McCaffrey DF, Ridgeway G, Morral AR. Propensity score estimation with boosted regression for evaluating causal effects in observational studies. Psychological Methods. 2004; 9:403–425. [PubMed: 15598095]

Meehl PE. Theoretical risks and tabular asterisks: Sir Karl, Sir Ronald, and the slow progress of soft psychology. Journal of Consulting and Clinical Psychology. 1978; 46:806–834.

Mercken L, Steglich C, Knibbe R, Hein de Vries H. Dynamics of friendship networks and alcohol use in early and mid-adolescence. Journal of Studies on Alcohol and Drugs. 2012; 73:99–110. [PubMed: 22152667]

Miller, WR.; Tonigan, JS.; Longabaugh, R. Test Manual (Vol. 4, Project Match Monograph Series). National Institute on Alcohol Abuse and Alcoholism; Rockville, MD: 1995. The drinker inventory of consequences (DrInc): An instrument for assessing adverse consequences of alcohol abuse..

Mouw T. Estimating the causal effect of social capital: A review of recent research. Annual Review of Sociology. 2006; 32:79–102.

Mundt MP, Mercken L, Zakletskaia L. Peer selection and influence effects on adolescent alcohol use: A stochastic actor-based model. BMC Pediatrics. 2012; 12:115–125. [PubMed: 22867027]

Muthén, LK.; Muthén, BO. Mplus User's Guide. 7th Ed.. Muthén and Muthén; Los Angeles, CA: 1998-2007.

Osgood WD, Ragan DT, Wallace L, Gest SD, Fienberg ME, Moody J. Peers and the emergence of alcohol use: Influence and selection processes in adolescent friendship networks. Journal of Research on Adolescence. 2013; 23(3):500–512.

Osgood WD, Wilson JK, Bachman JG, O'Malley PM, Johnston LD. Routine activities and individual deviant behavior. American Sociological Review. 1996; 61:635–55.

Peterson AC, Crockett L, Richards M, Boxer A. A self-report measure of pubertal status: Reliability, validity, and initial norms. Journal of Youth and Adolescence. 1988; 17:117–133. [PubMed: 24277579]

Pillow DR, et al. Confirmatory factor analyses examining attention deficit hyperactivity disorder symptoms and other childhood disruptive behaviors. Journal of Abnormal Child Psychology. 1998; 26:293–309. [PubMed: 9700521]

Poelen EAP, Engels RCME, Van Der Vorst H, Scholte RHJ, Vermulst AA. Best friends and alcohol consumption in adolescence: A within-family analysis. Drug and Alcohol Dependence. 2007; 88:163–173. [PubMed: 17127016]

Popper, K. Conjectures and refutations: The growth of scientific knowledge. Routledge & Kegan Paul; New York, NY: 1963.

Reifman A, Barnes GM, Dintcheff BA, Farrell MP, Uhteg L. Parental and peer influences on the onset of heavier drinking among adolescents. Journal of Studies on Alcohol. 1998; 59(3):311–317. [PubMed: 9598712]

Rosenbaum PR, Rubin DB. The central role of the propensity score in observational studies for causal effects. Biometrika. 1983; 70:41–55.

Rubin DB. Causal inference using potential outcomes: Design, modeling, decisions. Journal of the American Statistical Association. 2005; 100:322–331.

Scalco MD, Colder CR, Hawk LW, Read JP, Wieczorek WF, Lengua LJ. Internalizing and externalizing problem behavior and early adolescent substance use: A test of a latent variable interaction and conditional indirect effects. Psychology of Addictive Behaviors. in press.

Schafer, JL. Analysis of Incomplete Multivariate Data. Chapman; Boca Raton, FL: 1997.

Shalizi CR, Thomas AC. Homophily and contagion are generically confounded in observational social network studies. Sociological Methods & Research. 2011; 40:211–239. [PubMed: 22523436]

Snijders TAB, Bunt GG, Steglich CEG. Introduction to stochastic actor-based models for network dynamics. Social Networks. 2010; 32:44–60.

Steglich C, Snijders TAB, Pearson M. Dynamic networks and behavior: Separating selection from influence. Sociological Methodology. 2010; 40:329–393.

Stuart EA, Lee BK, Leacy FP. Prognostic score-based balance measures can be a useful diagnostic for propensity score methods in comparative effectiveness research. Journal of Clinical Epidemiology. 2013; 66:S84–S90. [PubMed: 23849158]

Sung M, Erkanli A, Angold A, Costello EJ. Effects of age at first substance use and psychiatric comorbidity on the development of substance use disorders. Drug and Alcohol Dependence. 2004; 75:287–299. [PubMed: 15283950]

Trucco EM, Colder CR, Wieczorek WF, Lengua LJ, Hawk LW Jr. Early adolescent alcohol use in context: How neighborhoods, parents, and peers impact youth. Development and Psychopathology. 2014; 26:425–436. [PubMed: 24621660]

Trucco EM, Colder CR, Wieczorek WF. Vulnerability to peer influence: A moderated mediation study of early adolescent intentions to drink. Addictive Behaviors. 2011; 36:729–736. [PubMed: 21420241]

Waldron HB, Turner CW. Evidence-based psychosocial treatments for adolescent substance use. Journal of Clinical Child and Adolescent Psychology. 2008; 37(1):238–261. [PubMed: 18444060]

Wichstrøm L. The impact of pubertal timing on adolescents' alcohol use. Journal of Research on Adolescence. 2001; 11:131–150.

Windle M. Parental, sibling, and peer influences on adolescent substance use and alcohol problems. Applied Developmental Science. 2000; 4:98–110.

Zucker, RA. Alcoholism; a life span perspective on etiology and course.. In: Sameroff, AJ., editor. Developmental psychopathology. 2 ed.. Kluwer Academic/Plenum Publishers; New York, NY: 2006. p. 569-585.

Table 1

Alpha's, Reporter, References, and Abbreviations for Confounders

Covariates	α	Reporter	Abbreviations
Descriptives			
Socioeconomic Index	NA	Parent	Socec
Age	NA	Parent	Age
Caucasian/minority	NA	Parent	Minority
Gender	NA	Parent	Gender
Marital Status	NA	Parent	Marital
Temperament (Ellis & Rothbart, 2001)			
Effortful Control	.89	Parent	EC
Negative Affect	.72	Parent	Neg Aff
Surgency	.84	Parent	Surg
Depressed Mood	.72	Parent	Dep Mood
Reinforcement Sensitivity (Colder et al., 2011)			
Fear (BIS)	.85	Parent	Fear (BIS)
Anxiety (BIS)	.62	Parent	Anx (BAS)
Reward Responsiveness (BAS)	.61	Parent	Rew (BAS)
Impulsivity/Fun Seeking (BAS)	.75	Parent	Imp (BAS)
Drive (BAS)	.69	Parent	Drive (BAS)
Disruptive Behavior (Pillow et al., 1998)			
DBD-Oppositional Defiant Disorder	.89	Parent	ODD
DBD-Conduct Disorder	.72	Parent	CD
DBD-ADHD-Impulsivity	.84	Parent	ADHD-imp
DBD-ADHD-Inattention	.91	Parent	ADHD-att
Parenting (Kodl & Mermelstein, 2004)			
Alcohol Specific Parenting	.79	Parent	Alc Parent
Parenting Efficacy	.88	Parent	Parent Eff
Parental Alcohol use (Miller, et al., 1995)			
Parental Alcohol Use	.90	Parent	Parent Alc
Parent Alcohol Consequences	.90	Parent	Par Alc Con
Pubertal Status (Peterson et al., 1998)	.70	Adolescent	Puberty
TR Peer Delinquency (Fergusson et al., 1999)	.74	Adolescent	Peer Delin
Adolescent Alcohol Use (Elliott & Huizinga, 1983)			
Alcohol Use with Parental Permission	NA	Adolescent	T Alc w- per
W1 Lifetime Alcohol Use	NA	Adolescent	T Alc
Peer Alcohol Use			

Covariates	α	Reporter	Abbreviations
W1 PR Peer Lifetime Alcohol Use	NA	Peer	PR P Alc
W1 TR Peer Alcohol Use	NA	Adolescent	TR P Alc

Note. α = Cronbach's alpha, NA = 1 item, BIS = Behavioral Inhibition System, BAS = Behavioral Activation System, DBD = Disruptive Behavior Disorder, ADHD = Attention Deficit Hyperactivity Disorder, PR = peer self-report, TR = target report, and W = Wave. The abbreviations in the last column are used in the balance tables (Online Resource 1).

Table 2

Odds Ratios for the Socialization and Selection Models Using Inverse Propensity Score Weights to Balance Groups

Socialization Comparisons	W2 Target AU	W3 Target AU	Stability
TR Initiation vs. Control	18.58	2.37	4.84
Age	1.03	1.32	
Gender	0.78	1.45	
Fear (BIS)	0.78	0.68	
Peer Delin	0.57	6.92	
PR Initiation vs. Control	2.44	2.49	6.10
Age	1.84	1.57	

Selection Comparison	W2 TR Peer AU	W3 TR Peer AU	Stability
TR Initiation vs. Control	17.97	2.84	3.44
Age	1.72	1.69	
Minor	1.47	0.99	
W1 TR P Alc	5.23	1.07	
T alc w- per	1.15	1.41	
Peer Delin	3.29	1.16	

Selection Comparison	W2 PR Peer AU	W3 PR Peer AU	Stability
TR Initiation vs. Control	3.02	2.14	2.75
Age	1.54	2.14	
T alc w- per	1.33	1.31	
Peer Delin	1.30	3.27	

Note. See Table 1 for abbreviations of Covariates. TR = target report, PR = Peer report. Column's 1 and 2 are odds ratios for each path model with outcome as column and IV and covariates as row. Column 3 is the odds ratio between W2 and W3 AU (for the socialization models, this is target AU and for the selection models it is peer AU).