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Dimensions of Adolescent Alcohol Involvement as Predictors of Young-Adult Major Depression*

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Abstract

Objective—Adolescent alcohol involvement may increase risk for young-adult depression; however, findings are mixed and important questions remain unanswered. Because alcohol involvement among teens is multidimensional, this study examined the extent to which four different adolescent alcohol dimensions (i.e., frequency of alcohol use, quantity of consumption, frequency of heavy episodic drinking, and frequency of problem use) were predictive of young-adult major depressive disorder (MDD).

Method—Participants in this prospective longitudinal study, which extended from age 11 to age 22, were 429 rural teens (including 222 girls) and their families. Self-reports of each dimension of adolescent alcohol involvement were obtained at ages 16 and 18. Depression diagnoses were obtained at age 22, using a structured interview. Analyses included adolescent depressed mood, measured via self-report at ages 16 and 18. Data were analyzed using confirmatory factor analysis and structural equation modeling.

Results—The multidimensional nature of adolescent alcohol involvement was best represented by a first-order problem-use factor and a second-order alcohol-intake factor comprised of quantity, frequency, and heavy drinking. After controlling for gender and depressed mood, adolescent problem use, but not alcohol intake, was a significant positive predictor of young-adult MDD.

Conclusions—Findings help clarify the link between alcohol involvement and depression and suggest that harm-reduction strategies may help prevent later mood disorders.

Alcohol involvement is common among teens, especially during late adolescence. In 2005, 75% of high school seniors reported having consumed alcohol at some point in their lifetimes (Johnston et al., 2006). Alcohol involvement is associated with a range of adverse outcomes. For instance, alcohol often plays a role in the three most common forms of mortality among youth: accidents (e.g., motor vehicle accidents), homicides, and suicides (Bonnie and O'Connell, 2003). The adverse effects of adolescent alcohol involvement can extend into early adulthood (Ellickson et al., 2003; Hill et al., 2000; Oesterle et al., 2004). Such involvement may have especially important consequences for mental health functioning during the young-adult years (Brook et al., 2002; Rohde et al., 2001). In particular, growing evidence suggests that alcohol may play a role in the onset and progression of a broad class of internalizing problems (Trim et al., 2007), including the development of clinical diagnoses of major depressive disorder (MDD; Brook et al., 2002).

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MDD is one of the most prevalent psychiatric conditions. In the United States, the lifetime and past-year prevalences of MDD among adults have been estimated to be 17% and 7%, respectively (Kessler et al., 2005a,b). *Depression*, which here refers to clinical diagnoses of MDD, is a serious public health concern. It increases risk for illness and health-compromising behaviors, including suicide (Fergusson and Woodward, 2002; Fombonne et al., 2001; Weissman et al., 1999). Depression also is associated with diminished psychosocial functioning, which can contribute to relationship difficulties (Fombonne et al., 2001; Weissman et al., 1999) and lost productivity in the workplace (Stansfeld et al., 1997).

Alcohol involvement and depression tend to co-occur (Dawson et al., 2005; Kessler et al., 2005b; Windle and Davies, 1999). Several possible explanations for the association between these outcomes have been proposed (Brook et al., 1998). It is possible that the association is spurious and that it is better explained by common predictors, such as shared familial factors (Coryell et al., 1992; Merikangas et al., 1985), that independently increase risk for both outcomes. Alternatively, one problem might influence the other. For example, individuals with preexisting subclinical depressive symptoms, who are known to be at risk for subsequent depression (Pine et al., 1999), may use alcohol to escape or cope with their depressive symptomatology (Johnson and Kaplan, 1990), according to the self-medication hypothesis (Hesselbrock and Hesselbrock, 1997). Instead, the adverse effects of alcohol consumption, which can impair brain development and disrupt neurocognitive functioning among adolescents (Tapert et al., 2004), may increase risk for depression either by altering the brain's natural reward system (Koob and Le Moal, 2001) or by disrupting psychosocial functioning in key developmental domains, such as school and family life (Baumrind and Moselle, 1985).

Growing evidence suggests that, if a causal relationship exists, it tends to flow from alcohol involvement to depression among young people (e.g., Brook et al., 2002; Hallfors et al., 2005). This evidence is consistent with a broader literature showing a stronger tendency for substance involvement to predict depression than for depression to predict substance involvement (e.g., Bovasso, 2001; Degenhardt et al., 2003; Goodman and Capitman, 2000; Wu and Anthony, 1999). Still, findings in this area of inquiry are equivocal. Some investigators examining the relationship between alcohol involvement and depression have reported null predictive effects (Chassin et al., 1999; Wells et al., 2004). Thus, important questions remain unanswered, and additional research is needed.

In particular, the aspects or dimensions of alcohol involvement that may be most salient in the prediction of depression are unknown. Alcohol involvement is multidimensional (Auerbach and Collins, 2006; Lewinsohn et al., 1996; Newcomb, 1992; Stein et al., 1988), including such aspects as frequency of use, quantity of consumption, frequency of heavy episodic drinking, and frequency of *problem use*, which refers to the experience of adverse consequences related to alcohol consumption (Stice et al., 1998). Different dimensions of alcohol involvement may have differential consequences for depression. Most investigators, however, have focused on only one alcohol dimension (e.g., a frequency measure) or have constructed measures that combine several alcohol dimensions into one scale (e.g., a quantity-frequency index).

A lack of consideration for the multidimensional nature of alcohol involvement may be a contributing factor to mixed findings in the literature. Simultaneously considering multiple alcohol dimensions and isolating those that play a role in the development of depression will enhance our understanding of possible pathways leading from specific aspects of alcohol involvement to depression. Furthermore, this enhanced understanding may aid in the development of effective prevention programs that target individuals who are elevated on key alcohol dimensions in an attempt to prevent the progression to depression.

This study was designed to investigate the effects of different dimensions of alcohol involvement in late adolescence on past-year MDD in early adulthood. Confirmatory factor analyses and structural equation modeling analyses of longitudinal data, collected from a sample of boys and girls followed from adolescence into early adulthood, were conducted to examine the structure of adolescent alcohol involvement and to test the extent to which different adolescent alcohol dimensions were related to young-adult depression. Specifically, it was hypothesized that adolescent frequency of alcohol use, quantity of consumption, frequency of heavy episodic drinking, and frequency of problem use would be independent but positively correlated factors.

Prior theory (e.g., Sadava, 1990) and research (Lewinsohn et al., 1996; Newcomb, 1992; Stice et al., 1998; Wills et al., 2002; Windle, 1996) have supported the distinction between alcohol consumption (e.g., frequency, quantity, and intensity of use) and problem drinking (i.e., alcohol use that produces adverse consequences). Thus, we also considered the possibility that frequency, quantity, and heavy episodic drinking could be indicators of a second-order alcohol-intake factor that is distinct from, but positively correlated with, problem use.

There is little theory to guide expectations about possible differential effects of adolescent alcohol dimensions on young-adult MDD. In general, studies suggest that both heavier alcohol involvement (Tapert et al., 2004; Windle and Davies, 1999) and problem use (Colder and Chassin, 1999) have more severe consequences for the healthy development of teenagers than more normative patterns of use. Therefore, these adolescent alcohol dimensions might emerge as particularly salient predictors of depression. Yet, because prior studies have not simultaneously considered multiple dimensions of alcohol involvement, the relative influence of these dimensions on MDD remains to be determined.

As a final consideration, alcohol involvement tends to be more prevalent among young men (Grant et al., 2004), whereas depression tends to be more prevalent among young women (Kessler et al., 2005a). Moreover, there may be gender differences in the links between alcohol involvement and depression (Poulin et al., 2005). Therefore, this study examined possible gender differences in the relationships under investigation.

Method

Data set

Analyses for this study were based on data collected as part of Project Family (Spath and Redmond, 2002), a longitudinal study of rural youth and their families. Some of these families were randomly assigned to participate in Preparing for the Drug Free Years (PDFY; now called Guiding Good Choices), a five-session universal parent-training intervention designed to prevent substance use. The PDFY program evaluation, which itself is part of a larger collaboration between investigators from the University of Washington and Iowa State University (see Spoth et al., 1998; Spoth et al., 2001), is described in greater detail in Kosterman et al. (2001). In the fall of 1993, a total of 883 families with sixth-grade students in rural communities of a midwestern state were invited to participate in the study. Of invited families, 49% ($N = 429$) agreed to participate and completed the baseline (Wave 1) assessment. Based on data collected from a prospective participation factor survey with a 90% response rate, comparisons between families that agreed and those that declined to participate in the study revealed minimal differences across a range of sociodemographic and psychosocial characteristics—providing support for the representativeness of the study sample (Spoth et al., 1997).

At the study's outset, the average age of the children was 11 years; the average age of the mothers and fathers was 37 years and 40 years, respectively. Fifty-two percent ($n = 222$) of

the target children were girls. Most families were dual parent in structure (83%, $n = 356$), and families had an average of three children when the study began. The majority of the mothers (61%) and fathers (58%) reported having some post-high-school education. In 1993, the median annual household income was \$32,000. As a reflection of the study region, most of the participants were white (>95%).

With gender measured at Wave 1, current analyses were based on Wave 5 (age 16) and Wave 6 (age 18) assessments of alcohol involvement. Certain alcohol dimensions were not measured in the earlier waves. As the adolescents aged, the decision was made to include a richer assessment of alcohol involvement. This richer assessment corresponded with the adolescents' increasing exposure to and experience with alcohol. In addition, because many psychiatric disorders develop in early adulthood, the participants were asked to complete a structured diagnostic instrument as young adults. Thus, analyses also incorporated MDD measured at Wave 7 (age 22). Sixty-nine percent ($n = 295$) of the teens participated in Wave 5, and 71% ($n = 305$) of the teens participated in Wave 6. In early adulthood, 73% ($n = 313$) of the target respondents participated at an average age of 21.56 years. Extensive attrition analyses have been conducted to compare assessment dropouts versus completers across a range of sociodemographic characteristics and psychosocial variables. Minimal differences have been revealed in these analyses. Evidence has been consistent, however, that more highly educated parents were more likely to stay in the study than less highly educated parents (Spath et al., 1998). Attrition analyses conducted on the measures of alcohol involvement and depression in the current study showed no significant differences between dropouts and completers.

Procedure

The families received information describing the assessments and the program components of the prevention trial and a packet of initial questionnaires to be completed individually by the parent(s) and the target children. The families then scheduled an in-home visit to complete additional assessments. The participants were compensated \$10/hour for their study involvement. Approximately 9 months later, similar procedures were used to collect follow-up information (Wave 2). Such procedures also were used to collect data at follow-up assessments roughly 21 months (Wave 3), 33 months (Wave 4), 51 months (Wave 5), and 75 months (Wave 6) after the baseline assessment, when students were in the 7th, 8th, 10th, and 12th grades, respectively. Current analyses focused on the 10th-grade (age 16) and 12th-grade (age 18) assessments. At each assessment, informed consent was obtained from the parents, and assent was obtained from the teens. All participants were assured that their responses would remain confidential. In the fall of 2004, computer-assisted telephone-interviewing procedures were used to collect information from the target respondents in early adulthood. The study procedures were approved by the Human Subjects Review Committees at Iowa State University and the University of Washington.

Measures

Adolescent alcohol involvement—The four dimensions of adolescent alcohol involvement were measured at ages 16 and 18. Specifically, to assess frequency of alcohol use, the teenage participants were asked to indicate how many times they had consumed beer, wine, wine coolers, or distilled spirits within the past month. The adolescents indicated their quantity of alcohol consumption by responding to the question “About how much (if at all) do you usually drink each time you drink?” on the following 5-point scale: (0) “I don’t drink alcohol,” (1) “one drink,” (2) “two drinks,” (3) “three or four drinks,” (4) “five or six drinks,” and (5) “more than six drinks.” The frequency of heavy episodic drinking was measured by asking teens to answer the question “During the past month, how many times have you had three or more drinks (beer, wine, or other liquor) in a row?” Finally, an assessment of problem use of alcohol (and other drugs) was created by averaging participants’ responses to four

questionnaire items that asked the teens to indicate how often their use of “alcohol, marijuana, or other drugs” caused them to behave in ways that they later regretted or hurt relationships with their parents, friends, and teachers ($\alpha = .84$). Response options were (0) “never,” (1) “rarely,” (2) “sometimes,” and (3) “very often.” Test-retest correlations for the alcohol measures across the 2-year interval from age 16 to age 18 were statistically significant and medium to large in magnitude, as reported in Table 1.

Descriptive frequency analyses indicated that 32% ($n = 94$) of the 295 teens who completed the 10th-grade assessment reported drinking alcohol at least once during the past month at age 16, and 50% ($n = 154$) of the 305 teens who completed the 12th-grade assessment reported drinking alcohol at least once during the past month at age 18. As indicated in Table 1, the respondents reported consuming an average of 1.46 drinks at age 16 and 2.33 drinks at age 18. Further analyses showed that 24% ($n = 71$) of the participants reported heavy episodic drinking at least once during the past month at age 16, and 41% ($n = 126$) reported heavy episodic drinking at least once during the past month at age 18. At age 16, 17% ($n = 51$) of the participating teens reported at least one problem-use symptom; at age 18, 29% ($n = 88$) of the participating teens reported at least one problem-use symptom.

Research has supported the validity and reliability of adolescent self-reports of alcohol involvement (Smith et al., 1995; Winters et al., 1990), and the alcohol measures used in this study are similar to those that have been used in other research projects (Elliott et al., 1989; Windle, 1996). Moreover, although our problem-drinking measure includes the possibility that adolescents could report problems with marijuana and other drugs, the use of illicit drugs was rare in this community sample of rural adolescents. For example, at age 18, only 30 teens reported having used marijuana, which was the most commonly used illicit drug, in the past year. Among those marijuana users, 18 reported using it five or fewer times. Alcohol was the most commonly used substance among the adolescent boys and girls in this sample. Moreover, additional analyses indicated that young-adult depression rates did not differ between past-year marijuana users and nonusers.

Adolescent depressed mood—Analyses included a control for adolescent depressed mood, which was measured with eight items from the Child Behavior Checklist–Youth Self-Report (Achenbach, 1991). Depressed mood is the most common depressive symptom (Lewinsohn et al., 1998), and it is a risk factor for subsequent depression (Devine et al., 1994). Participants were asked to indicate whether statements describing various feelings and behaviors were (0) “not true,” (1) “somewhat or sometimes true,” or (2) “very or often true” for them at any time within the preceding 6 months. Sample statements include “I feel worthless or inferior” and “I am unhappy, sad, or depressed.” Depressed-mood scales at age 16 ($\alpha = .85$) and age 18 ($\alpha = .84$) were computed as the average response to all items.

Young-adult MDD—MDD at age 22 was measured using a short form of the Diagnostic Interview Schedule (DIS; Robins et al., 1989). Responses to the interview questions were incorporated into a computer algorithm that was created by the current investigative team, with reference to the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV; American Psychiatric Association, 1994), to delineate those who met criteria for an MDD in the past 12 months. Thus, MDD was a dichotomous variable that was coded 1 for those who met criteria for the disorder in the past year and 0 for those who did not meet criteria for the disorder in the past year. The DIS is a commonly used instrument that has been shown to be reliable and valid (Leaf et al., 1991; Newman et al., 1996; Reinherz et al., 2000).

Gender—In the primary analyses, gender was included as a covariate. Gender was coded 1 for males and 0 for females. Correlations, means, and standard deviations for the study variables are reported in Table 1.

Data analyses

The data were analyzed with confirmatory factor analysis (CFA) and structural equation modeling (SEM) using Mplus 4.2 (Muthén and Muthén, 2006). Weighted least squares parameter estimates with mean- and variance-adjusted chi-square statistics were generated using the WLSMV estimator, given the categorical nature of the outcome and certain alcohol indicators. In addition, analyses incorporated advanced estimation procedures to account for missing data (Schafer and Graham, 2002)—resulting in a full analysis sample of 429 participants for the primary CFA and SEM analyses.

Some of the participants were involved in the substance-use preventive intervention described above during the early years of the project. To determine the appropriateness of the data for conducting covariance structure analyses to examine etiologic processes, a multiple-group model was estimated that constrained to equality across the control and intervention conditions all 66 covariances among the 12 observed variables. Results from this conservative test showed that the fit of the fully constrained model was not significantly worse than that of the completely unconstrained model ($\chi^2 = 74.61$, 66 df, $p = .22$; $N = 429$), indicating that the associations among variables were the same across conditions. Thus, the two groups were pooled for analysis. In addition, a program variable indexing intervention versus control status was not significantly associated with the variables in the current analyses, and supplemental analyses that included the program variable as a covariate resulted in the same findings as those reported below.

Results

Descriptive analyses indicated that 8% ($n = 24$) of the young-adult respondents reported meeting criteria for MDD. Although MDD was more common among young women (9.5%) than among young men (5.5%), the difference was not statistically significant ($\chi^2 = 1.77$, 1 df, $p > .05$; $n = 313$). Nonetheless, it is important to consider possible gender differences in the basic associations among variables as a preliminary step in model testing. To test for differences, a multiple-group analysis was conducted across gender groups in which all 55 covariances across the 11 observed variables (gender was not included as a variable) were constrained to equality for young men and young women. Results from this conservative test showed that the fit of the fully constrained model was acceptable, with only a marginally significant chi-square value ($\chi^2 = 73.91$, 55 df, $p = .05$; $n = 352$; Comparative Fit Index [CFI] = .98; Root Mean Square Error of Approximation [RMSEA] = .044). This finding demonstrates a high degree of correspondence between the basic covariances among variables for males and females. Thus, subsequent analyses were conducted on the total sample. Because gender was significantly associated with several variables in this study, as illustrated in Table 1, it was included as a covariate in the CFA and SEM analyses.

Confirmatory factor analyses

As a first step, confirmatory factor analyses were conducted to test the multidimensionality of adolescent alcohol involvement and to examine relationships among adolescent alcohol dimensions, adolescent depressed mood, young-adult major depression, and gender. Preliminary analyses, which are not reported because of space constraints, showed that the fit of a basic one-factor model of adolescent alcohol involvement was significantly worse than that of a four-factor model that included all dimensions as separate but correlated factors. For the present purposes, we began by specifying a model that estimated all covariances among five first-order latent variables (i.e., adolescent alcohol frequency, alcohol quantity, frequency of heavy episodic drinking, and problem use, as well as adolescent depressed mood), the manifest MDD outcome variable, and the manifest gender covariate. Latent variables were indicated by the age 16 and age 18 measures of each construct.

For example, alcohol frequency at age 16 and at age 18 was specified as indicators of a latent alcohol frequency construct. To identify the model, the latent variable variances were fixed at 1.0, and the two factor loadings for each construct were estimated but constrained to equality. In addition to estimating all covariances among latent variables and the MDD outcome variable, all within-time covariances among the residuals of the latent variable indicators were freely estimated (e.g., the residual of the age 16 alcohol frequency indicator with the residuals of the age 16 alcohol quantity, heavy episodic drinking, problem use, and depressed mood indicators).

Results showed that the fit between the data and the initial first-order factor model was acceptable ($\chi^2 = 20.88$, 11 df, $p < .05$; $N = 429$; CFI = .99; RMSEA = .046). To conserve space, detailed results from this model are not reported but are available on request. All factor loadings were positive and statistically significant, with standardized values ranging from .73 to .80. Correlations among the latent variables, the outcome, and the covariate in this model are reported in Table 2. As expected, correlations among the alcohol-involvement dimensions were high. In particular, correlations among the latent alcohol frequency, quantity, and frequency of heavy episodic drinking variables ranged from .87 to .96. Correlations of those factors with the latent problem-use variable were somewhat lower, ranging from .70 to .74. These findings suggest that a second-order model, in which frequency, quantity, and heavy episodic drinking serve as indicators of a higher order alcohol intake factor, might be tenable.

Accordingly, we specified an alternative second-order factor model, comparing its fit with that of the initial first-order factor model. To identify this alternative model, the constraints on the variances and factor loadings of the frequency, quantity, and heavy episodic drinking factors were released, and these latent variables served as indicators of a second-order alcohol-intake factor. The factor loading for alcohol frequency was fixed at unity to serve as a reference indicator, and the remaining two factor loadings were freely estimated. The variance of the alcohol-intake factor was freely estimated. The first-order problem-use and depressed-mood factors were parameterized in the same manner as described previously. All covariances among the second-order alcohol-intake factor, the first-order problem-use and depressed-mood factors, the MDD outcome variable, and the gender covariate were freely estimated, as were all within-time covariances among uniquenesses of the first-order latent-variable indicators.

The fit between the data and the second-order factor model was acceptable ($\chi^2 = 28.77$, 13 df, $p < .05$; $N = 429$; CFI = .99; RMSEA = .053). Moreover, results from a chi-square difference test showed that the fit of the second-order factor model was not significantly worse than that of the first-order factor model ($\chi^2 = 4.76$, 5 df, $p > .05$; $N = 429$). Because the second-order factor model is more restrictive and can fit only as well as or significantly worse than the first-order factor model, results from the chi-square difference test indicate that the second-order model is to be preferred as the most parsimonious model that demonstrates acceptable fit. It should be noted that, to obtain an accurate p value, chi-square statistics and degrees of freedom are mean- and variance-adjusted under WLSMV estimation. Therefore, calculation of the difference test is not straight-forward, as it is under maximum likelihood estimation. For this reason, difference tests were computed using the *diffest* option in Mplus 4.2.

Again, all first-order factor loadings were positive and statistically significant in the model, with standardized values ranging from .68 to .80. Factor loadings for the alcohol-intake factor were particularly high, ranging from .94 to .99. Correlations among the latent variables, the outcome, and the covariate in this model are reported in Table 3. As expected, there were positive and statistically significant correlations between alcohol intake and problem use, as well as between depressed mood and MDD. Interestingly, although alcohol intake was significantly associated with adolescent depressed mood, it was not associated with young-adult MDD. Additionally, although problem use and depressed mood were not associated with

one another in adolescence, problem use had a statistically significant positive association with MDD in early adulthood.

Structural equation modeling analyses

As a next step, the second-order CFA was re-parameterized by regressing young-adult MDD on adolescent alcohol intake, problem use, depressed mood, and gender to examine predictive effects on the outcome. The fit between the data and this initial prediction model was acceptable ($\chi^2 = 29.42$, 13 df, $p < .01$; $N = 429$; CFI = .99; RMSEA = .054). Consistent with results from the CFA, the longitudinal relationships between adolescent alcohol intake as well as gender and young-adult MDD were statistically nonsignificant. Thus, these nonsignificant paths were trimmed from the model to obtain a final parsimonious prediction model, which displayed acceptable fit statistics ($\chi^2 = 31.56$, 14 df, $p < .05$; $N = 429$; CFI = .99; RMSEA = .054).

Note that, because of the use of WLSMV estimation, degrees of freedom for the more parsimonious model increased by only one, in comparison with the more fully specified model, even though the former model included constraints on two structural paths. Results from this final model are depicted in Figure 1 (standardized estimates are displayed). Over and above the effect of earlier depressed mood on the outcome, adolescent problem use positively predicted young-adult MDD. Taken together, the predictors accounted for an estimated 29% of the variance in MDD.

Discussion

Analyses confirmed the multidimensionality of adolescent alcohol involvement. Results showed that alcohol involvement in this sample of rural teens was best conceptualized as two distinct but related dimensions, including a second-order alcohol-intake dimension (indicated by first-order alcohol frequency, alcohol quantity, and frequency of heavy episodic drinking factors) and a first-order problem-use dimension. This finding is consistent with prior research that has distinguished between *alcohol use*, defined as level of consumption, and *problem use*, defined as use leading to adverse consequences (Lewinsohn et al., 1996; Newcomb, 1992; Stice et al., 1998; Wills et al., 2002; Windle, 1996).

Results also indicated that adolescent alcohol dimensions were differentially related to young-adult MDD. Specifically, problem use positively predicted MDD, over and above the effect of adolescent depressed mood on the outcome. Although prior research has demonstrated a link between alcohol and depression (Brook et al., 2002; Dawson et al., 2005; Kessler et al., 2005b; Windle and Davies, 1999), this study provides a key contribution by identifying a particular dimension of alcohol involvement in adolescence that has a unique impact on risk for MDD in early adulthood. It is interesting that alcohol intake was unrelated to MDD; even the zero-order correlation between these constructs was nonsignificant. Alcohol consumption, including heavy episodic drinking, is relatively prevalent in late adolescence and, therefore, is somewhat normative (Johnston et al., 2006). Thus, the level of adolescent alcohol use, per se, may have little direct consequence for the development of depression in early adulthood. Instead, our results suggest that the experience of adverse use-related consequences increases risk for young-adult MDD.

Of course, adolescent alcohol use, regardless of how normative it might be in a given context, is not benign. Research has documented numerous deleterious effects of adolescent alcohol consumption (Ellickson et al., 2003; Hill et al., 2000; Oesterle et al., 2004). In this study, although alcohol intake did not predict later MDD, it was associated with elevated depressed mood in adolescence. It is possible that the effect of adolescent alcohol use on young-adult MDD is indirect, operating through elevated depressed mood in the teen years. Likewise, because a higher level of alcohol consumption increases the likelihood of experiencing adverse

use-related consequences (Newcomb, 1992; Stice et al., 1998), alcohol use may have an indirect effect on MDD through problem use. Indeed, alcohol intake was significantly correlated with problem use in the model.

An examination of these questions was beyond the scope of the current study, which adopted an analytic strategy tailored to examining the multidimensionality of alcohol involvement and differential prediction to young-adult MDD. Thus, additional research is needed to investigate possible mechanisms linking alcohol with depression, adopting an analytic strategy suited for testing mediational hypotheses (e.g., testing the temporal ordering from early alcohol intake to later depressed mood to later MDD). Likewise, because links between alcohol and depression may vary, depending on when during adolescence and adulthood these problems are measured, there is a need for longitudinal research that tests for developmental variations in predictive relations.

Interestingly, problem use was not associated with depressed mood in adolescence, even though it predicted MDD in young adulthood. As one explanation for this pattern of findings, the negative consequences of drinking may accumulate over the transition to early adulthood. Thus, they may compromise the successful completion of normative developmental tasks related to education, career, and romantic relationships, thereby promoting the development of depression (cf. Capaldi and Stoolmiller, 1999; Patterson and Capaldi, 1990). Alternatively, problem use may simply have a stronger relationship with clinical depression, as an indicator of more severe dysfunction, than with subclinical depressed mood (Graham et al., 2007). These alternative hypotheses should be investigated in future research.

In general, there were expected mean differences in the constructs across gender. For example, adolescent depressed mood was more common among girls than among boys. Likewise, young-adult MDD was more prevalent among women than among men, although this difference was not quite statistically significant. Male gender was positively correlated with certain indicators of alcohol involvement, including quantity of consumption and frequency of heavy episodic drinking at age 18.

Still, we found very little evidence for gender differences in the strength of associations among variables. Differences across boys and girls in the mean levels and prevalences of indicators of alcohol involvement and depression may be, in part, the result of gender-specific socialization processes. For example, typically boys are socialized to be more risk taking (Hagan et al., 1985). They often experience less monitoring and supervision (Steinberg et al., 1994), compared with girls—which may explain their tendency to be more elevated on externalizing problems, relative to internalizing problems. Despite these differences, the fundamental processes linking alcohol involvement and depression may be similar across gender groups. Yet, further research with additional samples of boys and girls followed from adolescence into young adulthood is needed before firm conclusions can be drawn.

In sum, whereas the level of alcohol intake was related to increased problem use, intake in itself did not predict MDD in the current analyses. This finding suggests that the key dimension in the link between alcohol involvement and depression may be the problems that result from alcohol use, such as regretting one's behavior and damaging one's relationships with significant others. Those who managed to drink without experiencing a high degree of these types of problems were not at increased risk for MDD. Risks are likely to remain, however, for other adverse consequences, such as criminality, school problems, and poor physical health (Ellickson et al., 2003; Hill et al., 2000; Oesterle et al., 2004). To test the degree to which these results hold when accounting for common predictors, such as shared familial risks for alcohol and depression (e.g., Coryell et al., 1992; Merikangas et al., 1985), further studies are needed.

The findings of this study may have implications for preventive intervention. For instance, because adolescent alcohol involvement is relatively prevalent, it might be beneficial to supplement existing universal prevention programs with programs that screen young boys and girls for signs of problem use, directing more targeted services toward those individuals who are in greatest need. In this regard, the current findings are consistent with harm-reduction strategies that seek not so much to encourage abstinence from alcohol as to limit the adverse consequences that can come from drinking (Marlatt and Witkiewitz, 2002).

In addition, prevention scientists might be able to take advantage of an opportunity to work toward reducing the incidence and prevalence of MDD among young adults by incorporating depression modules into existing adolescent alcohol- (and other substance-use) prevention curricula. Reducing adolescent problem alcohol use may not only limit the immediate consequences of such use, but may also provide a way to interrupt one potentially important pathway leading toward the development of MDD. It should be noted, however, that these prevention implications are tentative, because our analyses were unable to address predictors of the onset of initial MDD diagnosis.

Strengths of this study include the prospective longitudinal design, multidimensional assessment of adolescent alcohol involvement, diagnostic assessment of depression, and latent-variable analysis strategy. A primary limitation concerns the nature of the sample, which was predominantly white—a reflection of the rural midwestern region in which the study was conducted. Yet, use rates in this sample are comparable to national statistics. The extent to which the results might generalize to minority youth, as well as to boys and girls living in urban and suburban settings, is unknown. Attrition is another concern in this long-term longitudinal study. Accordingly, we conducted extensive attrition analyses and adopted an advanced strategy for handling missing data.

As mentioned previously, an additional limitation relates to the measurement of problem use in this study, which includes the possibility that some problems were the result of the use of marijuana and other drugs. Rates of marijuana use and other illicit drug use were low in this rural sample. Response biases, however, may have contributed to an underestimate of the rates of illicit drug use. Such underestimation would be a concern, because experiencing problem drinking in conjunction with problem illicit drug use may be a particularly potent predictor of depression. Future studies would benefit from more narrowly defined assessments of problem drinking and problem drug use.

Another important limitation is that, in our assessment of MDD, we were unable to account for the possibility that certain individuals may have experienced depression that was induced temporarily by alcohol intoxication or withdrawal (Schuckit, 2006). Future studies of the link between alcohol involvement and depression also would benefit from a more detailed level of assessment that can better discern the specific contexts and conditions under which diagnostic criteria are met. As a final consideration, all of the variables in the current analyses were based on adolescent self-reports. The findings may have been biased by an over-reliance on a single rater and a single assessment strategy.

Despite these limitations, this study provided a unique opportunity to identify the dimensions of adolescent alcohol involvement that are most salient in predicting young-adult depression. In this regard, adolescent problem use had a unique positive effect on young-adult MDD, over and above the effect of adolescent depressed mood on the outcome. Additional longitudinal analyses, such as those that test for mediators of the link between earlier alcohol use and later depression, promise to extend the current analyses. In the meantime, our results have implications for targeting youth in greatest need of intervention services and for interrupting a potentially important pathway leading to depression in early adulthood.

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FIGURE 1.

Final model examining adolescent alcohol dimensions as predictors of young adult major depression. (1) = reference indicator. Paths labeled with a common letter in parentheses indicate that the unstandardized estimates for these parameters were constrained to equality. Estimated time-specific correlations among the uniquenesses of the alcohol and depressed mood indicators are not reported but are available on request. Freq = frequency; quan = quantity; hvy = heavy; prob = problem; dep = depression; ALC = alcohol.

* $p < .05$; [†] $p < .01$; [‡] $p < .001$.

TABLE 1

Correlations, means, and standard deviations for the study variables

Variable	1	2	3	4	5	6	7	8	9	10	11	12
1. Gender, male												
2. Depressed mood, age 16	-.25 [‡]											
3. Depressed mood, age 18	-.24 [‡]	.61 [‡]										
4. Alcohol quantity, age 16	-.06	.11 [*]	.10									
5. Alcohol quantity, age 18	.14 [*]	.09	.10	.53 [‡]								
6. Alcohol frequency, age 16	-.03	.19 [†]	.22 [‡]	.63 [‡]	.43 [‡]							
7. Alcohol frequency, age 18	.08	.06	.07	.46 [‡]	.69 [‡]	.41 [‡]						
8. Heavy drinking, age 16	-.01	.11	.14 [*]	.64 [‡]	.38 [‡]	.78 [‡]	.37 [‡]					
9. Heavy drinking, age 18	.13 [*]	.01	.05	.45 [‡]	.66 [‡]	.43 [‡]	.82 [‡]	.42 [‡]				
10. Problem use, age 16	-.07	.08	-.02	.53 [‡]	.23 [‡]	.34 [‡]	.28 [‡]	.40 [‡]	.26 [‡]			
11. Problem use, age 18	.03	.11	.14 [*]	.42 [‡]	.43 [‡]	.30 [‡]	.34 [‡]	.29 [‡]	.36 [‡]	.44 [‡]		
12. MDD, age 22	-.08	.20 [†]	.15 [*]	.21 [†]	.06	.08	.03	.13 [*]	.00	.31 [‡]	.14 [*]	
Mean	0.48	0.29	0.27	1.46	2.33	0.44	0.78	0.31	0.65	0.27	0.44	0.08
SD	0.50	0.36	0.34	1.79	1.95	0.70	0.85	0.60	0.84	0.63	0.74	0.27

Note: MDD = major depressive disorder.

*
 $p < .05$;†
 $p < .01$;‡
 $p < .001$.

TABLE 2

Correlations among gender, first-order alcohol dimensions, depressed mood, and major depressive disorder

Variable	1	2	3	4	5	6
1. Gender, male						
2. Adolescent alcohol frequency	.04					
3. Adolescent alcohol quantity	.05	.93 [‡]				
4. Adolescent frequency of heavy episodic drinking	.10	.96 [‡]	.87 [‡]			
5. Adolescent problem use	-.03	.74 [‡]	.70 [‡]	.70 [‡]		
6. Adolescent depressed mood	-.31 [‡]	.29 [†]	.16	.16	.11	
7. Young adult major depressive disorder	-.08	.08	.19 [†]	.10	.27 [†]	.22 [‡]

†
 $p < .01$;‡
 $p < .001$.

TABLE 3

Correlations among gender, second-order alcohol intake, depressed mood, and major depressive disorder

Variable	1	2	3	4
1. Gender, male				
2. Adolescent alcohol intake (second-order factor)	.06			
3. Adolescent problem use	-.03	.73 [‡]		
4. Adolescent depressed mood	-.31 [‡]	.21 [*]	.11	
5. Young adult major depressive disorder	-.08	.13	.27 [†]	.22 [‡]

*
 $p < .05$;†
 $p < .01$;‡
 $p < .001$.