Examining Explanatory Mechanisms of the Effects of Early Alcohol Use on Young Adult Alcohol Dependence

KATARINA GUTTMANNOVA, PH.D.,[a,*] KARL G. HILL, PH.D.,[a] JENNIFER A. BAILEY, PH.D.,[a] JUNGEUN OLIVIA LEE, PH.D.,[a] LACEY A. HARTIGAN, M.ED.,[a] J. DAVID HAWKINS, PH.D.,[a] AND RICHARD F. CATALANO, PH.D.[a]

[a]*Social Development Research Group, School of Social Work, University of Washington, Seattle, Washington*

ABSTRACT. Objective: This study examined potential explanatory mechanisms linking childhood alcohol use onset and chronicity of adult alcohol dependence by testing the following three competing hypotheses: (1) a marker hypothesis, where early onset of alcohol use may be simply a marker for other factors that have been linked to both age at initiation and adult alcohol problems; (2) a compromised development hypothesis, where early alcohol initiation may interfere with adolescent development, which can lead to later alcohol problems; and (3) an increased substance use hypothesis, where early onset of alcohol use may lead to increased substance use in adolescence and, in turn, chronic alcohol dependence. **Method:** Data came from a longitudinal community sample of 808 participants recruited at age 10 in 1985. Participants were followed through age 33 in 2008 with 92% retention. **Results:** Childhood onset of alcohol use (before age 11), when compared with initiation during adolescence, predicted an increased chronicity of adult alcohol dependence, even after accounting for the hypothesized confounds from the marker hypothesis. In addition, adolescent compromised functioning did not mediate this relationship between early alcohol use and chronicity of adult dependence (Hypothesis 2), nor did adolescent substance use (Hypothesis 3). However, compromised functioning and substance use in adolescence predicted increased chronicity of alcohol dependence in young adulthood. **Conclusions:** Prevention efforts as early as the elementary grades should focus on delaying the onset of alcohol use and reducing substance use in adolescence as well as improving school functioning, reducing adolescent problem behaviors, and targeting adolescent peer networks. (*J. Stud. Alcohol Drugs, 73,* 379–390, 2012)

EARLY ONSET OF ALCOHOL USE has been linked to increased alcohol problems in adulthood. For example, progressively earlier initiation has been found to carry an increased risk for later alcohol misuse (Hawkins et al., 1999) and other adolescent alcohol-related problem behaviors (Gruber et al., 1996), as well as adult alcohol abuse and dependence (Grant et al., 2001). Results of studies based on retrospective reports suggest that early adolescence might be a vulnerable period for alcohol use onset, linking it with increased rates of lifetime alcohol problems (Dawson et al., 2008; DeWit et al., 2000; Grant and Dawson, 1997; Hingson et al., 2006). However, retrospective reports of initiation of various behaviors often suffer from "forward telescoping bias," placing the age at onset at a later time than it actually occurred (Johnson and Schultz, 2005). Few studies have used prospective, longitudinal data to test these links. Furthermore, as Donovan et al. (2004) concluded in the proceedings of the 2003 symposium of the Research Society on Alcoholism, there is a dearth of studies focusing on alcohol use among elementary students, or the "really underage" drinkers. Importantly, using prospective, longitudinal data, Guttmannova et al. (2011) found that very early onset of alcohol use (before age 11), when compared with initiation during early, mid-, and late adolescence, was related to an increased chronicity of alcohol dependence in young adulthood. Although these studies have documented the link between early alcohol use and adult alcohol use disorder, the mechanisms linking the two are not well understood.

Using the same sample, the present study expands on the findings of Guttmannova et al. (2011) and examines potential factors that may account for the link between pre-adolescent onset of alcohol use and increased chronicity of alcohol dependence in adulthood. Specifically, this study examined the following three competing hypotheses: (1) a marker hypothesis, (2) a compromised adolescent functioning hypothesis, and (3) an increased substance use hypothesis.

The marker hypothesis

One potential explanation of the link between early onset of alcohol use and later alcohol problems involves the marker hypothesis. Under this view, early alcohol use may simply be a marker or a symptom of risk factors that are salient predictors of adult alcohol problems (King and Chassin, 2007; McGue and Iacono, 2008; White, 1992). In other words, both adult alcohol dependence and early age at drinking onset may arise from a common vulnerability to alcohol problems such as shared familial vulnerability (genetic or environmental; Prescott and Kendler, 1999).

Received: August 15, 2011. Revision: January 5, 2012.

This research was supported by National Institute on Alcohol Abuse and Alcoholism Grant R01AA016960, National Institute on Drug Abuse Grants R01DA009679 and R01DA024411, and Robert Wood Johnson Foundation Grant 21548. These organizations had no further role in study design; in the collection, analysis, and interpretation of data; or in the writing of the report.

*Correspondence may be sent to Katarina Guttmannova at the Social Development Research Group, University of Washington, 9725 3rd Ave. N.E., Suite 401, Seattle, WA 98115, or via email at: kg27@u.washington.edu.

Because family environment has been identified as a predictor of both age at onset of alcohol use (Hawkins et al., 1997) and adult alcohol problems (Ellis et al., 1997; Maggs et al., 2008), we examined measures of general and alcohol-specific family risk. In particular, we included family functioning variables representing general family risk as well as measures of parental drinking representing alcohol-specific family risk as potential marker variables. We also considered as potential markers sociodemographic risk variables commonly linked to alcohol problems, including ethnicity, childhood poverty, and gender (Hasin et al., 2007; Johnston et al., 2008; Stinson et al., 1992; Treno et al., 2000). If findings show that age at onset of alcohol use is unrelated to later alcohol dependence once these variables are controlled, this would suggest that early onset of alcohol consumption is a symptom of other risk factors associated with adult alcohol psychopathology and therefore would provide support for the marker hypothesis.

Compromised adolescent functioning hypothesis

A competing explanation for the association between the early onset of alcohol use and young adult alcohol problems suggests that early alcohol initiation may disrupt adolescent development, which then increases the likelihood of adult alcohol use disorders. For example, adolescents who begin drinking early may consequently become exposed to social and physical environments that promote problem behaviors and exposure to other risk factors associated with maladaptive outcomes (Jessor, 1991). Or, early drinking may compromise future trajectories of youth development through its influence on their ability to achieve important milestones that constitute the building blocks of subsequent developmental periods, including the formation of positive peer relations and the acquisition of essential academic skills (DeWit et al., 2000; Masten et al., 2008; Schulenberg and Maggs, 2002). This study examined several socio-emotional and school functioning variables as well as environmental mediators of the relationships between very early alcohol use and later alcohol dependence.

Specifically, this study examined whether the relationship between pre-adolescent onset of alcohol use and adult alcohol dependence is mediated through impaired social functioning, such as increased problem behaviors and delinquency. Few studies have focused on the effects of very early alcohol use on negative outcomes in late adolescence. One study revealed that very early onset of drinking was related to more delinquency and behavior problems in late adolescence (Peleg-Oren et al., 2009). Yet the link between adolescent delinquency and young adult alcohol problems has been well established (Guo et al., 2001; Harford and Muthén, 2000). This study examined adolescent problem behavior as one of the potential mediating mechanisms between the very early onset of alcohol use and adult alcohol problems.

The formation of positive peer relations is among the key developmental tasks of adolescence. Peer social networks constitute an important environment in which youth development is embedded and can influence adolescent alcohol use and subsequent outcomes (Schulenberg and Maggs, 2002). Drinking peers can provide access to alcohol, model drinking behavior, and reinforce substance use (Catalano et al., 1996). Studies have shown that affiliating with substance-using peers predicted subsequent drinking (Fergusson et al., 1994), and higher levels of peer involvement in drinking were associated with increased levels of alcohol intake by youth (Duncan et al., 1995). Thus, this study examined whether the link between very early onset of drinking and adult alcohol dependence can be explained by greater involvement with peers who engage in general antisocial behaviors as well as drinking behavior specifically, and who provide opportunities for continued antisocial behavior.

Finally, educational achievement constitutes one of the stepping stones for a well-adjusted, productive life and is one of the key tasks of adolescence. However, school achievement can be disrupted by early alcohol use (Crosnoe, 2006), and compromised educational development can also contribute to adult alcohol problems (Crum et al., 2006; Schulenberg et al., 1994). Thus, this study also examined school functioning as a potential mediator of the association between very early onset and adult alcohol dependence.

Increased substance use hypothesis

Perhaps the simplest explanation for the relationship between pre-adolescent onset of alcohol use and increased chronicity of alcohol dependence in adulthood may be that youths who begin using alcohol early continue or even escalate their drinking during adolescence, thus increasing the likelihood or chronicity of later alcohol use disorders. For example, studies indicate that youth with early onset of alcohol use are more likely to become heavy drinkers in adolescence and to be diagnosed with alcohol use disorders in adulthood (Ellickson et al., 2003; Hingson et al., 2006). Therefore, we include a measure of adolescent heavy episodic drinking as a possible mediator of the link between early alcohol initiation and the chronicity of adult alcohol dependence. We also examine the possible mediating effects of adolescent tobacco, marijuana, and other illicit drug use, which have been linked to both adolescent alcohol initiation and adult alcohol dependence (Merline et al., 2008).

Method

Sample

We used data from the Seattle Social Development Project, a longitudinal study that has followed 808 youth from elementary school to age 33. Participants were recruited in

the fall of 1985, when they were approximately 10 years of age, from 18 Seattle public elementary schools serving high-crime areas. Of all 1,053 fifth-grade students in these schools, 808 (77%) consented to participate and were assessed in the fall of 1985, spring of 1986, and then every year through 10th grade, again in 12th grade, and every 3 years thereafter through age 33. For an additional description of the sample, see Guttmannova et al. (2011), of which this is a follow-up.

This study focused on pre-adolescent onset versus adolescent alcohol initiation. Therefore, only individuals who initiated alcohol use before the legal age of 21 (87.4% of the Seattle Social Development Project sample) were included. Of the 706 participants retained in analyses, 367 (52%) were male, 351 (49.7%) were White, 184 (26.1%) were African American, 131 (18.6%) were Asian American, and 40 (5.7%) were Native American. More than half of the analysis sample (n = 359; 50.8%) came from low-income families (as indicated by their participation in the free school lunch program in Grades 5, 6, or 7).

A portion of the sample was exposed to a multicomponent preventive intervention in elementary grades, consisting of teacher training, parenting classes, and social competence training for children (see Hawkins et al., 1999, for a description and analysis of the intervention and effects). Although differences in prevalences and means have been observed between intervention and control groups, prior analyses have shown few differences in the covariance structures of the groups (Abbott et al., 1991; Catalano et al., 1996; Huang et al., 2001). To test possible differences in etiology between the groups, we examined a multiple-group covariance structure model constraining the covariance parameter estimates between all predictors and outcomes in the study to be equal across intervention groups. This constrained model fit the data well (e.g., root mean square error of approximation [RMSEA] = .02; comparative fit index [CFI] = .99), and the results suggested no substantial between-group differences in the relationships of interest in this report, supporting a single-group analysis involving participants from all intervention conditions.

Measures

Outcomes. The primary outcome in this study involved adult alcohol dependence problems. Participant-reported *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition* (DSM-IV; American Psychiatric Association, 1994), diagnostic criteria for alcohol dependence were assessed using the short form of the Diagnostic Interview Schedule (Robins et al., 1981). Consistent with the DSM-IV diagnostic criteria, alcohol dependence diagnosis was given when three or more of these criteria were met. Alcohol dependence was assessed at ages 21, 24, 27, 30, and 33 (1996 through 2008, respectively). Between the ages of 21 and 33, annual rates of alcohol dependence ranged from 7.7% to 13.5%, which is higher than those reported in national samples (Grant, 1994), but expected, given this higher risk, urban sample. Chronicity of alcohol dependence was assessed by modeling it as a latent variable with five categorical indicators indexing presence or absence of the diagnosis at each of the five adult time points. Thus, the variable indicates the shared variance in dependence diagnoses between the ages of 21 and 33.

Early alcohol use initiation. Alcohol use initiation was the primary predictor variable and its assessment was based on self-reports of having ever drunk alcohol (asked between 1985 and 1999). For the first 4 years, the respondents were asked if they ever drank alcohol. This question was modified in 1990 to include the qualifier "other than a sip or two." This wording change did not result in a significant change in the patterns of initiation (Kosterman et al., 2000). A prospective measure of age at alcohol initiation was calculated based on the respondents' exact age at each interview and defined as the earliest age at which respondents reported having drunk alcohol. Early alcohol use was represented by a single dummy variable coded as 1 for those who began using alcohol before age 11 (n = 76 or 10.8% of the analytic sample) and 0 for those who began drinking at a later point in adolescence (n = 630). Initiation before age 11 represents onset in childhood, before the transition to adolescence (Bronfenbrenner, 1979; Harter et al., 1992; Rudolph et al., 2001). This study is an extension of Guttmannova et al. (2011), who found that onset before age 11 was a more vulnerable period for alcohol initiation compared with onset during later periods in adolescence. A recent set of U.S. national surveys (e.g., Partnership Attitude Tracking Study) estimated the prevalence of alcohol use at 10% among children younger than age 11 (Donovan, 2007; Donovan et al., 2004). This rate is comparable to that reported in our study.

Measures examining the marker hypothesis

Family functioning. Youth reported on two indicators of family functioning at baseline. Family management (α = .67) was assessed using six questions that tapped into family practices, such as "The rules in my family are clear." Family bonding (α = .63) was assessed using five questions tapping into emotional relationship with family members, such as "Do you share your thoughts and feelings with your mother?" Higher scores on both measures indicate more positive family functioning.

Parental drinking. Parental drinking was assessed in a series of parent-reported questions about the frequency of their and their spouse's drinking when children were in 5th–10th grade. Responses were averaged over time. The average parental drinking across Grades 5–10 was included among the observed control variables (α = .89). A variable measuring parental drinking over time was preferred to a variable based on a single time point because it more likely captured

problem drinking. In sensitivity analyses including parental drinking in Grade 5 only, results were analogous to those presented here.

Confounds and background variables. Control variables used to test the marker hypothesis included self-report of gender (coded as male = 1, female = 0) and ethnicity (dummy variables for African American, Asian American, Native American, and "other" ethnicity, with European American as the reference group). We also controlled for childhood poverty as defined by participation in the National School Lunch/School Breakfast program collected from participants' school records (coded as 1 = eligible for free school lunch between Grades 5 and 7; 0 = not eligible).

Measures examining the compromised development hypothesis variables

An adolescent social functioning latent factor was created using three indicators measuring delinquency, externalizing behavior problems, and behavioral disinhibition. Delinquency was assessed at each wave between Grades 5 and 12 by five questions in which children were asked whether they have ever engaged in delinquent activities, such as taking something that did not belong to them or breaking into a building without permission. The responses were averaged within wave and then averaged over time (the mean of wave-specific reliability coefficients was $\alpha = .63$). Externalizing behavior problems were reported between fifth and eighth grade by teachers using the Teacher Report Form of the Child Behavior Checklist (Achenbach and Edelbrock, 1986). For each year, externalizing behavior problem scores were computed based on the scoring manual (see Achenbach and Rescorla, 2001). The composites were averaged over time (mean $\alpha = .96$). Behavioral disinhibition was reported by youth using a five-item scale between Grades 8 and 12 (see Hill et al., 2010). Questions assessed the number of times adolescent respondents had done things like "upset or annoyed adults just for the fun of it" or "done something dangerous because someone dared you to do it." The responses were averaged within waves and then averaged over time (mean $\alpha = .77$).

An adolescent antisocial peer latent factor was created using three indicators measuring antisocial behavior by respondents' peers, peer-related antisocial opportunities, and peer involvement in drinking alcohol. Peer antisocial behavior was reported by respondents using seven items from 5th to 12th grade. The questions assessed whether one's friends did things to get them into trouble with the teacher or the police (e.g., stealing, selling drugs, vandalism). Responses were averaged within wave and then averaged over time (mean $\alpha = .69$). Peer antisocial opportunities were reported by youth using six items assessed between Grades 7 and 12. The items included whether friends had asked or expected respondents to do things that could get them in trouble with their parents, the school, or the police. Mean peer-related antisocial opportunities composites were computed at each wave and then averaged over time (mean $\alpha = .61$). Peer drinking was reported by youth between 5th and 12th grade using four items asking about each of the youth's three best friends at the time. The questions assessed whether the best friends had ever tried alcohol, whether they had drunk alcohol in the past year, and how many times they had gotten drunk in the past month. Mean peer drinking composites were computed at each wave and then averaged over time (mean $\alpha = .75$).

An adolescent school functioning latent factor was created using indicators measuring students' grades and achievement scores (school records). Achievement scores were the average of the reading, language, and math subtests on the California Achievement Test, a frequently used standardized achievement battery with excellent internal consistency and reliability coefficients on verbal and quantitative subscales (Wardrop, 1989). California Achievement Test scores from Grades 5–8 were averaged over time ($\alpha = .95$). Grades were measured using six indicators. Two mean grade point average scores were computed based on all available school records for Grades 6–8 and Grades 9–10 ($\alpha = .94$ and $\alpha = .90$, respectively). Furthermore, for Grades 5–8, students were asked how their grades compared with those of their classmates, and the student-reported composite score was computed by averaging these responses over time ($\alpha = .71$). Similarly, for Grades 6–8 and 9–12, students were asked how they thought their grades were that year, and student-reported grade point averages for middle school and high school were computed ($\alpha = .76$ and $\alpha = .70$, respectively). Finally, for Grades 6–8, parents were asked what their children's grades were in the given school year, and these responses were averaged over time ($\alpha = .80$).

Measures examining the adolescent substance use hypothesis

An adolescent substance use latent factor was created using three indicators that measured adolescent marijuana and tobacco use and adolescent heavy episodic drinking. Adolescent substance use and heavy episodic drinking were reported by youth at each wave between 7th and 12th grade. Participants were asked about the number of times in the past month they had used marijuana and tobacco and their responses were averaged over time. In addition, a variable was created indicating chronicity of heavy episodic drinking (defined as having five or more drinks in a row) and was computed as a proportion of assessment years participants reported heavy episodic drinking out of assessment years available during adolescence.

Analysis

Adolescent socio-emotional and school functioning variables as well as their adolescent substance use and anti-

TABLE 1. Estimated effects of background variables and individual mediator factors on the chronicity of alcohol dependence

	Chronicity of alcohol dependence							
	Model 1		Model 2		Model 3		Model 4	
Variable	Est.	*p*	Est.	*p*	Est.	*p*	Est.	*p*
Early onset (<11 years)[a]	**.53****	.002	**.46***	.012	**.45***	.010	**.46***	.010
Gender								
Male	**.38****	.003	**.42****	.003	.17	.199	**.38****	.003
Ethnicity[b]								
African American	-.31	.056	-.25	.141	-.24	.139	-.10	.541
Native American	.38	.115	.41	.099	.41	.096	.29	.230
Asian American	-.12	.502	-.23	.214	.00	.983	-.12	.514
Poverty								
Free/reduced lunch	.10	.467	.07	.635	.03	.842	-.01	.933
Family								
Management	-.03	.672	-.06	.362	-.05	.442	-.04	.535
Bonding	-.04	.499	-.07	.293	-.03	.603	-.07	.299
Parental drinking	.05	.459	.08	.213	.06	.349	.04	.482
Model 1								
Peers	**.39*****	.000	–	–	–	–	–	–
Model 2								
School	–	–	**-.19****	.001	–	–	–	–
Model 3								
Problem behavior	–	–	–	–	**.38*****	.000	–	–
Model 4								
Substance use	–	–	–	–	–	–	**.41*****	.000

Notes: Estimates (est.) are standardized coefficients, continuous predictors are standardized on y and x, dichotomous predictors only on y. **Bold** indicates statistical significance. [a]Reference group is alcohol initiation later in adolescence; [b]reference group is European American.
*$p < .05$; **$p < .01$; ***$p < .001$.

social peer factors were related to the DSM-IV diagnoses of adult alcohol dependence using structural equation modeling. Specifically, we used a multiple causes and multiple indicators model to represent latent variables intervening between a set of observed background variables predicting a set of observed outcome variables. All analyses were conducted in Mplus 6 (Muthén and Muthén, 1998–2010). The robust means and variance adjusted weighted least squares (WLSMV; Muthén et al., 1997) estimator was used to compute parameter estimates to account for the categorical nature of several latent variable indicators (e.g., the indicators of the chronicity of the alcohol dependence diagnosis). To maximize the use of available data and minimize bias, the missing data option within the WLSMV estimator was used. The correlations among the exogenous variables, although not explicitly shown in the model figure, were accounted for in model estimation (Muthén and Muthén, 1998–2010). The model fit was evaluated using the chi-square statistic, as well as the CFI and the RMSEA, wherein CFI values close to or above .95 and RMSEA values below .08 represent reasonably good fit (Browne and Cudeck, 1993; Hu and Bentler, 1999).

Results

Preliminary analysis of variance and chi-square difference tests examined differences in the demographic and risk covariates by the early alcohol use onset variable in the analysis sample (not shown). Ethnicity and childhood poverty were significantly related to early alcohol use initiation. In general, a greater proportion of European Americans (14.2%) than African, Native, and Asian Americans (8.7%, 5.0%, and 6.1%, respectively) initiated the use of alcohol before age 11. Those who had experienced childhood poverty were less likely to initiate alcohol use before age 11. Those who initiated alcohol use before age 11 had higher levels of parental drinking than those who initiated later in adolescence. No other demographic and risk covariates differed by age at alcohol use initiation.

Marker hypothesis

To test the marker hypothesis, we examined the effect of onset of alcohol use before age 11 on the chronicity of alcohol dependence with and without the hypothesized observed predictors and background controls. On a bivariate level, pre-adolescent onset of alcohol use was positively related to the chronicity of alcohol dependence ($\beta = .44$, $p < .05$). This effect persisted after we included the background and sociodemographic controls ($\beta = .45$, $p < .05$) and when we added measures of family functioning and parental drinking ($\beta = .45$, $p < .05$). Thus, the marker hypothesis was not supported in our analyses. Model fit was acceptable, $\chi^2(41) = 65.83$, $p < .01$, CFI = .95, RMSEA = .03. All factor loadings of indi-

TABLE 2. Standardized factor loadings from Models 1–4

Variable	Factor loadings			
	Model 1	Model 2	Model 3	Model 4
Chronicity of alcohol dependence				
Alcohol dependence 1996	.65	.57	.60	.64
Alcohol dependence 1999	.80	.76	.78	.76
Alcohol dependence 2002	.86	.88	.88	.90
Alcohol dependence 2005	.89	.94	.90	.89
Alcohol dependence 2008	.88	.87	.89	.87
Model 1: Peers				
Peer drinking	.72	–	–	–
Peer antisocial behavior	.91	–	–	–
Peer antisocial opportunities	.80	–	–	–
Model 2: School				
CAT achievement	–	.51	–	–
GPA 5–8 comparative (SR)	–	.64	–	–
GPA 6–8 (SR)	–	.84	–	–
GPA 9–10 (SR)	–	.72	–	–
GPA 6–8 (PR)	–	.83	–	–
GPA 6–8 (SCR)	–	.92	–	–
GPA 9–10 (SCR)	–	.79	–	–
Model 3: Problem behavior				
Delinquency	–	–	.87	–
Behavioral disinhibition	–	–	.70	–
Externalizing behavior problems	–	–	.62	–
Model 4: Substance use				
Tobacco	–	–	–	.48
Marijuana	–	–	–	.56
Heavy episodic drinking	–	–	–	.74

Notes: All factor loadings are significant at the $p < .001$ level; CAT = California Achievement Test; GPA = grade point average; SR = student report; PR = parent report; SCR = school report.

cators of the alcohol dependence outcome were statistically significant, with standardized values ranging from .60 to .91 (not shown).

Competing explanations

Table 1 summarizes the structural regression estimates (which can be interpreted as partial regression coefficients in regular multiple regression) for the chronicity of alcohol dependence by the four explanatory factors denoting compromised adolescent functioning (Models 1–3) and increased substance use (Model 4) hypotheses. Table 2 summarizes the standardized factor loadings for the indicators of the outcome and the individual latent variable mediators. Table 3 displays associations between the observed background and risk covariates and the four latent factors representing

TABLE 3. Estimated effects of background variables on hypothesized mediators

Variable	Peers Model 1		School functioning Model 2		Problem behavior Model 3		Substance use Model 4	
	Est.	*p*	Est.	*p*	Est.	*p*	Est.	*p*
Early onset (<11 years)[a]	-.22	.109	.00	.986	-.01	.930	-.04	.802
Gender								
Male	**.33*****	.000	**-.43*****	.000	**.86*****	.000	**.31****	.004
Ethnicity[b]								
African American	**.41*****	.000	**-.54*****	.000	**.25****	.007	-.13	.326
Native American	.31	.069	**-.49****	.002	.25	.160	**.51****	.005
Asian American	**-.57*****	.000	**.59*****	.000	**-.88*****	.000	**-.55****	.003
Poverty								
Free/reduced lunch	.13	.158	**-.45*****	.000	**.31*****	.000	**.38****	.001
Family								
Management	**-.15****	.001	**.11****	.002	**-.09***	.030	**-.11***	.029
Bonding	-.05	.276	-.03	.516	-.08	.062	.01	.852
Parental drinking	.09	.037	.01	.832	.06	.190	.10	.087

Notes: Estimates (est.) are standardized coefficients, continuous predictors are standardized on y and x, dichotomous predictors only on y. **Bold** indicates statistical significance. [a]Reference group is alcohol initiation later in adolescence; [b]reference group is European American.
*$p < .05$; **$p < .01$; ***$p < .001$.

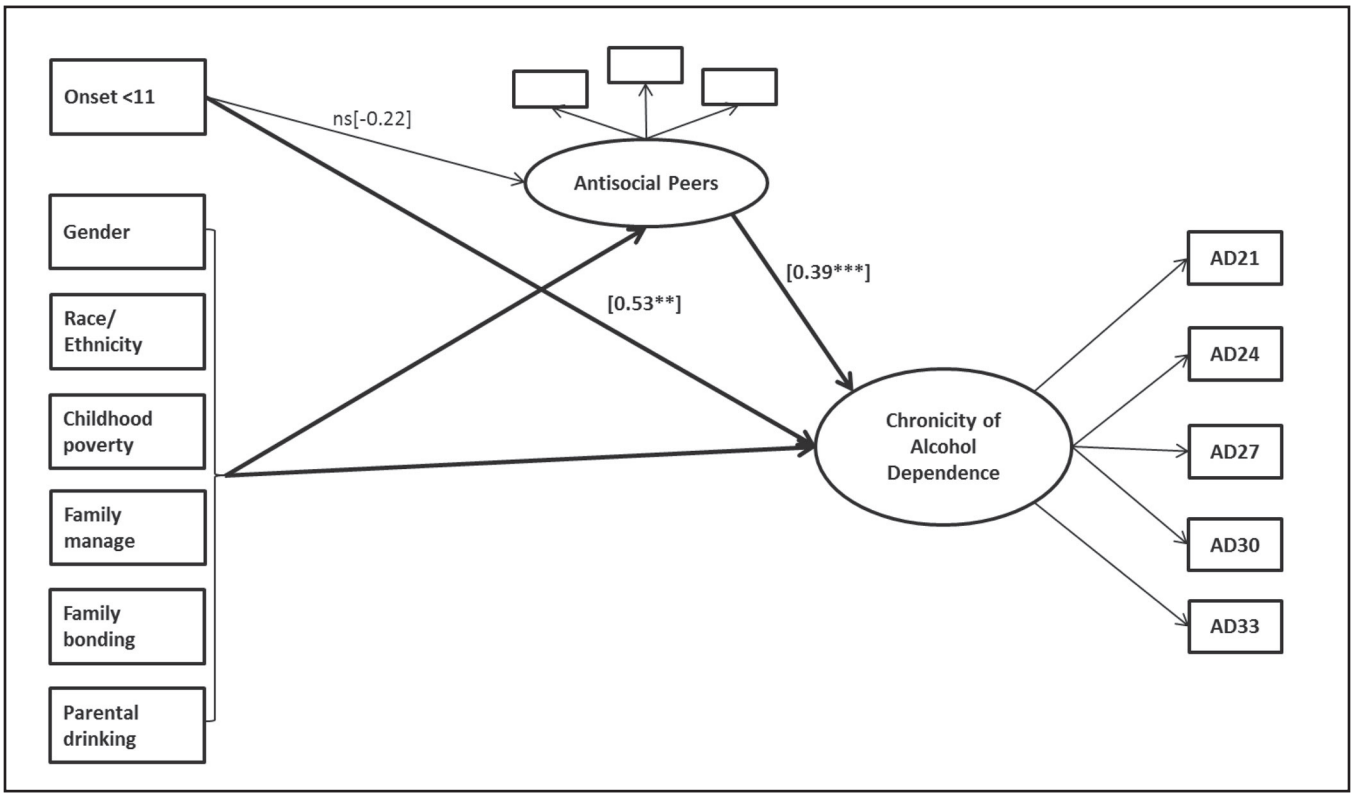

FIGURE 1. Structural equation model for Model 1—adolescent antisocial peers. Family manage = family management; AD = alcohol dependence; ns = not significant.

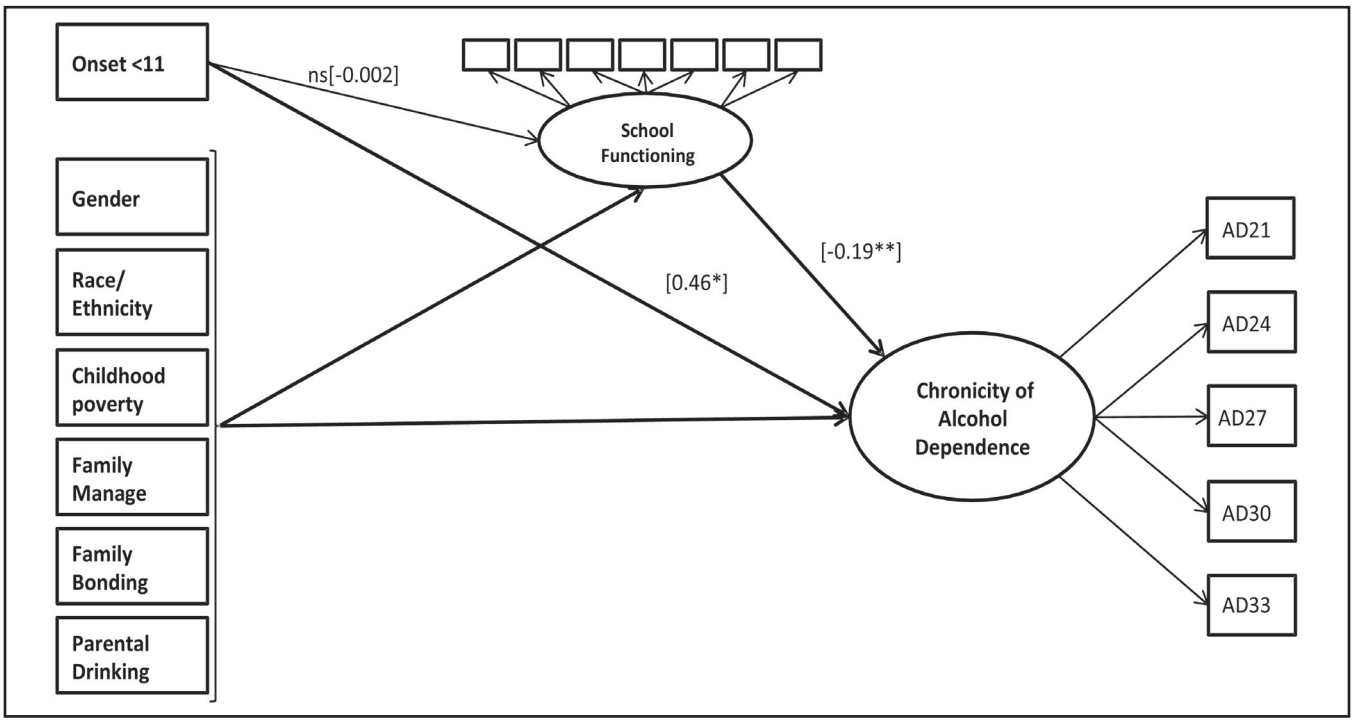

FIGURE 2. Structural equation model for Model 2—adolescent school functioning. Family manage = family management; AD = alcohol dependence; ns = not significant.

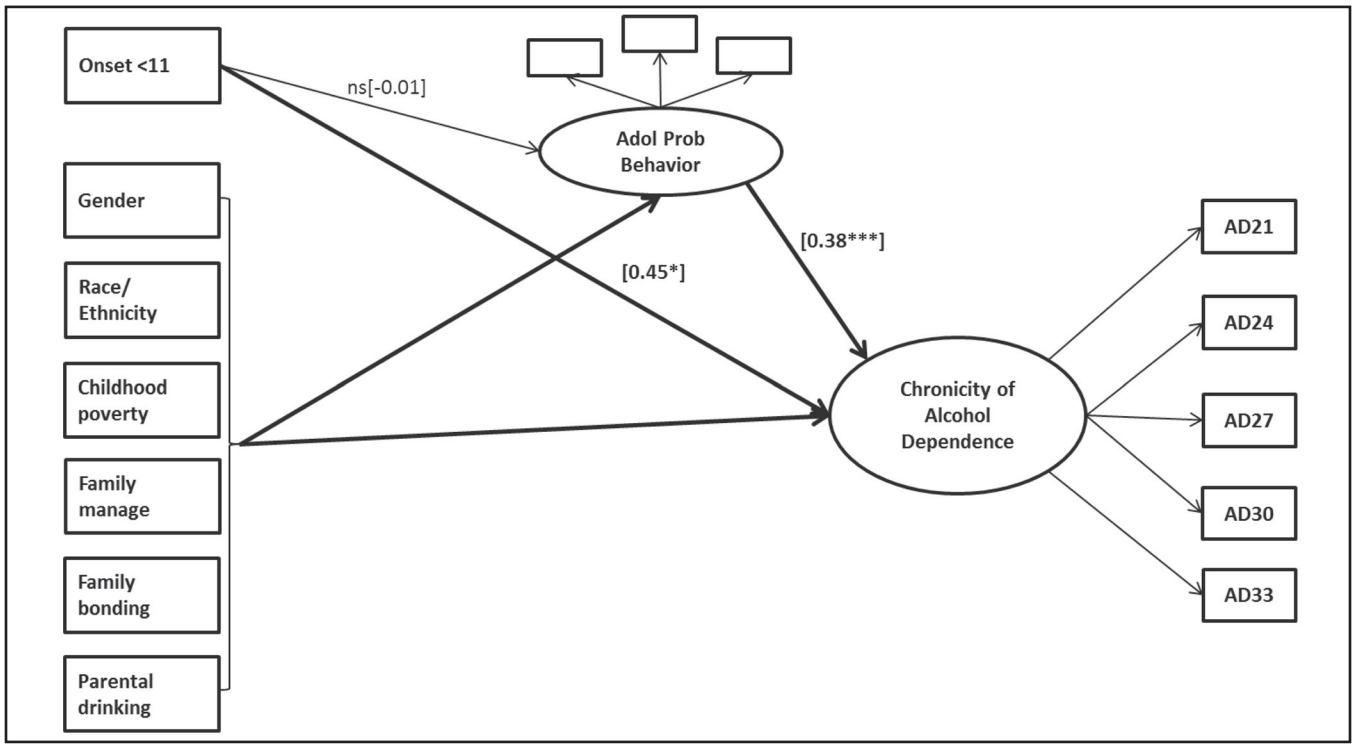

FIGURE 3. Structural equation model for Model 3—adolescent problem behavior. Family manage = family management; adol prob behavior = adolescent problem behavior; AD = alcohol dependence; ns = not significant.

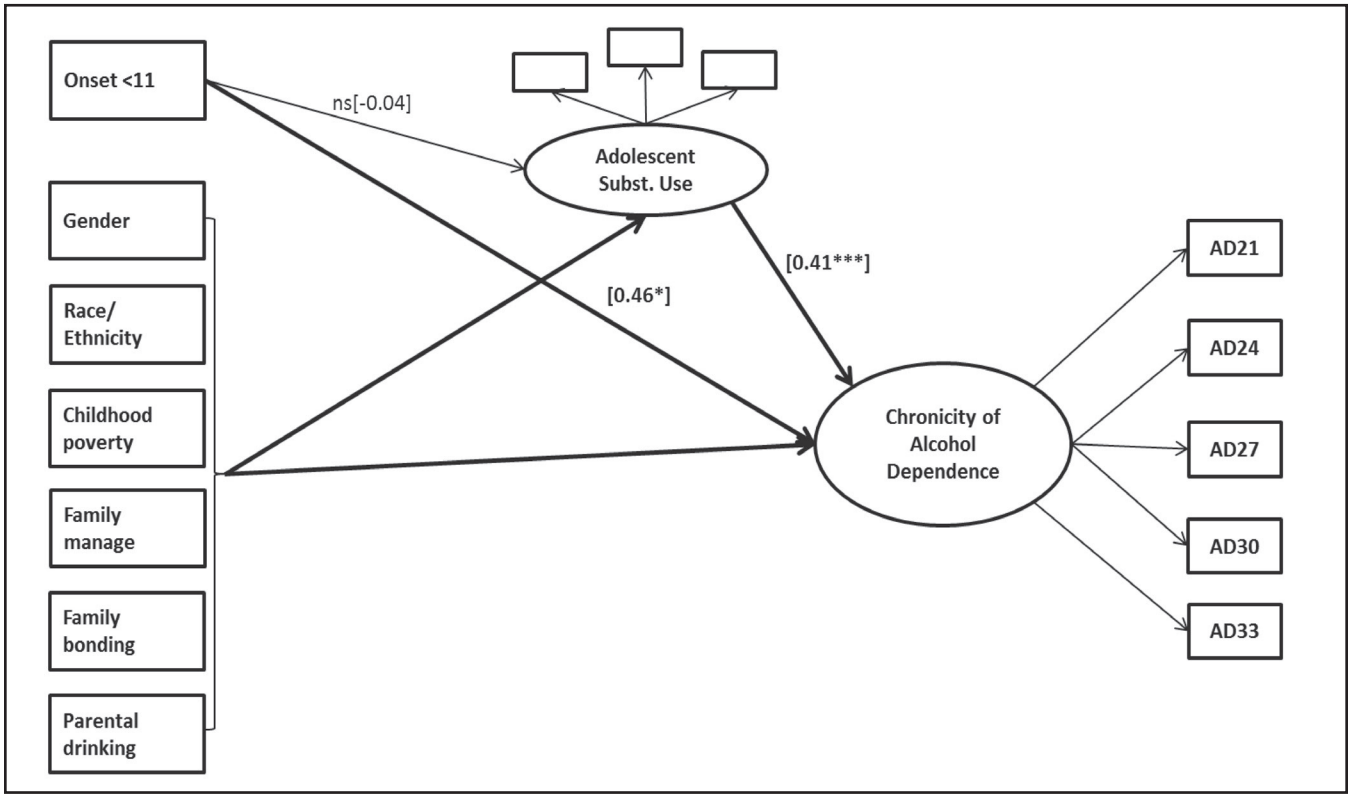

FIGURE 4. Structural equation model for Model 4—adolescent substance use. Family manage = family management; adolescent subst. use = adolescent substance use; AD = alcohol dependence; ns = not significant.

adolescent functioning and substance use. Figures 1–4 correspond to Models 1–4, respectively, and highlight the most salient findings.

The compromised adolescent functioning hypothesis was tested using the antisocial peers factor (Model 1), $\chi^2(73) = 177.62$, $p < .01$, CFI = .91, RMSEA = .05; the school functioning factor (Model 2), $\chi^2(143) = 261.30$, $p < .01$, CFI = .95, RMSEA = .04; and the problem behavior factor (Model 3), $\chi^2(73) = 152.629$, $p < .01$, CFI = .93, RMSEA = .04. Controlling for other variables in the model, each of the three factors significantly predicted the chronicity of adult alcohol dependence diagnosis ($\beta = .39$, $p < .001$; $\beta = -.19$, $p < .01$; $\beta = .38$, $p < .001$, respectively). However, none of the factors mediated the effect of early alcohol use initiation on the chronicity of alcohol dependence. About 27.3% of the variance in the chronicity of alcohol dependence was explained by the peer model, 17.1% by the school functioning model, and 24% by the problem behavior model.

The increased substance use hypothesis was tested using the adolescent substance use factor (Table 1, Model 4), $\chi^2(73) = 130.144$, $p < .01$, CFI = .95, RMSEA = .03. Again, although adolescent substance use was significantly and positively related to the chronicity of alcohol dependence in adulthood ($\beta = .41$, $p < .001$), it did not mediate the positive relationship between late childhood initiation of alcohol use and the chronicity of alcohol dependence. In a series of robustness checks, we also operationalized substance use as an alcohol-specific factor representing alcohol misuse at age 18. Again, we did not find evidence of mediation for this adolescent functioning factor. This model explained 24.7% of the variance in alcohol dependence chronicity.

Discussion

This study examined potential explanatory mechanisms of the association between very early age at alcohol use onset and chronicity of adult alcohol dependence by testing three competing hypotheses: (1) a marker hypothesis, which states that the link may be an artifact of other risk factors; (2) a compromised functioning hypothesis, which states that very early alcohol initiation may interfere with adolescent development, which could lead to later alcohol problems through a greater exposure to risky environments, involvement in risky behaviors, or compromised school functioning; and (3) an increased adolescent substance use hypothesis, which posits that preadolescent onset of alcohol use may lead to increased substance use in adolescence, which may in turn lead to adult alcohol dependence. None of these three hypotheses was supported: in each model the association between very early alcohol use and the chronicity of alcohol dependence in adulthood remained unchanged with the inclusion of control variables and potential mediators.

These three hypotheses (marker, compromised functioning, increased substance use) were all well operationalized, yet none accounted for the relationship between early drinking and adult dependence. The lack of mediation observed here is consistent with results from Mason et al. (2011), who examined the potential mechanisms of the link between the early onset of alcohol use (defined as drinking at or before age 13) and adolescent alcohol problems at age 15. In that study, the authors found that low self-regulation, peer deviance, and continued alcohol use did not mediate the small but significant positive link between early onset of alcohol use and alcohol problems in mid-adolescence. If not these mechanisms, then what could explain the association between very early alcohol initiation and the chronicity of adult alcohol dependence?

One possibility is that the association is accounted for by a common biological or genetic vulnerability. There is evidence from behavior and molecular genetic studies of a genetic influence on early alcohol use onset and also on adult alcohol dependence (Hopfer et al., 2005; Kaufman et al., 2007; Zucker, 2006). For example, individual variations in the dopaminergic, serotonergic, or GABAergic systems might give rise to both early onset of alcohol use and alcohol problems in adulthood (van der Zwaluw and Engels, 2009). The link between early alcohol use and later alcohol problems could also arise from a common genetic liability to disinhibitory psychopathology (McGue and Iacono, 2008; Young et al., 2000). Furthermore, in the Mason et al. (2011) study described above, alcohol use at age 13, although unrelated to the change in self-regulation later in adolescence, was related to low self-regulation at baseline, suggesting the possibility of a common neurobehavioral disinhibitory antecedent. Future studies should test this common biological or genetic vulnerability hypothesis.

It is also possible that early passive exposure to alcohol explains both preadolescent onset drinking and adult alcohol dependence. Prenatal exposure to alcohol has been linked to both early adolescent alcohol problems (Baer et al., 1998) and young adulthood alcohol use disorders (Alati et al., 2005; Baer et al., 2003). Interestingly, these effects have been seen to persist even after controlling for familial history of alcohol problems (Baer et al., 1998, 2003). In their review of the literature, Spear and Molina (2005) suggested that preadolescent alcohol use initiation may be a biological consequence of fetal, infantile, or even early childhood exposure to ethanol's chemosensory and pharmacological influences, which can change an individual's responsiveness to alcohol later in development. Thus, the link between early initiation of alcohol use and the chronicity of adult alcohol dependence could simply be attributable to even earlier, albeit passive, exposure to alcohol. The present study does not contain information about prenatal or early childhood exposure to alcohol or other substances. Future studies using different data sets should examine this early exposure hypothesis.

Furthermore, although most explanatory frameworks of the link between early alcohol use and later alcohol use

disorders involve mechanisms originating in late childhood or early adolescence (Donovan et al., 2004), a few relatively recent studies point to the importance of earlier developmental pathways (i.e., in early and middle childhood) in the link between early onset of alcohol use and later alcohol use problems. Prior studies suggest that early childhood alcohol schemas (Zucker et al., 1995) and early and middle childhood internalizing (Hussong et al., 2011; Zucker, 2008) as well as disinhibited or externalizing (for a review, see Zucker, 2008) symptomatology may be important. Again, our study does not include measures from the early and middle childhood periods; however, these links should be examined in future studies using samples that include both information on alcohol use in childhood and its potential precursors from earlier developmental periods.

As in the earlier study by Guttmannova et al. (2011), the results of this study should be interpreted with the following additional limitations in mind. The annual adult alcohol dependence problems were assessed every 3 years between ages 21 and 33 and therefore represent repeated snapshots of alcohol dependence, which might underestimate actual prevalence. Furthermore, alcohol use in adolescence was illegal at the time of assessment and thus could be subject to underreporting. However, response bias in substance use reporting can be minimized in the context of longitudinal studies where trust and rapport are developed over years as in this study (Del Boca and Darkes, 2003; Langenbucher and Merrill, 2001).

Although specific factors accounting for the relationship between early alcohol use and adult dependence were not identified in the present study, current analyses did identify important predictors of the chronicity of alcohol dependence. The finding that each of the four adolescent functioning domains predicted the chronicity of alcohol dependence in young adulthood is important and consistent with existing literature. For example, we found that adolescent problem behaviors—defined by delinquency, behavioral disinhibition, and externalizing behavior problems—predicted greater chronicity of alcohol dependence in young adulthood (Alati et al., 2006; Clapper et al., 1995; D'Amico et al., 2005; Kuperman et al., 2001). Similarly, we found that greater substance use—including alcohol, marijuana, and tobacco—in adolescence and greater association with antisocial peers were also predictive of increased chronicity of adult alcohol dependence (Bonomo et al., 2004).

Although these hypothesized explanatory mechanisms did not mediate the effects of pre-adolescent onset of alcohol use on the chronicity of alcohol dependence in young adulthood, they joined with the very early onset of alcohol use in making individuals more vulnerable to alcohol dependence, ultimately accounting for a substantial proportion of variance in the chronicity of alcohol dependence in young adulthood. Thus, prevention efforts should focus on delaying the onset of alcohol use, reducing substance use in adolescence, as well as improving school functioning, reducing adolescent problem behaviors, and targeting adolescent peer networks.

Acknowledgments

The authors gratefully acknowledge Seattle Social Development Project panel participants for their continued contribution to the longitudinal study. We also acknowledge the Social Development Research Group Survey Research Division for their hard work maintaining high panel retention and the Social Development Research Group editorial and administrative staff for their editorial and project support.

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