# Adult Transition From At-Risk Drinking to Alcohol Dependence: The Relationship of Family History and Drinking Motives

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**Background:** Prospective studies have not previously examined whether a family history of alcoholism and drinking motives conjointly predict a diagnosed DSM-IV alcohol abuse or dependence in adults, despite a large literature that each is associated with alcohol consumption. The focus of this study is the conjoint, prospective examination of these risk factors in a 10-year longitudinal study of adults who were at-risk drinkers at baseline.

**Methods:** Prospective, population-based cohort of drinkers aged 18 or older from a Northeastern U.S. area initially evaluated for history of alcohol use disorders and drinking motives in 1991 to 1992. New onset dependence was studied in those who never met the criteria for alcohol dependence at baseline ($n$ = 423), and new onset abuse was studied in those who never met the criteria for alcohol abuse at baseline ($n$ = 301) and who did not develop dependence during the follow-up.

**Results:** Family history significantly interacted with 2 baseline drinking motives in predicting new onsets of DSM-IV alcohol dependence: drinking to reduce negative affect (OR 3.38; 95% CI 1.05, 10.9) and drinking for social facilitation (OR 3.88; CI 1.21, 12.5). Effects were stronger after conditioning the drinking motives on having a positive family history of alcoholism. In contrast, in predicting new onsets of alcohol abuse, drinking motives did not have direct effects or interact with family history.

**Conclusions:** Those who drank to reduce negative affect or for social facilitation at baseline were at greater risk of alcohol dependence 10 years later if they also had a family history of alcoholism. These results suggest an at-risk group that can be identified prior to the development of alcohol dependence. Further, the findings suggest utility in investigating the interaction of drinking motives with measured genetic polymorphisms in predicting alcohol dependence.

**Key Words:** Alcohol Dependence, Alcohol Abuse, Reasons for Drinking, Drinking Motives, Family History of Alcoholism.

MANY PROSPECTIVE STUDIES of adolescents and young adults have examined predictors of problem drinking (Fothergill and Ensminger, 2006; Grant et al., 2001; Harford and Muthen, 2001; Jackson and Sher, 2003, 2005; Rutledge and Sher, 2001), but fewer studies have prospectively addressed predictors of adult-onset alcohol use disorders (AUDs). Although many young adults drink at their highest lifetime levels while in their early 20's, some continue to drink heavily and develop an AUD after these years. Data from the U.S. National Epidemiologic Survey on Alcohol and Related Conditions indicate that the majority of Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV) alcohol dependence cases begin at age 20 or later (Hasin et al., 2007). Therefore, prospective studies of the onset of AUDs in adults are also important in addressing etiology.

Family history is an established risk factor for alcohol dependence (Cotton, 1979; Dawson et al., 1992; Goodwin, 1979; Harford and Muthen, 2001; Harford et al., 1992). For example, data from the Collaborative Study on the Genetics of Alcoholism showed a 2-fold increased risk of alcohol dependence in the relatives of probands compared to controls (Nurnberger et al., 2004). Family history does not, in itself, demonstrate genetic etiology, but community-based twin studies have established genetic heritability estimates for alcohol dependence of 45 to 64% (Heath et al., 1997; Knopik et al., 2004). Therefore, family history remains important as an initial proxy for genetic influences, allowing preliminary exploration of whether genetic factors may interact with other risk factors.

Motivations are also important risk factors for alcohol-related behaviors. Four main types of drinking motives have been identified: (1) drinking to obtain social rewards or

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enhance social interactions; (2) drinking to enhance positive mood; (3) drinking to reduce negative mood; and (4) drinking to avoid social rejection and conform to social norms (Cooper, 1994; Cooper et al., 1995). Drinking to reduce negative affect includes drinking to cope with negative emotions and is associated with drinking alone (Cooper, 1994), whereas drinking for social interaction and positive enhancement is associated with heavy drinking in situations where it is tolerated (Cooper, 1994; Cooper et al., 1995). In cross-sectional studies of adults, social drinking motives were strongly associated with alcohol problems (Conway et al., 2003; Engels et al., 2005; Mann et al., 1987). Reduction of negative affect and enhancement of positive affect were associated with drinking involvement in numerous studies (Cooper et al., 1995; Jackson and Sher, 2003, 2005; Mann et al., 1987; Rutledge and Sher, 2001). In 553 Dutch adolescents and adults, social enhancement motives were more strongly associated with drinking than motives related to coping or conforming to social norms (Engels et al., 2005). Most studies examined the relationship of drinking motives to drinking frequency and intensity rather than a diagnosed AUD (Abbey et al., 1993; Mann et al., 1987). However, a twin study of drinking motivations showed that drinking to enhance social interaction predicted a DSM-IV diagnosis of alcohol abuse or dependence (Prescott et al., 2004).

Only 2 previous studies conjointly addressed family history of alcoholism and drinking motives. Both of these were cross-sectional, included only adolescents, and addressed only drinking per se, not alcohol abuse or dependence. In one (Mann et al., 1987), drinking for motives such as tension reduction and personal dissatisfaction (all generally aspects of negative affect) had a stronger effect in the presence of family history of alcoholism, while social motives were unrelated to high-risk drinking (Mann et al., 1987). In the other (Chalder et al., 2005), coping motives but not social or enhancement motives, significantly interacted with parental alcohol problems to predict drinking quantity, while social, enhancement and conformity motives interacted with parental alcohol problems to predict increased drinking frequency. These 2 studies suggest that family history and specific drinking motives conjointly influence drinking behavior. However, in both of these cross-sectional studies, drinking motivations were not assessed prior to measuring alcohol consumption. Thus, because drinking motivations can be altered by heavy alcohol consumption (Cox and Klinger, 1988), the direction of the effect was left unclear. Further, drinking motives are likely to differ in adolescents and adults, and understanding the risk for adult AUDs is also of high public health significance. Finally, factors affecting drinking per se may differ considerably from factors affecting DSM-IV alcohol abuse or dependence. Therefore, prospective studies of these issues are needed in adult samples where AUDs were carefully assessed.

Drinking motives and alcohol expectancies are not always well differentiated. Some studies primarily examined alcohol expectancies, the beliefs that drinking alcohol will reduce negative affect or enhance positive emotions (Goldman and Rather, 1993; Smith, 1994). Other studies examined both expectancies and motivations in the same study (Engels et al., 2005; Galen and Rogers, 2004). However, expectancies have been hypothesized to precede drinking motives in the sequence of events leading to drinking behavior and drinking problems (Cooper et al., 1995; Leigh, 1990; Read et al., 2003), and to alcohol abuse or dependence (Schuckit and Smith, 2000), making drinking motives immediately proximal to drinking outcomes (Cooper et al., 1995) and therefore important to understand in the etiology of alcohol dependence.

Previously, we investigated drinking motives in a community sample of adult at-risk drinkers (Carpenter and Hasin, 1998a,b). In a cross-sectional analysis at baseline, DSM-IV alcohol dependence was associated with drinking to reduce negative affect, for enjoyment (positive affect enhancement), for social facilitation and in response to social pressure, while DSM-IV alcohol abuse was associated only with drinking for enjoyment and in response to social pressure (Carpenter and Hasin, 1998b). After 1 year of follow-up (Carpenter and Hasin, 1998a), drinking to reduce negative affect at baseline was the only drinking motive that prospectively predicted new onsets of DSM-IV alcohol dependence. In other studies of this sample, parental history of alcoholism was associated with alcohol dependence at baseline and predicted new onsets of dependence at 1-year follow-up (Hasin et al., 2001). However, drinking motives and family history were never investigated conjointly in this sample to determine whether or how they interacted in predicting the onset of subsequent alcohol dependence or abuse.

Addressing the conjoint relationship of drinking motives and family history to AUDs must be done prospectively for the causal direction to be clear. In such studies, the drinking motives must be assessed prior to the development of an AUD. The previous studies that addressed the conjoint relationship were cross-sectional, and did not examine an AUD as the outcome of interest. Very few studies of family history and drinking motives have been conducted in a community-based sample of adults, and none have explored the interactions between these risk factors. Further, they have not examined alcohol dependence separately from alcohol abuse. Here, we report on a 10-year prospective study of family history and motives for drinking as risk factors for new onsets of alcohol dependence and abuse in a sample of male and female adult household residents whose drinking at baseline placed them at risk for an AUD. Thus, the study focused on a critical stage in the pathway to AUDs, the transition from risk-level drinking to onset of AUD. We first examined transition from at-risk drinking to dependence, enabling us to include abuse at baseline as a potential risk factor for dependence. We then examined transition to abuse among those who never developed dependence.

## METHODS

### *Sample Design, Procedures, and Participants*

The study has been described in detail previously (Hasin et al., 1996, 1997b, 2001). Original study aims included investigation of the natural history of alcohol consumption, abuse and dependence among at-risk drinkers (Hasin et al., 1996, 1997b, 2001). To create the sample in 1991 to 1992, households in a New Jersey county near lower Manhattan were designated by random digit dialing. Within these households, household members age 18 to 65 were enumerated, and among these, randomly selected household members were screened for eligibility. Eligibility included at least 1 occasion of ≥5 drinks in the past 12 months. In 1991 to 1992, 24.5% of U.S. adults drank ≥5 drinks at least once in the past 12 months, and 41.8% drank ≥5 drinks ever (Dawson et al., 1995); 5+ drinks is now part of the NIAAA guidelines to signal at-risk drinking (National Institute on Alcohol Abuse and Alcoholism, 2004). Household and person eligibility status was determined for 81% of the randomly dialed numbers (Hasin et al., 1997a). Of those eligible, 92.0% ($n$ = 962) were personally interviewed for the study after procedures were fully explained and participants gave written informed consent. Interviewer training followed procedures used in national surveys (Grant et al., 2004; Hasin and Grant, 2002). At all interviews, after full explanation of procedures, informed consent was obtained for in-person interviews and verbal consent for telephone interviews, as approved by the New York State Psychiatric Institute Institutional Review Board.

Of the original participants, 777 (81%) completed a baseline self-administered questionnaire (SAQ) that included drinking motivations. Those who completed the SAQ did not differ significantly from those that did not on lifetime DSM-IV alcohol abuse/dependence ($\chi^2$ = 2.39, $p$ = 0.12), gender ($\chi^2$ = 0.15, $p$ = 0.70) mean age (t-test −0.48, $p$ = 0.63) completion of high school ($\chi^2$ = 2.61, $p$ = 0.11), baseline full-time employment ($\chi^2$ = 2.48, $p$ = 0.12), drinking frequency ($\chi^2$ = 0.02, $p$ = 0.90), binge drinking ($\chi^2$ = 1.85, $p$ = 0.17), drinking before age 15 ($\chi^2$ = 1.63, $p$ = 0.20) or drug use history ($\chi^2$ = 0.005, $p$ = 0.94). They did differ significantly on being Caucasion (yes 83% vs. no 72%, $\chi^2$ = 12.5, $p < 0.001$) and having a family history of alcohol problems (yes 84.6% vs. no 78.5%, $\chi^2$ = 5.49, $p$ = 0.02).

After 1 year, 846 of the original participants were re-interviewed, and at the 10-year follow-up, 809 of the original participants were reinterviewed, mainly by phone (36 died, 4 abroad, 1 too ill; the rest unlocated or refused). Those who were reinterviewed were more likely to be female, high school graduates and work full-time than those who did not participate in the 10-year follow-up. No significant differences were seen for marital status, age at drinking onset, family history of alcoholism, history of drug use, or drinking frequency. Of the 777 who returned the SAQ at baseline, 658 (84.7%) were re-interviewed and had both a baseline SAQ and 10-year diagnosis information available. Compared to the 119 who were not reinterviewed, the 658 who were reinterviewed were significantly more likely to be white, female, work full-time, have a high school education and have a lifetime diagnosis of alcohol abuse or dependence ($p < 0.05$). Of these 658 with a baseline SAQ and a 10-year follow-up interview, 235 (35.7%) had a current and/or past diagnosis of DSM-IV alcohol dependence at baseline, leaving 423 respondents who remained at risk of developing alcohol dependence. We analyzed onset of dependence in these 423 respondents. After excluding those who ever had dependence or had developed dependence at the 10-year follow-up, 301 respondents remained who had never met the criteria for a diagnosis of alcohol dependence and who had not met criteria for alcohol abuse at baseline. We analyzed onset of abuse in these 301 respondents.

### *Outcome Measures: Alcohol Use Disorders*

DSM-IV alcohol abuse and dependence were assessed with the NIAAA Alcohol Use Disorder and Associated Disabilities Interview Schedule - DSM-IV Version (AUDADIS-IV), a fully-structured diagnostic interview for nonclinician interviewers. The AUDADIS-IV includes an extensive list of symptom questions that operationalize DSM-IV criteria for alcohol abuse and dependence. In contrast to other epidemiologic instruments that use DSM-IV alcohol abuse as a screener for DSM-IV alcohol dependence (Kessler and Ustun, 2004), the AUDADIS-IV ascertains data on all criteria for DSM-IV alcohol abuse and dependence criteria independently, allowing full identification of alcohol dependent individuals regardless of whether they met criteria for abuse. The discriminant, concurrent, convergent, construct and population validity of the AUDADIS-IV alcohol and drug use disorder diagnoses have been well documented, including work done in this sample as well as in a World Health Organization/National Institutes of Health (WHO/NIH) reliability and validity study (Grant et al., 1995, 2003; Hasin et al., 1996, 1997a; World Health Organization, 1992). In the present study, those who met the DSM-IV criteria for alcohol dependence at the 10-year follow-up interview were coded as 1 and 0 otherwise. We excluded from the study of new onset alcohol dependence those with a DSM-IV alcohol dependence diagnosis at baseline. Those who met the DSM-IV criteria for alcohol abuse at the 10-year point were coded as 1 and 0 otherwise. We excluded from the study of new onset alcohol abuse those with either an alcohol abuse diagnosis at baseline or those who developed dependence during the 10-year follow-up.

### *Measures of Predictors*

Family history of alcohol problems was a binary variable based on whether the participant had a parent or sibling with a history of problem drinking. This has been shown to have good reliability (Grant et al., 1995; Hasin et al., 1997a). Because few respondents had children in the age of risk for alcohol problems at the baseline interview, and because parents often do not know about substance use in their children (Fisher et al., 2006), children were not included in the family history variable.

The Reasons for Drinking Scale (RDS) (Carpenter and Hasin, 1998b) is a 35 item Likert-style questionnaire of drinking motives whose items have values ranging from 1 (agree strongly) to 5 (disagree strongly). Items are reverse-coded during scoring so that higher scores represent higher agreement with the item. It consists of 4 factors (Carpenter and Hasin, 1998a,b): (1) drinking for social facilitation (Cronbach's $\alpha$ = 0.87), (2) drinking to reduce negative affect ($\alpha$ = 0.83), (3) drinking because of social pressure ($\alpha$ = 0.76) and (4) drinking for enhancement of positive affect (enjoyment) ($\alpha$ = 0.67). The scale used here is based on social learning constructs as applied to substance use (Hilton, 1987). These constructs have been shown to predict DSM-III alcohol abuse and dependence criteria (Cooper et al., 1988; Smith et al., 1993).

Potentially confounding binary variables included gender (male = 1) (Crum et al., 2005; Hasin et al., 2007), race (nonwhite = 1) (Harford and Muthen, 2001; Hasin et al., 2007), marital status (unmarried = 1) (Crum et al., 2005; Harford and Muthen, 2001; Hasin et al., 2007), early initiation of regular drinking (before age 15 = 1) (Grant and Dawson, 1997), an alcohol abuse diagnosis at baseline (yes = 1) (Hasin and Grant, 2004), and history of drug use (yes, indicating any illicit drug or any prescription drug on his or her own at least 12 times = 1) (Crum et al., 2005; Hasin et al., 2007). Potentially confounding continuous variables included age at time of baseline interview (Crum et al., 2005; Hasin et al., 2007), age at initiation of regular drinking (Hingson et al., 2006; Pitkanen et al., 2005), and frequency of drinking at least 5 drinks at 1 time (binge drinking) (Hasin et al., 2001) in the year prior to baseline. Age was log transformed to improve normality. Age at initiating regular drinking was

dichotomized based on the increased risk of dependence in those who begin drinking prior to age 15 (Grant and Dawson, 1997) in the bivariate analysis and used as a continuous variable to test for differences in distributions between groups.

### *Analysis*

Descriptive statistics including t-tests and incidence odds ratios (IORs) were used to compare characteristics of those with and without new onsets of alcohol dependence at the 10-year follow-up. Spearman correlation coefficients for non-normal continuous variables and tetrachoric correlations for binary variables were used to examine relationships between the RDS variables. We used IORs from logistic regression models to estimate effects of predictors on new onset dependence and abuse. Predictors significant at the 95% confidence level in bivariate analyses were evaluated for inclusion in multivariable models. Each of the RDS variables displayed very different non-normal distributional forms. Because of the highly skewed and kurtotic distributions of the RDS variables, they were dichotomized at the medians obtained from the baseline sample that responded to the SAQ (MacCallum, 2002).

We first explored the relationship between family history and each RDS variable in bivariate models using 4 groups (a 2×2 analysis) obtained from combining the binary family history variable and each binary RDS variable. We used dummy variables to represent these 4 groups: (1) negative family history and below the median of the RDS variable; (2) negative family history and above the median on the RDS variable; (3) positive family history and below the median of the RDS variable; and, (4) positive family history and above the median on the RDS variable. To estimate effects across the different groups on new-onset alcohol dependence, we used the group with no family history and below the RDS median score as the reference group and calculated IORs to compare each of the 3 other groups to this reference group.

Next, in order to determine the interaction of family history with reasons for drinking in predicting alcohol dependence and abuse, we used logistic regression models, both unadjusted and adjusted for potential confounders. The interaction term was defined as the cross-product of the binary family history variable and the binary RDS variable dichotomized at the median (a 2×2 analysis). In this case, the reference group included either those positive for family history or those who scored above the median on the RDS. By doing so, we compared those with a family history and being above the median on the RDS to the reference group, which in this case contained everyone else. Ideally, the next step after obtaining a significant interaction would be to conduct separate analyses of the RDS effects within the family history positive and family history negative groups. However, as the number of new onsets did not allow sufficient power for this type of analysis, we sought a different analytic approach to conduct the final step of further evaluating the group differences identified by the significant interactions in the logistic regression models.

The approach we used for this final step of the analysis was to compare the magnitude of the association with dependence between those scoring low and high on the RDS in the background of a positive family history. We conditioned reasons for drinking on a family history of alcoholism, including only cross-product interactions that were significant in the first analysis. The approach taken was designed to address the question of how strongly the relationship between the alcohol disorder used as the outcome (alcohol dependence or abuse) and the RDS variable depended on a family history of alcohol problems. We did this by creating 2 dummy variables, 1 representing respondents with RDS above the median and positive family history, and the other representing respondents with RDS below the median and positive family history. In contrast to the reference group described above, the reference group in this analysis consisted of those who reported no family history of alcoholism, regardless of their reason for drinking score. This resulted in 3 mutually exclusive groups. We used this procedure for each RDS variable.

Because of individual heterogeneity in the RDS variables and the clinical implications of identifying at-risk individuals, analyses were done using a separate model for each RDS variable. Nearly half (45%) of the population scored above the median on only 1 (21%) or 2 (24%) of the 4 variables. Therefore, we hypothesized that these reasons might predict dependence differently in a background of family history and we might lose information by combining the social facilitation and enjoyment reasons into a positive motive and the negative affect and social pressure into a negative motive.

The same analyses were conducted for new onset diagnoses of abuse at the 10-year follow-up, excluding those who developed dependence during follow-up. All analyses were performed in SAS, version 9.1 (SAS institute, Inc., Cary, NC) and all confidence intervals are at the 95% confidence level and *p*-values are 2-sided with significance set at .05.

## RESULTS

### *The Sample at Baseline*

The mean age of the 423 respondents without a lifetime diagnosis of DSM-IV alcohol dependence at baseline in 1991 to 1992 was 34.0 years (se 11.6; range 18 to 67). Women constituted nearly half (46.8%) of the sample, illustrating success in creating a gender-balanced sample. Of the sample, 83.9% were white, with the remainder largely African-American. Nearly all (94.8%) had finished high school, 73.5% worked full-time and 42.6% were married. A current diagnosis of alcohol abuse was found in 11.8%, 41.1% had a history of drug use, 17.5% had ever had a major depressive episode and 34.0% had a family history of alcohol problems in first-degree relatives (parents or siblings). At baseline, 14.7% of the respondents drank ≥5 drinks per occasion at least weekly and 28.8% drank ≥5 drinks per occasion at least twice a week.

At baseline, RDS medians were: drinking for social facilitation, 2.86 (range 1.0 to 5.0), to reduce negative affect, 1.50 (range 1.0 to 4.8), for positive effect (enjoyment), 3.50 (range 1.0 to 5.0), and because of social pressure, 1.33 (range 1.0 to 4.0). Spearman correlations between the RDS variables ranged from 0.21 to 0.51. Among these, the highest correlations were found between negative affect and social facilitation (0.49), and negative affect and social pressure (0.51). After median splits, tetrachoric correlations ranged from 0.20 to 0.60. Negative affect remained most highly correlated with social facilitation (0.56) and social pressure (0.60). Tetrachoric correlations between enjoyment and negative affect were 0.36; social facilitation and enjoyment, 0.44; social facilitation and social pressure, 0.41; and enjoyment and social pressure, 0.20.

### *The Sample at 10-Year Follow-Up: New-Onset Alcohol Dependence*

Compared to those without new-onset dependence at 10 years, those with new-onset dependence were younger (mean and SD: 30.2 ± 9.6 and 34.7 ± 11.8, $p < 0.01$) and had a greater frequency of 5 or more drinks per month in the

**Table 1.** Baseline Characteristics of New Jersey Community Residents That Did ($n$ = 66) and Did Not ($n$ = 357) Develop First-Time Onset of Alcohol Dependence at 10-Year Follow-Up

| Respondent characteristic | Not dependent at follow-up $n$ (%) | Dependent at follow-up $n$ (%) | OR (95% CI) |
|---|---|---|---|
| Gender | | | |
| Female | 166 (46.5) | 32 (48.5) | 1.00 |
| Male | 191 (53.5) | 34 (51.5) | 0.92 (0.55, 1.56) |
| White race | | | |
| Yes | 300 (84.0) | 55 (83.3) | 1.00 |
| No | 57 (16.0) | 11 (16.7) | 0.95 (0.47, 1.93) |
| Married | | | |
| Yes | 157 (44.0) | 23 (34.8) | 1.00 |
| No | 200 (56.0) | 43 (65.2) | 1.47 (0.85, 2.54) |
| Began drinking at age <15 | | | |
| No | 322 (90.2) | 62 (93.9) | 1.00 |
| Yes | 35 (9.8) | 4 (6.1) | 0.59 (0.20, 1.73) |
| History of drug use | | | |
| No | 219 (61.3) | 30 (45.4) | 1.00 |
| Yes | 138 (38.7) | 36 (54.6) | 1.90 (1.12, 3.23) |
| Abuse diagnosis at baseline | | | |
| No | 320 (89.6) | 53 (80.3) | 1.00 |
| Yes | 37 (10.4) | 13 (19.7) | 2.12 (1.06, 4.25) |
| Lifetime depression | | | |
| No | 303 (84.9) | 46 (69.7) | 1.00 |
| Yes | 54 (15.1) | 20 (30.3) | 2.44 (1.34, 4.44) |
| Family history of alcoholism | | | |
| No | 245 (68.6) | 34 (51.5) | 1.00 |
| Yes | 112 (31.4) | 32 (48.5) | 2.06 (1.21, 3.51) |
| Reasons for Drinking Scale: social facilitation | | | |
| < median of 2.86 | 219 (61.3) | 30 (45.4) | 1.00 |
| ≥ median of 2.86 | 138 (38.7) | 36 (54.6) | 1.90 (1.12, 3.23) |
| Reasons for Drinking Scale: enjoyment | | | |
| < median of 3.50 | 214 (59.9) | 30 (45.4) | 1.00 |
| ≥ median of 3.50 | 143 (40.1) | 36 (54.6) | 1.80 (1.06, 3.05) |
| Reasons for Drinking Scale: negative affect | | | |
| < median of 1.50 | 183 (51.3) | 24 (36.4) | 1.00 |
| ≥ median of 1.50 | 174 (48.7) | 42 (63.6) | 1.84 (1.07, 3.17) |
| Reasons for Drinking Scale: social pressure | | | |
| < median of 1.33 | 181 (50.7) | 25 (37.9) | 1.00 |
| ≥ median of 1.33 | 176 (49.3) | 41 (62.1) | 1.69 (0.98, 2.89) |
| Family history and social facilitation (< or ≥ median) | | | |
| No family history, < median | 152 (42.6) | 20 (30.3) | 1.00 |
| No family history, ≥ median | 93 (26.0) | 14 (21.2) | 1.14 (0.55, 2.37) |
| Family history, < median | 67 (18.8) | 10 (15.2) | 1.13 (0.50, 2.55) |
| Family history, ≥ median | 45 (12.6) | 22 (33.3) | 3.72 (1.86, 7.42) |
| Family history and enjoyment (< or ≥ median) | | | |
| No family history, < median | 142 (39.8) | 17 (25.7) | 1.00 |
| No family history, ≥ median | 103 (28.8) | 17 (25.8) | 1.38 (0.67, 2.83) |
| Family history, < median | 72 (20.2) | 13 (19.7) | 1.51 (0.69, 3.28) |
| Family history, ≥ median | 40 (11.2) | 19 (28.8) | 3.97 (1.89, 8.34) |
| Family history and negative affect (< or ≥ median) | | | |
| No family history, < median | 122 (34.2) | 16 (24.2) | 1.00 |
| No family history, ≥ median | 123 (34.4) | 18 (27.3) | 1.12 (0.54, 2.29) |
| Family history, < median | 61 (17.1) | 8 (12.1) | 1.00 (0.41, 2.47) |
| Family history, ≥ median | 51 (14.3) | 24 (36.4) | 3.59 (1.76, 7.31) |
| Family history and social pressure (< or ≥ median) | | | |
| No family history, < median | 124 (34.7) | 14 (21.2) | 1.00 |
| No family history, ≥ median | 121 (33.9) | 20 (30.3) | 1.46 (0.71, 3.03) |
| Family history, < median | 57 (16.0) | 11 (16.7) | 1.71 (0.73, 4.00) |
| Family history, ≥ median | 55 (15.4) | 21 (31.8) | 3.38 (1.60, 7.14) |

12 months prior to the baseline interview (mean ± SD: dependence 5.9 ± 2.2; no dependence 4.7 ± 2.3; $p < 0.0001$). Those who did and did not develop new-onset DSM-IV alcohol dependence also differed significantly on history of drug use, having a current DSM-IV alcohol abuse diagnosis at baseline, history of depression, and family history of alcohol problems (Table 1). However, the age at onset of regular drinking did not differ significantly between the groups (mean ± SD: dependence 18.0 ± 3.7; no dependence 17.5 ± 2.9; $p = 0.09$).

In bivariate analyses (Table 1), having a family history of alcoholism, drinking for social facilitation, drinking for enjoyment and drinking to reduce negative affect were significantly associated with new onset DSM-IV alcohol dependence, but drinking because of social pressure was not (Table 1). The associations between drinking for social facilitation and new-onset alcohol dependence became much more striking after creating 4 mutually exclusive groups representing the 4 possible combinations of family history (positive or negative) and drinking for social facilitation (above or below the median; Table 1). The same pattern was also found for the remaining 3 drinking motives variables (Table 1). In analyses designed to examine the differences in each group compared to the reference group, individuals with a new onset dependence diagnosis were much more likely to have a positive family history of alcoholism and be above the median on each RDS variable compared to those who did not develop alcohol dependence (Table 1). Because family history was not associated with any of the drinking motives, a mediation model of the relationship between family history, drinking motives and onset of an alcohol disorder was ruled out.

Because of the significant associations that family history and the drinking motives (RDS variables) showed with new-onset dependence (Table 1), interactions between family history and each RDS variable were tested in separate logistic regression models (Table 2). History of drug use and depression were not significant in multivariable models and did not influence the effect sizes of the variables of interest. Thus, we chose to drop them from the models. However, because gender and race are generally adjusted for in studies of AUDS and to permit comparison with other studies, we chose to leave these covariates in the models even though they were not significant and did not influence on the variables of interest. Main effects models without the interactions are provided for those interested (Table 2). After adjusting for gender, race, age and binge drinking, logistic regression models showed that family history interacted significantly with drinking for social facilitation and drinking to reduce negative affect (Table 2), effects that were strengthened after controlling for potential confounding.

Table 3 shows the breakdown of the specific effects of social facilitation and drinking to reduce negative affect conditioned on family history of alcohol problems. After conditioning social facilitation and drinking to reduce negative affect on family history of alcohol problems, being above the median score on drinking for social facilitation or drinking to reduce negative affect greatly elevated the relative risk for new onset alcohol dependence, a relationship that was strengthened after adjustment for covariates.

**Table 2.** Incidence Odds Ratios (IORs) and 95% Confidence Intervals From Univariate and Multivariable Main Effects and Interaction Models of Family History, Reason for Drinking and the Interaction of Family History and Reason for Drinking[a] in Predicting New Onset Alcohol Dependence in 423 New Jersey Residents

| Variable | Unadjusted model IOR (95% CI) | Adjusted model[b] IOR (95% CI) |
|---|---|---|
| Model 1: main effects | | |
| Positive family history | 1.98 (1.16, 3.38) | 2.29 (1.30, 4.04) |
| Social facilitation | 1.82 (1.07, 3.11) | 1.89 (1.07, 3.31) |
| Model 1: interaction effects | | |
| Positive family history | 1.13 (0.50, 2.55) | 1.14 (0.49, 2.65) |
| Social facilitation | 1.14 (0.55, 2.37) | 1.05 (0.49, 2.24) |
| Family history[a] social facilitation | 2.86 (0.94, 8.70) | 3.88 (1.21, 12.5) |
| Model 2: main effects | | |
| Positive family history | 2.06 (1.21, 3.52) | 2.34 (1.33, 4.12) |
| Reduce negative affect | 1.84 (1.06, 3.18) | 1.71 (0.97, 3.01) |
| Model 2: interaction effects | | |
| Positive family history | 1.00 (0.41, 2.47) | 1.11 (0.44, 2.82) |
| Reduce negative affect | 1.12 (0.54, 2.29) | 1.02 (0.49, 2.14) |
| Family history[a] negative affect | 3.22 (1.03, 10.0) | 3.38 (1.05, 10.9) |
| Model 3: main effects | | |
| Positive family history | 2.11 (1.23, 3.60) | 2.38 (1.36, 4.19) |
| Enjoyment | 1.84 (1.08, 3.15) | 1.35 (0.76, 2.38) |
| Model 3: interaction effects | | |
| Positive family history | 1.51 (0.69, 3.28) | 1.55 (0.69, 3.48) |
| Enjoyment | 1.38 (0.67, 2.83) | 0.93 (0.43, 1.97) |
| Family history[a] enjoyment | 1.91 (0.65, 5.61) | 2.31 (0.75, 7.13) |
| Model 4: main effects | | |
| Positive family history | 2.05 (1.20, 3.50) | 2.33 (1.32, 4.11) |
| Social pressure | 1.68 (0.97, 2.88) | 1.61 (0.92, 2.85) |
| Model 4: interaction effects | | |
| Positive family history | 1.71 (0.73, 4.00) | 1.93 (0.80, 4.66) |
| Social pressure | 1.46 (0.71, 3.03) | 1.40 (0.66, 2.98) |
| Family history[a] social pressure | 1.35 (0.45, 4.04) | 1.38 (0.44, 4.27) |

[a]Reason for drinking variable based on median split.
[b]Adjusted for gender, race, age and binge drinking.

### *New-Onset Alcohol Abuse*

Among those who had never met the criteria for a diagnosis of alcohol abuse at baseline and never developed alcohol dependence ($n = 301$), 73 had new-onset abuse by the 10-year follow-up. Bivariate analyses showed being male ($p < 0.0001$) being younger ($p = 0.04$), being a high school graduate ($p = 0.04$), drinking for enjoyment ($p = 0.02$), drinking prior to age 15 ($p = 0.04$), having a history of drug use ($p < 0.001$) and binge drinking ($p < 0.0001$) significantly predicted new onset alcohol abuse. Importantly, family history of alcoholism was not associated with alcohol abuse. In multivariable models controlling for race, age and education, only being male (OR 2.58; CI 1.38, 4.83), history of drug use (OR 2.10; CI 1.17, 3.80) and binge drinking (OR 1.35; CI 1.18, 1.54) remained significantly associated with alcohol abuse. Binge drinking explained the association of abuse to both drinking for enjoyment and drinking prior to age 15; with binge drinking in the model, these 2 variables became nonsignificant. No interactions between reasons for drinking and family history were observed.

## DISCUSSION

In a community sample of at-risk adult drinkers, this prospective study examined the conjoint relationship between motivations for drinking at baseline and family

**Table 3.** Univariate and Multivariable Models of Reasons for Drinking at Baseline Predicting New Onset Alcohol Dependence at 10 Years in 423 New Jersey Residents Without a Diagnosis of Alcohol Dependence at Baseline Conditioning Reasons for Drinking on Having a Positive Family History of Alcohol Problems[a]

| | IOR (95% CI) |
|---|---|
| Model 1: drinking for social facilitation | |
| *Unadjusted model:* | |
| No family history of alcoholism | reference |
| Positive family history and < median | 1.08 (0.51, 2.29) |
| Positive family history and ≥ median | 3.52 (1.89, 6.57) |
| *Adjusted model:* | |
| No family history of alcoholism | reference |
| Positive family history and < median | 1.12 (0.51, 2.47) |
| Positive family history and ≥ median | 4.54 (2.31, 8.93) |
| Male | 0.75 (0.42, 1.34) |
| White | 0.87 (0.41, 1.87) |
| Age | 0.26 (0.10, 0.66) |
| Binge drinking | 1.29 (1.13, 1.46) |
| Model 2: drinking to reduce negative affect | |
| *Unadjusted model:* | |
| No family history of alcoholism | reference |
| Positive family history and < median | 0.95 (0.42, 2.15) |
| Positive family history and ≥ median | 3.39 (1.86, 6.20) |
| *Adjusted model:* | |
| No family history of alcoholism | reference |
| Positive family history and < median | 1.10 (0.47, 2.58) |
| Positive family history and ≥ median | 3.79 (2.00, 7.18) |
| Male | 0.82 (0.46, 1.46) |
| White | 0.98 (0.45, 2.10) |
| Age | 0.27 (0.11, 0.69) |
| Binge drinking | 1.26 (1.11, 1.43) |

[a]Having a positive family history and being below the median on the Reasons for Drinking Scale (RDS) was coded as 1 and 0 otherwise. A positive family history and being at or above the median on the RDS was coded 1 and 0 otherwise.

history in predicting new onsets of DSM-IV alcohol dependence or abuse during the 10-year follow-up. The results showed that family history modified the relationship of drinking motivations to alcohol dependence. Specifically, in the final interaction models, family history significantly moderated the effects of 2 drinking motives: drinking to reduce negative affect and drinking for social facilitation. This was a strong effect: in the presence of a positive family history, those above the median on drinking for social facilitation and drinking to reduce negative affect had a 4-fold increase in risk for new-onset alcohol dependence compared to those below the median on these scales. In contrast, those with a positive family history who were below the median on the drinking motives scales had no increase in risk for alcohol dependence.

Our study is the first to prospectively report that adults with a family history of alcoholism and high scores (i.e., above the median) on the RDS were more likely to become alcohol dependent. Cross-sectional studies of adolescents showed similar results: among adolescents, drinking to cope with personal dissatisfaction was associated with greater alcohol use, but only among those with a positive family history of alcoholism (Chalder et al., 2005; Mann et al., 1987). These cross-sectional studies of drinking among adolescents differed in design, population, and drinking outcome variables from our study. However, the consistency of results on the heightened risk for alcohol dependence from drinking to cope with negative emotions among those with a family history of alcohol problems suggests that this relationship is important and merits further attention and study.

In the interaction models, inclusion of age and frequency of binge drinking (Table 2) had different effects depending on the reason for drinking analyzed. In the model that included drinking for social facilitation, inclusion of age and frequency of binge drinking only slightly altered the IOR of the main effects (family history and RDS variable), but strengthened the interaction term between family history and social facilitation, increasing it by 35.0%. However, age and binge drinking increased the IOR for the cross-product interaction term between family history and negative affect by only 5.0% (Table 2). Therefore, younger age and binge drinking appear mainly to affect the relationship between having a positive family history of alcoholism and drinking for social facilitation in predicting alcohol dependence, but not the relationship between family history and drinking to cope with negative affect.

With only 66 cases of new onset dependence, power was lacking to examine a threshold effect for the interaction between positive family history and the RDS variables. The striking difference in IORs between those below and above the median among those with a positive family history suggested that a threshold might exist. We re-analyzed the data using a 75th percentile cut-off rather than the median for each RDS variable. If the relationship had been linear, use of the 75th percentile should have strengthened the interaction term, but this did not occur, suggesting that all those above the median were contributing equally to the increased risk of dependence in the presence of a positive family history. This analysis further supported the use of a median as a cutpoint that captures a low and high level of motivation without loss of information.

Drinking motives predicted alcohol dependence, but not alcohol abuse in our study. If those who drink for enjoyment are more likely to drink outside the home, leading to hazardous use [one of the most common ways to receive a diagnosis of DSM-IV alcohol abuse (Hasin and Paykin, 1999)], then the lack of relationship of this motive to alcohol abuse might seem surprising. The bivariate analyses of abuse showed a positive association with drinking for enjoyment, but lost significance in the multivariable model that included frequency of binge drinking at baseline. In the case of dependence, inclusion of binge drinking frequency in the multivariable model acted independently in predicting dependence, but also strengthened the interaction of family history with drinking motives. Thus, binge drinking seems to largely account for a DSM-IV diagnosis of alcohol abuse, while the contributing factors to alcohol dependence are more complex, as might be expected given the common understanding of alcohol dependence as a complex disorder. Future work should continue to differentiate between abuse and dependence as outcome variables in addressing familial or genetic etiology.

Our result that in the presence of a family history of alcoholism, drinking to reduce negative affect and drinking for social facilitation markedly increases an individual's risk for developing alcohol dependence was found in a sample where all respondents (male and female) drank 5+ drinks at least once in the prior year. This information could be further explored for clinical preventive purposes in medical treatment settings. For example, in NIAAA Clinician's Guide to helping patients that drink too much (http://pubs.niaaa.nih.gov/publications/Practitioner/CliniciansGuide2005/clinicians_guide.htm, accessed 09/25/07, NIAAA, 2005) one of the key indicators of at-risk drinking for men is drinking 5+ drinks in a day. If medical patients report such drinking but do not yet meet criteria for alcohol dependence, clinicians could ask a few additional questions to establish whether patients have a family history, and whether they drink to feel better when feeling low, and/or to facilitate social interaction. If the responses to these questions are also positive, patients could be cautioned that they are at substantially increased risk of becoming alcohol dependent. Thus, the information from this study has the potential to serve an important clinical preventive function. Future studies will be needed to transform and validate the measures used in this study into brief questions easily posed by a clinician in a medical interview.

The limitations of this study include the self-reported nature of family history, although such reports have been shown to be reliable in previous studies (Hasin et al., 1997a; Prescott et al., 2005). Also, not all respondents completed the SAQ that included the drinking motives questionnaire at baseline. While respondents that did and did not complete the SAQ were similar on most variables, the difference on race/ethnicity suggests that the study results may not generalize to African-American samples. Finally, some of our variables, such as age at onset of regular drinking, may be subject to recall bias, as is always the case in cohorts not followed since childhood. However, the prospective study design improved considerably on previous cross-sectional examinations of the variables we studied. We note also that the results of this paper may not be generalizable to younger samples. This study was specifically designed to address whether drinking motivations predict new onset dependence in adults because there is a lack of such studies in the literature. However, our results were quite consistent with the earlier cross-sectional studies in adolescents and young adults, suggesting some degree of generalizability.

Another possible limitation is the overlap found in the drinking motives. Unfortunately, we were unable to classify individuals using mutually exclusive groups because some respondents scored high on several drinking motives. To address the effects of multiple drinking motives, we investigated whether the risk of new onset dependence increased with the number of drinking motivations, using a count variable of number of motive scores above the median. The risk increased with each additional motive in adjusted models (IRR 1.31; CI 1.06, 1.61). The interaction term with family history was significant (IOR: 2.31; 1.31, 4.08) but the effect was smaller than for the individual motives. When drinking for social facilitation and to reduce negative affect were used in the same model as main effects, the effect sizes were somewhat reduced and social facilitation became insignificant (social facilitation: IOR 1.71, CI 0.94, 3.12; negative affect 1.42, CI 0.78, 2.60), although each showed its effects most strongly in the presence of family history in any case.

A substantial strength of this study was the ability to prospectively examine predictors of new-onset cases using data on drinking motives obtained at baseline in a group of adult, high-risk drinkers. Motivations were measured prior to any diagnosis of abuse or dependence. The 10-year follow-up provides an adequate trajectory for the risk of dependence in the adult years. The effects observed in this study for dependence were strong and consistent, with reasonable precision around the estimators. This study also examined new onset abuse in those without new onset dependence. Very different risk factors emerged compared to those found for dependence, including being male, a history of drug use and binge drinking, with no associations with family history of alcoholism. These risk factors suggest a considerably different underlying mechanism for the development of alcohol abuse, adding to the literature indicating that combining these categories for purposes of diagnosis will result in an alcohol variable with considerably heterogeneity, undesirable in etiologic research.

This longitudinal study demonstrates an interaction between drinking motives and family history in predicting new onsets of DSM-IV alcohol dependence in a community sample of adults. In addition, it suggests that in the presence of a family history of alcoholism, scoring high on scales that measure drinking to reduce negative affect and drinking for social facilitation puts an individual at much higher risk for dependence than those who score lower on these scales. This result indicates that at least some of those who have a positive family history and are at risk of becoming alcohol dependent can be identified early, when intervention might prevent dependence. Finally, further exploration of the interaction of drinking motives with measured genetic polymorphisms in predicting the onset of DSM-IV alcohol dependence may provide much-needed clarification on the role of genetic and cognitive-motivational factors in the etiology of alcohol dependence.

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