

Developmental Psychology

Academic Achievement as a Moderator of Genetic Influences on Alcohol Use in Adolescence

Aprile D. Benner, Natalie Kretsch, K. Paige Harden, and Robert Crosnoe

Online First Publication, December 2, 2013. doi: 10.1037/a0035227

CITATION

Benner, A. D., Kretsch, N., Harden, K. P., & Crosnoe, R. (2013, December 2). Academic Achievement as a Moderator of Genetic Influences on Alcohol Use in Adolescence. *Developmental Psychology*. Advance online publication. doi: 10.1037/a0035227

BRIEF REPORT

Academic Achievement as a Moderator of Genetic Influences on Alcohol Use in Adolescence

Aprile D. Benner, Natalie Kretsch, K. Paige Harden, and Robert Crosnoe
University of Texas at Austin

Prior research suggests a link between academic performance and alcohol use during adolescence, but the degree to which this association reflects actual protective effects continues to be debated. We investigated the role of genetic factors in the association between academic achievement and adolescent alcohol use and whether achievement might constrain the translation of genetic influences on drinking into actual behavior (a Gene \times Environment interaction). Analysis of twin data from Add Health ($n = 399$ monozygotic and dizygotic twin pairs) revealed not only common genetic factors underlying the association between achievement and alcohol consumption but also evidence for a gene–environment interaction. Specifically, the protective effect of achievement operated by moderating heritability of alcohol use, which was particularly salient for adolescents at high genetic risk for alcohol use.

Keywords: alcohol use, academic achievement, genetics, adolescent development

By the end of high school, 70% of U.S. adolescents have consumed alcohol, and almost one quarter have engaged in binge drinking (Johnston, O'Malley, Bachman, & Schulenberg, 2012). Historic trends in this risky behavior have spurred a great deal of research that, in addition to documenting its many negative short- and long-term repercussions, has elucidated its etiology (Brown et al., 2008; Hawkins, Catalano, & Miller, 1992). In particular, the role of high school academic performance in drinking—academic struggle as a risk factor, academic success as a protective factor—has generated much attention (Crosnoe, 2006), revealing interesting developmental insights that need to be better understood.

In general, the evidence suggests that academic performance, primarily higher grades but also advanced coursework, school engagement, and educational expectations, is associated with lower levels of alcohol use (Bryant, Schulenberg, O'Malley, Bachman, & Johnston, 2003; Bryant & Zimmerman, 2002; Maggs,

Patrick, & Feinstein, 2008; Schulenberg, Bachman, O'Malley, & Johnston, 1994). Yet, the degree to which this association reflects actual protective effects continues to be debated. One issue is the potential for bidirectionality in this association, with drinking behavior affecting academic achievement (i.e., abstaining promoting academic progress over time; Crosnoe, Benner, & Schneider, 2012; Jeynes, 2002; King, Meehan, Trim, & Chassin, 2006; Staff, Patrick, Loken, & Maggs, 2008). Although this reverse pattern does happen, studies attempting to adjudicate between the two directions have clearly illustrated that the path from academic performance to substance use is stronger than the reverse (Bryant, Schulenberg, Bachman, O'Malley, & Johnston, 2000; Crosnoe, 2006). Another issue is the potential for unmeasured confounds to create the appearance of a link between academics and drinking even if the former does not influence the latter. Of particular concern are confounds related to heritable traits within families, given that both academic behavior and alcohol use have strong genetic components (Cleveland & Wiebe, 2003; Petrill & Wilkerson, 2000).

The purpose of this research brief was to provide a preliminary exploration of the role that heritable traits play in the association between academic performance and adolescent drinking. Is this association free of genetic influences, driven by shared genetic influences, or something more complex and interactive? Drawing on the twin sample in the National Longitudinal Study of Adolescent Health (Add Health), this exploration is intended to provide some context for interpreting the extant literature while also encouraging new work in an area that is both theoretically grounded and relevant to public health intervention.

Linking Academic Achievement to Alcohol Use

Two classic theories of deviant behavior are often used to explain why adolescents' academic experiences might be related to

Aprile D. Benner, Department of Human Development and Family Sciences, University of Texas at Austin; Natalie Kretsch and K. Paige Harden, Department of Psychology, University of Texas at Austin; Robert Crosnoe, Department of Sociology, University of Texas at Austin.

The authors acknowledge the support of National Institute on Drug Abuse Grant R03DA032018, awarded to Aprile D. Benner; National Institute on Alcohol Abuse and Alcoholism Grant R21AA020045-01, awarded to Robert Crosnoe; and National Institute of Child Health and Human Development Grant R24 HD42849, awarded to the Population Research Center, University of Texas at Austin. Opinions reflect those of the authors and not necessarily those of the granting agencies.

Correspondence concerning this article should be addressed to Aprile D. Benner, Department of Human Development and Family Sciences, University of Texas at Austin, 1 University Station Stop A2702, Austin, TX 78712. E-mail: abenner@prc.utexas.edu

their alcohol use. The first, social control, asserts that ties to conventional institutions reduce the propensity to engage in antisocial behavior (Hirschi, 1969). According to this theory, adolescents who do well academically develop a greater stake in school as a social institution and consequently have more to lose by engaging in behaviors, like drinking, that could jeopardize their standing in school. The second theory, differential association (Sutherland, 1947), views antisocial behavior as a product of social learning, where the ecological organization of adolescents' daily lives (e.g., neighborhoods, families, schools, peer groups) exposes them to different kinds of messages that support or discourage antisocial activities (Catalano, Oesterle, Fleming, & Hawkins, 2004). Given the curricular and organizational segregation of students in schools by academic performance as well as the strong academic homophily (i.e., attraction to similar others) among adolescents, academic achievement tends to select young people into peer settings with fewer positive messages about drinking and fewer opportunities to drink (Cleveland & Wiebe, 2003; Crosnoe & Riegle-Crumb, 2007).

Taken together, these theories (along with more psychological perspectives such as social learning theory; Bandura, 1977) suggest that doing well in school raises the stakes for drinking and limits opportunities to engage in this risky behavior. Yet, this interpretation only holds to the extent that the association between academic performance and adolescent drinking actually captures a protective effect. Over the years, research has lent more support to this protective effect by teasing out the bidirectionality in this association and by identifying and controlling for any number of confounds (Bryant et al., 2000; Crosnoe, 2006). We argue that exploring the genetic influences on both academic achievement and adolescent alcohol use and the possibility that these influences may be related to each other further advances the field.

Focusing on Genetic Influences

One possible role of heritable traits in the association between academic achievement and adolescent drinking is that shared genetic influences completely account for any observed link between the two and, therefore, that no protective effect actually exists. Previous behavioral genetic research has revealed overlapping genetic influences between phenotypes related to the ones we examine here, including nicotine use and educational attainment (McCaffery et al., 2008), academic mastery and alcohol dependence (Kiecolt, Aggen, & Kendler, 2013), and verbal ability and alcohol dependence (Latvala et al., 2009).

Another possibility—one more in line with developmental theory (e.g., systems perspectives; Lerner, 2006) and the theoretical underpinnings of this research—is that the potential for academic success to raise the stakes for drinking and decrease opportunities to drink might moderate adolescents' genetic propensities to drink. If so, then academic achievement would have the biggest protective effect on adolescents at greatest genetic risk for alcohol use. Without the constraints on behavior that academic success creates, adolescents genetically predisposed to alcohol use would follow through on this predisposition. In contrast, the academically related stakes and opportunities of drinking would make less difference to adolescents lacking strong genetic proclivities to drink, as they would be less likely to drink regardless of the potential ramifications of drinking or opportunities to drink.

In this study, therefore, we used twin data to disentangle genetic and environmental underpinnings of academic achievement, adolescent drinking, and the connection between the two, both reflecting and extending theoretical insights about social control and opportunity. After assessing the extent to which the association between academic achievement and drinking was accounted for by shared genetic influences, we explored whether academic achievement might constrain the translation of genetic influences on drinking into actual behavior, that is, a Gene \times Environment interaction in which heritable variation in drinking is reduced among high-achieving adolescents.

Method

Data

Add Health is a nationally representative longitudinal study of adolescents in the United States, with data collected across four waves between 1994 and 2008. It used a school-based sampling procedure, randomly selecting schools across strata defined by region, type, size, and racial composition. The In-School Survey was administered to all present students in the schools to create a sampling frame for the Wave I In-Home Interview ($N = 20,744$). Three follow-up In-Home Interviews were completed. The In-Home Interview sampling deliberately targeted sibling pairs to allow for genetically informed analyses of adolescent behavior. Siblings of different genetic relatedness were identified in the initial In-School Survey and then recruited for the Wave I sample. The demographic composition of the Add Health sibling sample is comparable to the full sample (Jacobson & Rowe, 1999).

Given our interest in Gene \times Environment interactions, we used the Wave I sibling sample. First, we selected twins only to control for age differences between different kinds of sibling pairs in the sample. Twins were classified as monozygotic (MZ) or dizygotic (DZ) based on self-reported zygosity as well as responses to four questions assessing similarity of appearance and frequency of being mistaken for one's twin. These items have been validated with analysis of genetic markers (Harris, Halpern, Smolen, & Haberstick, 2006). Second, only high school students were selected, as academic curricula are more stratified and cumulative in high school (vs. middle school), and norms and opportunity structures are more supportive of alcohol use later in adolescence (Schulenberg & Maggs, 2002). These filters resulted in a sample of 399 twin pairs (in Grades 9–11 in Wave I), with 798 adolescents total (M age = 16.45, $SD = .93$). Our final sample included 163 MZ pairs (85 male, 78 female), 135 same-sex DZ pairs (71 male, 64 female), and 101 opposite-sex DZ pairs. The racial/ethnic composition was 54% White, 24% African American, 15% Latino, and 7% other races/ethnicities.

Measures

For the independent variable, academic achievement, adolescents reported their most recent grades in English, math, history, and science on a 1 (*A*) to 4 (*D or lower*) scale. Grades were reverse coded (1 = *D or lower*, 4 = *A*) and averaged to create an overall grade point average (GPA; $M = 2.78$, $SD = 0.77$). Transcript data were collected in Wave III, but, due to attrition between Waves I

and III, we did not use these data to measure achievement. Prior work with the full Add Health sample has revealed that adolescents' self-reported grades are highly correlated with grades from official school transcripts ($r = .88$; see Langenkamp, 2009). For the dependent variable, alcohol use, we followed Add Health conventions (Johnson, 2004; Resnick et al., 1997) by using adolescents' self-report data on whether they had ever drunk alcohol and, if so, how often in the past month. Responses ranged from 0 (*none*; included abstainers) to 6 (*nearly everyday*).

Analysis Plan

To begin, we examined phenotypic associations between alcohol use and academic achievement using group comparisons. Mixed-effects models accounted for twins clustered in pairs. Individuals with GPAs of 3.0 or higher were classified as high achievers. We compared alcohol use among high versus lower achievers and the GPAs of abstainers (those who reported no alcohol use) with nonabstainers. In addition, we examined GPA and alcohol use stratified by gender, race/ethnicity, and grade level.

Next, we conducted a series of structural equation models (SEMs) using twin data on GPA and alcohol use in Mplus (Muthén & Muthén, 1998–2012). Full information maximum likelihood accounted for all missing item-level data. Model fit was assessed through root-mean-square error of approximation (RMSEA) and chi-square values (Steiger, 1990), and nested models were compared by differences in chi-square and log-likelihood values. Three sets of analyses were performed: (a) a bivariate twin model to estimate genetic and environmental associations between academic achievement and alcohol use, (b) a Gene \times Environment interaction model that tested whether academic achievement conditioned genetic influences on alcohol use, and (c) an alternative Gene \times Environment interaction model that tested whether alcohol use conditioned genetic influences on academic achievement.

The twin design (see Neale & Cardon, 1992) used in this study focused on sources of variation in a given phenotype. This model partitioned variance in each phenotype into three components: additive genetic effects (A), shared environmental effects (C; family-level environmental influences that make siblings more

similar than unrelated individuals), and nonshared environmental effects (E; environmental influences that make siblings different from each other, plus measurement error). Collectively, these components are often referred to as *ACE components*. On the basis of genetic theory, correlations between additive genetic effects were set to 1.0 for MZ twins and 0.5 for DZ twins. By definition, correlations between shared environmental effects were set to 1.0, and correlations between nonshared environmental effects were set to 0 for all twin pairs. Paths from the ACE components to the observed variables were estimated, and the ACE components were fixed to have a mean of 0 and a standard deviation of 1.

For the bivariate twin model, the regression path labeled bA in Figure 1 tests whether genes related to academic achievement also influenced alcohol use; that is, to what extent did the association between achievement and alcohol use reflect common genetic effects? The regression path bC tested whether family-level environmental influences on achievement also influenced alcohol use. Together, the bA and bC paths can be interpreted as a “between-twin pair” effect of academic achievement; that is, did twin pairs who differed from other twin pairs in average achievement also differ in average alcohol use? The regression path labeled bE tested whether nonshared environmental influences on achievement also influenced alcohol use. This path can be interpreted as the “within-twin pair” effect of achievement on alcohol use; that is, did twins who differed in academic achievement also differ in their alcohol use? Because opposite-sex twins were included in the sample, all models regressed both phenotypes (achievement and alcohol use) on gender (1 = *female*, 0 = *male*); our selection of same-age, same-race twins effectively controlled for age and race.

Next, a Gene \times Environment interaction model tested whether academic achievement conditioned genetic influences on alcohol use (see Figure 2). That is, does achievement constrain youth from acting on genetic proclivities to drink? Here, we tested for moderation of genetic influences on alcohol use by achievement, net of genetic and environmental associations between achievement and alcohol use. We allowed all sources of variance in alcohol use to be moderated by achievement. This full moderation model, for one twin only, is shown in Figure 2.

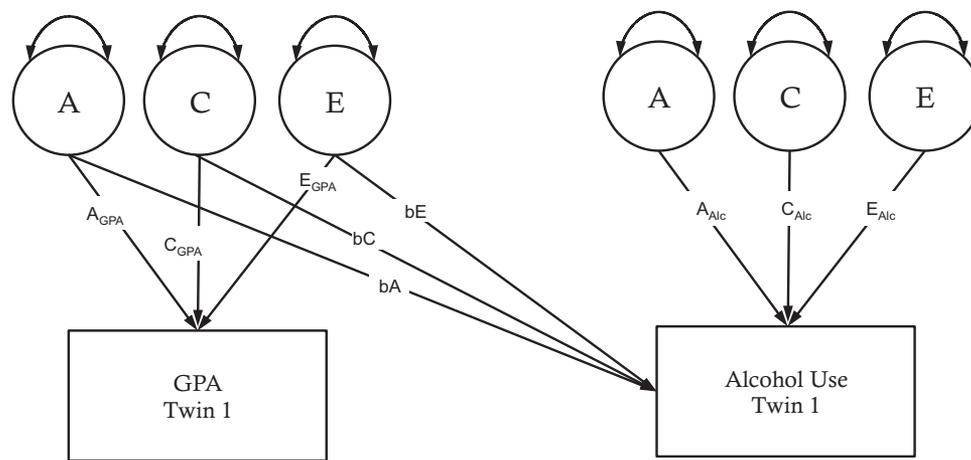


Figure 1. Basic ACE model of academic achievement and alcohol use. A = additive genetic effects; C = shared environmental effects; E = nonshared environmental effects; GPA = grade point average.

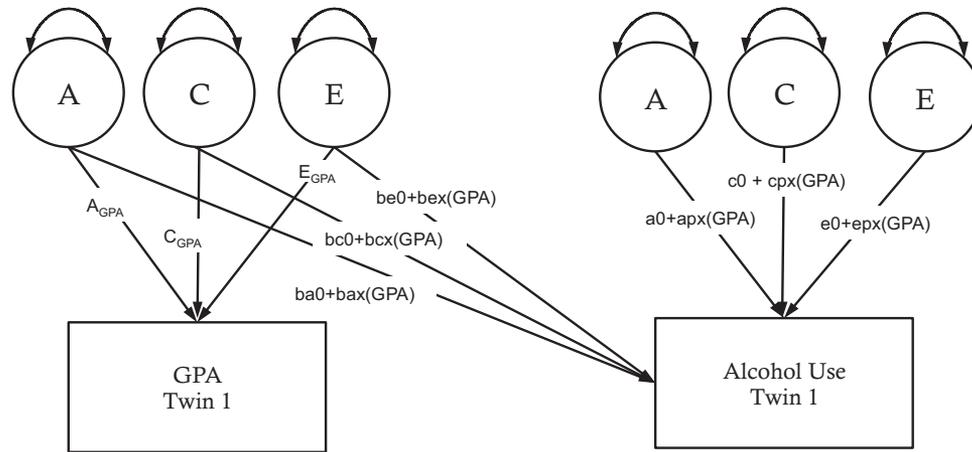


Figure 2. Interaction between academic achievement and genetic influences on alcohol use. A = additive genetic effects; C = shared environmental effects; E = nonshared environmental effects; GPA = grade point average.

A significant interaction term on the genetic variance in alcohol use, labeled apx , would indicate a Gene \times Environment ($G \times E$) interaction. Our hypothesis that genetic influences on alcohol use would decrease as academic achievement increased would be supported by a significant negative interaction. A significant positive interaction would indicate the opposite, that genetic influences on alcohol use were greater among high-achieving youth. Likewise, significant interaction terms on the C and E paths would suggest that achievement moderated shared and/or nonshared environmental influences on alcohol use. We also allowed the regression paths b_A , b_C , and b_E to be moderated by alcohol use, as van der Sluis, Posthuma, and Dolan (2012) have shown that estimating interactions on the cross-paths between moderator and phenotype is necessary to avoid false-positive estimates of $G \times E$. Finally, as a means of exploring bidirectional associations between academic achievement and alcohol use, we estimated a second interaction model in which the ordering of our two variables was switched, with alcohol use modeled as the predictor and moderator and academic achievement modeled as the outcome.

The strengths and limitations of the twin method have been exhaustively described elsewhere, but we note a few assumptions here. The equal environmental assumption presumes that MZ twins are treated no more similarly than DZ twins. Several empirical studies have directly tested this assumption and found support for its validity (e.g., Kendler, Neale, Kessler, Heath, & Eaves, 1993) and have questioned its impact on heritability estimates (Conley & Rauscher, 2011). The twin model also assumes that there is random mating in the parent generation and that MZ twins share 100% of their inherited segregating genes. MZ twin differences in gene expression or acquired variation in DNA sequence (e.g., mutation, retrotransposition events), therefore, are not captured in the "A" factor of a twin model. It should be noted that alternative approaches, such as adoption studies (Bouchard & McGue, 1981) and genome-wide complex trait analysis (Trzaskowski, Yang, Visscher, & Plomin, 2013), have revealed estimates of heritability that are consistent with twin studies. Furthermore, violations of assumptions may influence estimates of main genetic effects more than interaction effects (e.g., Loehlin, Harden, & Turkheimer, 2009).

Results

Table 1 presents mean levels of alcohol use and GPA in the full sample and stratified by gender, race, grade level, level of achievement, and alcohol abstainer status. Results from a mixed-effects model accounting for twins clustered in pairs show that high achievers reported significantly less alcohol use than lower achievers. Likewise, abstainers reported significantly higher GPAs than nonabstainers. More frequent alcohol use was associated with being White and being older (11th vs. 9th or 10th grade). African American and Latino/a students and boys had lower GPAs than their White and female peers.

We then examined twin pair correlations for academic achievement and alcohol use (see Table 2). We simply describe the pattern of correlations here, as the role of sampling error (including

Table 1
Descriptive Statistics for Key Study Variables

Variable	n (%)	Alcohol	GPA
		M (SD)	M (SD)
Full sample	798	1.08 (1.45)	2.78 (.77)
Females	385 (48.2)	1.03 (1.41)	2.91 (.79) _a
Males	413 (51.8)	1.14 (1.50)	2.67 (.73) _b
White	434 (54.4)	1.21 (1.48) _a	2.85 (.78) _a
Black	190 (23.8)	0.76 (1.39) _b	2.55 (.70) _b
Hispanic	119 (14.9)	1.20 (1.52)	2.43 (.77) _b
Other	55 (6.9)	0.50 (0.89)	2.90 (.75) _a
Higher achievers	390 (49.7)	0.90 (1.33) _a	3.43 (.36) _a
Lower achievers	395 (50.3)	1.23 (1.53) _b	2.15 (.49) _b
Grade 9	298 (36.1)	0.98 (1.44) _a	2.67 (.80) _a
Grade 10	258 (32.6)	0.95 (1.44) _a	2.87 (.73) _b
Grade 11	242 (31.3)	1.35 (1.48) _b	2.82 (.76)
Abstainers	412 (52.2)	0.00 (0.00) _a	2.89 (.76) _a
Nonabstainers	377 (47.8)	2.24 (1.32) _b	2.67 (.77) _b

Note. High achievement is defined as a GPA ≥ 3.0 . Subscripts denote significant differences between groups at $p < .05$. Groups with the same subscript or with no subscript are not significantly different. GPA = grade point average.

relevant p values) is assessed in our subsequent, more rigorous SEMs. For alcohol use, the correlation for MZ twins ($r = .53$) was higher than that of all DZ twins ($r = .30$) or same-sex DZ twins ($r = .28$). We found a similar pattern for academic achievement; differences between MZ and DZ correlations, however, were less pronounced. In examining cross-trait correlations, both within individuals (i.e., correlations between Twin 1's alcohol use and Twin 1's GPA) and across twin pairs (i.e., correlations between Twin 1's alcohol use and Twin 2's GPA), we found that the cross-twin cross-trait correlations were as high as within-person cross-trait correlations. Finally, the cross-trait cross-twin correlations in DZ twin pairs were comparable in magnitude to those correlations in MZ twin pairs, consistent with shared environmental influences that affect both phenotypes.

Results from the bivariate twin models are summarized in Table 3. The initial bivariate model had good fit, $\chi^2(31) = 29.76$, $p = .53$, RMSEA = .00. The variances of the ACE components in the bivariate model indicated that 59.8% of the variance in academic achievement was due to additive genetic influences, 15.6% was due to shared environmental influences, and 24.7% was due to nonshared environmental influences. Higher academic achievement was associated with lower alcohol use through a shared genetic pathway ($bA = -.42$, $p < .05$). Residual variance in alcohol use (unique of academic achievement) was due to nonshared environmental (47.3%) and additive genetic effects (38.7%), with minimal shared environmental influences (13.9%).

Although the cross-trait, cross-twin correlations suggested a shared environmental contribution to the association between alcohol use and academic achievement, none of the shared environmental parameters were significant in the full model. This finding may have occurred because the current sample size was underpowered to detect small shared environmental influences. Consequently, we fit a trimmed model that fixed shared environmental influences on academic achievement and alcohol use and the

shared environmental cross-path to zero. Results from this trimmed model are shown in Table 3. Again, academic achievement and alcohol use were associated through a shared genetic pathway ($bA = -.31$, $p < .01$). This "AE" model did not fit the data significantly worse than the original model ($\Delta\chi^2 = 1.26$, $\Delta df = 3$, $p = .74$). In addition, we fit another trimmed model that fixed all genetic parameters to zero. This "CE" model (results not tabulated) did in fact fit significantly worse, $\chi^2(34) = 42.73$, $\Delta\chi^2 = 12.97$, $\Delta df = 3$, $p = .005$. Thus, for the sake of parsimony, the "AE" model was carried forward for our subsequent, more complicated models.

In the next step, we fit two interaction models to test whether academic achievement conditioned genetic influences on alcohol use. Results from the interaction models are summarized in Table 4. The first interaction model revealed a significant negative interaction between achievement and genetic influence on alcohol use ($apx = -.30$, $p < .01$). This effect is illustrated in Figure 3. Additive genetic variation accounted for 59% of the variance in drinking among low-achieving adolescents but only 28% among high-achieving adolescents.

A second interaction model was tested, in which alcohol use was the predictor and moderator and achievement was the outcome. In this model, none of the interaction terms on the ACE components of academic achievement were significant, indicating that alcohol use did not moderate genetic or environmental influences on achievement.

Discussion

In summary, we found a phenotypic association between higher academic achievement and lower alcohol use that was consistent with previous research. An examination of cross-twin, cross-trait correlations suggested that unmeasured family-level factors account for the association between achievement and alcohol use.

Table 2
Sibling Pair Correlations for Alcohol Use and Grade Point Average

Variable	Full sample	MZ twins	DZ twins		
			All DZ twins	Same-sex	Opposite-sex
Alcohol Twin 1/Alcohol Twin 2	.40** ($n = 393$) [.31, .48]	.53** ($n = 162$) [.40, .63]	.30** ($n = 231$) [.17, .41]	.28** ($n = 133$) [.11, .43]	.35** ($n = 98$) [.16, .51]
GPA Twin 1/GPA Twin 2	.54** ($n = 389$) [.47, .61]	.63** ($n = 160$) [.52, .67]	.48** ($n = 229$) [.38, .58]	.56** ($n = 132$) [.43, .67]	.38** ($n = 97$) [.19, .53]
Alcohol Twin 1/GPA Twin 2	-.15** ($n = 392$) [-.25, -.05]	-.14 ($n = 160$) [-.29, .01]	-.16* ($n = 232$) [-.28, -.03]	-.08 ($n = 133$) [-.24, .10]	-.25* ($n = 99$) [-.42, -.05]
Alcohol Twin 2/GPA Twin 1	-.15** ($n = 387$) [-.25, -.05]	-.12 ($n = 162$) [-.27, .03]	-.17** ($n = 225$) [-.30, -.04]	-.26** ($n = 131$) [-.41, -.09]	-.01 ($n = 94$) [-.21, .19]
Alcohol Twin 1/GPA Twin 1	-.16** ($n = 389$) [-.26, -.06]	-.16* ($n = 162$) [-.30, 0.00]	-.17* ($n = 227$) [-.29, -.04]	-.14 ($n = 131$) [-.31, .03]	-.20 ($n = 96$) [.38, 0.00]
Alcohol Twin 2/GPA Twin 2	-.15** ($n = 390$) [-.24, -.05]	-.09 ($n = 160$) [-.24, .06]	-.19* ($n = 230$) [-.31, -.06]	-.20* ($n = 133$) [-.36, -.03]	-.18 ($n = 97$) [.36, .03]

Note. MZ = monozygotic; DZ = dizygotic; GPA = grade point average. Numbers of complete pairs are shown in parentheses. 95% confidence intervals are shown in brackets.

* $p < .05$. ** $p < .01$.

Table 3

Unstandardized Parameter Estimates From Bivariate Models of Academic Achievement and Alcohol Use ($N = 399$ Twin Pairs)

Variable	Full ACE model		Trimmed AE model	
	Unstandardized	Standardized	Unstandardized	Standardized
Model fit indices				
χ^2 (df , p)	29.76 (31, .53)		31.02 (34, .61)	
RMSEA	.00		.00	
Variance in GPA				
A_{GPA}	.82* [.68, .96]	$h^2 = 59.8\%$.85* [.78, .91]	$h^2 = 71.6\%$
C_{GPA}	.21 [-.30, .72]	$c^2 = 15.6\%$		
E_{GPA}	.34* [.29, .39]	$e^2 = 24.65\%$.34* [.29, .38]	$e^2 = 28.4\%$
Regression parameters				
Genetic path (bA)	-.42* [-.78, -.06]	$\beta = -.29$ [-.53, -.04]	-.31* [-.46, -.16]	$\beta = -.21$ [-.31, -.11]
Shared environmental path (bC)	.35 [-1.33, 2.03]	$\beta = .24$ [-.91, 1.39]		
Nonshared environmental path (bE)	.06 [-.14, .25]	$\beta = .04$ [-.10, .17]	.04 [-.15, .22]	$\beta = .03$ [-.10, .15]
Variance in alcohol use				
A_{Alc}	.83* [.42, 1.24]	$h^2 = 38.7\%$	1.00* [.88, 1.13]	$h^2 = 50.1\%$
C_{Alc}	.30 [-1.97, 2.56]	$c^2 = 13.9\%$		
E_{Alc}	1.02* [.91, 1.12]	$e^2 = 47.3\%$	1.00* [.91, 1.10]	$e^2 = 49.9\%$

Note. A = additive genetic effects; C = shared environmental effects; E = nonshared environmental effects; RMSEA = root-mean-square error of approximation; GPA = grade point average. 95% confidence intervals are shown in brackets.

* $p < .05$.

Subsequent bivariate twin models indicated that these family-level factors were genetic; that is, the same genetic factors that promoted achievement also appeared to deter drinking. Moreover, we found evidence for a $G \times E$ interaction, with genetic influence on alcohol use suppressed among higher achieving adolescents.

The context for these findings is the growing body of research highlighting that more academically successful adolescents are less likely to drink, research that has generated theoretical discussions of risk and protection and school-based intervention and prevention efforts targeting public health concerns. Yet, there remain questions about the nature of the underlying link between achievement and alcohol use. Investigations using advanced methods to account for both directionality and other confounds has documented the achievement-to-substance use association (Bryant et al., 2000; Crosnoe, 2006), and our findings provide further evidence of the pathway from achievement to drinking using a

genetically informed approach. The research reported here suggests that, although the association between achievement and drinking is largely due to shared genetic influences, a protective effect is also in play, albeit one that emerges for only those with the highest propensity to drink. That is, for adolescents who have stronger genetic predispositions to engage in alcohol use, high achievement protects them from actually acting on this predisposition.

The protective effect that we observed—essentially, environment-constraining genetics—is theoretically meaningful. By showing exactly where the balance of academic stakes and behavioral opportunities is strongest, our work informs both social control and differential association theories as well as other perspectives (e.g., social learning theory) relevant to the social etiology of substance use. Moreover, these findings align with behavioral genetic research showing that genetic influences on substance

Table 4

Unstandardized Parameter Estimates From Interaction Models of Academic Achievement and Alcohol Use ($N = 399$ Twin Pairs)

Variable	Achievement \rightarrow Alcohol use		Alcohol use \rightarrow Achievement	
	Estimate	[95% CI]	Estimate	[95% CI]
Genetic and environmental paths from predictor to outcome				
Main effect of genes (ba0)	-.41 (.09)*	[-.59, -.24]	-.25 (.09)*	[-.42, -.08]
Gene \times Predictor interaction (bax)	-.06 (.09)	[-.24, .12]	.01 (.08)	[-.14, .16]
Main effect of nonshared environment (be0)	.05 (.10)	[-.14, .24]	.04 (.05)	[-.06, .13]
Nonshared Environment \times Predictor interaction (bex)	-.06 (.13)	[-.32, .20]	-.02 (.06)	[-.13, .09]
Residual genetic and environmental influences on outcome				
Main effect of genes (a0)	.92 (.09)*	[.75, 1.09]	.81 (.04)*	[.73, .89]
Gene \times Predictor interaction (apx)	-.30 (.10)*	[-.49, -.11]	-.02 (.04)	[-.09, .05]
Main effect of nonshared environment (e0)	1.00 (.06)*	[.87, 1.12]	.33 (.03)*	[.28, .38]
Nonshared Environment \times Predictor interaction (epx)	-.11 (.07)	[-.25, .04]	.02 (.03)	[-.05, .08]

Note. Achievement is the predictor in the first column and outcome in the second. The residual genetic and environmental influences on the outcomes in the lower portion of the table are unique of the predictors. For moderation analyses, achievement was centered at the grand mean of the sample, 2.71 (on a 4.0 scale). CI = confidence interval.

* $p < .05$.

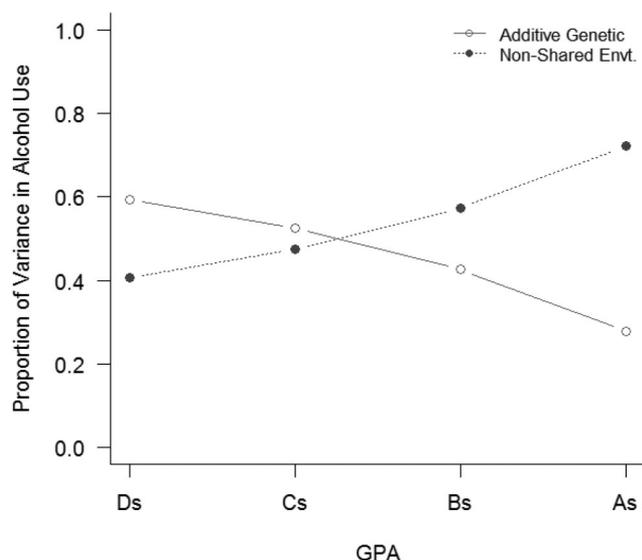


Figure 3. Proportion of variance in alcohol use due to additive genetics (h^2) and nonshared environmental (Env.) influences (e^2) by achievement level. Results based on model parameters summarized in Table 3. GPA = grade point average.

use are suppressed in environments that impose high social control (see Kendler et al., 2012). For example, genetic influence on substance use declines in the presence of high-parental monitoring (Dick et al., 2007), religious upbringing (Koopmans, Slutske, van Baal, & Boomsma, 1999), and positive marital relationships (Dick et al., 2006). Such environments limit opportunities for substance use and impose social norms and expectations that are incompatible with heavy use. Our results suggest that the environmental context of high achievement operates similarly. The high stakes tied to academic achievement in combination with more limited opportunities to drink associated with achievement seem to be particularly relevant for adolescents at greatest risk to engage in alcohol use. How this protective effect plays out (i.e., stronger for those most at risk) is in line with developmental theory that suggests the transactions among systems internal and external to the adolescent are inextricably linked (e.g., systems theory; Lerner, 2006). Those transactions between what is going on inside the bodies of adolescents and what is going on in the settings of their lives are difficult to study directly, but figuring out how they work is essential to testing and refining theory.

Beyond its implication for theory, this study also has relevance for policy and practice. Our findings suggest that promoting achievement may be one promising method of intervention and prevention for youth predisposed to drink. Given prior research highlighting the academic benefits of school bonding, extracurricular activities, and other school engagement processes (Anderman, 2002; Fredricks & Eccles, 2006; Furlong et al., 2003), the findings reported here suggest that these activities might also limit at-risk adolescents' likelihood of engaging in risky behaviors. The likely benefits of interventions targeting achievement rather than substance use are further bolstered by the fact that our tests of directionality revealed that the $G \times E$ interaction extended in only one direction. Alcohol use and achievement were not more

strongly associated among youth with stronger genetic propensities to achieve, echoing prior work favoring the achievement-to-substance use pathway more so than the alternative substance-use-to-achievement pathway (Bryant et al., 2000; Crosnoe, 2006). We believe that further work replicating the findings reported here using larger, longitudinal samples will strengthen the call for such academically targeted, dual-purpose intervention and prevention efforts.

Although consistent with both social control and differential association theories, the mechanisms underlying the observed protective effect of academic achievement on drinking remain unclear, and elucidating these mechanisms is critical for intervention efforts. Importantly, academic achievement was not randomly assigned; as an individual-differences variable in an observational study, academic achievement is a proxy for the array of contextual (e.g., peer groups, time use, parental engagement) and intrapersonal (e.g., impulsivity, conscientiousness, cognitive ability) variables that differ between high-achieving and low-achieving adolescents. It remains to be determined which of the features of academic achievement are the most potent constraints on genetic predispositions toward drinking. Prior research has revealed both academic stakes and behavioral opportunity structures play key roles in adolescents' engagement in risky behaviors (Crosnoe, 2006; Matsueda, 1982) and that the effects of externalizing and internalizing behaviors on alcohol initiation are generally indirect (Geels, Vink, van Beijsterveldt, Bartels, & Boomsma, 2013). Future research should address this potential limitation by thoughtfully integrating both stakes and opportunities as mechanisms into models to identify the most potent processes by which high achievement may limit adolescents' alcohol use. Genetically informed mediational models provide a promising avenue, as they enable researchers to integrate path analyses with decompositions into genetically and environmentally mediated effects (see, e.g., Tucker-Drob & Harden, 2012).

Our sample size ($n < 400$ pairs) may have been underpowered to detect shared environmental effects (Hanscombe et al., 2012). The magnitudes of the raw cross-twin cross-trait correlations in MZ and DZ twins suggest that there may be shared environmental contributions to the association between alcohol use and achievement that could not be differentiated in our study. A key strength of Add Health, relative to many other U.S. and European twin samples, is its economic and racial/ethnic diversity. Still, by the standards of behavioral genetics, it contains a small number of twins. Large twin samples that represent population diversity and have sufficient power for sophisticated analyses of gene-environment interplay remain a lacuna in developmental research. Despite this limitation, the key goal of this study was not to decompose the main effect of academic achievement into genetic and shared environmental effects but rather to assess the interaction between achievement and unique genetic influences on alcohol use.

This line of work also should be extended to later periods of development. We specifically targeted middle adolescence, as it is a time when substance use is likely to be initiated and when academic factors can delay the initiation and frequency of substance use (Johnston et al., 2012). Yet, whether achievement serves a similar protective factor later in the developmental life course, such as in late adolescence or young adulthood, is unknown. Prior research shows that the association between aca-

dem achievement and alcohol use actually switches direction, such that higher achievement is associated with lower levels of substance use in adolescence but with higher rates of substance use in young adulthood (Crosnoe & Riegle-Crumb, 2007; Maggs et al., 2008). Furthermore, evidence suggests that genetic influences play an increasingly strong role in alcohol use and abuse from adolescence into young adulthood (Dick, 2011). Taken together, this emerging body of work suggests that the protective effects of achievement may be time limited, but further empirical studies are needed to directly examine the validity of this hypothesis. Longitudinal designs with genetically informed data offer a promising avenue for exploring the persistence of protective effects across adolescence and early adulthood.

Adolescents are initiating alcohol use at an earlier age and are engaging in more drinking behaviors than they did two decades ago (Geels et al., 2012). Such behaviors are a public health concern given the plethora of research that highlights the negative effects of drinking across the life course (Krohn, Lizotte, & Perez, 1997; Oesterle et al., 2004). In the current study, we explored drinking behaviors at a critical developmental period and elucidated an important protective factor that mitigated alcohol use in those with genetic predispositions for drinking. Such work is in line with the call to examine positive youth development and protective (rather than risk) factors in development (Lerner et al., 2013). By identifying the genetic components that link achievement to alcohol use and determining who benefits most from academic success, we hope that this study will stimulate the additional research needed to more effectively inform policy and practice in developmentally appropriate ways.

References

- Anderman, E. M. (2002). School effects on psychological outcomes during adolescence. *Journal of Educational Psychology, 94*, 795–809. doi:10.1037/0022-0663.94.4.795
- Bandura, A. (1977). *Social learning theory*. Oxford, England: Prentice Hall.
- Bouchard, T. J., & McGue, M. (1981). Family studies of intelligence: A review. *Science, 212*, 1055–1059. doi:10.1126/science.7195071
- Brown, S. A., McGue, M., Maggs, J., Schulenberg, J., Hingson, R., Swartzwelder, S., . . . Murphy, S. (2008). A developmental perspective on alcohol and youth age 16 to 10 years of age. *Pediatrics, 121*(Suppl. 4), S290–S310. doi:10.1542/peds.2007-2243D
- Bryant, A. L., Schulenberg, J., Bachman, J. G., O'Malley, P. M., & Johnston, L. D. (2000). Understanding the links among school misbehavior, academic achievement, and cigarette use: A national panel study of adolescents. *Prevention Science, 1*, 71–87. doi:10.1023/A:1010038130788
- Bryant, A. L., Schulenberg, J. E., O'Malley, P. M., Bachman, J. G., & Johnston, L. D. (2003). How academic achievement, attitudes, and behaviors relate to the course of substance use during adolescence: A 6-year, multiwave national longitudinal study. *Journal of Research on Adolescence, 13*, 361–397. doi:10.1111/1532-7795.1303005
- Bryant, A. L., & Zimmerman, M. A. (2002). Examining the effects of academic beliefs and behaviors on changes in substance use among urban adolescents. *Journal of Educational Psychology, 94*, 621–637. doi:10.1037/0022-0663.94.3.621
- Catalano, R. F., Oesterle, S., Fleming, C. B., & Hawkins, J. D. (2004). The importance of bonding to school for healthy development: Findings from the Social Development Research Group. *Journal of School Health, 74*, 252–261. doi:10.1111/j.1746-1561.2004.tb08281.x
- Cleveland, H. H., & Wiebe, R. P. (2003). The moderation of adolescent-to-peer similarity in tobacco and alcohol use by school levels of substance use. *Child Development, 74*, 279–291. doi:10.1111/1467-8624.00535
- Conley, D., & Rauscher, E. (2011). The equal environments assumption in the post-genomic age: Using misclassified twins to estimate bias in heritability models. *National Bureau of Economic Research* (Working Paper No. 16711), 1–27.
- Crosnoe, R. (2006). The connection between academic failure and adolescent drinking in secondary school. *Sociology of Education, 79*, 44–60. doi:10.1177/003804070607900103
- Crosnoe, R., Benner, A. D., & Schneider, B. (2012). Drinking, socioemotional functioning, and academic progress in secondary school. *Journal of Health and Social Behavior, 53*, 150–164. doi:10.1177/0022146511433507
- Crosnoe, R., & Riegle-Crumb, C. (2007). A life course model of education and alcohol use. *Journal of Health and Social Behavior, 48*, 267–282. doi:10.1177/002214650704800305
- Dick, D. M. (2011). Developmental changes in genetic influences on alcohol use and dependence. *Child Development Perspectives, 5*, 223–230. doi:10.1111/j.1750-8606.2011.00207.x
- Dick, D. M., Agrawal, A., Schuckit, M. A., Bierut, L., Hinrichs, A., Fox, L., . . . Begleiter, H. (2006). Marital status, alcohol dependence, and GABRA2: Evidence for gene-environment correlation and interaction. *Journal of Studies on Alcohol, 67*, 185–194.
- Dick, D. M., Viken, R., Purcell, S., Kaprio, J., Pulkkinen, L., & Rose, R. J. (2007). Parental monitoring moderates the importance of genetic and environmental influences on adolescent smoking. *Journal of Abnormal Psychology, 116*, 213–218. doi:10.1037/0021-843X.116.1.213
- Fredricks, J. A., & Eccles, J. S. (2006). Is extracurricular participation associated with beneficial outcomes? Concurrent and longitudinal relations. *Developmental Psychology, 42*, 698–713. doi:10.1037/0012-1649.42.4.698
- Furlong, M. J., Whipple, A. D., St. Jean, G., Simental, J., Soliz, A., & Punthuna, S. (2003). Multiple contexts of school engagement: Moving toward a unifying framework for educational research and practice. *The California School Psychologist, 8*, 99–113.
- Geels, L. M., Bartels, M., van Beijsterveldt, T. C. E. M., Willemsen, G., van der Aa, N., Boomsma, D. I., & Vink, J. M. (2012). Trends in adolescent alcohol use: Effects of age, sex, and cohort on prevalence and heritability. *Addiction, 107*, 518–527. doi:10.1111/j.1360-0443.2011.03612.x
- Geels, L. M., Vink, J. M., van Beijsterveldt, T. C. E. M., Bartels, M., & Boomsma, D. I. (2013). Developmental prediction model for early alcohol initiation in Dutch adolescents. *Journal of Studies in Alcohol and Drugs, 74*, 59–70.
- Hanscombe, K. B., Trzaskowski, M., Haworth, C. M., Davis, O. S., Dale, P. S., & Plomin, R. (2012). Socioeconomic status (SES) and children's intelligence (IQ): In a UK-representative sample SES moderates the environmental, not genetic, effect on IQ. *PloS ONE, 7*, e30320. doi:10.1371/journal.pone.0030320
- Harris, K. M., Halpern, C. T., Smolen, A., & Haberstick, B. C. (2006). The National Longitudinal Study of Adolescent Health (Add Health) twin data. *Twin Research and Human Genetics, 9*, 988–997. doi:10.1375/twin.9.6.988
- Hawkins, J. D., Catalano, R. F., & Miller, J. Y. (1992). Risk and protective factors for alcohol and other drug problems in adolescence and early adulthood: Implications for substance use prevention. *Psychological Bulletin, 112*, 64–105. doi:10.1037/0033-2909.112.1.64
- Hirschi, T. (1969). *Causes of delinquency*. Berkeley: University of California Press.
- Jacobson, K. C., & Rowe, D. C. (1999). Genetic and environmental influences on the relationships between family connectedness, school

- connectedness, and adolescent depressed mood. *Developmental Psychology*, 35, 926–939. doi:10.1037/0012-1649.35.4.926
- Jeynes, W. H. (2002). The relationship between the consumption of various drugs by adolescents and their academic achievement. *American Journal of Drug and Alcohol Abuse*, 28, 15–35. doi:10.1081/ADA-120001279
- Johnson, M. K. (2004). Further evidence on adolescent employment and substance use: Differences by race and ethnicity. *Journal of Health and Social Behavior*, 45, 187–197. doi:10.1177/002214650404500205
- Johnston, L. D., O'Malley, P. M., Bachman, J. G., & Schulenberg, J. E. (2012). *Monitoring the Future national survey results on drug use, 1975–2011: Volume I, secondary school students*. Ann Arbor: Institute for Social Research, The University of Michigan.
- Kendler, K. S., Chen, X., Dick, D., Maes, H., Gillespie, N., Neale, M. C., & Riley, B. (2012). Recent advances in the genetic epidemiology and molecular genetics of substance use disorders. *Nature Neuroscience*, 15, 181–189. doi:10.1038/nn.3018
- Kendler, K. S., Neale, M. C., Kessler, R. C., Heath, A. C., & Eaves, L. J. (1993). A test of the equal-environment assumption in twin studies of psychiatric illness. *Behavior Genetics*, 23, 21–27. doi:10.1007/BF01067551
- Kiecolt, J., Aggen, S. H., & Kendler, K. S. (2013). Genetic and environmental influences on the relationship between mastery and alcohol dependence. *Alcoholism: Clinical and Experimental Research*, 37, 905–913. doi:10.1111/acer.12058
- King, K. M., Meehan, B. T., Trim, R. S., & Chassin, L. (2006). Marker or mediator? The effects of adolescent substance use on young adult educational attainment. *Addiction*, 101, 1730–1740. doi:10.1111/j.1360-0443.2006.01507.x
- Koopmans, J. R., Slutske, W. S., van Baal, G. C., & Boomsma, D. I. (1999). The influence of religion on alcohol use initiation: Evidence for genotype X environment interaction. *Behavior Genetics*, 29, 445–453. doi:10.1023/A:1021679005623
- Krohn, M. D., Lizotte, A. J., & Perez, C. M. (1997). The interrelationship between substance use and precocious transitions to adult statuses. *Journal of Health and Social Behavior*, 38, 87–103. doi:10.2307/2955363
- Langenkamp, A. G. (2009). Following different pathways: Social integration, achievement, and the transition to high school. *American Journal of Education*, 116, 69–97. doi:10.1086/605101
- Latvala, A., Castaneda, A. E., Perälä, J., Saarni, S. I., Aalto-Setälä, T., Lönnqvist, J., . . . Tuulio-Henriksson, A. (2009). Cognitive functioning in substance abuse and dependence: A population-based study of young adults. *Addiction*, 104, 1558–1568. doi:10.1111/j.1360-0443.2009.02656.x
- Lerner, R. M. (2006). Developmental science, developmental systems, and contemporary theories of human development. In R. M. Lerner (Ed.), *Theoretical models of human development: Vol. 1. Handbook of child psychology* (pp. 1–17). Hoboken, NJ: Wiley.
- Lerner, R. M., Agans, J. P., Arbeit, M. R., Chase, P. A., Weiner, M. B., Schmid, K. L., & Warren, A. E. A. (2013). Resilience and positive youth development: A relational developmental systems model. In S. Goldstein & R. B. Brooks (Eds.), *Handbook of resilience in children* (pp. 293–308). New York, NY: Springer Science + Business Media.
- Loehlin, J. C., Harden, K. P., & Turkheimer, E. (2009). The effects of assumptions about parental assortative mating and genotype-income correlation on estimates of genotype-environment interaction in the National Merit Twin Study. *Behavior Genetics*, 39, 165–169. doi:10.1007/s10519-008-9253-9
- Maggs, J. L., Patrick, M. E., & Feinstein, L. (2008). Childhood and adolescent predictors of alcohol use and problems in adolescence and adulthood in the National Child Development Study. *Addiction*, 103, 7–22. doi:10.1111/j.1360-0443.2008.02173.x
- Matsueda, R. L. (1982). Testing control theory and differential association: A causal modeling approach. *American Sociological Review*, 47, 489–504. doi:10.2307/2095194
- McCaffery, J. M., Papandonatos, G. D., Lyons, M. J., Koenen, K. C., Tsuang, M. T., & Niaura, R. (2008). Educational attainment, smoking initiation and lifetime nicotine dependence among male Vietnam-era twins. *Psychological Medicine*, 38, 1287–1298. doi:10.1017/S0033291707001882
- Muthén, L. K., & Muthén, B. O. (1998–2012). *Mplus user's guide* (6th ed.). Los Angeles, CA: Author.
- Neale, M. C., & Cardon, L. R. (1992). *Methodology for genetic studies of twins and families*. New York, NY: Kluwer Academic/Plenum. doi:10.1007/978-94-015-8018-2
- Oesterle, S., Hill, K. G., Hawkins, J. D., Guo, J., Catalano, R. F., & Abbott, R. D. (2004). Adolescent heavy episodic drinking trajectories and health in young adulthood. *Journal of Studies on Alcohol*, 65, 204–212.
- Petrill, S. A., & Wilkerson, B. (2000). Intelligence and achievement: A behavioral genetic perspective. *Educational Psychology Review*, 12, 185–199. doi:10.1023/A:1009023415516
- Resnick, M. D., Bearman, P. S., Blum, R. W., Bauman, K. E., Harris, K. M., Jones, J., . . . Udry, R. (1997). Protecting adolescents from harm: Findings from the National Longitudinal Study on Adolescent Health. *Journal of the American Medical Association*, 278, 823–832. doi:10.1001/jama.1997.03550100049038
- Schulenberg, J., Bachman, J. G., O'Malley, P. M., & Johnston, L. D. (1994). High school educational success and subsequent substance use: A panel analysis following adolescents into young adulthood. *Journal of Health and Social Behavior*, 35, 45–62. doi:10.2307/2137334
- Schulenberg, J. E., & Maggs, J. L. (2002). A developmental perspective on alcohol use and heavy drinking during adolescence and the transition to young adulthood. *Journal of Studies on Alcohol* (Suppl. 14), 54–70.
- Staff, J., Patrick, M. E., Loken, E., & Maggs, J. L. (2008). Teenage alcohol use and educational attainment. *Journal of Studies on Alcohol and Drugs*, 69, 848–858.
- Steiger, J. H. (1990). Structural model evaluation and modification: An interval estimation approach. *Multivariate Behavioral Research*, 25, 173–180. doi:10.1207/s15327906mbr2502_4
- Sutherland, E. H. (1947). *Principles of criminology* (4th ed.). Philadelphia, PA: Lippincott.
- Trzaskowski, M., Yang, J., Visscher, P. M., & Plomin, R. (2013). DNA evidence for strong genetic stability and increasing heritability of intelligence from age 7 to 12. *Molecular Psychiatry*. Advance online publication. doi:10.1038/mp.2012.191
- Tucker-Drob, E. M., & Harden, K. P. (2012). Intellectual interest mediates Gene × Socioeconomic Status interaction on adolescent academic achievement. *Child Development*, 83, 743–757.
- van der Sluis, S., Posthuma, D., & Dolan, C. V. (2012). A note on false positives and power in G × E modeling of twin data. *Behavior Genetics*, 42, 170–186. doi:10.1007/s10519-011-9480-3

Received April 4, 2013

Revision received September 13, 2013

Accepted October 16, 2013 ■