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The Interplay of Peer, Parent, and Adolescent Drinking

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Abstract

Objective—To explore variability in the link between peer and adolescent drinking by parental drinking. Stress and differential susceptibility perspectives led to hypotheses that adolescents with drinking parents would be more reactive to peer drinking but also to peer abstention.

Methods—Using data from the National Longitudinal Study of Adolescent to Adult Health, regressions estimated whether the association between peer alcohol use and increased drinking among adolescents was moderated by parental drinking. A regions of significance test identified the level of peer drinking that predicted adolescent drinking in the context of parental drinking.

Results—Adolescents with binge-drinking parents were more likely to increase drinking at every level of peer drinking, supporting the stress perspective; such adolescents did not accrue benefits from abstaining peers, going against the differential susceptibility perspective.

Conclusions—Far from monolithic, peer influences on adolescent risky behaviors may require family environments and genetic predispositions conducive to those behaviors.

The norms and behaviors of peers are central to alcohol use in adolescence, but not all adolescents are equally susceptible to such peer influences (Johnston et al. 2011; Ennett et al. 2006). As such, studying the link between peer drinking and adolescent drinking has been a central part of efforts to curb risky behaviors and understand adolescent behavior more broadly (Crosnoe, Muller, and Frank 2004; Kandel 1996). A stress process model (Pearlin 1981) suggests that the influence of social environments on problem behaviors is amplified among those already most vulnerable to engaging in these behaviors. Consequently, peer influences on drinking may vary based on whether adolescents are already vulnerable to drinking due to other formative influences, including, for example, their parents. Indeed, parents can model problematic drinking behavior for their children, increase opportunities and motivations for their children to drink, and pass down genetically heritable traits associated with drinking to their children (Chassin et al. 2013; Dick et al. 2006). Thus, the interplay of peer and parent drinking, not just each on its own, is likely to factor into whether and how much adolescents drink.

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In this spirit, this study explores the degree to which parental problem drinking moderates the link between peer drinking and adolescent drinking over time. Integrating theory across medical sociology and developmental psychology, we posit and test two hypotheses. Following the stress response perspective (and the closely connected diathesis stress perspective), the first hypothesis is that a negative peer environment would matter most to adolescent drinking in the context of a family vulnerability, so that the link between peer drinking and adolescent drinking would be magnified when parents drink. The differential susceptibility perspective makes this same prediction but, at the same time, contends that people in vulnerable circumstances will be more reactive to *both* negative and positive influences in their environmental contexts—more affected by risks and getting more from resources. Following this perspective, the alternate hypothesis is that the link between having peers who drink and adolescent drinking will be stronger among adolescents whose parents are problem drinkers, as will the link between having abstaining peers and adolescent drinking.

These hypotheses will be tested by examining how the interaction of peer drinking and parental drinking predicts changes in adolescent drinking over time with data on adolescents in the National Longitudinal Study of Adolescent to Adult Health (Add Health). The adjudication between the stress and differential susceptibility hypotheses is supported by a region of significance test that points to the thresholds at which a predictor becomes a significant risk or protective factor for an outcome in the presence of a moderator (Fraley 2014; Roisman et al. 2012). This research can identify unique subsets of the adolescent population in need of support and bring new conceptual and methodological tools to the sociological study of health.

Peer and Parental Influences on Adolescent Drinking

That adolescents tend to look highly similar to their peers in terms of drinking is well-established (Crosnoe 2006; Schulenberg et al. 1999; Kandel 1996; Aseltine 1995). The heightened sensitivity to social influence that is a hallmark of adolescence—reflecting a combination of individuation from parents and socioemotional and brain development—increases the degree to which adolescents are attuned to and shaped by what their peers are doing (Steinberg 2008). Adolescents, therefore, are susceptible to the influence of alcohol-using peers, above and beyond the powerful selection effects of homophily. As such, researchers have increasingly turned their attention to the variability within this general pattern—if adolescents are generally influenced by their peers in terms of drinking, are there specific groups of adolescents who are not or who are even more than usual? Elucidating such variability helps to identify adolescents most and least at risk (Crosnoe 2006; Crosnoe et al. 2004).

Two otherwise similar adolescents may differ in whether their drinking is influenced by peers depending on, for example, their contexts or their own developing personal characteristics (Paschall, Lipperman-Kreda, and Grube 2014; Crosnoe and Riegle-Crumb 2007). A stress model—stress process in medical sociology, diathesis stress in developmental psychology—provides a lens through which to view this variability because it considers differential exposure/vulnerability as possible explanations for adverse outcomes.

Indeed, the stress model asserts that some adolescents are more reactive to stressors (e.g., peer behaviors) because of prior histories, genetic predispositions, or current circumstances (e.g., parental behaviors). Thus, some adolescents are more vulnerable to the influences of alcohol-using peers than others, and they will be more likely to go along with peers while others resist or are unaffected.

Although the vulnerability central to the stress model can come in many forms, we focus on parental problem drinking, which is directly associated with adolescent drinking via multiple mechanisms. Parents model alcohol use and provide opportunities for adolescents to drink through their permissive attitudes towards drinking or the availability of alcohol in their homes (Abar, Abar, and Turrissi 2009; Komro et al. 2007). Parents also influence personal development via the intergenerational transmission of genes specific to alcohol use and/or addiction or related behaviors and conditions (e.g., risk-taking or impulsivity) that may increase the likelihood of drinking (Dick et al. 2006; Whitfield et al. 2004). Consequently, parents who drink tend to have adolescents who drink through complex developmental processes that connect social life and biology (Chassin et al. 2013; Chassin, Pitts, and Prost 2002).

Notably, the consideration of vulnerability and reactivity in the stress model shifts attention away from the direct influences of peers and parents on adolescent drinking to the interplay between these two influences. In other words, we are not testing if peer and parental drinking are both associated with adolescent drinking on their own but instead whether the association between peer drinking and adolescent drinking is moderated by parental problem drinking. In line with the stress model, the first of our hypotheses is that adolescents who are exposed to problem drinking in the home may be vulnerable to drinking within their peer groups and, therefore, drink more. If drinking is problematic, they would be at a double disadvantage.

A Different Kind of Parent-Peer Interplay

The stress model focuses on the interplay between personal vulnerability and environmental risk and how these amplify each other, but the other side of the interplay between the peer and parental drinking may be more positive. The differential susceptibility model, which has been prominent in developmental research on psychopathology (Belsky and Pluess 2009), highlights this positive side. It contends that vulnerable youth are more reactive to their social environments in general—they are more at risk in the face of negative influences but have more to gain from positive resources. In other words, they suffer when environments are unhealthy and benefit when environments are healthy (Belsky, Bakermans-Kranenburg, and van IJzendoorn 2007). Applying this model to the issues at hand, adolescents who are vulnerable to drinking because their parents are problem drinkers will have unhealthier behavior when immersed in peer contexts characterized by high levels of drinking but will have healthier behavior when they are surrounded by peers who abstain from drinking. In the former case, peers facilitate the translation of vulnerability into drinking behavior, and, in the latter, peers block or buffer it.

In line with this model, our examination of the moderating role of parental problem drinking in the link between peer and adolescent drinking is guided by a second hypothesis, which is that having peers who drink will be more positively associated with adolescent drinking when parents problem drink while having peers who do not drink will be more negatively associated with adolescent drinking when parents problem drink. As described below, the regions of significance test developed as part of the theoretical discussion of diathesis stress and differential susceptibility in developmental psychopathology will be leveraged to consider this interplay among adolescents, peers, and parents in the etiology of a major public health issue.

Methods

Data and Sample

Add Health followed a nationally representative sample of adolescents beginning in 1994 with an in-school survey of 90,118 students in 132 middle and high schools across the U.S. (Harris et al., 2009). The in-school survey was used to create a nationally representative sample of 20,745 students for the core in-home interview sample. Wave I in-home interviews were conducted in 1995 when students were aged 11 to 18. Additional in-home interviews were conducted in 1996 (Wave II; ages 12 to 18, with Wave I high school seniors excluded), 2001-2002 (Wave III; ages 18 to 26), and 2007-2008 (Wave IV; ages 24 to 32).

Given our focus on adolescence, we used data from Waves I and II, necessarily excluding high school seniors in Wave I, who were purposely dropped from the core Add Health sampling frame in Wave II. We retained all youth with valid longitudinal sampling weights (necessary to adjust for study design effects and to correct for differential attrition across waves). The final sample included 13,568 students, with all missing data within this sample estimated through full-maximum likelihood estimation (FIML).

Measures

Table 1 presents descriptive statistics for all study variables.

Adolescent drinking—Consistent with past research with Add Health (e.g., Crosnoe, Benner, and Schneider 2012; Johnson 2004), self-reports were used to measure alcohol use at Waves I and II. Adolescents reported how often they drank alcohol in the past year, with responses ranging from 0 (none) to 6 (daily or nearly daily). The Wave II version served as the outcome, the Wave I version as the lagged control variable.

Peer drinking—Respondents in the in-school survey nominated up to five female and five male friends, most of whom also participated in Add Health. This network design allowed for the characteristics of most nominated friends to be measured directly from those friends' survey responses (Crosnoe et al. 2004; Cavanagh 2004; Haynie 2001; Moody 2001). Peer drinking, therefore, was the mean of self-reported alcohol use (same responses described above) for all nominated friends in the sample. This network measure minimizes problems associated with adolescents' overestimation of their peers' similarity to them and their incomplete information about their peers (Kandel 1996). At the same time, the network

measures are imperfect, particularly given the high rate of missingness. A few schools that participated in Add Health did not allow the in-school survey to be administered, and these respondents were missing friendship nominations. Missingness also occurred when friends were nominated outside Add Health schools or did not participate in Add Health for some reason. FIML (described below) helps to address these biases. Further, a sensitivity check replicated all analyses for the sub-sample of respondents with valid peer data; and, the results were consistent with the full sample results.

Parent problem drinking—In the Wave I parent survey, parents self-reported their frequency of various types of drinking. From these reports, we created a binary variable differentiating between parents who had recent experiences with binge drinking (i.e., has had five or more drinks in one sitting in the last month) and those who had not.

Covariates—A set of characteristics was measured to account for sociodemographic variability and spurious factors. These covariates included adolescent gender (1 = female), grade in school, parent's education (1 = less than high school, 2 = high school graduate, 3 = some post-secondary education, 4 = college graduate, 5 = post-graduate degree), family structure (1 = lives with both biological parents), and race/ethnicity (dummy variables for non-Hispanic White, non-Hispanic Black, Hispanic, non-Hispanic Asian, and other/multi-racial).

To gauge different contexts of peer drinking, we also controlled for several peer characteristics. Involvement with friends was the sum of four items (1 = yes) that gauged the frequency with which adolescents had gone to the friend's house, talked on the telephone with the friend, spent time with the friend outside of school, and hung out with the friend. This value was averaged across all nominated friends (Cavanagh 2004). Further, data from the in-school survey were used to capture peer academic engagement in three ways. For each of these three measures, we created an individual-level measure and then aggregated it across all nominated peers. The individual-level measures used this aggregation process were: grade point average (self-reported grades in core subjects on conventional four-point scale), school attachment (average of self-reported degree to which adolescents felt close to people at their school, a part of their school, and happy to be at their school in the past year on a scale from 1, strongly disagree, to 5, strongly agree), and educational expectations (adolescents' estimations that they will graduate from college on a scale from 0, no chance, to 8, it will happen).

Analytical Strategy

This study considered variability in the link between peer drinking (focal predictor) and adolescent drinking (outcome) according to parental problem drinking (moderator). To examine this variability, we estimated a series of models regressing Wave II adolescent drinking on measures of peer drinking, parent drinking, and the interaction between the two (note: both peer and parent measures were grand mean-centered). These models controlled for the full set of covariates as well as the Wave I measure of adolescent drinking, creating a lagged modeling strategy that effectively captured changes in drinking across waves (Glazerman, Levy, and Myers 2003). These regression models were estimated in Mplus

(Muthén and Muthén 2006). FIML estimated exogenous variance for missingness, so that all cases in the sample were retained even if they had missing data on individual variables. Moreover, we employed the cluster feature in Mplus to account for students being nested within schools in the sampling frame as well as the longitudinal sampling weight to address differential probability of being included in the frame and differential cross-wave attrition from the sample.

Our pair of hypotheses concerned the different forms that a significant peer drinking x parent drinking interaction might take. We explored this form using the regions of significance examination. Conventionally, significant interactions are interpreted with a simple slope analysis that calculates the association between the predictor and outcome at high and low levels of the moderator. Recently, Roisman and colleagues (2012) put forward a statistical test, regions of significance that gauges the significance of the full range of values for the interaction. Here, we computed the regions of significance for the moderation of the association between peer drinking and adolescent drinking by parent drinking using an online calculator employing the Johnson-Neyman technique (Preacher, Curran, and Bauer 2006). This tool (see <http://www.yourpersonality.net/interaction/>) was developed by Fraley as a supplement to the review of interaction effects by Roisman and colleagues (2012). By inputting the intercept, regression coefficients for the predictor, moderator, and interaction term, variances of the predictor, moderator, and interaction term, covariances of the predictor and moderator, and degrees of freedom, the application produces a graph plotting the interaction and calculates regions of significance with respect to the predictor and the moderator.

This regions of significance test gives evidence for differential susceptibility if the interaction term involves crossed lines and the association between the outcome and moderator is significant for both low and high values of the predictor; in this case, if an adolescent with drinking parents had lower drinking than other youth when peers abstained and had greater drinking than other youth when peers drank. On the other hand, if the regions of significance determines that the association between the outcome and moderator is significant only for high values of the predictor regardless of whether the interaction lines cross, it points to a stress model; in this case, an adolescent with problem-drinking parents drank more than other youth when peers drank but did not seem to derive any more benefit than other youth from having abstaining peers.

As a sensitivity check (results not shown), we compared the three peer academic engagement variables to the peer drinking variable, given that prosocial behaviors among peers (e.g., being engaged in school) better tapped the idea of peer resources in the differential susceptibility perspective than the absence of a problem behavior among peers (e.g., peer abstention). We estimated these parental drinking x peer interactions and interpreted them with the regions of significance test. This sensitivity check revealed the same pattern as peer drinking.

Results

Peer, Parent, and Adolescent Drinking

To begin, we examined whether a risk factor (peer drinking) and a vulnerability factor (parental problem drinking) were each independently and interactively associated with adolescent drinking. Table 2 presents the results. Model 2 included the measure of drinking among peers as a predictor of Wave II adolescent drinking along with all covariates and the Wave I measure of adolescent drinking, Model 2 added the parental drinking measure, and Model 3 added the interaction between drinking among peers and parental drinking.

In Models 1-2, both peer drinking and parent drinking significantly predicted adolescent drinking at Wave II, net of Wave I adolescent drinking ($b = .06$, $p < .05$ for peer drinking in Model 1; $b = .24$, $p < .001$ for parental drinking in Model 2). Every point increase on the scale of drinking among peers was associated with an increase in adolescent drinking between Waves I and II of about 5% of a standard deviation on the Wave II drinking distribution. The difference between having a parent who had recently engaged in binge drinking and one who had not was equivalent to about 15% of a standard deviation on that distribution.

Stress and Differential Susceptibility

In addition to the main effects of drinking among peers and parental drinking, their interaction was significant and positive ($b = .18$, $p < .05$). To interpret this interaction, we followed the conventional approach of calculating predicted Wave II drinking levels among adolescents with different combinations of drinking among peers (one standard deviation below and above the mean) and parental drinking (any recent experience with binge drinking vs. no such experience) with all other variables held to their sample means/modes. Figure 1 graphically depicts these predicted levels.

When parents had no recent history of binge drinking, adolescent drinking at Wave II (net of Wave I drinking and the covariates) did not differ depending on the level of drinking among peers. In other words, peer drinking and adolescent drinking were not associated with each other among adolescents with parents who did not binge drink. Yet, among adolescents with binge-drinking parents, a gap opened up between those whose peers drank and those whose peers did not, with the former drinking more. Thus, in line with the stress model, adolescents with binge-drinking parents appeared to be more susceptible to the influences of drinking peers than other adolescents, who did not seem to be all that susceptible.

Graphing the interaction between peer and parent drinking indicated that vulnerability to drinking peers differed between adolescents with and without parents who had a recent history of binge drinking. What it did not do was identify at what point on the spectrum of peer drinking this increased vulnerability became meaningful. The differential susceptibility hypothesis contended that moderation could have two poles—heightened vulnerability to the negative influences of peers who drank *and* to the positive influences of peers who did not. The graph in Figure 1 suggests that such differential susceptibility was likely not occurring, as there seemed to be no point along the continuum of drinking among peers at which the children of binge-drinking parents had lower levels of drinking than the children of other

parents (i.e., the interaction lines did not cross). Even without using the region of significance test, we could be reasonably sure that the interaction between drinking among peers and parental drinking fit the stress model better than the differential susceptibility model. The region of significance test, however, could still be informative by shedding light on the level of peer drinking that was significantly associated with adolescent drinking among adolescents with binge-drinking parents.

Figure 2 presents the interaction between peer and parent drinking in a different form than Figure 1. Based on the online calculator developed by Fraley (Roisman et al. 2012), it shows the region in which drinking among peers was significantly associated with adolescent drinking in the focal group of adolescents with binge-drinking parents (note that the units on the X and Y axes refer to standard deviation increments).

In this figure, adolescent drinking at Wave II tended to increase as peer drinking increased. The simple slope for adolescents with binge-drinking parents was .24 ($p < .001$). The simple slope for students with parents who had no recent history of binge drinking was also marginally significant but of a smaller magnitude (.06, $p < .10$). This difference in slopes confirmed the observed pattern in Figure 1 that the link between drinking among peers and adolescent drinking was stronger for adolescents with binge-drinking parents. The shaded region indicates the level at which the difference in slopes became statistically significant. At all points of drinking among peers above a Z-score of -1.135, adolescents with binge-drinking parents had significantly higher levels of drinking than those without such parents. A Z-score of -1 relates to one standard deviation below the mean of peer drinking, effectively a value of zero on the peer drinking scale (i.e., the adolescents' peers were non-drinkers). In other words, adolescents with binge-drinking parents became more vulnerable to having drinking peers than other adolescents once the level of drinking among those peers reached 1 standard deviation below the mean of peer drinking; essentially, having peers who drank at any level. That the crossover point on peer drinking (at a Z-score of -1.290) was below the lower bound of the region of significance is the best evidence for the stress model.

In terms of our hypotheses, therefore, we found support for the stress perspective but not for the differential susceptibility perspective. Adolescents with binge-drinking parents were more susceptible than other youth to what appeared to be negative influences of drinking among peers (i.e., high levels of drinking among peers associated with more adolescent drinking) but not to positive influences of peer abstention (i.e., peer abstention from drinking associated with less adolescent drinking). Furthermore, this apparent vulnerability of adolescents with binge-drinking parents to having peers who drank held for just about any level of peer drinking.

Discussion

Adolescence is a critical life course stage during which individuals undergo rapid social, psychological, and physical development. As they individuate from parents and establish peer networks, adolescents differ in how they respond to their social environments. One peer-influenced behavior that has received public and academic attention is adolescent drinking. Adolescents tend to drink more when their peers drink through a mixture of

selection and socialization, but not all adolescents follow this pattern. We explored such variability by examining parental drinking as a moderator of the link between peer and adolescent drinking.

The results of this study did demonstrate a moderating role of parental problem drinking in this oft-studied association. Young people with parents who binge drank were more likely to drink if their peers were also drinkers. This pattern—closely aligned with the predictions of stress response and diathesis stress models—was not paired with a different but related pattern; namely, the prediction of the differential susceptibility model that parental drinking would increase the positive association between peer drinking and adolescent drinking but also the negative association between peer abstention and adolescent drinking. Instead, region of significance testing showed that adolescents with binge-drinking parents were more vulnerable to drinking than other youth when their peers were drinkers—at any level—and did not appear to derive any more benefit than other youth from having peers who did not drink. Sensitivity analyses comparing peer academic engagement variables to peer drinking were performed to account for the fact that the absence of drinking in a peer group is not necessarily a peer resource. Tapping into three aspects of peer academic engagement as potential resources in the peer context, however, we again found support for a stress perspective, with no evidence of differential susceptibility. These results highlight three important themes.

First, some adolescents may be less susceptible to potentially negative norms and values among peers and better able to self-regulate based on the modeling, influence, and genetic heritability of their parents. This finding corresponds to research by Steinberg and Monahan (2007) showing that, although adolescents are generally more susceptible to peers than adults are, they may still develop resistance to peer influences when they have internal or external resources on which to draw. As a result, different processes may be at work among peers when particular vulnerabilities are, or are not, present. Given the limited nature of the peer and family data in our analyses, we were unable to understand processes within and among peer groups or intra-family dynamics that could explain the elevated risk for drinking among adolescents with peers and parents who drank. Future research, however, needs to both unpack and contextualize these mechanisms.

Second, potential peer risks may be more important to understand than potential peer protective resources in the context of family vulnerabilities. At least in our sample of adolescents, moderation by parental drinking pointed to risky peer groups seeming to be worse for adolescents with parents who drank, not prosocial peer groups seeming to be better for them (both in terms of the absence of peer drinking or the presence of pro-academic norms among peers). Importantly, the lack of evidence for the positive side of differential susceptibility in this scenario is at odds with other research on drinking, such as a study by Olfson and colleagues (2014) indicating that associating with non-drinking peers was beneficial to teens with a genetic predisposition to drink. We are not ready to rule out that differential susceptibility is at work in the interplay of parents and peers and its role in adolescent drinking. Instead, we argue that a more extensive investigation of variability is in order. Among adolescents with binge-drinking parents, having abstaining peers might not matter in general, but it might matter to some. The key is to identify, in a systematic way, the

ones for which it matters—in terms of other sources of vulnerability. Another possibility is that the differential susceptibility only emerges for some dimensions of adolescent drinking, such the initiation of drinking or developmental increases and declines in drinking as adolescence unfolds (Chassin et al. 2013; Albert et al. 2013). Certainly, as adolescents transition to adulthood, drinking becomes more normative and the influences of peers and parents may take on new forms. Similarly, perhaps gender or socioeconomic status condition these pathways. Adolescent social life is gendered, for example, and friendship expectations differ between girls and boys, as does the acceptability and social benefits/costs of drinking. Drinking also plays more of a social role and influences social status as socioeconomic status increases (Crosnoe, 2011; Clark and Ayers 1993). Future research should shed light on which adolescents are most vulnerable to peers' risky behaviors when their family contexts also expose them to risk.

Third, the potentially dynamic and multi-dimensional role of parental drinking in adolescent drinking needs to be unpacked. Here, we saw that something long considered to be the most important factor in adolescent drinking (i.e., association with peers who drank) only mattered in the face of parental drinking. Why parental drinking had such a role was difficult to adjudicate in conventional designs. Clearly, it has a heritability component, but the behavioral influences of parents on adolescents goes beyond heritability (Handley and Chassin 2013). Genetically informed designs—behavioral genetics ACE models, for example, or models incorporating genetic traits known to raise the likelihood of drinking—can provide insight into these issues and need to be better leveraged in social science research on health behavior (Watson et al. 2012; Dick et al. 2006). At the same time, social and psychological mechanisms by which parental drinking may be associated with adolescent drinking above and beyond genetic links need to be studied more explicitly. Is the channel of such influence about values, opportunities, self-medication, or something else? Moving forward with this line of research, however, scholars should consider alternative ways to measure parental drinking. One limitation of our analyses was the operationalization of parental drinking as binge drinking over a short time period. Exposing adolescents to binge drinking may be qualitatively different than exposing adolescents to casual drinking, and the consistency of that exposure likely also matters.

Theoretically, the relevance of this research on the interplay among parent, peer, and adolescent drinking goes beyond the specific conceptualizations of stress and differential susceptibility models. It also speaks to the transactional emphasis of many general perspectives on human development, such as developmental systems and life course (Johnson, Crosnoe, and Elder 2011), that highlight how behavioral trajectories reflect the ongoing bidirectional relations between what is going on inside youth (e.g., biological and psychological processes) and what is going on outside them (e.g., proximate and distal contextual process) and how these transactions vary across diverse segments of the population. Research influenced by and informing such theoretical perspectives can then guide policy intervention by identifying who needs help in finer-tuned ways.

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Figure 1.
Moderation of the Association between Drinking among Peers and Adolescent Drinking by
Parental Drinking

Figure 2.
Regions of significance in interaction between drinking among peers and parental drinking
as a predictor of adolescent drinking

Table 1

Descriptive Statistics for Study Variables

	Mean	SD	%
Drinking Variables			
Adolescent (Wave II)	1.091	1.519	
Adolescent (Wave I)	1.076	1.474	
Peer (Wave I)	1.084	1.202	
Parent binge (Wave I)			12.76
Covariates			
Gender (female)			51.27
Grade level	9.339	1.465	
Parent education			
Less than high school			12.86
High school graduate			29.78
Some post-secondary			20.51
College graduate			24.26
Post-graduate degree			12.60
Family structure (two-parent)			53.10
Race/ethnicity			
Non-Hispanic white			51.75
Non-Hispanic black			19.62
Hispanic			16.94
Non-Hispanic Asian			6.21
Other/ multi-racial			5.49
Number of friends	3.038	2.591	
Involvement with friends	2.109	1.066	
Friends' GPA	2.858	0.710	
Friends' school attachment	3.702	0.817	
Friends' educational expectations	6.486	1.924	

Note: $n = 13,568$; responses for adolescent and peer drinking variables range from 0 to 6 with increasing frequency of alcohol use; school attachment is a scale from 1 to 5 with increasing attachment; educational expectations is estimations of graduating from college on a scale from 0 to 8 with increasing likelihood.

Table 2

Results of Models Predicting Wave II Adolescent Drinking

	<i>Estimate (SE)</i>		
	Model 1	Model 2	Model 3
Peer drinking	0.062 (0.028)	* 0.061 (0.028)	* 0.061 (0.028)
Parent binge drinking		0.242 *** (0.060)	0.236 *** (0.060)
Peer drinking x parent drinking			0.183 * (0.083)
<i>Covariates</i>			
Female	-0.140 (0.044)	** -0.134 (0.044)	** -0.135 (0.045)
Grade	0.189 (0.014)	*** 0.192 (0.014)	*** 0.192 (0.014)
Parent education	0.027 (0.021)	0.031 (0.021)	0.028 (0.021)
Two-bio parent household	-0.182 (0.033)	*** -0.162 (0.033)	*** -0.158 (0.034)
Alcohol use at Wave I	0.004 (0.013)	0.003 (0.013)	0.004 (0.013)
Race/ethnicity (ref: non-Hispanic white)			
Hispanic	-0.134 (0.065)	* -0.125 (0.064)	† -0.118 (0.064)
Non-Hispanic black	-0.532 (0.055)	*** -0.528 (0.055)	*** -0.516 (0.054)
Non-Hispanic Asian	-0.599 (0.100)	*** -0.581 (0.098)	*** -0.581 (0.101)
Other/multi-racial	-0.048 (0.093)	-0.046 (0.093)	-0.042 (0.094)
Number of friends	0.046	*** 0.045	*** 0.045

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	<i>Estimate (SE)</i>		
	Model 1	Model 2	Model 3
Involvement with friends	(0.008) 0.217 *** (0.019)	(0.007) 0.213 *** (0.019)	(0.008) 0.214 *** (0.020)
Friends' GPA	(0.038) -0.189 *** (0.038)	(0.038) -0.188 *** (0.038)	(0.038) -0.183 *** (0.038)
Friends' school attachment	(0.030) -0.004 (0.030)	(0.031) -0.004 (0.031)	(0.030) -0.003 (0.030)
Friends' educational expectations	(0.012) 0.016 (0.012)	(0.012) 0.017 (0.012)	(0.012) 0.016 (0.012)

n = 13,568;
† *p* < .10,
* *p* < .05,
** *p* < .01,
*** *p* < .001.