Depressed Mood in Childhood and Subsequent Alcohol Use Through Adolescence and Young Adulthood

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Context: Despite prior evidence supporting cross-sectional associations of depression and alcohol use disorders, there is relatively little prospective data on the temporal association between depressed mood and maladaptive drinking, particularly across extended intervals.

Objective: To assess the association between depressed mood in childhood and alcohol use during adolescence and young adulthood by mood level and sex and race/ethnicity subgroups.

Design: Cohort study of individuals observed during late childhood, early adolescence, and young adulthood.


Participants: Two successive cohorts of students from 19 elementary schools have been followed up since entry into first grade (1985, cohort I [n=1196]; 1986, cohort II [n=1115]). The students were roughly equally divided by sex (48% female) and were predominantly African American (70%). Between 1989 and 1994, annual assessments were performed on students remaining in the public school system, and between 2000 and 2001, approximately 75% participated in an interview at young adulthood (n=1692).

Main Outcome Measures: Among participants who reported having used alcohol, Cox and multinomial regression analyses were used to assess the association of childhood mood level, as measured by a depression symptom screener, with each alcohol outcome (incident alcohol intoxication, incident alcohol-related problems, and DSM-IV alcohol abuse and dependence).

Results: In adjusted regression analyses among those who drank alcohol, a high level of childhood depressed mood was associated with an earlier onset and increased risk of alcohol intoxication, alcohol-related problems during late childhood and early adolescence, and development of DSM-IV alcohol dependence in young adulthood.

Conclusions: Early manifestations associated with possible depressive conditions in childhood helped predict and account for subsequent alcohol involvement extending across life stages from childhood through young adulthood.

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tend across life stages. Using a birth cohort of youth from New Zealand, Fergusson and Woodward assessed outcomes during young adulthood associated with adolescent depression and found that the association with alcohol use disorders was explained by social, personal, and familial characteristics. Rao and colleagues observed a sample of female adolescents into early adulthood and reported that substance use disorders predicted depression, but not the reverse.

Psychological theories used to help explain these associations include the general concept of using alcohol for self-medicating depressive symptoms or general tension reduction and the stress-response dampening model. Social learning theory might explain roles played by alcohol expectancies, coping skills, and self-efficacy in the regulation of drinking, especially in the presence of stress or mood symptoms. Life-course developmental models suggest that both social adaptational status (the interaction between social task demands and how one's responses are judged) and psychological well-being form a foundation for mental health and predict subsequent behavioral patterns, such as alcohol consumption. Furthermore, there may be common underlying genetic as well as social or environmental factors that are associated with increased risk of both disorders. Family history of alcoholism as well as depression may be influential. Other conditions may explain familial associations. In a male twin sample, evidence indicated that antisocial personality disorder may account for the genetic association found between depression and alcohol dependence. Chassin et al found that externalizing but not internalizing symptoms mediated the effects of a parental history of alcohol disorders.

In the current study, we used data gathered from 2 cohorts originally assessed in the first grade and subsequently followed up through adolescence and young adulthood. Prior analyses of these cohorts have shown that depressed mood in childhood is associated with alcohol initiation without parental permission. Our objective here was to extend these findings among alcohol drinkers to improve our understanding of the temporal association of depressed mood with subsequent risk associated with several alcohol outcomes. Using a depression symptom screener, we examined varying levels of nondiagnostic depressed mood to assess whether alcohol involvement might covary with these levels. Specifically, we hypothesized that any level of depressed mood in childhood would be associated with an increased risk of alcohol intoxication and the development of alcohol-related problems during adolescence and that this association would extend into young adulthood, possibly accounting for the occurrence of alcohol abuse or dependence. Because prior evidence indicates that these associations may differ for some subgroups, we completed separate analyses by sex and race/ethnicity.

**METHODS**

**STUDY POPULATION**

The data for these analyses were derived from a prospective study, which began in 1985-1986, conducted within the context of a randomized trial of 2 primary school interventions directed toward academic achievement and classroom behavior. A total of 2311 students were sampled during 2 successive school years in first grade classrooms of 19 elementary schools in an urban mid-Atlantic region of the United States (1985, cohort I [n=1196]; 1986, cohort II [n=1115]). Parental consent was obtained from 95% of the original cohort. Additional details on the study design and data collection are available elsewhere.

Between 1989 and 1994, trained interviewers administered face-to-face assessments annually in private locations at each school. Interviewers were young adults of both sexes from predominantly minority racial/ethnic backgrounds, similar to the racial/ethnic composition of the study sample. The interviews conducted in 1990 are considered the baseline interviews for this current study, because 1990 was the first year that depressed mood was assessed by face-to-face interviews. Of this baseline sample, 92.8% were interviewed again in 1991 (90.7% of cohort I and 94.9% of cohort II), 84.7% were interviewed again in 1992 (83.1% of cohort I and 86.3% of cohort II), and 75.7% were interviewed again in 1993 (74.6% of cohort I and 76.7% of cohort II). In 1994, limited resources were focused on following up youths in cohort II so that they would be followed up to a similar mean age as the first cohort (30.7% of cohort I and 69.0% of cohort II). A total of 77.0% of the baseline sample had at least 3 years of follow-up between 1991 and 1994. Child assent was obtained at the time of each interview. A certificate of confidentiality was secured, and the study was approved by the Johns Hopkins Bloomberg School of Public Health Committee on Human Research.

Funding for follow-up was not available during the high school years of these 2 cohorts. Nevertheless, it was possible to start the follow-up and tracing processes again so that between 2000 and 2001, approximately 75% of the pupils surviving from the original sample of 2311 participants, including 134 incarcerated individuals, could be traced and interviewed in face-to-face assessments in young adulthood (n=1692). An additional 12% were successfully located but either chose not to be interviewed (n=142) or were not available (eg, they lived out of state with no telephone number or had military postings overseas [n=133]). The mean age for young adulthood follow-up was 21.0 years.

We assessed individuals at 2 separate life stages, defined here by length of follow-up (Figure 1): (1) during late childhood/early adolescence (age range, 10-16 years [n=1392]) and (2) during young adulthood (age range, 19-24 years [n=1920]). The analysis for the late childhood/early adolescence follow-up involved exclusion of 919 youths from the original list of 2311 students enrolled in first grade (526 youths who did not have ≥1 interviews subsequent to 1990 as well as 393 youths who had not initiated alcohol use and therefore were not at risk of transitioning to the alcohol outcomes). The analyses for the young adulthood follow-up included all the individuals who reported alcohol use (1392 individuals who reported alcohol use during late childhood/early adolescence and an additional 328 individuals who reported ever having used alcohol when interviewed during young adulthood). Excluded from the young adulthood analyses were the 61 individuals who never used alcohol (as reported on the young adulthood interview), 245 individuals who were not followed up either during adolescence or young adulthood, and 85 individuals who had not begun drinking at their last late childhood/early adolescence contact. Participants with missing follow-up information were more likely to be white and to have paid for lunch as opposed to receiving reduced or free lunch during first grade. In the young adulthood assessment, men were also more likely to be missing than women (32% vs 23%).

**MEASURES**

Four facets of depressed mood were assessed, based on 4 questions that asked about being in a bad mood, feeling sad, feel-
ing crabby or cranky, and feeling like crying "all the time or almost all of the time." Four items quizzed whether each feeling had ever occurred every day or almost every day for 2 weeks or for the prior 2 weeks if the mood problem was described as current.\(^a\)\(^b\)\(^c\)\(^d\) We refer to a high level of depressed mood only if (1) at least 3 of the 4 feelings had been experienced all of the time or almost all of the time or (2) at least 2 of the 4 feelings were described as being current (within the prior 2 weeks) as well as having occurred for at least 2 weeks in the past. Low depressed mood refers to mood in youths who (1) had expressed no more than 1 of the feelings at any time or (2) had experienced 2 of the 4 feelings (current or in the past but not both). The reference category for the high- and low-level depressed mood classifications were youth who did not report any of these mood features at the time of the 1990 assessment. Our reason for the mood categories was to distinguish those with multiple features or evidence for chronicity in the high-level group from those in the low-level group with only 1 feeling and little evidence of persistence of depressed mood. We hypothesized that those with greater burdens of depressed mood, as evidenced by the greater number of features and recurrence or persistence of these feelings, would be associated with the highest risk for alcohol involvement. This measure of depressed mood has adequate internal consistency (Cronbach \(\alpha = 0.7\)) and predictive validity with respect to an adapted version of the Composite International Diagnostic Interview, University of Michigan version (CIDI-UM),\(^6\) module of major depression (relative to those without depressed mood by the time of the 1990 interview, high-depressed mood level is associated with CIDI-UM major depression assessed during the 1993-1994 interview [odds ratio, 3.17; 95% confidence interval, 1.68-5.95; \(P < .001\)]. However, the measure is a screener questionnaire and is not intended to be used to diagnose depression. Furthermore, the features measured may indicate a range of psychopathology.

Information on alcohol use was based on responses to the question asked each year (1989-1994) regarding whether the student had "ever drunk beer, wine, wine coolers, or any other drink with alcohol in it, even just a sip or taste." Alcohol intoxication was assessed yearly by the question, "How old were you the first time you got drunk or high on alcohol?" Individuals with intoxication before or at the 1990 interview were excluded to assess new-onset intoxication. Data on the occurrence and age at onset of alcohol-related problems were also assessed through 1994. The alcohol-related problem construct was represented by 2 groups of indicators: (1) having social difficulties because of drinking, such as trouble in school, at home, or with the police, or (2) having health or physical problems related to using alcohol, such as a "sick stomach," feeling "panicky," or feeling a "hunger to use" alcohol. Individuals with alcohol-related problems before or at baseline were removed to focus this inquiry on incident problem drinking.

The interview assessment for the DSM-IV\(^a\)\(^b\) classifications of lifetime alcohol abuse and dependence in the young adulthood follow-up was an amended version of the approach used in the United States National Comorbidity Survey Replication and in the World Mental Health Surveys Initiative. The amendment involved the addition of this global alcohol problems screening question: "Was there ever a time in your life when people told you they thought you drank too much or when you thought you might have a drinking problem?" This was added in an effort to increase sensitivity for clinically significant alcohol dependence. Reed et al\(^5\) provide more detail on this approach (Web-only Appendix available at http://www.archgenpsychiatry.com). Because of the relatively short developmental interval for these new dependence cases (during late adolescence to young adulthood), we included the entire group of individuals who met 3 or more dependence criteria in our definition (70.5% also self-reported 12-month symptom clustering). When we excluded those without self-reported clustering, substantive conclusions were unchanged.

Covariates included in the analyses were sex and race/ethnicity (where applicable in stratified analyses, African American individuals vs all other races/ethnicities [\(\geq 90\%\) white]), caregiver history of heavy drinking and/or illegal drug use (yes=1, no=0), youth report of peer alcohol use (yes=1, no=0), age at baseline (continuous variable), level of conduct problems, and neighborhood disadvantage. Caregiver history of heavy drinking and/or illegal drug use was self-reported by the adult participants. Peer drinking was assessed with 1 question asking whether participants had any friends who drank alcohol. The presence of conduct problems was assessed using the 11-item Baltimore Conduct Problems and Delinquency Scale, which assesses delinquent and antisocial behavior. This measure was previously described\(^6\) and is an adaptation of a self-report measure developed by Elliott et al\(^8\) for the National Survey of Delinquency and Drug Use. In our work, the number of items was increased to provide full coverage of the DSM criteria for conduct disorder. Responses from the 4-point Likert scale (not at all to \(\geq 3\) times in the prior year) were summed, and high and low categories were characterized (above and below the median scale score for this sample). Psychometric analyses suggest good to excellent

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**Figure 1.** Flowchart of cohorts and sample used for data analyses. Data are from an epidemiologic sample originally recruited at entry into first grade in 1985-1986 from an urban mid-Atlantic public school system (N=2311).
STATISTICAL ANALYSIS

After exploratory data analyses, we completed initial and multiple regression models to assess the strength of the associations between depressed mood levels and the risk of each of the subsequent alcohol-related outcomes. Cox proportional hazard ratios were calculated for time to first alcohol-related problem and first episode of alcohol intoxication at the late childhood/early adolescence follow-up. Multinomial regression was used to examine the association of childhood depressed mood with the occurrence of DSM-IV alcohol use disorders at the young adulthood follow-up. In lieu of survival analysis with the young adulthood outcomes, we used multinomial regression because of a violation of the proportionality assumption of the Cox model for these outcomes, with too few cases to stratify by age groups. A single multinomial regression model was used, with the outcome having 4 levels: alcohol abuse, alcohol dependence, no alcohol disorder (reference category), and a category for those with missing information for the diagnostic criteria. For all regression models, stratified analyses were completed by sex and race/ethnicity.

Potential confounding was constrained in these analyses via statistical adjustment. To take into account various occurrences of missing information and to keep the representativeness of the original sample intact, we created dummy-coded covariate categories to denote subgroups with missing covariate data. This approach stabilizes the number of participants contributing information to the regression analyses. An alternative approach to handling missing values (multiple imputation) did not alter the results. We also included a covariate for receipt of subsidized lunch during first grade in the multiple regression analyses because this characteristic was associated with attrition. To hold constant the participants’ first grade intervention status, this covariate was also included in the adjusted analyses. Lastly, a variant of the Huber-White sandwich estimator of variance was used to obtain robust SEs and variance estimates in all regression analyses to accommodate the initial sample design and clustering of students within schools.

RESULTS

Among the 2311 first graders, a total of 1785 completed at least 1 follow-up interview after 1990. Because those followed up in late childhood/early adolescence and young adulthood come from the same original study population, the distributions are similar (Table 1). Half of the participants are female and most are African American. At the time of the baseline interview for these analyses, the students were aged a mean of 10.0 years (SD, 0.8 years). Forty percent had a caregiver history of either heavy drinking or drug use. Most had received free or subsidized lunches in first grade.

In Table 2, we present the distribution of depressed mood level at baseline, stratified by sex and race/ethnicity. Among those followed up in late childhood/early adolescence, 20% had been in the low-depressed mood category at baseline in 1990, and 12% were at the high level. Among those followed up in young adulthood, 16% and 10% had been in the low and high baseline categories of depressed mood, respectively. Contrary to our prior hypotheses, there was no appreciable association of sex or race/ethnicity with the baseline assessment of depressed mood.

As presented in Table 3, by the time of the late childhood/early adolescence follow-up, 16% of the youths had experienced at least 1 episode of alcohol intoxication and 22% had experienced alcohol-related problems for the first time. These frequency differences may occur because some underage drinking problems do not require drinking to intoxication. Similar drinking outcome proportions were found for each of the sex and race/ethnicity subgroups. By young adulthood, 12% had developed alcohol abuse, with a greater cumulative incidence proportion of abuse occurring among the male (16%) and other race/ethnicity (18%) subgroups. A total of 9.5% had developed alcohol dependence, with a greater cumulative incidence proportion for males (14%) and for individuals in the other race/ethnicity category (13%). For reasons discussed by Degenhardt et al., this estimate for DSM-IV alcohol dependence should be regarded as possibly conservative, as the clinical significance criterion was in place. Drinkers without evidence of maladaptive or hazardous alcohol problems were not counted in the numerators for these estimates.

Kaplan-Meier survival curves were estimated for each of the alcohol-related outcomes by depressed mood. Age at first intoxication was earliest for those with high depressed mood at baseline (log-rank test, $\chi^2 = 18.21, P < .001$) (Figure 2). The smoothed hazard estimates indicate proportionality, which helped motivate use of the Cox model. Similar findings were found for Kaplan-Meier curves assessing age at first alcohol problem (log-rank test, $\chi^2 = 10.91, P = .001$) (Figure 2). The smoothed hazard estimates indicate proportionality, which helped motivate use of the Cox model. Similar findings were found for Kaplan-Meier curves assessing age at first alcohol problem (log-rank test, $\chi^2 = 10.91, P = .001$). With respect to the young adulthood follow-up outcomes, no appreciable association was found for age at first clinical feature of alcohol abuse in relation to baseline depressed mood level (log-rank test, $\chi^2 = 2.55, P = .28$). However, with respect to the first alcohol dependence criterion, individuals with high baseline depressed mood started the process earlier (log-rank test, $\chi^2 = 10.24, P = .006$) (Figure 3).
In Table 4, we present the unadjusted and adjusted hazard ratios based on a comparison of the subgroups’ onset of alcohol intoxication and alcohol-related problems as assessed during the late childhood/early adolescence follow-up. Table 5 presents odds ratios for the occurrence of alcohol abuse and dependence observed at the young adulthood follow-up. Based on covariate-adjusted Cox models, the estimated hazards for alcohol intoxication at the late childhood/early adolescence follow-up among individuals with high childhood depressed mood were roughly 2-fold greater than values observed for youths without depressed mood. High-depressed mood level at baseline was also associated with the onset of alcohol-related problems among the male and other race/ethnicity subgroups. An estimated 2-fold increased risk of alcohol dependence was associated with those who had high childhood depressed mood relative to those without (Table 5); for individuals in the other race/ethnicity subgroup, this association was attenuated with wider confidence intervals. Childhood depressed mood was not associated with risk of developing alcohol abuse by the time of the young adulthood follow-up. Because our analyses were restricted to those who drank alcohol, which theoretically could introduce bias, we also repeated these regression analyses including the nondrinkers as part of the reference group in the Cox models and as a separate outcome in the multinomial regression analyses. These supplemental analyses did not lead to any differences in substantive conclusions.

Consistent with our hypotheses in this prospective study, depressed mood as assessed in childhood helped to predict and may account for increased risk of several manifestations of alcohol involvement. For predictive associations, the strength of the associations increased with greater severity of depressed mood, but the risk estimates for the subgroup at a lower level of baseline depressed mood were not statistically different from estimates for children without depressed mood. Higher depressed mood in childhood also signaled earlier onset of alcohol involvement, and in general congruent risk ratios were observed for the sex and race/ethnicity subgroups we studied. For the assessment of mood level with first report of alcohol-related problems, males and participants in the other race/ethnicity category had the strongest associations. The vulnerability to increased risk for alcohol involvement extended across both life stages studied and was independent of potential causal determinants of maladaptive drinking, such as family income in childhood, heavy drinking among caregivers, peer alcohol use, level of conduct problems, and neighborhood disadvantage. Possible explanations for these findings include motives to use alcohol for stress reduction. Children with early mood symptoms may be more likely to subsequently use alcohol as a method for coping with negative affect. In addition, the association of childhood depressed mood with later alcohol involvement may be mediated or moderated by alcohol-related expectancies. Familial factors or processes, with both genetic and/or environmental substrates, may influence the development of affective symptoms as well as the occurrence of maladaptive drinking patterns. These processes may be specific for alcohol involvement relatively early in life.

Our findings are consistent with clinical and population-based cross-sectional data and extend findings from prior prospective analyses. However, our findings...
differ from prior reports that indicate that adolescent depression predicts alcohol disorders only in the presence of conduct disorder symptoms (data for this interaction are not shown).27 Our results may differ because we examined mood at an earlier life stage (in childhood as opposed to adolescence). In our analyses, early depressed mood level predicted all but 1 of the alcohol involvement outcomes. We found no association with alcohol abuse. Early depressed mood appears to predict the transition from high-risk drinking to alcohol dependence but not to the occurrence of alcohol abuse without dependence. Similar findings have been documented in some prior data. In analyses of the National Comorbidity Survey, using retrospective reports from cross-sectional data,
mood disorders were positively associated with alcohol dependence but negatively associated with alcohol abuse among men. In the European Study of the Epidemiology of Mental Disorders project, alcohol abuse had the weakest association with depression among all the comorbid patterns assessed. Some investigators speculate that field research assessments of the DSM alcohol abuse construct may reflect nonpathologic drinking patterns that occur inextricably with adolescent or young adult drinking experiences, particularly for young male drinkers.

One of the issues to consider in understanding these associations is the role of concurrence and persistence of depressed mood and drinking. These study estimates may challenge the idea that depressed mood must take place concurrently with drinking behaviors to affect the development of some alcohol-related outcomes. Furthermore, depressive conditions that occur in childhood may increase vulnerability for maladaptive drinking outcomes, whereas similar conditions that occur later in life may not be as strongly associated. This window of vulnerability may be in place for some but not all comorbid associations. Childhood mood disturbances occur at a significant stage for neurobiological development. During this critical period, mood disturbances may inhibit the normal progression of a child’s ability to develop mechanisms to cope with stress and negative expectations and may increase the likelihood that skills to handle stress-related occurrences are poorly developed or remain immature. Early manifestations of depressive conditions can also be difficult to recognize by families and health care providers. It is typically quite a challenge to determine whether these features are clinically relevant. Our assessment of depressed mood queried about “being crabby,” having “frequent crying spells,” and “being sad.” These feelings are common to many children, and it may be difficult for families and clinicians to recognize when they may indicate a psychiatric disturbance or when further evaluation is warranted. Although early depressive feelings may be a potential predictor of alcohol involvement, other psychopathologic conditions are also associated with risk for alcohol disorders, including disruptive behaviors, other substance use, anxiety, and affective disorders.

There are several limitations in our analyses that should be mentioned. First, residual confounding may be present (eg, familial history of psychopathology [depression, anxiety, alcohol dependence, or antisocial personality] or participant history of childhood trauma). For example, family history may be associated with early occurrence of depressive conditions and increased vulnerability for alcohol involvement owing to genetic predispositions as well as familial influences, such as modeling behavior, provision of support, and parental monitoring. However, we were able to include information on report of illicit drug use and heavy drinking in the caregiver, which might help to hold constant these potential confounding characteristics. Second, our assessment of depressed mood in childhood did not include a structured psychiatric interview and thus cannot be regarded as diagnostic of depression. In addition, the measure used identified features that may be present in a range of psychopathology. However, as we discussed previously, this measure had adequate internal consistency and was associated with major depression (evidence of predictive validity) assessed by CIDI-UM. Third, as mentioned previously, our assessment of alcohol dependence focused on clinically significant dependence syndrome. As discussed by Degenhardt et al, this use of clinical significance criteria may miss some individuals who developed a milder form of alcohol dependence. Fourth, as with any prospective study, particularly one extending across long intervals, there was participant attrition and missing information. We have addressed these concerns by using 2 statistical strategies: including covariates for missing data in the regression models and multiple imputation. Yet, the potential for bias remains. Fifth, generalizability of the findings from this sample of school-attending youths rests on replication of the findings elsewhere.
Table 4. Risk of Alcohol Intoxication and Alcohol-Related Problems During Late Childhood/Early Adolescence by Level of Childhood Depressed Mood Among Youth Reporting Alcohol Use Based on Cox Regression Analyses

<table>
<thead>
<tr>
<th>Childhood Depressed Mood</th>
<th>Alcohol Intoxication</th>
<th>Alcohol Problems</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Total</td>
<td>Boys</td>
</tr>
<tr>
<td></td>
<td>HR (95% CI)</td>
<td>P Value</td>
</tr>
<tr>
<td>Unadjusted</td>
<td></td>
<td></td>
</tr>
<tr>
<td>None (Reference)</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Low</td>
<td>1.65 (1.15-2.37)</td>
<td>.007</td>
</tr>
<tr>
<td>High</td>
<td>2.28 (1.55-3.37)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Adjusted</td>
<td></td>
<td></td>
</tr>
<tr>
<td>None (Reference)</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Low</td>
<td>1.59 (1.10-2.29)</td>
<td>.01</td>
</tr>
<tr>
<td>High</td>
<td>1.99 (1.33-2.97)</td>
<td>.001</td>
</tr>
</tbody>
</table>

Abbreviations: CI, confidence interval; HR, hazard ratio.

*Adjusted for sex and race (when applicable), age, caregiver history of heavy drinking and/or illegal drug use, peer drinking, neighborhood disadvantage, conduct problems, intervention status, subsidized lunch, and clustering within schools. To avoid biases resulting from nonrandom deletion of participants within our prospective study cohorts and to keep the representativeness of the original sample intact, thereby stabilizing the number of participants contributing information to the regression analyses, we created separate categories for subgroups with missing information for each covariate included in each multivariate model.

Table 5. Estimated Risk of Alcohol Abuse and Alcohol Dependence in Young Adulthood by Level of Childhood Depressed Mood Among Youth Reporting Alcohol Use Based on Multinomial Regression Analyses

<table>
<thead>
<tr>
<th>Childhood Depressed Mood</th>
<th>Alcohol Abuse</th>
<th>Alcohol Dependence</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Total</td>
<td>Males</td>
</tr>
<tr>
<td></td>
<td>OR (95% CI)</td>
<td>P Value</td>
</tr>
<tr>
<td>Unadjusted</td>
<td></td>
<td></td>
</tr>
<tr>
<td>None (Reference)</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Low</td>
<td>0.96 (0.61-1.52)</td>
<td>.86</td>
</tr>
<tr>
<td>High</td>
<td>0.53 (0.25-1.16)</td>
<td>.11</td>
</tr>
<tr>
<td>Adjusted</td>
<td></td>
<td></td>
</tr>
<tr>
<td>None (Reference)</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Low</td>
<td>0.94 (0.57-1.53)</td>
<td>.79</td>
</tr>
<tr>
<td>High</td>
<td>0.58 (0.27-1.22)</td>
<td>.15</td>
</tr>
</tbody>
</table>

Abbreviations: CI, confidence interval; OR, odds ratio.

*One multinomial regression model was used to assess alcohol abuse and alcohol dependence as separate outcomes, with alcohol drinkers without either disorder as references.

*Adjusted for sex and race (when applicable), age, caregiver history of heavy drinking and/or illegal drug use, peer drinking, neighborhood disadvantage, conduct problems, intervention status, subsidized lunch, and clustering within schools. To avoid biases resulting from nonrandom deletion of participants within our prospective study cohorts and to keep the representativeness of the original sample intact, thereby stabilizing the number of participants contributing information to the regression analyses, we created separate categories for missing information for each covariate included in each multivariate model.

*Clinically significant alcohol dependence syndrome.

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Notwithstanding these limitations, the study contributes new information about childhood depressed mood and subsequent alcohol outcomes as well as possible childhood predictors of alcohol involvement that may extend across adolescence into young adulthood. The effects of internalizing symptoms on transitional and sequential stages of drinking will need to be explored in future research. In addition, this research can be extended from this initial platform of evidence in an exploration of the potential moderating and mediating influences attributable to other drug use, externalizing behaviors, and expectancies associated with drinking alcohol. Whether and how the observed mood manifestations change, persist, or progress through one’s life stages deserves scrutiny. Co-occurring transitions in drinking behavior may have important implications for identifying and targeting high-risk groups for prevention and early intervention efforts.

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Additional Information: The Appendix is available online at http://www.archgenspsychiatry.com.

REFERENCES


34. Maisto SA, Connors GJ, Zywiak WH. Alcohol treatment, changes in coping skills, self-efficacy, and levels of alcohol use and related problems 1 year following treatment initiation. Psychol Addict Behav. 2000;14(3):257-266.


84. Tambs K, Harris JR, Magnus P. Genetic and environmental contributions to the correlation between alcohol consumption and symptoms of anxiety and depression: results from a bivariate analysis of Norwegian twin data. *Behav Genet*. 1997;27(3):241-250.


90. Elkins IJ, McGue M, Iacono WG. Prospective effects of attention-deficit/hyperactivity disorder, conduct disorder, and sex on adolescent substance use and abuse. *Arch Gen Psychiatry*. 2007;64(10):1145-1152.


