

Density of Familial Alcoholism and Its Effects on Alcohol Use and Problems in College Students

Christy Capone and Mark D. Wood

Background: Previous studies of family history of alcoholism (FHA) in college students have typically relied on dichotomous indices of paternal drinking. This study examined the prevalence of FHA and its effects on alcohol use and problems using a density measure in a sample ($n = 408$) of college students.

Methods: Undergraduate students completed an anonymous survey in exchange for course credit. Data was collected between 2005 and 2006.

Results: Using a density measure of FHA, we observed an overall prevalence rate of 65.9% and a rate of 29.1% for FHA in both first and second-degree relatives. Structural equation modeling (SEM) was used to investigate relations among FHA, alcohol use/problems and previously identified etiological risk factors for alcohol use disorders (AUD). Results indicated a significant positive association between FHA and alcohol-related problems and this relationship was mediated by age of onset of drinking, behavioral undercontrol and current cigarette use. Behavioral undercontrol also mediated the relationship between gender and alcohol problems. Additionally, FHA was associated with an earlier age of onset of drinking and this was related to greater alcohol use.

Conclusions: Assessing density of FHA in future trajectory research may capture a greater number of students at risk for acute alcohol-related problems and/or future development of AUDs. Future preventive interventions with this population, which should begin well before the college years, may benefit from considering personality factors and incorporating smoking cessation to help identify at-risk students and assist those who wish to cut down on their alcohol use but find that smoking acts as a trigger for increased drinking.

Key Words: Alcohol, Family History of Alcoholism, College Students, Smoking.

IT IS WELL documented that most college students will “mature out” of heavy drinking by young adulthood, while, for others, this pattern of drinking will persist (e.g., Jackson et al., 2001; O’Neill et al., 2001; Schulenberg et al., 1996). Several studies examining the course of binge drinking during adolescence and the college years provide consistent evidence of distinct trajectories during these developmental periods (e.g., Hill et al., 2000a; Schulenberg et al., 1996; Tucker et al., 2003). Among those who do not “mature out” of heavy drinking are individuals at risk for development or persistence of alcohol use disorders (AUDs). For example, individuals with an earlier onset of binge drinking may be at risk for alcohol problems consistent with Cloninger’s (1987) Type II or Babor and colleagues (1992) Type B alcoholism, characterized by greater severity of dependence, higher levels of comorbid psychopathology and polydrug use, and family

history of alcoholism (Chassin et al., 2002). Since membership in different trajectory groups is not easily discernible during the college years (Schulenberg et al., 1996), the ability to not only identify, but also to predict, who will belong to these “at-risk” trajectory groups is clearly imperative. Indeed, an examination of risk factors related to trajectory group or drinking patterns is an essential part of informing new interventions and refining existing ones.

One of the most widely recognized risk factors for the development of problematic drinking is familial alcoholism. There is substantial evidence that individuals with a family history of alcoholism (FHA) are at greater risk for developing alcohol use disorders themselves (Dawson et al., 1992; Schuckit, 1998, 2000; Sher et al., 1991). Two recent longitudinal studies, utilizing high-risk designs (i.e., oversampling on positive family history status), found significant associations between FHA and risk of AUD (Chassin et al., 2002). At the same time, some researchers make a distinction between the utility of a dichotomous (present vs. absent) family history construct and the notion of familial *density*, with the latter being a more useful index of genetic liability for alcohol dependence. In fact, individuals with only 1 affected relative (even a parent) may be at relatively low risk when compared with those with greater density of alcoholism among relatives (Hill and Yuan, 1999). Thus, while previous studies

From the Department of Psychology, University of Rhode Island, Kingston, Rhode Island.

Received for publication November 9, 2007; accepted April 12, 2008.

Reprint requests: Christy Capone, PhD, Center for Alcohol and Addiction Studies, Brown University, Box G-121S-4, Providence, RI 02912; Fax: 401-863-6647; E-mail: Christy_Capone@brown.edu

Copyright © 2008 by the Research Society on Alcoholism.

DOI: 10.1111/j.1530-0277.2008.00716.x

examining FHA provide valuable information regarding its relationship to binge drinking, their findings may be limited by reliance on a dichotomous indicator of parental, typically paternal, alcoholism.

By contrast, when assessing familial density of alcoholism, first, second, and third degree relatives are typically included, resulting in a multilevel construct (e.g., Chermack et al., 2000). Using this method, Dawson et al. (1992) looked at probability of past year alcohol dependence among drinkers from a nationally representative household sample and found that the odds of dependence increased dramatically with greater familial density. Specifically, in comparison to those with no family history of alcoholism, individuals with alcoholism in second or third degree relatives had 45% greater odds of dependence, those with alcoholism in first degree relatives had 86% greater odds, and those with the greatest density (first and second or third degree relatives) had the greatest odds of dependence (167%). Notably, these odds ratios remained consistent after controlling for gender, age, race, and socioeconomic status.

Research examining the prevalence of FHA specifically among college students is limited. Most college drinking studies that incorporate assessment of FHA do so in the context of a high-risk design (e.g., Marlatt et al., 1998; Sher et al., 1991). Moreover, it is often difficult to make comparisons between non-high-risk studies due to the discrepant methods of measuring FHA. For example, Perkins and Berkowitz (1991) measured FHA by asking about formal diagnosis and treatment of AUD and observed an overall prevalence rate of approximately 15%. Weitzman and Wechsler (2000), in a national sample of college students ($n = 17,592$), assessed parental drinking only and used the term "problem drinker" to infer FHA status, yielding a 10% prevalence rate. By contrast, national household studies (e.g., Dawson et al., 1992; Harford, 1992) have typically utilized density measures of FHA, resulting in higher prevalence estimates, approximately 38% overall in Harford's study. To our knowledge, research examining familial density of alcoholism among college students is lacking.

While there is compelling evidence that familial density is a strong influence in the etiology of alcohol use disorders, the mechanisms underlying this relationship are less clear, leading to the question, *how* does familial alcoholism affect the drinking behavior of offspring? While this question has been widely researched in studies assessing alcoholism among first-degree relatives, much less research has been conducted using measures of familial density. This was an important objective of the current study.

Underlying Mechanisms of Familial Alcoholism

Alcoholism is a complex disorder with both genetic and environmental components. It is estimated that genes explain between 40 and 60% of the variance in alcoholism risk and that the genetic influences are themselves heterogeneous (Schuckit, 2000). Research examining FHA among first-

degree relatives suggests several mechanisms by which FHA affects drinking outcomes. First, reduced sensitivity to the effects of alcohol has been implicated in the etiology of alcohol dependence. For example, Schuckit (1994, 1998) found that *level of response* (LR) to alcohol mediated the relationship between FHA and alcoholism risk among sons of alcoholics. Additionally, there is evidence that LR is genetically influenced among men (Newlin and Thomson, 1991); however, evidence that female offspring exhibit a lower level of response to alcohol has not been as strong (Evans and Levin, 2003; Heath et al., 1999). Second, there is considerable evidence that adolescents with FHA have an earlier *onset of alcohol use* than their non-FHA peers (Dawson, 2000; Hill and Yuan, 1999; Hill et al., 2000b), potentially setting the stage for development of an AUD in early adulthood. Third, Sher et al. (1991) found that *behavioral undercontrol*, a construct incorporating such personality characteristics as impulsivity, aggressiveness, and extraversion, mediated the relationship between paternal alcoholism and drinking outcomes among offspring. Finally, the comorbidity between alcohol use disorders and *tobacco dependence* is amply documented (Bien and Burge, 1990; Myerhoff et al., 2006; Sher et al., 1996) and previous research has shown a link between FHA and both alcohol and tobacco use disorders among college students (Jackson et al., 2000). These findings have been explained in the context of a common vulnerability to both disorders as well as a pattern of reciprocal influence in which each affects the other over time (Sher et al., 1996). To date, associations among density of FHA, alcohol use and these previously identified constructs have not been examined in a college student sample.

The present study addresses unresolved issues regarding assessment of FHA and sampling. Our use of a density measure is in contrast to most previous research with college students that assessed paternal alcoholism only (Evans and Levin, 2003; Schuckit, 1998; Sher et al., 1991) and our inclusion of a mixed gender sample contrasts with earlier studies that included male offspring only (e.g., Schuckit, 1998) based on the recently refuted assumption that heritability for alcoholism is greater among males (Finn et al., 2000). Indeed, there is ample evidence that genetic influences on alcoholism are as strong for women as for men (Heath, 1995; Kendler et al., 1994).

The primary aims of the present study were twofold: 1) to assess the prevalence of family history of alcoholism in a college student sample using a multilevel construct of familial density; and 2) to investigate the direct and indirect (i.e., mediational) effects of familial alcoholism on alcohol use and problems. We hypothesized that a higher overall prevalence of FHA would be observed in the current sample due to our use of a more refined measure. We further hypothesized that significant direct associations between FHA and alcohol use and problems would be observed and that these effects would be mediated by one or more of the following: age of onset of drinking, level of response to alcohol, behavioral undercontrol and cigarette use.

METHODS

Participants

Participants were 408 undergraduate college students at a mid-size university in the Northeastern United States. The average age was 18.8 (SD = 1.17) years; 71.8% of the sample were women, 87.2% were White, and the majority were freshmen (68.6%) or sophomores (20.34%). Comparison of the current sample with the entire undergraduate population at the study university indicated that underclassmen were overrepresented in the current sample, as were women (71.8% vs. 57%) and Caucasian students (87.2% vs. 75.7%).

Regarding alcohol use, 96.3% of participants reported drinking alcohol at some time during their lifetime and 90.7% had consumed more than 1 drink on a single occasion during the past year. Average age of onset of any alcohol use was 14.9 years (SD = 2.42) and age of first drunk episode was 16.1 years (SD = 1.62). For the present analyses, we included only those who reported current (i.e., past month) alcohol use. Therefore, 89 participants were excluded due to nondrinker (no alcohol use in the past month) status ($n = 86$) and unknown FHA status ($n = 3$), resulting in a sample of 319 current drinkers. Among those, the mean number of drinks per month was 74.4 (SD = 80.3) for men and 31.7 (SD = 32.3) for women. The most frequently endorsed alcohol-related consequences for both men and women were hangovers (72.8%), feeling physically ill (68.1%), and blackouts (58.6%).

Procedure

Data for this study was collected during the Fall 2005 and Spring 2006 semesters. Undergraduate students enrolled in introductory psychology and communication courses were invited to participate in a study about college student "health behaviors." Small groups ranging from 5 to 25 students provided informed consent and completed an anonymous paper-and-pencil survey. Students received course credit for their participation in the study. All study procedures were approved by the Internal Review Board (IRB) of the institution.

Measures

The survey consisted of 168 items. Many of the items have been adapted from previous measures, but some were created specifically for this study, as comparable items are not currently available in the extant literature.

Family History of Alcoholism. A density construct of familial alcoholism (FHA) was created consistent with methodology utilized by Dawson et al. (1992). Participants were queried regarding alcoholism among first-degree relatives by 3 separate items asking, "Do you think your biological [mother/father/siblings] is/was a problem drinker or alcoholic?" Participants were also asked about alcohol problems in other blood relatives (i.e., grandparents, aunts, uncles, cousins). In addition to an overall prevalence rate and 1 for paternal alcoholism, these items yielded 4 categories of FHA: 1) FH-Neg (no alcoholism in any biological relatives); 2) FHP-1 (alcoholism in second or third degree relatives only); 3) FHP-2 (alcoholism in first degree relatives only); and 4) FHP-3 (alcoholism reported in both first and second or third degree relatives).

Alcohol Use. Participants were queried regarding several aspects of their drinking history, including age of onset of any alcohol use, current quantity and frequency of consumption, and peak number of drinks on 1 occasion. These items were adapted from our previous work assessing college student drinking (e.g., Read et al., 2005). A composite alcohol use variable (monthly quantity \times frequency) was derived for the present analyses.

Alcohol-Related Consequences. Problems related to the use of alcohol were assessed using an abbreviated 17-item version of the Young Adult Alcohol Problems Screening Test (YAAPST; Hurlbut and Sher, 1992). Participants were asked to rate the past year frequency of several common negative consequences of alcohol use, including drinking and driving, feeling physically sick, neglecting responsibilities, and unwanted sexual behaviors. Items were rated on a scale ranging from 0 (*never*) to 4 (*3 or more times in the past year*). Coefficient alpha for this measure was 0.90.

Mechanisms of Familial Alcoholism. Level of response to alcohol (LR) was assessed with a 4-item version of the Self-Rating of the Effects of Alcohol (SRE; Schuckit et al., 1997a). Participants were asked to estimate the number of standard drinks needed to experience 4 different effects of alcohol: 1) recognition of any effect; 2) dizziness or slurred speech; 3) poor motor coordination (e.g., stumbling while walking); and 4) passing out. All response options were continuous. Participants were instructed to base their responses on the first 5 drinking occasions of their lifetime. The SRE has demonstrated good test-retest reliability and has been found to correlate strongly with alcohol challenge results (Schuckit et al., 1997b).

Two scales were used to assess behavioral undercontrol (BU): the Psychoticism (P) scale from the Eysenck Personality Questionnaire, Revised (EPQ-R; Eysenck, 1988) and the Impulsive Sensation Seeking (ImpSS) scale from Zuckerman and Kuhlman's Personality Questionnaire (ZKPQ; Zuckerman et al., 1993). Both scales, embedded within larger personality measures, assess impulsivity and willingness to take risks. Response options were dichotomous (true/false). The EPQ has demonstrated good reliability and validity, and the P scale in particular has been shown to predict diagnoses of AUDs both cross-sectionally and prospectively (Sher et al., 2000). Similarly, the ZKPQ has demonstrated good internal consistency and the ImpSS scale has been shown to correlate strongly with substance abuse disorders (Zuckerman and Cloninger, 1996). In our sample, coefficients alpha for the P and ImpSS scales were 0.69 and 0.73, respectively. Finally, we assessed current tobacco use (quantity \times frequency). Items were of our own construction but mirror those used in prior research (e.g., Sher et al., 1991).

RESULTS

Preliminary Analyses

All data analyses were conducted using the Statistical Analysis System (SAS), version 9.1 (SAS Institute, Cary, N.C., 2002 to 2003). First, univariate statistics were computed on all continuous variables in order to detect any irregularities in the data. Due to elevated levels of skewness and kurtosis on the composite alcohol use variable, scores of "far outliers" (10 in total) were recoded to equal one greater than the largest non-far-outlying value (Tabachnick and Fidell, 2001). These adjustments resulted in acceptable skewness (less than 2) and kurtosis (less than 4) values. All other variables included in the current analyses were normally distributed.

Prevalence of FHA

Overall, 66.42% of participants indicated alcohol problems among first, second, or third degree relatives (see Table 1). This general prevalence rate can be divided into 4 levels (e.g., Dawson et al., 1992). Accordingly, 33.6% of participants were classified as FH-Neg (no alcoholism in any biological

Table 1. Family History of Alcoholism According to Different Methods of Measurement

	Percentage	<i>n</i>
Overall prevalence (all levels)	66.42	269
Paternal history only	22.06	90
Density FHA construct		
No family history	33.58	136
2nd degree relatives only	35.06	142
1st degree relatives only	2.22	9
1st and 2nd degree relatives	29.14	118

relatives), 35.1% were FHP-1 (alcoholism in second or third degree relatives only), 2.2% were FHP-2 (alcoholism in first degree relatives only), and 29.1% were FHP-3 (alcoholism reported in both first and second or third degree relatives).

Effects of FHA on Alcohol Use and Problems

We used structural equation modeling (SEM) to investigate the direct and indirect effects of FHA on alcohol use and problems. As a first step, bivariate correlations were computed for the relevant variables (see Table 2). Next, we estimated 2 nested path models in order to investigate hypothesized mediators of relations between FHA and alcohol use/problems. Identical models were estimated separately for alcohol use and problems. In all SEM models, we analyzed the covariance matrix using maximum likelihood (ML) estimation. Gender and FHA were modeled as covaried exogenous variables. Alcohol use and problems were modeled as endogenous variables with measures of age of onset of alcohol use (ONSET), level of response to alcohol (LR), behavioral undercontrol (BU), and tobacco use (SMOKE) as potential mediating variables (see Fig. 1). BU was modeled as a latent variable with 3 indicators – the P scale from the EPQ (Eysenck, 1988), and the Imp and SS items from the ZKPQ (Zuckerman et al., 1993). All other variables were manifest.

Alcohol Use Models. The direct effects model, which examined relations between FHA, gender, and alcohol use, yielded a significant chi square value ($\chi^2 = 119.34$ [22], $p < 0.0001$), suggesting poor model fit. However, since

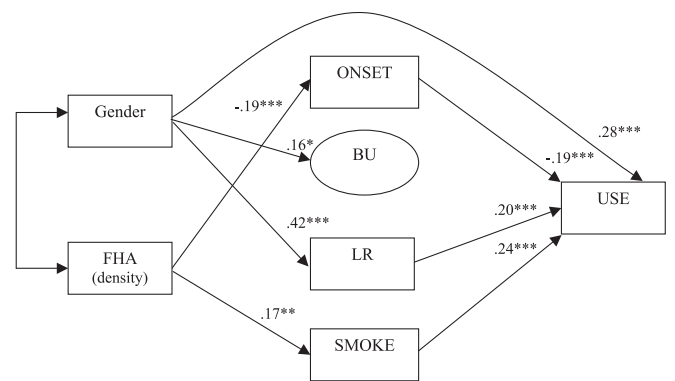


Fig. 1. Mediation model of alcohol use with density FHA construct. Standardized path coefficients are presented. Only significant paths are depicted. * $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$. ONSET = age of onset of alcohol use; BU = behavioral undercontrol; LR = level of response to alcohol; SMOKE = current cigarette use. Gender was coded as 1 = female, 2 = male; higher scores on FHA measure reflect greater familial density.

chi-square values are sensitive to sample size and often significant in larger samples (Bollen, 1990), we examined additional indices of model fit. These also indicated poor overall fit [Comparative Fit Index (CFI) = 0.75, root-mean-square error of approximation (RMSEA) = 0.11]. Inspection of the structural paths revealed a significant direct effect of gender on alcohol use ($\beta = 0.36$, $p < 0.001$), with men reporting higher levels of drinking. In contrast to our hypotheses, the association between FHA and alcohol use was nonsignificant.

Regarding associations between the independent variables and hypothesized mediators, a significant effect of gender was observed on level of response to alcohol ($\beta = 0.42$, $p < 0.001$) and behavioral undercontrol ($\beta = 0.16$, $p < 0.01$) with men reporting lower response to alcohol's effects and higher levels of impulsivity. Additionally, direct effects of FHA were observed on 2 of the mediators, age of onset of drinking and tobacco use such that greater levels of FHA were associated with earlier onset of drinking ($\beta = -0.19$, $p < 0.001$) and higher levels of smoking ($\beta = 0.16$, $p < 0.01$). This model accounted for 13.0% of the variance in alcohol use.

Next, we tested a mediation model according to criteria described by Baron and Kenny (1986) and MacKinnon

Table 2. Correlations Among Exogenous and Endogenous Variables (SEM Models)

Variables	1	2	3	4	5	6	7	8	9	10
1. Familial alcoholism	—									
2. Gender	-0.01	—								
3. Age of onset	-0.14**	0.03	—							
4. Impulsiveness	0.11*	0.11*	-0.11*	—						
5. Sensation-seeking	0.09	0.09	-0.17***	0.52***	—					
6. Psychoticism	0.15**	0.21***	-0.13**	0.46***	0.39***	—				
7. Level of response	0.06	0.41***	-0.13*	0.11*	0.10	0.15**	—			
8. Tobacco use	0.16***	-0.06	-0.22***	0.13**	0.15**	0.13**	-0.01	—		
9. Alcohol use	0.03	0.35***	-0.29***	0.23***	0.17**	0.19***	0.35***	0.26***	—	
10. Alcohol problems	0.11*	0.09	-0.26***	0.29***	0.36***	0.22***	0.16**	0.36***	0.58***	—

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$.

(1994) which state that a variable may be considered a mediator when 4 criteria are satisfied: 1) there is a significant direct effect between an independent variable and a dependent variable; 2) there is a significant relationship between an independent variable and the proposed mediator; 3) there is a significant relationship between the mediator and the dependent variable; and 4) a reduction in the direct path from the independent to the dependent variable occurs in the presence of the indirect (mediated) effect.

Accordingly, the mediation model included paths from the 4 hypothesized mediators (ONSET, LR, BU, SMOKE) to alcohol use. This model provided a superior fit to the data as compared with the direct model ($\chi^2 = 51.95$ [18], $p < 0.0001$, CFI = 0.92, RMSEA = 0.07, χ^2 difference score = 67.39 [4], $p < 0.0001$). Inspection of the regression paths revealed significant associations between 3 of the mediators, ONSET ($\beta = -0.19$, $p < 0.001$), LR ($\beta = 0.20$, $p < 0.001$), and SMOKE ($\beta = 0.24$, $p < 0.001$), and alcohol use. Specifically, an earlier onset of drinking, decreased sensitivity to the effects of alcohol and greater tobacco use were all associated with higher levels of drinking.

Alcohol Problems Models. The same models were then tested with alcohol problems as the outcome (see Fig. 2). Consistent with the previous models, the direct model showed a poor fit to the data ($\chi^2 = 133.44$ [22], $p < 0.0001$, CFI = 0.73, RMSEA = 0.12). Gender ($\beta = 0.13$, $p < 0.01$) and FHA ($\beta = 0.11$, $p < 0.05$) were both significantly associated with alcohol problems such that men and individuals with greater density of FHA reported greater alcohol-related problems. Associations between the independent and mediator variables were comparable to the alcohol use models with the addition of a significant relationship between FHA and BU ($\beta = 0.16$, $p < 0.05$) in this model. The direct model accounted for 3% of the variance in alcohol problems.

The mediation model provided good overall fit to the data ($\chi^2 = 51.75$ [18], $p < 0.001$, CFI = 0.92, RMSEA = 0.07, χ^2 difference score = 81.69 [4], $p < 0.0001$). Three of the

mediators, SMOKE ($\beta = 0.27$, $p < 0.001$), BU ($\beta = 0.29$, $p < 0.001$), and ONSET ($\beta = -0.16$, $p < 0.01$) were related to increased alcohol-related problems. This model accounted for 21% of the variance in alcohol problems.

In applying the criteria for mediation described above, none of the relations involving alcohol use satisfy all 4 criteria largely due to the lack of a direct effect of FHA on use. However, several relations in the alcohol problems model indicate the presence of mediation. Namely, the direct model indicated a significant association between the independent (FHA and gender) and dependent (alcohol problems) variables, as well as a significant relationship between the independent variable (FHA) and 3 mediators (ONSET, BU, SMOKE). A significant relationship between gender and one of the mediators (BU) was also observed. The mediation model showed a significant relationship between the mediators and the dependent variable and the formerly significant direct effects of FHA and gender on problems became nonsignificant in the presence of the mediators.

In order to further tease apart these relations and determine which mediator(s) influence the direct effects of FHA and gender, we tested the mediation models including 1 mediator at a time. Our results indicated that BU mediates the effects of both FHA and gender on alcohol problems, and that SMOKE and ONSET mediate the effects of FHA but not gender. Taken together, results of our SEM analyses confirm the hypothesis that behavioral undercontrol, cigarette use, and age of onset of drinking mediate the relationship between familial alcoholism and alcohol-related problems in college students.

As noted, in contrast to our expectations, none of the relations between FHA and alcohol use satisfy all the criteria for mediation described above. However, FHA was significantly related to age of onset of drinking, which, in turn, was significantly associated with increased alcohol use. While the lack of a direct effect of FHA on alcohol use does not satisfy full criteria for mediation as detailed by Baron and Kenny (1986) and others (MacKinnon, 1994), it should be noted that the methodology used in the present analyses represents a highly conservative approach. In fact, these relations would satisfy criteria for mediation according to other approaches (Collins et al., 1998) that do not require the presence of a direct effect.

DISCUSSION

The present study adds to the extant literature in several ways. To our knowledge, this study represents the first investigation of a density measure of familial alcoholism in a college student sample. In addition, we extend previous research through the examination of the effects of this construct on alcohol use and problems, as well as known etiological risk factors for AUDs. Our findings provide further evidence of the role of behavioral undercontrol, smoking and age of onset of drinking in relations between FHA and alcohol-related problems in this population. Additionally, our results provide evidence for the influence of personality factors such as

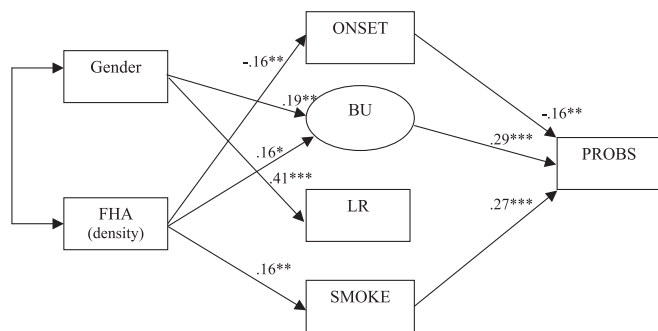


Fig. 2. Mediation model of alcohol problems with density FHA construct. Standardized path coefficients are presented. Only significant paths are depicted. * $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$. ONSET = age of onset of alcohol use; BU = behavioral undercontrol; LR = level of response to alcohol; SMOKE = current cigarette use. Gender was coded as 1 = female, 2 = male; higher scores on FHA measure reflect greater familial density.

behavioral undercontrol in the relationship between gender and alcohol problems.

Consistent with our hypotheses, a higher prevalence of FHA was observed in this sample, approximately 66% overall, as compared with previous research with college students (Weitzman and Wechsler, 2000) and a national household sample (Harford, 1992). Also consistent with our hypotheses, FHA was significantly related to alcohol-related problems such that higher levels of familial density were associated with increased problems. This association was mediated by 3 previously identified etiological risk factors – age of onset of drinking, behavioral undercontrol, and current cigarette use – commensurate with previous research linking FHA with these constructs (Dawson, 2000; Hill and Yuan, 1999; Jackson et al., 2000; Sher et al., 1991, 2000). Notably, these relationships were evident in the absence of a high-risk study design, suggesting robustness of these associations.

There were some unexpected findings in this study. Contrary to our expectations, we did not find a direct effect of FHA on alcohol use. One potential explanation may be that, in lieu of a high-risk design that oversamples for those with FHA, a larger sample is needed in order to detect relatively small FHA effects (Baer, 2002). The fact that we did not oversample for positive FHA status likely explains why we did not replicate previous research (Sher et al., 1991) demonstrating significant effects of FHA on alcohol use. Additionally, it is possible that FHA is more relevant to alcohol problems than alcohol use per se, particularly among college students where relatively heavy drinking is somewhat normative.

In contrast to previous research (Schuckit, 1994), we did not observe significant associations between FHA and level of response to alcohol. In addition to our non-high-risk study design, another potential explanation for this finding concerns the observed gender differences in this construct (i.e., men reported lower levels of response). Since men were underrepresented in our sample, it is possible that we were not able to accurately model these relations. Notably, Schuckit's (1994) study included only men; therefore, further research with mixed gender samples is needed in order to clarify the relationship between FHA and LR, as well as LR's influence on risk of AUD. However, in the present study, we did not observe a significant bivariate association between FHA and LR when we examined correlations exclusively for male participants.

The overall prevalence rate of 66% observed in the current study is in marked contrast to the 10% prevalence rate for parental "problem drinking" in a national sample of college students (Weitzman and Wechsler, 2000). This discrepancy was anticipated and is most likely due to our use of a more inclusive measure of FHA. However, Harford's (1992) assessment of prevalence of FHA in a national household sample, utilizing a similar familial density FHA construct as in the present study, yielded a lower overall prevalence rate (40.3% among 18 to 29 year olds). This finding may be partially due to the fact that women are overrepresented in the current

sample (approximately 72% vs. 57% women in Harford's study) and women have been shown to report higher prevalence of FHA than men (Dawson et al., 1992; Harford, 1992).

While the elevated overall prevalence of FHA in the current study may reflect high sensitivity and low specificity of measurement (e.g., Bradley et al., 1998), it is important to note that over 29% of participants reported alcohol problems in both first and second-degree relatives. This sizeable group represents those students at highest risk for developing alcohol problems themselves (Dawson et al., 1992; Perkins and Berkowitz, 1991). This finding has implications for targeted preventive interventions with college students, especially with women, whose drinking and related consequences have been shown to be particularly affected by FHA (Sher et al., 1991).

Limitations

These findings should be considered in light of several limitations, perhaps chief among them being the nature of the sample. Participants were recruited from a single, ethnically homogenous public university. As such, the results may not generalize to the U.S. college student population and, in particular, to more diverse universities. Second, this study utilizes cross-sectional data. Therefore, the relationships among constructs can best be viewed as 1 moment in time. Future longitudinal studies of drinking trajectories would allow for stronger inferences regarding potential causality. Finally, these data were derived solely from self-report measures. However, we attempted to minimize threats to validity by assuring participants of the anonymity of their responses and the nonevaluative nature of the survey. Also, recent research found no evidence of systematic underreporting of alcohol use by college students (Laforge et al., 2005).

Implications and Conclusions

Findings from the present study offer several implications for future etiological research and preventive interventions. First, assessing FHA more broadly in future trajectory research may capture a greater number of students at risk for acute alcohol-related problems and/or future development of AUDs. Similarly, including assessment of personality factors such as behavioral undercontrol, particularly among men, may also help to identify at-risk students. Third, given the compelling evidence from this study and previous research showing significant associations between early onset of drinking and higher levels of alcohol use in college, it is clear that future prevention efforts should begin well before the college years.

Regarding future preventive interventions with college students, FHA status may help to identify students who may benefit most from early alcohol-focused preventive interventions, given the evidence that FHA⁺ students are less likely to transition out of heavy drinking (Jackson et al., 2001). Additionally, traditional individualized feedback approaches may profit from incorporating a module regarding smoking

cessation. This may serve dual purposes: targeting smoking may help to identify students who are at risk for developing problems with alcohol later on (especially those with FHA) and may aid students who wish to cut down on their alcohol use but find that smoking acts as a trigger for increased drinking.

In sum, the current study provides both conceptual and practical contributions to the college drinking literature. Our finding that density of familial alcoholism is associated with alcohol-related problems in a college student sample extends previous etiological research. Additionally, our findings that behavioral undercontrol, smoking and early onset of drinking are important factors in relations between familial alcoholism and alcohol problems may further enhance our understanding of the etiology of alcohol-related consequences in this population. These findings may also help to shape future preventive efforts aiming to reduce alcohol use and problems among college drinkers. Specifically, consideration of cigarette use and familial density of alcoholism, particularly among women, may enhance the efficacy of targeted preventive interventions.

ACKNOWLEDGMENT

We wish to thank Drs. Brian Borsari and John Hustad for their very helpful comments on a previous version of this manuscript.

REFERENCES

- Babor TF, Hofmann M, DelBoca FK, Hesselbrock VM, Meyer RE, Dolinsky ZS, Rounsaville B (1992) Types of alcoholics: I. Evidence for an empirically derived typology based on indicators of vulnerability and severity. *Arch Gen Psychiatry* 49:599–608.
- Baer JS (2002) Student factors: understanding individual variation in college drinking. *J Stud Alcohol Suppl* 14:40–53.
- Baron RM, Kenny DA (1986) The moderator-mediator variable distinction in social psychological research: conceptual, strategic, and statistical considerations. *Pers Soc Psychol* 51:1173–1182.
- Bien TH, Burge R (1990) Smoking and drinking: a review of the literature. *J Addict* 25:1429–1454.
- Bollen KA (1990) Overall fit in covariance structure models: two types of sample size effects. *Psych Bull* 107:256–259.
- Bradley KA, Boyd-Wickizer J, Powell SH, Burman ML (1998) Alcohol screening questionnaires in women: a critical review. *J Am Med Assoc* 280:166–171.
- Chassin L, Pitts SC, Prost J (2002) Binge drinking trajectories from adolescence to emerging adulthood in a high-risk sample: predictors and substance abuse outcomes. *J Consult Clin Psychol* 70:67–78.
- Chermack ST, Stoltenberg SF, Fuller BE, Blow FC (2000) Gender differences in the development of substance-related problems: impact of family history of alcoholism, family history of violence and childhood conduct problems. *J Stud Alcohol* 61:845–852.
- Cloninger CR (1987) Neurogenetic adaptive mechanisms and alcoholism. *Science* 236:410–416.
- Collins LM, Graham JW, Flaherty BP (1998) An alternative framework for defining mediation. *Multivariate Behav Res* 33:295–312.
- Dawson DA (2000) The link between family history and early onset alcoholism: earlier initiation of drinking or more rapid development of dependence? *J Stud Alcohol* 61:637–646.
- Dawson D, Harford TC, Grant BF (1992) Family history as a predictor of alcohol dependence. *Alcohol Clin Exp Res* 16:572–575.
- Evans SM, Levin FR (2003) Response to alcohol in females with a paternal history of alcoholism. *Psychopharmacology* 169:10–20.
- Eysenck HJ (1988) *Eysenck Personality Questionnaire-Revised*. Educational and Industrial Testing Services, San Diego, CA.
- Finn PR, Sharkansky EJ, Brandt KM, Turcotte N (2000) The effects of familial risk, personality, and expectancies on alcohol use and abuse. *J Abnorm Psychol* 109:122–133.
- Harford TC (1992) Family history of alcoholism in the United States: prevalence and demographic characteristics. *Br J Addict* 87:931–935.
- Heath AC (1995) Genetic influences on alcoholism risk. *Alcohol Health Res World* 19:166–171.
- Heath AC, Madden PAF, Bucholz KK, Dinwiddie SH, Slutske WS, Bierut LJ, Rohrbach JW, Statham DJ, Dunne MP, Whitfield JB, Martin NG (1999) Genetic differences in alcohol sensitivity and the inheritance of alcoholism risk. *Psychol Med* 29:1069–1081.
- Hill SY, Shen S, Lowers L, Locke J (2000b) Factors predicting the onset of adolescent drinking in families at high risk for developing alcoholism. *Biol Psychiatry* 48:265–275.
- Hill KG, White HR, Chung I-J, Hawkins JD, Catalano RF (2000a) Early adult outcomes of adolescent binge drinking: person- and variable-centered analyses of binge drinking trajectories. *Alcohol Clin Exp Res* 24:892–901.
- Hill SY, Yuan H (1999) Familial density of alcoholism and onset of adolescent drinking. *J Stud Alcohol* 60:7–17.
- Hurlbut SC, Sher KJ (1992) Assessing alcohol problems in college students. *J Am Coll Health* 41:49–58.
- Jackson KM, Sher KJ, Gotham HJ, Wood PK (2001) Transitioning into and out of large-effect drinking in young adulthood. *J Abnorm Psychol* 110:378–391.
- Jackson KM, Sher KJ, Wood PK (2000) Prospective analysis of comorbidity: tobacco and alcohol use disorders. *J Abnorm Psychol* 109:679–694.
- Kendler KS, Neale MC, Heath AC, Kessler RC, Eaves LJ (1994) A twin-family study of alcoholism in women. *Am J Psychiatry* 151:707–715.
- Laforge RG, Borsari B, Baer JS (2005) The utility of collateral informant assessment in college alcohol research. *J Stud Alcohol* 66:479–487.
- MacKinnon DP (1994) Analysis of mediating variables in prevention and intervention research, in *Scientific Methods in Prevention Research*, NIDA Research Monograph 139 (Cazares A, Beatty LA eds) Vol DHHS Publication No. 94-3631, pp 127–153. U.S. Government Printing Office, Washington, DC.
- Marlatt GA, Baer JS, Kivlahan DR, Dimeff LA, Larimer ME, Quigley LA, Somers JM, Williams E (1998) Screening and brief intervention for high-risk college student drinkers: results from a 2-year follow-up assessment. *J Consult Clin Psychol* 66:604–615.
- Myerhoff DJ, Tizabi Y, Staley JK, Durazzo TC, Glass JM, Nixon SJ (2006) Smoking comorbidity in alcoholism: neurobiological and neurocognitive consequences. *J Am Coll Health* 30:253–264.
- Newlin DB, Thomson JB (1991) Chronic tolerance and sensitization to alcohol in sons of alcoholics. *Alcohol Clin Exp Res* 15:399–405.
- O'Neill SE, Parra GR, Sher KJ (2001) Clinical relevance of heavy drinking during the college years: cross-sectional and prospective perspectives. *Psychol Addict Behav* 15:350–359.
- Perkins HW, Berkowitz AD (1991) Collegiate COAs and alcohol abuse: problem drinking in relation to assessments of parent and grandparent alcoholism. *J Couns Dev* 69:237–240.
- Read JP, Wood MD, Capone C (2005) A prospective investigation of relations between social influences and alcohol involvement during the transition into college. *J Stud Alcohol* 66:23–34.
- Schuckit MA (1994) Low level of response to alcohol as a predictor of future alcoholism. *Am J Psychiatry* 151:184–189.
- Schuckit MA (1998) Biological, psychological and environmental predictors of the alcoholism risk: a longitudinal study. *J Stud Alcohol* 59:485–494.
- Schuckit MA (2000) Genetics of the risk for alcoholism. *Am J Addict* 9:103–112.
- Schuckit MA, Smith TL, Tipp JE (1997a) The Self-Rating of the Effects of Alcohol (SRE) form as a retrospective measure of the risk for alcoholism. *Addiction* 92:979–988.

- Schuckit MA, Tipp JE, Smith TL, Wiesbeck GA, Kalmijn J (1997b) The relationship between Self-Rating of the Effects of alcohol and alcohol challenge results in ninety-eight young men. *J Stud Alcohol* 58:397-404.
- Schulenberg J, O'Malley PM, Bachman JG, Wadsworth KN, Johnston LD (1996) Getting drunk and growing up: trajectories of frequent binge drinking during the transition to young adulthood. *J Stud Alcohol* 57:289-304.
- Sher KJ, Bartholow BD, Wood MD (2000) Personality and substance use disorders: a prospective study. *J Consult Clin Psychol* 68:818-829.
- Sher KJ, Gotham HJ, Erickson DJ, Wood PK (1996) A prospective, high-risk study of the relationship between tobacco dependence and alcohol use disorders. *Alcohol Clin Exp Res* 20:485-492.
- Sher KJ, Walitzer KS, Wood PK, Brent EE (1991) Characteristics of children of alcoholics: putative risk factors, substance use and abuse, and psychopathology. *J Abnorm Psychol* 100:427-448.
- Tabachnick BG, Fidell LS (2001) *Using Multivariate Statistics*. 4th ed. Allyn and Bacon, Boston.
- Tucker JS, Orlando M, Ellickson PL (2003) Patterns and correlates of binge drinking trajectories from early adolescence to young adulthood. *Health Psychol* 22:79-87.
- Weitzman ER, Wechsler H (2000) Alcohol use, abuse, and related problems among children of problem drinkers: findings from a national survey of college alcohol use. *J Nerv Ment Dis* 188:148-154.
- Zuckerman M, Cloninger CR (1996) Relationship between Cloninger's, Zuckerman's, and Eysenck's dimensions of personality. *Pers Individ Dif* 21:283-285.
- Zuckerman M, Kuhlman DM, Joireman J, Teta P, Kraft M (1993) A comparison of three structural models for personality: the Big Three, the Big Five, and the Alternative Five. *J Pers Soc Psychol* 65:757-768.