Racial Differences in the Transactional Relationship Between Depression and Alcohol Use From Elementary School to Middle School

ERICA L. BIRKLEY, PH.D.,[a] TAMIKA C. B. ZAPOLSKI, PH.D.,[b,*] & GREGORY T. SMITH, PH.D.[c,d]

[a]*Department of Psychological Sciences, Purdue University, West Lafayette, Indiana*
[b]*Department of Psychology, Indiana University Purdue University at Indianapolis, Indianapolis, Indiana*
[c]*Department of Psychology, University of Kentucky, Lexington, Kentucky*
[d]*Center on Drug and Alcohol Research, University of Kentucky, Lexington, Kentucky*

ABSTRACT. Objective: The aim of this investigation was to test hypothesized reverse prospective relationships between alcohol consumption and depressive symptomatology as a function of race among youth. **Method:** In a two-wave prospective study, 328 European American, 328 African American, and 144 Hispanic American youth were studied at the end of fifth grade (last year of elementary school) and the end of sixth grade (first year of middle school). **Results:** A positive correlation was observed between alcohol consumption and depressive symptoms among all youth. However, the predictive relationship differed based on race. For European American and Hispanic American youth, depressive symptom levels at the end of elementary school predicted alcohol consumption at the end of the first year of middle school, but the converse relationship was not observed. For African American youth, the opposite pattern was found. Alcohol consumption at the end of elementary school predicted depressive symptom levels at the end of the first year of middle school, and the converse relationship was not observed. **Conclusions:** These findings suggest the possibility that etiological relationships between depression and alcohol use vary by race, thus highlighting the importance of considering race when studying the risk process. (*J. Stud. Alcohol Drugs, 76,* 799–808, 2015)

THE RELATIONSHIP BETWEEN DEPRESSIVE symptomatology and alcohol has been well documented in youth (Kelder et al., 2001; Kubik et al., 2003), as are alcohol use disorders and major depressive disorder in adults (Lynskey, 1998; Swendsen & Merikangas, 2000). Yet, debate remains as to whether (a) depression predates and predicts alcohol use, (b) the reverse is true, or (c) reciprocal prediction exists between the two forms of dysfunction. This study reports the first longitudinal test of the hypothesis that this relationship varies by race. We studied European American (EA), African American (AA), and Hispanic American (HA) youth making the transition from elementary school to middle school. We briefly review evidence concerning the directionality of the relationship, the importance of the transition to middle school, and the argument for racial differences in this process.

Temporal nature of the depression–problem drinking relationship

Hussong and colleagues (2011) argued that there is an internalizing pathway to substance use, such that heightened negative affect places an individual at risk for subsequent substance use problems. Indeed, depressive symptoms or the presence of negative affect predicts alcohol consumption (Kidorf & Lang, 1999; Swendsen et al., 2000), and risk for developing alcohol use disorders is substantially increased by a prior major depressive disorder episode (Kuo et al., 2006; Prescott et al., 2000). The self-medication theory has provided rationale for this phenomenon, suggesting that individuals engage in alcohol use to provide relief from distress (Goldman, 1999; Khantzian, 1997).

A similar pathway has also been found among youth (e.g., Hussong et al., 2008; King et al., 2004). Caspi and colleagues (1996) found that anxiety and depression symptoms in childhood predicted substance use in late adolescence and early adulthood. Alcohol expectancy theory provides one possible explanation for this phenomenon (Smith & Goldman, 1994). Alcohol expectancies can be defined as one's learning history about the outcomes of alcohol use (Goldman et al., 1999). Thus, among youth, alcohol use may be facilitated by positive expectancies that alcohol will reduce negative affect, positive beliefs about alcohol as a means of coping or reducing tension, and the use of alcohol to aid in interpersonal deficits (Hussong et al., 2011).

There is also support for the converse relationship, that alcohol use is predictive of subsequent depressive symptoms. This relationship has been observed among adults (Boden & Fergusson, 2011; Wang & Patten, 2002) and youth (Brook et al., 2002; Hallfors et al., 2004). Among the possible ex-

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*Correspondence may be sent to Tamika C. B. Zapolski at the Department of Psychology, Indiana University Purdue University at Indianapolis, Indianapolis, IN 46202, or via email at: tzapolsk@iupui.edu.

planations for this relationship is that of an indirect effects process, in which negative consequences related to alcohol consumption increase the risk for experiencing negative mood states (Hasin & Grant, 2002; Swendsen & Merikangas, 2000).

It is also possible that the relationship is reciprocal. Hartka and colleagues (1991) conducted a meta-analysis of adult studies and found support for a reciprocal relationship between alcohol consumption and depressive symptomatology. Among adolescents, although alcohol consumption and depressive symptoms are related, empirical support for reciprocal pathways of prediction has not been found (Fleming et al., 2008; Hooshmand et al., 2012). Moreover, existing research has not tested the hypothesis that the temporal relationship between alcohol consumption and depressive symptoms varies by race.

Transition to middle school

To best advance understanding of etiological processes, it is important to investigate the depression–drinking relationship in youth at the very beginning of drinking experience, such as the first experience of drinking beyond a sip or taste of alcohol. The transition from elementary school to middle school is an ideal period of study for two specific reasons. First, rates of drinking increase dramatically during this age period (Donovan & Molina, 2011), and the percentage of youth who had consumed more than a few sips of alcohol approximately doubles from age 11 (the average age of fifth graders) to age 12 years (Donovan, 2007).

Second, both alcohol consumption and depressive symptomatology among youth this age are of considerable importance in predicting current and future dysfunction. For youth at age 12 years, reports of having consumed alcohol one or more days in the preceding year had a sensitivity of 1.0 and a specificity of .95 (girls) or .94 (boys) in the concurrent prediction of any past-year *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition* (American Psychiatric Association, 1994), alcohol use disorder symptoms (Chung et al., 2012). Early consumption is also a significant predictor of diagnostic status in later adolescence and adulthood (DeWit et al., 2000; Grant & Dawson, 1997). The early experience of depressive symptoms is also associated with long-term negative effects. Youth who experience depressive symptoms during adolescence (ages 14–18 years) often struggle with symptoms throughout their lives (Lewinsohn et al., 1999). Similarly, youth diagnosed with major depressive disorder during adolescence are at two- to threefold increased risk of major depressive disorder during adulthood (Pine et al., 1998; Weissman et al., 1999). Thus, it is crucial to identify and understand the developmental pathways between alcohol and depressive symptoms during this important developmental period.

Racial differences in the prospective relationship between depressive symptoms and drinker status

It is possible that the transactional relationship between negative mood and alcohol use differs as a function of race. Empirically, the finding that depressive symptomatology predicts subsequent alcohol use appears to have relied heavily on EA youth samples. For example, the participants in the King et al. (2004) study in which depression predicted subsequent drinking were almost entirely EAs (97.9% as reported in Iacono et al., 1999). In comparison, the racial composition in the Hallfors and colleagues (2004) study in which alcohol use was predictive of subsequent depressive symptoms was more diverse, with 16.7% of participants identifying themselves as AA and 12.1% identifying themselves as HA. Moreover, the Gilman and Abraham (2001) adult study that found a reciprocal relationship between alcohol use and depressive symptoms also consisted of a diverse sample of participants (EA, 70%; AA, 20%; HA, 8%).

In addition, we know that drinking patterns tend to differ as a function of race. AA youth generally report higher rates of abstinence and engage in lower levels of use (e.g., Wallace et al., 2003) and show slower increases in rates of drinking (e.g., Chen & Jacobson, 2012) compared with their EA peers. Those AA youth who do drink tend to experience more negative social consequences from use compared with their EA peers at similar levels of alcohol use (e.g., Bailey & Rachal, 1993; Barnes & Welte, 1986).

It has been proposed that one reason for racial differences in alcohol consumption among youth is differences in cultural norms, such that AA culture includes more restricted norms of acceptable alcohol use, more negative attitudes toward alcohol use, and stronger beliefs in the dangers of alcohol consumption compared with mainstream American culture (see the review by Zapolski et al., 2014). As a consequence, AA youth who engage in drinking behaviors tend to experience greater social sanctions from their peers and parents related to their alcohol use than do their EA counterparts (Zapolski et al., 2014). For these reasons, we hypothesized that for AA youth, the relationship would be such that alcohol use would increase the risk for subsequent depressive symptoms. Consistent with Hasin and Grant (2002), we believe that for AA youth, alcohol consumption indirectly increases the risk for depressive symptoms because of the experience of negative consequences resulting from alcohol use.

Concerning HA children, regrettably there is little research on the competing pathways to risk for alcohol use and depressive symptomatology. A positive relationship between alcohol use and depressive symptoms has been reported for this group (Alva, 1995; Kelder et al., 2001). Some authors have proposed that substance use among HA youth can be a response to emotional distress (Félix-Ortiz et al., 1995), suggesting a similar pathway between alcohol use and depressive symptoms as that postulated for EA youth.

Current study

Based on these considerations, we hypothesized that depressive symptom endorsement in fifth-grade children would predict alcohol consumption for EA and HA youth, but the converse would not be true. We hypothesized the opposite relationship for AA youth: that alcohol consumption in fifth grade would predict subsequent increases in depression symptom endorsement, but the converse would not be true. We conducted this test using a two-wave longitudinal design, measuring both depressive symptom endorsement and drinker status in the spring of fifth grade (the last year of elementary school) and the spring of sixth grade (the first year of middle school). We sought to test the predictive relationships during this developmental transition to observe these processes before the accumulation of ongoing experiences with alcohol. Support for our hypotheses will clarify the observation of cross-sectional relationships between depressive symptoms and alcohol use that may reflect different processes for youth of different racial backgrounds.

Although pubertal onset was not a focus of our study, it is important to consider in any model concerning the transition into middle school. Early pubertal onset, often defined as occurring before 75% of one's peers (Lynne-Landsman et al., 2010), predicts early alcohol use and related behaviors (Dick et al., 2000; Westling et al., 2008). Its presumed influence is thought to reflect biological, social, and contextual factors, and even to represent parental psychopathology (Dick et al., 2000; Ellis & Garber, 2000). We thus included early pubertal onset in our model test.

Two other demographic variables included in our model are socioeconomic status (SES) and sex. We included SES because apparent racial differences may, at times, be more attributable to SES differences than racial differences (e.g., Caetano, 1984; Jones-Webb et al., 1997). We included sex because in older adolescents, depression scores are generally higher among girls (Garrison et al., 1990), and a stronger relationship between depressive symptoms and drinking behavior has been observed among girls than in boys (Edwards et al., 2014; Poulin et al., 2005). The possible impact of sex differences in children this young and over a 1-year window may be low, but we controlled for sex nevertheless.

Method

Participants

Participants were drawn from an ongoing parent study examining risk for alcohol, smoking, and disordered eating among youth sampled from urban, suburban, and rural school districts in the Southeast. Each family with a child in one of 23 elementary schools was sent a letter, through the U.S. Mail, introducing the study. A passive consent procedure was used: parents were invited to notify the study office or their school if they did not want their child to participate. For children whose parent did not opt them out of the study, active assent was obtained from each participant. This procedure was requested by the participating school systems and approved by the university institutional review board. Of 1,988 fifth graders in the participating schools, 1,906 participated in the study (95.9%). Reasons for nonparticipation included declination of consent from parents, declination of assent from children, and language or cognitive difficulties (see Settles et al., 2014, for further information about the parent study).

For the current study, we used all 328 AA and 144 HA children in the parent sample and randomly selected 328 EA children from the full sample of EA participants (N = 1,174). Thus, the sample for the current study included 800 children. We used this smaller number of EA participants to make sure that tests of invariance between groups did not favor one group due to differences in sample size. The subsample used for the study was equally divided between boys (EA = 48.4%, AA = 48.7%, HA = 50.0%) and girls. When we conducted model tests on the full sample, path coefficients differed slightly, but all tests of significance yielded the same results.

Measures

Demographic and background questionnaire. Participants were asked to provide demographic information by indicating their current age (in years), sex, and racial/ethnic background.

The Pubertal Development Scale. This scale assesses pubertal development and consists of five questions for each sex, using a 4-point Likert scale (Petersen et al., 1988). Estimates of internal consistency were very similar across race: Wave 1 α values ranged from .85 to .88 for girls and .90 to .93 for boys. At Wave 2, α = .99 for both sexes in all races. Scores on the Pubertal Development Scale correlate highly with physician ratings (Coleman & Coleman, 2002). As is common (e.g., Culbert et al., 2009), we used an available dichotomous classification in the current study, with mean scores above 2.5 indicative of pubertal onset.

The Drinking Styles Questionnaire. This scale was used to measure self-reported drinking. The Drinking Styles Questionnaire (Smith et al., 1995) measures drinking frequency with a single item. Children were classified as positive for drinking if they reported ever having consumed at least one drink, defined as "more than just a sip or a taste." We dichotomized the item to reflect drinker versus nondrinker status. This assessment method has proven stable over time and with good evidence for its validity (Settles et al., 2014; Smith et al., 1995).

Center for Epidemiological Studies–Depression Scale (CES-D). The CES-D is a 20-item scale measuring depression, with scores ranging from 0 to 60. The items are an-

swered on a scale of four frequency ratings (*less than 1 day* to *most or all [5–7] days*). Higher CES-D scores indicate greater severity of depressive symptoms. We used scores on a continuum to model the degree of depressive symptomatology (Chabrol et al., 2002; Garrison et al., 1991). There is good evidence of internal consistency and construct validity for the CES-D in adolescent samples (Radloff, 1991; Roberts et al., 1991). In the current study, $\alpha = .85$ at Wave 1 and .88 at Wave 2, skew values were 1.51 at Wave 1 and 1.67 at Wave 2, and kurtosis values were 2.72 at Wave 1 and 3.79 at Wave 2. Values for these variables were virtually identical across race.

Socioeconomic status. This variable was constructed using the 2000 U.S. Census data. Residential addresses of the 800 youth were matched with the census tract coding for April 1, 2000 (we used 2000 census data because data for this study were collected in 2008 and 2009, before the 2010 census). Census tracts are small, relatively permanent statistical subdivisions of a county and are uniquely numbered in each county with a numeric code (U.S. Census Bureau Geography, 2013). Four variables were used to characterize the SES of participants by census tract: percentage of individuals with education lower than a high school diploma, adult unemployment rate, adult poverty rate, and child (under age 18 years) poverty rate. A factor analysis resulted in a one-factor solution that explained 75.1% of the variance, with an eigenvalue of 3.01. The factor loadings of the four items ranged from 0.56 to 0.98. The internal consistency reliability estimate was high ($\alpha = .85$).

Procedure

Questionnaire administration procedures. Administration of the majority of questionnaires was completed during school hours. The few children who had moved completed the measures either at their new school or by mail. Completion time for the questionnaires was 60 minutes or less. Participants were provided small incentives for participation (i.e., keychain, pencil, rubber bracelet).

Data analytic method

Participation attrition. Of the sample of 800, 306 EA, 309 AA, and 128 HA youth participated in the spring of sixth grade (Wave 2), for a retention rate of 93% (the retention rate for the full sample of 1,906 was 92%). Individuals who participated in both waves of the study did not differ from those who participated in only one wave on any demographic, criterion, or trait variable. We therefore concluded that data were missing at random and imputed missing values using the expectation maximization procedure (Enders, 2006) and thus were able to use all data (Muthén & Muthén, 2010).

Path model. We constructed a two-wave path analysis to test prospective predictive relationships between depressive symptoms and drinker status. All constructs were modeled as measured variables. The model is depicted in Figure 1. Wave 1 pubertal status, drinker status, and CES-D score were each allowed to predict Wave 2 drinker status and CES-D score. We also modeled cross-sectional associations between each variable at Wave 1 and between disturbance terms at Wave 2. With this analytic design, we tested whether the hypothesized prediction from Wave 1 depressive symptoms to Wave 2 drinker status was present above and beyond both prior drinker status and pubertal onset. We also tested whether the prediction from Wave 1 drinker status to Wave 2 depressive symptoms was present above and beyond both prior depressive symptoms and pubertal onset. SES was also included in the model. Because, as we describe below, it was unrelated to any variable within race, we constrained its relationship to each Wave 2 outcome to zero. Sex was not included as a covariate; we separately tested for invariance by sex within race (also described below).

After testing whether this omnibus model fit the data well, we ran two additional models to test for group differences. The first imposed only the same structure of correlations and pathways for each group. The second imposed constraints designed to test our specific hypothesis of reverse prediction by race. For this second model, we constrained three pathways to be equal across group: the path from Wave 1 depression to Wave 2 drinking, the path from Wave 1 drinking to Wave 2 depression, and the Wave 2 covariance between drinking and depression. If these additional constraints are not justified (i.e., are inconsistent with the data), then the model specifying the constraints will fit significantly worse than the unconstrained model, which was our first model (Hoyle & Smith, 1994). Thus, if the constraints result in a decrement in model fit, one concludes that the prediction pathways did differ across groups. A difference in prediction pathways is what we hypothesized. We also tested invariance between groups on pathways other than those central to this model test; results are available on request.

In addition, within the EA and AA racial groups, we tested whether the model fit differently as a function of sex. We did not conduct this test with the HA group because we considered the sample size by sex (72 boys and 72 girls) too small to produce stable results.

Because we had a dichotomous endogenous variable, we used the weighted least squares means and variance adjusted (WLSMV) estimator (Muthén & Muthén, 2010). To measure model fit, we relied on four common fit indices: the comparative fix index (CFI), the Tucker–Lewis Index (TLI), the root mean square error of approximation (RMSEA), and the weighted root mean square residual (WRMR). To test whether imposition of equality constraints produced decreases in model fit, we relied on two indices. The first is based on the chi-square value produced for each model. One can calculate the chi-square of the difference between the constrained and unconstrained models; a significant chi-square value indi-

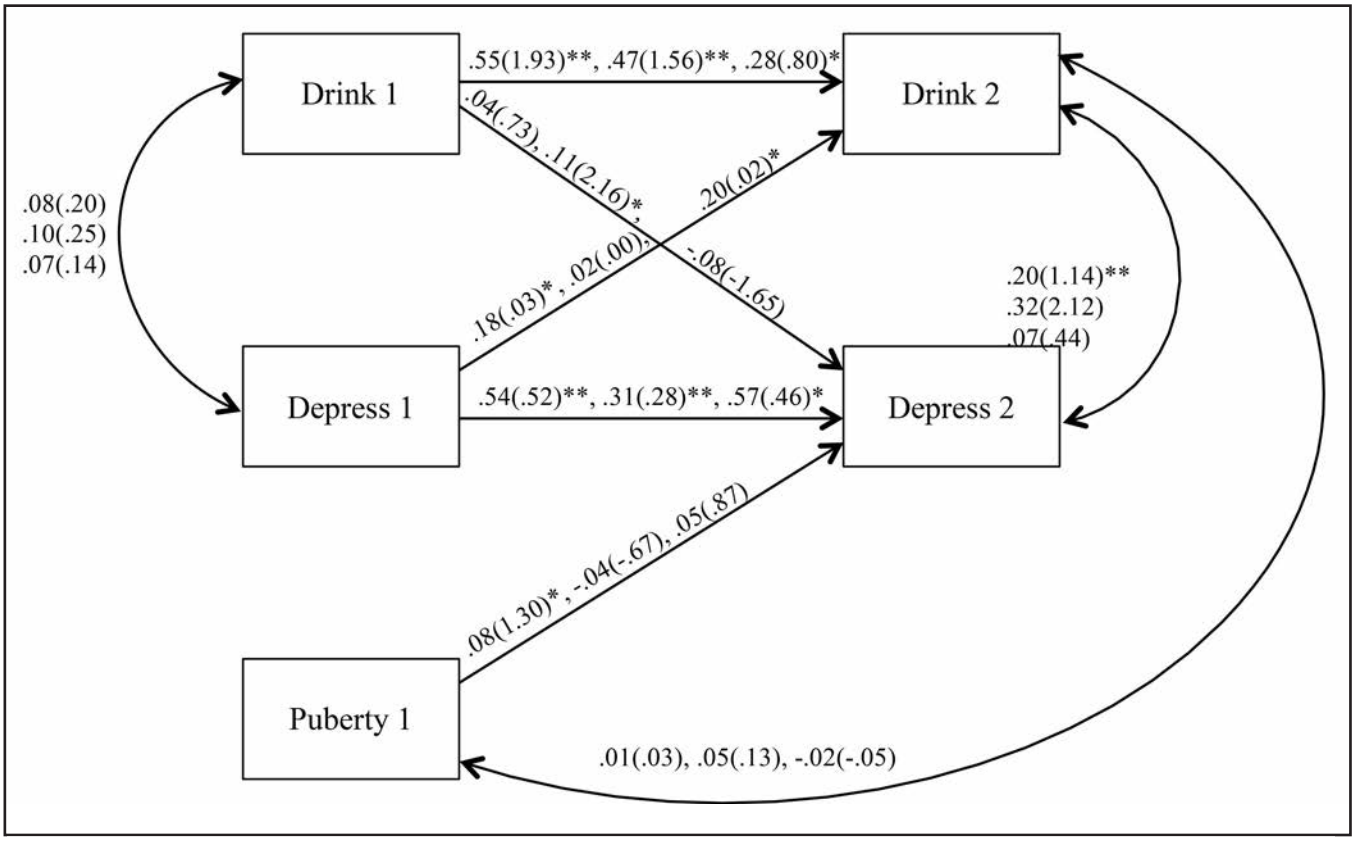

FIGURE 1. Depiction of a path analysis representing the pathways to drinker status (Drink) and depression (Depress) for European American, African American, and Hispanic American youth. Only hypothesized pathways are presented. Not included in the figure, for ease of presentation, are disturbance terms and error terms. Standardized coefficients are presented first, followed by the unstandardized coefficients in parentheses. The first set of coefficients is for the European American sample, the second for the African American sample, and the third for the Hispanic American sample. Measured, not latent, variables were studied.
*$p < .05$; **$p < .01$.

cates that the constrained model fits significantly worse than the unconstrained model, thus indicating that the constraints are not justified. The second index is the difference between the constrained and unconstrained CFI value: a drop of .01 or greater is thought to reflect a meaningful reduction in model fit (Kline, 2005).

Results

Tests of school-specific effects

To rule out school-specific effects, intraclass coefficients for each study variable were calculated using school membership ($N = 23$) as the nesting variable. There were no significant effects on any variable based on school membership. Interclass correlations ranged from 0.03 to 0.00.

Descriptive statistics

Table 1 lists descriptive statistics for SES, depression scores, and drinker status by race and sex within race. On average, HA and AA youth resided in neighborhoods with higher rates of unemployment, adult poverty, and child poverty and in neighborhoods with higher rates of individuals who did not have a high school diploma compared with EA youth ($p < .001$). AA youth resided in more disadvantaged neighborhoods than did HA youth ($p < .001$). The three groups reported comparable rates of depressive symptoms and drinker status. In addition, 24.8% of the children had experienced pubertal onset by Wave 1 (the percentage did not differ by race). Because this represents almost precisely the upper quartile, we defined pubertal onset by Wave 1 as early pubertal onset in comparison with one's peers.

Correlations among sex, pubertal status, drinker status, and depression symptom scores

Tables 2, 3, and 4 present the correlations among study variables. Because sex, pubertal status, and drinker status are dichotomous, correlations among those three variables are phi coefficients, and correlations between any of those

TABLE 1. Descriptive statistics for socioeconomic status, depression, and drinking by race

Variable	European American % (*SD*) or *M* (*SD*)	African American % (*SD*) or *M* (*SD*)	Hispanic American % (*SD*) or *M* (*SD*)
Socioeconomic status			
Less than high school diploma	14.48% (9.32)[a]	20.13% (10.65)[b]	18.83% (9.25)[b]***
Unemployment rate	2.65% (1.78)[a]	4.42% (3.29)[b]	3.55% (2.27)[b]***
Adult poverty rate	6.87% (5.54)[a]	11.82% (8.80)[b]	9.27% (6.24)[b]***
Child poverty rate	9.88% (7.75)[a]	17.14% (12.30)[b]	14.01% (8.96)[b]***
CES-D depressive symptoms			
Depression Wave 1	26.42 (7.47)	27.88 (8.11)	27.64 (8.93)
Depression Wave 2	25.60 (7.28)	26.78 (7.05)	25.32 (7.52)
Drinker status			
Drinker status Wave 1	14.94%	14.02%	16.67%
Drinker status Wave 2	16.16%	12.50%	19.44%

Notes: European Americans (n = 328); African Americans (n = 328); Hispanic Americans (n = 144). CES-D scores range from 0 to 60, with higher scores indicating greater severity of depressive symptoms. Groups with different superscripts differed significantly across socioeconomic status indicators: ***$p < .001$.

variables and other variables are point biserial correlations. SES was unrelated to any study variable within race.

For EA youth (Table 2), females were more likely to report depression at Waves 1 and 2. Pubertal onset was also related to depressive symptoms and drinker status at both waves. Depression at Wave 1 was related to drinker status at both waves. For AA youth (Table 3), depressive symptoms at Wave 2 were related to drinker status at Waves 1 and 2. For HA youth (Table 4), males were more likely to report drinker status at Wave 1. Pubertal onset was associated with drinker status at Wave 1. Depressive symptoms at Wave 1 were related to drinker status at Wave 2.

Tests of racial differences in prospective prediction between drinker status and depressive symptoms

We first tested an omnibus model that imposed no equality constraints across racial groups. The model fit the data well, $\chi^2(15) = 10.28$, $p > .80$; CFI = 1.0; TLI = 1.0; RMSEA = .00; WRMR = .78. To test our hypothesis of racial group differences in the specific pathways of Time 1 depression to Time 2 drinker status and Time 1 drinker status to Time 2 depression, we next tested a model that constrained three pathways: those two time-lagged pathways and the Time 2 correlation between depression and drinker status. This constrained model fit significantly worse than the unconstrained model: $\chi^2(21) = 23.71$, $p = .31$; CFI = .99; TLI = .99; RMSEA = .02; WRMR = 1.18. The chi-square difference test indicated a significant drop in model fit: $\chi^2(6) = 13.43$, $p < .05$. The drop in CFI equaled 1.0 and there were increases in both the RMSEA and WRMR. This finding indicates that there are different relationships between drinker status and depressive symptoms by race in this sample (Figure 1).

Tests of sex differences in prospective prediction between drinker status and depressive symptoms by race

For the EA children alone, when testing the same model for both sexes but without a constraint of equality of predictive pathway across sex, the model fit well: $\chi^2(11) = 7.33$, $p > .77$; CFI = 1.0; TLI = 1.0; RMSEA = .00; WRMR = .54. When we constrained all predictive pathways and the disturbance term to be equal for boys and girls, the fit of the model did not decline. The chi-square difference test was not significant, and neither the CFI nor TLI dropped with the constraint. We found no evidence that the predictive associations varied by sex among EA children.

For the AA children alone, again testing the same model for both sexes but without a constraint of equality of predictive pathway across sex, the model fit well: $\chi^2(11) = 5.31$, $p > .86$; CFI = 1.0; TLI = 1.0; RMSEA = .00; WRMR = .56. When we constrained all predictive pathways and the

TABLE 2. European American bivariate correlation matrix

	SEX	PUB1	SES	DP1	DP2	DS1	DS2
SEX	–	.05	-.04	.18***	.18***	-.10	-.05
PUB1		–	.04	.16**	.18***	.21***	.15**
SES			–	-.06	-.07	-.02	.01
DP1				–	.53***	.13*	.20***
DP2					–	.14*	.21***
DS1						–	.61***
DS2							–

Notes: n = 328. Sex = girls higher; pub = pubertal onset; SES = socioeconomic status; lower income is higher; DP = depressive symptoms; DS = drinker status; 1 = Time 1; 2 = Time 2. *$p < .05$; **$p < .01$; ***$p < .001$.

Table 3. African American bivariate correlation matrix

	SEX	PUB1	SES	DP1	DP2	DS1	DS2
SEX	–	-.04	-.03	.07	.08	.04	-.01
PUB1		–	.02	.03	-.03	.05	.04
SES			–	.02	.04	-.04	-.01
DP1				–	.30***	.07	.04
DP2					–	.12*	.25***
DS1						–	.46***
DS2							–

Notes: n = 328. Sex = girls higher; pub = pubertal onset; SES = socioeconomic status; lower income is higher; DP = depressive symptoms; DS = drinker status; 1 = Time 1; 2 = Time 2.
*p < .05; ***p < .001.

disturbance term to be equal for boys and girls, the fit of the model did not decline. The chi-square difference test was not significant, and neither the CFI nor TLI dropped with the constraint. We found no evidence that the predictive associations varied by sex among AA children.

Discussion

The key finding of this study was reverse longitudinal predictive relationships between depressive symptoms and drinker status as a function of race during the transition from elementary school to middle school. For EA and HA children, depressive symptoms at the end of elementary school predicted drinker status at the end of the first year of middle school, above and beyond prior drinker status, sex, and early pubertal onset; however, elementary school drinking did not predict subsequent depressive symptoms for either group. The opposite was true for AA children: Drinker status at the end of elementary school predicted depressive symptom levels at the end of the first year of middle school, above and beyond prior depressive symptoms, sex, and early pubertal onset, but elementary school depressive symptoms did not predict drinking for this group of youth.

These findings may prove important for theory and clinical application, particularly for this developmental period. Regarding theory, there are multiple implications specific to the emergence of drinking behavior in youth. Considering first EA and HA youth, it is important to appreciate the developmental context in which depressive symptoms predicted subsequent drinker status. This finding presumably does not mean that sixth graders are drinking to self-medicate as some adults do (Khantzian, 1997; Sher & Grekin, 2007). Instead, the process might reflect an initial exploration of alcohol's putative effects that is fostered by one's learning history about the effects of alcohol, as highlighted in expectancy theory (Smith & Goldman, 1994), or more specifically, extensive modeling that alcohol makes one feel better in many ways (Zucker et al., 2008). Perhaps distressed EA and HA youth are more likely to try alcohol because they have learned to anticipate that drinking will help them to feel better (e.g., Tomlinson & Brown, 2012).

We found the reverse predictive relationship for AA youth. We hypothesized this prediction based on a review by Zapolski and colleagues (2014) highlighting evidence of more conservative drinking norms and lower levels and rates of alcohol use among AA youth compared with their EA peers. As a consequence, AA youth who engage in drinking behaviors tend to experience greater social sanctions from their peers and parents related to their alcohol use (Zapolski et al., 2014). Thus, it is possible that the prospective relationship from drinking to depressive symptom endorsement reflects a process in which one's behavior is more fully outside the norm for one's group and is disapproved of, resulting in higher levels of distress. This possibility is consistent with the theory of the indirect effect of alcohol on depressive symptoms (Hasin & Grant, 2002). It is also possible that simple engagement in a behavior that is both unusual and negative in one's group leads to worse feelings about oneself. However, the current study provides no empirical information on these hypothesized mechanisms for the relationship

Table 4. Hispanic American bivariate correlation matrix

	SEX	PUB1	SES	DP1	DP2	DS1	DS2
SEX	–	.06	-.03	.04	.20	-.27***	-.04
PUB1		–	.04	.05	.10	.18*	.05
SES			–	.12	.10	.09	.13
DP1				–	.54***	.02	.17*
DP2					–	.01	.16
DS1						–	.25**
DS2							–

Notes: n = 144. Sex = girls higher; Pub1: early pubertal onset; SES = socioeconomic status; lower income is higher; DP = depressive symptoms; DS = drinker status; 1 = Time 1; 2 = Time 2.
*p < .05; **p < .01; ***p < .001.

between fifth-grade drinker status and sixth-grade depressive symptom endorsement.

Concerning clinical application, any alcohol consumption at this early age is important clinically because even one drink in the past year is highly associated with the concurrent presence of alcohol use disorder symptoms (Chung et al., 2012), and early consumption predicts future alcohol problems and diagnosable disorders (Ellickson et al., 2003; Pitkänen et al., 2005). Even though the number of youth drinking is relatively small (mean of 16% across groups), drinking during this developmental transition predicts current and future dysfunction. This is also true for depressive symptomatology (Lewinsohn et al., 1999; Thapar et al., 2012).

Although sex differences among depressive symptoms and drinker status were observed for each group, the strength and direction of the predictive relationship did not differ based on sex for EA and AA youth analyzed. Perhaps (a) there really are no sex differences in this process within each of these two racial groups, and other factors explain adult sex differences in alcohol consumption for the two groups; (b) such differences do exist but are only detectable with larger sample sizes; or (c) sex differences in alcohol use for negative reinforcement emerge later in development. The current study does not enable us to distinguish among these possibilities. The sample size of HA youth was too small to do multigroup tests by sex, so we could not test for sex differences within HA youth. There is a need for further work on the relationship between drinking and depression among youth as a function of both sex and race.

Although our two-wave design is a strength of the study because it identified important differences in the direction of prediction between alcohol and depressive symptomology that would be masked by cross-sectional correlational analysis, the short time frame is also a weakness. We have no information on whether the observed racial differences predict later differences in drinking behavior or depressive experience. A second weakness was the inability to fully assess externalizing dysfunction. However, we were able to control for prior drinking and early pubertal onset, which have been shown to predict subsequent drinking and other forms of externalizing behavior. Third, we relied on questionnaire assessment. Although there is good evidence for the validity of the measures we used, it is possible that interview assessments could have provided more precise information because of the opportunity to answer questions and clarify concepts. Fourth, we did not assess the context of drinking behavior. Fifth, we analyzed measured variables rather than latent variables. The use of measured variables means that some error variance remained in our measurement. However, the reliability estimates of each measure used were quite high, indicating little measurement error. Sixth, although we tested predictions of temporal relationships, we did not conduct a rigorous test of causal processes. We cannot infer from this study alone that fifth-grade depressive symptoms caused an increase in drinking 1 year later for EA and HA youth, nor can we infer that alcohol consumption in fifth grade caused an increase in depressive symptoms 1 year later for AA youth.

In summary, the current study provides the first evidence for reversed prospective prediction between depressive symptoms and alcohol consumption as a function of race. We found this difference during the important developmental transition into middle school. The presence of racial differences at this young age suggests the importance of considering race when evaluating risk processes in general. More broadly, successful prediction from depressive symptomatology to drinking for EA and HA youth, and from drinking to depressive symptomatology for AA youth, may prove important as researchers unravel the etiological processes relating drinking behavior and internalizing dysfunction.

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