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Age of Alcohol Drinking Onset: Precursors and the Mediation of Alcohol Disorder

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ABSTRACT. This study explored early alcohol drinking onset (ADO), its precursors, and the mechanisms by which it leads to later alcohol disorder. Data came from the National Longitudinal Survey of Youth with ADO items from 1982 and 1983, and alcohol symptoms from 1989 and 1994. Drinking began earlier for respondents who were male, younger, non-Hispanic, non-African-American, and later born, and for those not living with both parents at age 14, ever charged with an illegal act, and with a family history of alcohol problems, lower academic aptitude, or less frequent religious attendance (n = 8165). Early ADO predicted 1994 abuse and dependence even after controlling for such potential mediators as 1987 self-esteem, 1989 alcohol disorder, and 1992 depression (n = 5643). *[Article copies available for a fee from The Haworth Document Delivery Service: 1-800-HAWORTH. E-mail address: <docdelivery@haworthpress.com> Website: <http://www.HaworthPress.com>*

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INTRODUCTION

Causal Role of Early Drinking?

Recent research has suggested an association between the age of alcohol drinking onset (ADO) and later alcohol misuse and other health problems (Grant & Dawson, 1997). For example, respondents who begin drinking at earlier ages appear more likely to drive drunk, and to have alcohol-related motor-vehicle accidents (Hingson, Heeren, Levenson, Jamanka, & Voas, 2002). However, the causal mechanism connecting ADO to later alcohol misuse remains unclear. Early drinking may have a greater adverse influence, compared to later drinking that begins when one has greater maturity, social resources, and coping skills. Research linking ADO to alcohol abuse, independent of the contribution of familial alcoholism history, is consistent with such a causal association (Grant, 1998). Even without definitive evidence, the putative causal relationship between early drinking onset and later alcohol abuse has provided the rationale for preventive school, family, and community interventions aimed at delaying the onset of drinking (Hawkins, Catalano, & Miller, 1992).

On the other hand, the observed associations may be explained by confounding variables. Precursors of ADO (e.g., ethnicity, gender, intelligence, criminal activity) might also account for later alcohol abuse or dependence, regardless of when the individual starts drinking (Prescott & Kendler, 1999). Under this scenario, even if social interventions could postpone the start of drinking, their effects on adult abuse would be nil. Additionally, other variables believed to strongly influence the development of adult alcohol disorder, such as educational achievement (Muthen & Muthen, 2000), may mediate the relationship between ADO and later disorder, again rendering such interventions ineffective.

Prior studies of ADO and alcohol misuse have often used cross-sectional designs that measure recalled ADO and current alcohol misuse at the same point in time. However, older respondents may have difficulty recalling their age of drinking onset, and younger respondents, who are closer to their ADO, may not be able to provide information about the risk of adult alcohol misuse. Moreover, such cross-sectional studies are vulnerable to the rival hypothesis that people with current drinking

problems report earlier ADO in an effort to explain their situation compared to people without current alcohol problems.

Longitudinal studies can counter this explanation by measuring ADO at a time point close to drinking onset and alcohol abuse at a later time point. Few longitudinal studies exist, and some of these are based on European samples that may not generalize to the American case (e.g., Hammer & Pape, 1997; Plant, Peck, & Samuel, 1985). For example, Andersson and Magnusson (1988) measured ADO in a sample aged 14-16 and measured alcohol abuse with archival health records collected when respondents were in their 20s. They concluded that early drinking habits were of limited importance in the development of adult alcohol problems. In contrast, analyses of a large American panel found that ADO predicted later alcohol abuse and dependence up to 12 years later (Grant, Stinson, & Harford, 2001).

Research Challenges

Confirmation of the link between ADO and later alcohol misuse raises two issues. First, what factors predict ADO? Variables that reflect characteristics of the respondents or their environments prior to onset of drinking (e.g., gender) can potentially cause early alcohol use, though some plausible causes of early drinking must be examined carefully since their timing relative to ADO may be uncertain (e.g., religious observance). Any precursors of ADO become potential confounders of the association between ADO and later alcohol disorder.

Second, after controlling for potentially confounding variables, by what mechanisms might ADO influence later alcohol misuse? Early drinking may influence intervening variables such as educational attainment, family formation, self-esteem, depression, or early-adult alcohol misuse that in turn lead to later alcohol abuse or dependence. The present analyses will check the roles of potential mediating variables in order to illuminate the mechanism linking early ADO and adult alcohol problems.

METHOD

Sample

The National Longitudinal Survey of Youth (NLSY) is based on a large nationally representative sample of youth aged 14 to 22 years old in 1979. The panel, consisting of 12,686 respondents in 1979, has been

followed annually through 1994 and every other year thereafter. In 1996 the NLSY reported an 86.7% retention rate (Center for Human Resource Research, 1999).

Assessment of the predictors of early ADO is based on 8,165 respondents who had complete data on ADO and its possible precursors. About half (50.6%) were male, 81.4% were non-Hispanic/non-African American (12.8% were African American), 76.0% reported living with both mother and father when they were 14 years old, and 49.0% reported a negative family alcoholism history. Parental education averaged 12.6 years (SD = 3.0).

Analyses of the mediators of the relationship between early ADO and later alcohol misuse are based on 5,643 respondents who had complete data on ADO, contextual variables, and alcohol disorder in 1989 and 1994. The demographic profile of this sample is presented in Table 1 and closely resembles the sample described above for the study of ADO precursors.

The NLSY supplies a set of sampling weights for each survey year that are intended to provide an estimate of the number of individuals represented by each respondent. These weights are used to adjust the sample for attrition and for the over-sampling of African Americans, Hispanics, and economically disadvantaged whites. These weights were used in all analyses presented in this paper.

Age of Alcohol Drinking Onset (ADO)

Respondents who reported ever having a drink were asked three age-at-first-drinking items, one in 1982 and two in 1983. In 1982, respondents were asked, "How old were you when you first started drinking?" This item also included a follow-up probe asked at the interviewer's discretion: "For example, having two or more drinks a week?" In 1983, the respondents were asked: "How old were you when you first began drinking alcoholic beverages on a regular basis, that is, at least once or twice a month?" Respondents who indicated that they had drunk alcohol on a monthly basis were asked: "How old were you when you first began drinking alcoholic beverages at least once or twice a week?"

We used all of the ADO items, both to reflect agreement across multiple responses and to minimize missing cases in order to increase power especially for the small earliest ADO group (less than 5% of the sample). We used the reported age for respondents whose ADO came from one response or from multiple responses that all agreed. When there was disagreement among the responses, we used the youngest

TABLE 1. Descriptive Statistics for Study Variables (n = 5,643)

Age at Alcohol Drinking Onset (ADO) [a]	Mean	%	St. Dev.
12 years or younger old		4.6	
13 years to 15 years old		23.4	
16 years or older		72.0	
1994 DSM-IV Alcohol Classification			
Non-Drinker[b]		37.0	
Alcohol Abuse		6.9	
Alcohol Dependence		6.3	
No Alcohol Abuse/Dependence		49.8	
Contextual Variables			
% Male		51.9	
Ethnicity			
Hispanic		5.6	
African American		12.2	
non-Hispanic/non-African American		82.1	
Religious Attendance at 14 yrs: >1 time/wk		8.7	
Ever Charged with Illegal Act ’80		9.5	
Family History of Alcoholism			
1st degree, biological		14.7	
2nd degree, biological		21.7	
1st & 2nd degree, biological		12.1	
Non-Biological Relative		3.0	
Family History Negative		48.4	
Birth Order: 1st born		21.6	
Live with both Mother/Father at 14 yrs.		76.1	
Age in years ’94	32.7		2.3
Parental Years of Education	12.7		3.0
Aptitude (AQFT) ’80	50.8		28.3
Mediating Variables			
Rosenberg Self-Esteem ’87	33.9		4.1
1989 DSM-IV Alcohol Classification			
Non-Drinker[a]		20.0	
Alcohol Abuse		10.0	
Alcohol Dependent		7.3	
No Alcohol Abuse/Dependence		62.7	
Years of Education ’94	13.5		2.4
Number of Children ’94	1.3		1.2
Number of Different Jobs ’94	9.3		5.4
Not Married ’94		16.8	
CES-Depression ’92	8.7		8.7

[a] Respondents were asked how old they were when they first started drinking alcohol in 1982 and again in 1983. See Methods section for definition of age at Alcohol Drinking Onset (ADO).
[b] Non-Drinkers did not report drinking alcohol in the 30 days preceding the interview.

reported age, which favored reports by respondents at their first (1982) interview and minimized the forward telescoping effect in which older respondents report events later than they really occurred (Crawley & Pring, 2000). This strategy was used to define ADO for use in our analytic models because it provides a larger sample size (n = 5,643) than use of any one of the ADO items. Final models were replicated using each ADO item, i.e., 1982 weekly ADO (n = 4,869), 1983 weekly ADO (n = 3,263), and 1983 monthly ADO (n = 4,757).

Test-retest reliability for exact year of ADO was low (intra-class correlation coefficient of 1982 and 1983 reports of weekly drinking = .36). To allow for adjacencies in reported ADO and for possible nonlinear patterns of association with ADO, we assigned the respondents to categories based on spans of years: 12 or younger, 13 to 15, and 16 or older (including those reporting never had a drink). These age groups had two advantages. The number of respondents in each group provided adequate cell sizes for subgroup analyses, and these age groups correspond roughly to school segments in which prevention programs are often mounted: elementary (grades K-6), middle (7-9), and high (10-12) school.

Alcohol Disorder

The 1989 and 1994 waves included alcohol items that were similar to each other and to the criteria of the DSM-IV diagnostic categories of alcohol abuse and alcohol dependence (American Psychiatric Association, 1994). Using these items for these two years, we assigned respondents to the following four categories: alcohol dependent, alcohol abusing, drinking but not disordered, and non-drinking in the past month (details of the NLSY items used to assign these categories are available on request). Rather than discarding the non-drinking respondents, we retained them in part to maximize the study's statistical power. More importantly, keeping them separate from the not-disordered drinkers helps illuminate the full range of drinking outcomes that might follow early ADO from dependence to moderate drinking to complete abstinence.

The NLSY participants indicated, in both the 1989 and 1994 interviews, the frequency with which they had experienced each of the alcohol symptoms. Following Harford and Grant's (1994) method of handling the "duration" criterion for abuse, an abuse symptom had to have occurred two or more times in the past year to be counted. Although DSM-IV criteria do not include the DSM-IIIR duration requirement, they often include references to "often," "persistent," or

"continued." For consistency with the abuse criteria and in an effort to yield realistic prevalence rates, dependence symptoms also had to be reported as occurring two or more times in the past year.

The 15 alcohol dependence items had satisfactory internal consistency in both 1989 and 1994 (Cronbach alpha = .87 for each year). The eight alcohol abuse symptoms had somewhat lower inter-item consistency (.78 in 1989, .75 in 1994). There was significant test-retest stability of the 1989 and 1994 DSM-IV categories, even adjusted for chance agreement (kappa = .49, $p < .001$). We retained the distinction between abuse and dependence, rather than combining them into a single alcohol disorder category, in part to check for possible etiological differences related to tolerance or withdrawal (American Psychiatric Association, 1994) and in part for comparability with other studies (e.g., Grant et al., 2001).

Contextual Variables

Table 1 describes additional variables used in these analyses. Precursors to ADO were required to be temporally prior to and likely associated with ADO. Some demographic variables are clearly prior to the onset of drinking (e.g., ethnicity and gender), but other variables are less clearly prior because they were asked only in the first two years of the survey. For example, aptitude as measured by the Armed Forces Qualifications Test (AFQT), and ever charged with an illegal act, were both asked in the 1980 interview. Other variables referred to the respondent's circumstances when he/she was 14 years old, which in some cases was after, rather than prior to, age of alcohol drinking onset. For example, items on whether respondents lived with both parents, religious affiliation, and frequency of attendance were asked in 1979.

Familial history of alcoholism is unique because it was asked in 1988, the only "precursor" measured after report of ADO in 1982 and 1983. This variable is coded following Jennison and Johnson (1998) into the following mutually exclusive categories: first-degree biological relative but not second-degree, second-degree biological relative but not first-degree, both first- and second-degree biological relatives, non-biological relative, and family history negative. Family history of alcoholism, and other variables for which the temporal ordering with ADO is ambiguous, are referred to as "plausible" precursors in our analyses.

We investigated four types of possible mediators between early ADO and later alcohol abuse or dependence: family context, educational/occupational performance, psychological well-being, and prior history of alcohol disorder (MacKinnon, Warsi, & Dwyer, 1995). Family charac-

teristics included marital status, cumulative number of separations/divorces, and number of children in the household in 1994. Educational performance was measured by years of education in 1994, and the individual's job stability was measured by the number of different jobs ever held and the number of interview weeks the respondent was unemployed as of 1994. Psychological well-being was measured in 1987 by the Rosenberg self-esteem scale (Rosenberg, 1965) and depression by the CES-D scale in 1992 (Radloff, 1977). Potentially mediating alcohol disorder was represented by the 1989 DSM-IV categorization.

Analyses

The precursor analysis used multinomial logistic regression to evaluate the predictors of ADO as coded into three groups: 12 years or younger, 13 to 15 years old, and 16 years or older (the latter group served as the reference category). The contextual variables included variables that were clearly temporally prior to ADO, such as gender, and those that were plausible precursors to ADO, such as religious affiliation and family history of alcoholism.

The mediation analysis used multinomial logistic regression, with the group with no alcohol dependence/abuse in 1994 serving as the reference group. The analysis was divided into three models. The first model estimated the association between ADO and later alcohol abuse and/or dependence, after statistically controlling for confounding variables. The second model tests the hypothesis that the variables measured prior to 1990 (self-esteem in 1987 and DSM-IV abuse or dependence in 1989) might mediate the relationship between early ADO and later alcohol abuse or dependence. The third model adds the potential mediators collected after 1990 that might have been influenced by ADO or indirectly from ADO via earlier self-esteem or alcohol misuse. We also checked the possible moderating effect of all these variables on the association between early ADO and later alcohol abuse/dependence, but none of the interactions between ADO and the tested variables reached significance.

RESULTS

Precursors of ADO

Defining ADO using the decision rules reported in the methods section, multinomial logistic regression was used to model the odds of

ADO at 12 years or younger and ADO at 13-15 years (both relative to ADO at 16 years or older) as a function of variables that are clearly precursors of early ADO (Table 2, Model 1). When adjusting for the other variables included in Model 1, early ADO is reported more by males than females, younger rather than older respondents, later born as opposed to first-born respondents, and non-Hispanic Whites rather than African Americans (for ADO at 13-15 years, only).

Model 2 (Table 2) shows the relationship between early ADO and variables considered "plausible" precursors because their temporal ordering with ADO is ambiguous. When adjusting for other variables included in the model, the odds of early ADO decreased with increasing aptitude and more frequent religious attendance, while the odds of early drinking increased for respondents who were ever charged with an illegal act. Respondents who reported living with both their mother and father when they were 14 years old were less likely to report ADO at ages 13-15 years. A family history of alcoholism, including first- and second-degree biological relatives, compared to a negative family history, is significantly associated with early drinking. However, the presence of non-biological alcoholic relatives was also associated with increased odds of drinking onset at ages 13-15 years old.

Mediators of the ADO–Alcohol Disorder Association

Before conducting this analysis, we examined the relationship between each of the hypothesized mediators and ADO using a single factor analysis of variance (followed by a Bonferroni procedure for all possible pairwise comparisons among the ADO groups) or a chi-square. Respondents who reported an ADO of 16 years or older had significantly more education, fewer children, and fewer jobs ever reported when compared to each of the early ADO groups. Depression was significantly lower in the 16-years-or-older ADO group when compared to the 13-15 year-old ADO group. Respondents who reported ADO of 16 years or older were more likely to be married and less likely to be classified as alcohol abusive or dependent in 1989 using the DSM-IV, compared to the early ADO groups. There was no significant difference in self-esteem among the ADO groups. Although eliminated as a potential mediator, self-esteem was retained in subsequent analyses because it significantly predicted 1994 alcohol disorder and was required to avoid mis-specification error.

Multinomial logistic regression was used to model 1994 alcohol disorder: dependence; abuse; no alcohol dependence/abuse; or non-drinker

TABLE 2. Precursors to Age of Alcohol Drinking Onset: Multinomial Logistic Regression (n = 8,165)

	Odds Ratios for ADO[a]			
	Model 1: Precursors to ADO		Model 2: Plausible Precursors to ADO	
Predictor	12 years or younger (n = 367)	13 - 15 years old (n = 1,820)	12 years or younger (n = 367)	13 - 15 years old (n = 1,820)
Male	3.02**	1.96**	2.74**	1.91**
Age in years (1979)	0.90**	0.88**	0.88**	0.87**
Ethnicity[b]				
Hispanic	0.79	0.99	0.65	0.87
African American	0.81	0.59**	0.65*	0.49**
Birth Order[c]				
Not First Born	1.44**	1.44*	1.38*	1.40
Missing	1.35	1.26	1.42	1.29
Family History of Alcoholism (1980)[d]				
1st degree biological			1.60**	1.23*
2nd degree biological			1.24	1.17*
1st & 2nd degree biological			1.91**	1.55**
Non-biological relative			1.80	1.56**
Aptitude in 1980 (AFQT)			0.99**	0.99**
Lived with both Mother & Father when 14 years old (1979)			0.86	0.87*
Religious attendance once/wk or more often (1979)			0.72*	0.77**
Ever charged with illegal act (1980)			3.60**	2.10**
Likelihood Ratio Chi-Square (df)		427.79 (12)**		729.80 (28)**
Cox-Snell R^2		.051		.086

* p < .05 ** p < .01

[a] Reference group is ADO at 16 years or older (n = 5,978).

[b] Reference group is non-Hispanic/non-African American.

[c] Reference group is first-born.

[d] Reference group is family history negative.

(in the 30 days preceding the interview). The group with no alcohol dependence/abuse was used as the reference group in these analyses. As seen in Table 3, Model 1 estimates the effect of ADO controlling for several variables identified as potential confounders (i.e., prior to both ADO and later development of alcohol dependence/abuse). When adjusting for these variables, the odds of both abuse and dependence were greater for males than females and for those who had a first-degree biological relative. The odds of abuse were less for older respondents, and the odds of dependence fell with increasing aptitude but rose for those who reported ever having been charged with an illegal act. Being a nondrinker was negatively related to being male, years of parental education, aptitude, and beginning drinking between 13 and 15, and positively related to frequent religious observance. Paradoxically, abstinence was positively associated with beginning drinking at 12 or younger and with having relatives with a history of alcohol problems. These and other variables that display such counterintuitive associations with the abstinence outcome will be revisited in the discussion section.

After adjusting for these confounding variables, the odds of later alcohol abuse and dependence were higher for those who first began drinking at 12 years or younger when compared to those who first started drinking at 16 years or older (OR = 1.71 and OR = 1.66, respectively, both $p < .05$). For the respondents who reported an ADO of 13-15 years (compared to 16 years or older), the odds of alcohol abuse and dependence were also greater (OR = 1.61 and OR = 1.89, both $p < .01$). Interestingly, ADO of 12 or younger was associated with increased odds of nondrinking in 1994 (OR = 1.53), but ADO of 13-15 was associated with decreased odds of later abstinence (OR = .79), a curvilinear pattern that held throughout later models.

Model 2 adds 1989 DSM-IV abuse/dependence as a potential mediator of the early ADO-with-later alcohol misuse relationship, and it controls for 1987 self-esteem. If early drinking causes individuals to begin alcoholic careers, then controlling for interim abuse or dependence (1989) should reduce the influence of ADO (1982/83) on later disorder (1994). Nevertheless, ADO at 13-15 years remained significantly associated with alcohol abuse and dependence in 1994 despite the inclusion of 1989 DSM-IV as a potential mediator (coded 1 = either alcohol abuse or dependence in 1989, 0 = neither abuse nor dependence). In contrast, DSM-IV in 1989 appeared to mediate the relationship between ADO at 12 years or younger and later alcohol abuse and dependence; i.e., the inclusion of 1989 alcohol disorder dropped the association below significance. But the effect sizes of the association of ADO at 12 or younger

TABLE 3. Age at Drinking Onset (ADO) and 1994 DSM-IV Alcohol Dependence and Abuse in NLSY Adult Sample (n = 5,643)

	Odds Ratios for DSM-IV in 1994[a]								
	Model 1			Model 2			Model 3		
	Non-Drinker	Abuse	Dependent	Non-Drinker	Abuse	Dependent	Non-Drinker	Abuse	Dependent
Predictor Variable[b]									
Male	0.53**	1.85**	1.60**	0.56**	1.52**	1.31*	0.59**	1.45**	1.28
Age (years) '79	1.04*	0.90**	0.96	1.04**	0.92**	0.99	1.02	0.94*	0.99
Parental years of Education	0.94**	1.00	0.99	0.95**	1.00	0.99	0.95**	0.99	0.99
Aptitude AFQT Score '80	0.99**	1.00	0.99**	0.99**	0.99	0.99**	0.99**	1.00	1.00
Frequency of Religious Attendance '79									
More than 1x/week	1.70**	0.78	0.92	1.67**	0.84	1.03	1.67**	0.90	1.11
Family History of Alcoholism[c]									
1st degree, biological	1.15	1.41*	1.43*	1.21*	1.21	1.18	1.20*	1.16	1.11
2nd degree, biological	1.17*	1.66**	1.12	1.21*	1.47**	0.98	1.21*	1.43*	0.97
1st and 2nd degree, biological	1.45**	1.19	2.30**	1.49**	1.05	1.99**	1.50**	0.94	1.76**
Non-biological relative	1.36	1.72	0.71	1.36	1.56	0.64	1.34	1.41	0.55
Ever charged with illegal act '80	1.04	1.34	2.26**	1.05	1.16	1.87**	1.04	1.00	1.60**

Age at drinking Onset[d]									
12 or younger	1.53**	1.71*	1.66*	1.58**	1.54	1.49	1.59**	1.40	1.37
13 to 15 years old	0.79**	1.61**	1.89**	0.81**	1.39**	1.64**	0.80**	1.33*	1.52**
Rosenberg Self-Esteem '87				0.97**	0.98	0.93**	0.97**	0.99	0.96*
DSM-IV Alcohol Abuse/ Dependent '89				0.48**	4.30**	5.18**	0.48**	4.21**	4.89*
Years of Education '94							0.99	0.93*	0.85**
# children in household '94							1.11**	0.90	0.85*
Number of different jobs ever held as of '94							0.99	1.02*	1.01
Not Married in '94							1.14	1.91**	2.03**
CES-D Depression '92							1.01*	1.00	1.03**
Likelihood Ratio (df)	760.44 (36)**			1246.61 (42)**			1418.59 (57)**		
Cox and Snell R^2	0.126			0.198			0.222		

*p <.05 **p < .01

[a] Reference group for DSM-IV is recent drinkers, but no alcohol dependence or abuse (n = 2,089). Sample size in the alcohol Abuse group was n = 388, 354 in the alcohol Dependent group, and 2,812 non-drinkers. Non-drinkers in 1994 did not report having a drink in the 30 days preceding the interview.
[b] The following variables were tested, but were found to be non-significant: ethnicity; birth order of respondent; number of siblings; type of residence in 1979; whom respondent lived with at age 14; whom respondent lived with at age 18; ever divorced 1990-1994; and number of interview weeks unemployed 1990-1994. Interactions between ADO and all other study variables were tested and none reached the p = .05 level of significance.
[c] Reference group is family history negative.
[d] Reference group is 16 years or older.

with both abuse and dependence in 1994 remained high and comparable to those involving ADO from 13 to 15, suggesting that the loss of significance may be related to the lower power in the smaller earliest drinking group.

Model 3 includes potential mediators that occurred after 1990 and might be influenced by earlier self-esteem or alcohol misuse. These variables, although significantly associated with later alcohol misuse, did not mediate the relationship between ADO at 13-15 years and later alcohol misuse. The odds of both abuse and dependence were greater for those who were not married in 1994 and decreased with increasing education. There was no association between the number of marital separations or divorces and alcohol abuse or dependence. The odds of dependence (not abuse) increased with increasing depression, decreased with higher self-esteem, and decreased with more children in the household. The individual's job instability as measured by the number of jobs ever held was significantly associated with greater odds of abuse (not dependence), but there was no association between number of weeks unemployed and either alcohol abuse or dependence.

These models were replicated (not shown) using the individual ADO items as described in the Methods section. Despite the smaller sample sizes when using the items separately, these analyses revealed the same general pattern of findings with minor variations. Using the 1982 and 1983 weekly items to define ADO, ADO at 12 years or younger remained significantly associated with later alcohol dependence when controlling for DSM-IV measured in 1989 (OR = 3.63 for 1983 weekly and OR = 1.91 for 1982 weekly, both $p < .05$). When using the 1982 weekly item to define ADO, ADO at 12 years or younger remained significantly associated with later alcohol abuse when controlling for DSM-IV in 1989 (OR = 2.08, $p < .01$). These replications tend to support the conclusion that interim alcohol disorder (1989) does not fully mediate the link from very early ADO (12 or younger) to later alcohol disorder (1994) any more than it mediates the link from 13-to-15 ADO to later disorder.

DISCUSSION

Precursors of ADO

Several factors that were clear precursors to ADO had expected associations (see Table 2, Model 1). Consistent with the universally reported

gender difference in alcohol misuse, males were almost three times as likely as females to report drinking before age 13 and almost twice as likely to report drinking at ages 13 to 15. Later born respondents reported earlier drinking than first-borns, perhaps reflecting their greater opportunity to model the behavior of older siblings. African-American respondents were significantly less likely to report early drinking than their non-Hispanic, non-African American peers. Perhaps reflecting the forward telescoping memory effect (Crawley & Pring, 2000), respondent's age at interview was negatively correlated with reported ADO.

Other variables were considered "plausible" precursors because of their ambiguous temporal relationship to ADO. Among these variables, frequency of religious observance at age 14 years, living with both mother and father when 14 years old, ever having been charged with an illegal act, and aptitude were all associated in the expected ways with ADO (see Table 2, Model 2). Although these four variables were collected prior to the ADO measures, it is possible that for some of the respondents with the earliest age of ADO, drinking onset may have occurred at the same time or even before the "precursor." Another variable, not collected prior to report of ADO, was the presence of relatives with alcohol problems, which was positively associated with early ADO. Whether this variable can be interpreted as a proxy for genetic vulnerability is unclear. A similar pattern holds for non-biological relatives consistent with a social contagion explanation rather than a genetic one. Thus, the findings for these variables require replication in child samples where the predictor variables can be measured unambiguously before initial drinking. However, taken together, these analyses point to a set of risk factors that might serve to target prevention programs. Moreover, they provide an interesting corollary to the work of Malone, Iacono, and McGue (2002), which found adolescent substance misuse to be closely related to other problem behaviors. However, their finding of a strong genetic contribution (via the quantity of the father's alcohol consumption) further suggests that the NLSY measures of familial alcohol use may not accurately capture genetic effects.

Mediators of the ADO "Effect" on Alcohol Misuse

The present analyses confirm that ADO measures collected in 1982 or 1983 can predict alcohol disorder measured in 1994, replicating the findings of Grant et al. (2001). These associations appear both statistically and socially significant. Early drinking predicts not merely an increase in mild symptoms of alcohol misuse but quasi-clinical levels of

both alcohol abuse and dependence. Moreover, the magnitudes of these effects are substantial, which is impressive in light of our statistical control for numerous potential confounders. The robust relationship between ADO and adult alcohol disorder also holds across various subgroups, as evidenced by the lack of interactions with variables such as gender or ethnicity.

In order to illuminate the mechanism by which ADO influences later abuse or dependence, we introduced potential mediators that could account for the ADO-with-alcohol-disorder associations. Although several potential mediators were themselves significantly related to both ADO and alcohol disorder, their inclusion did not fully account for the ADO effects. One cluster reflected the individual's functioning in family contexts. The number of children was negatively associated with dependence, and the absence of a spouse increased the odds of both abuse and dependence. Another cluster represented the individual's educational resources and employment stability. The number of different jobs ever held was positively associated with alcohol abuse but not dependence in 1994, and education was associated with decreased risk of both dependence and abuse. A third cluster reflected psychological well-being. Depression and self-esteem (the only one of these variables not associated with ADO) were both associated in the expected (opposite) directions with dependence (although not abuse). Controlling for these variables might be overly conservative in that they may function not only as causes but also as effects of chronic alcohol misuse. For example, depression may be co-morbid with alcohol disorder and a result of earlier alcohol misuse, rather than a mediator connecting ADO and alcohol misuse. Most dramatically, even prior alcohol disorder measured in 1989 fails to explain the connection between ADO and later alcohol misuse. Despite all these controls, ADO at 13-15 remained linked to later adult abuse and dependence with earlier ADO (12 or under) exhibiting a similar although in some analyses statistically nonsignificant pattern.

In the final model of Table 3, several variables predicted both increased risk of abuse or dependence and also decreased odds of abstinence (e.g., being male, ADO ages 13-15, earlier DSMIV alcohol abuse or dependence, fewer children in the household). Still other variables were intuitively related either to abuse/dependence or to abstinence while being statistically unrelated to the other (e.g., age, parental years of education, aptitude, illegal acts, years of education, number of jobs, not married). In contrast to these unsurprising associations, several variables predicted both abstinence and abuse or dependence in the *same*

direction: family history of alcohol problems, ADO age 12 or younger, self-esteem, and depression. These unexpected associations require replication to assure that they are not statistical artifacts, but their appearance here argues for the inclusion of an abstinence category in future studies as opposed to excluding nondrinkers or combining them with moderate drinkers. We suspect that these associations reflect a real rather than artifactual pattern. People with a history of very early drinking, familial alcohol history, low self-esteem, and high depression are, as these data confirm, at higher risk of alcohol misuse. We speculate that many of these at-risk people who do not currently (1994) report alcohol symptoms may have reduced their risk by becoming abstinent rather than engaging in moderate drinking. Perhaps these individuals include former alcoholics who are following (at least in the prior month) the abstinence prescribed by such widespread treatment regimes as Alcoholics Anonymous. Although the variables needed to check this speculation are not available in the present data set, future research might usefully test this hypothesis.

Implications for Research and Practice

This design cannot rule out the rival hypothesis that unmeasured factors cause both ADO and later alcohol disorder. Future research might usefully search for such variables among earlier childhood and family influences. However, reliance on any such correlational method will never prove definitive. A more convincing demonstration could come from a study that manipulates ADO (i.e., through social interventions). Children randomly assigned to treated and untreated conditions could be followed into adulthood to verify that experimentally delayed ADO leads to decreased risk of alcohol disorder.

However, such programs must first demonstrate that they can produce significant delays in initial alcohol usage. Although some studies report success in this regard (Hansen & Graham, 1991; Johnson et al., 1998), others report no significant effect (e.g., Cohen & Rice, 1995; Dielman, Shope, Leech, & Butchart, 1989). Still other studies find beneficial effects (i.e., decreased drinking) for some groups but not others (e.g., benefits for children with no prior alcohol use but not for children with some prior use; Loveland-Cherry, Ross, & Kaufman, 1999). Beyond such demonstrations of delayed ADO, there must be follow-ups showing that the respondents with experimentally delayed ADO also had lower rates of alcohol problems when measured in adulthood. Although now largely lacking, such evidence could provide a better foun-

dation for the future expansion of such interventions. In addition, such research might help identify the mediating factors that vary as a result of the ADO interventions (e.g., attitudes towards drinking, resistance to peer influence) and that in turn help explain consequent effects on alcohol misuse.

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