# Female Offspring of Alcoholic Individuals: Recent Findings on Alcoholism and Psychopathology Risks: Symposium Presented at the Research Society on Alcoholism, 2004, Vancouver Aruna Gogineni, Chair

Aruna Gogineni, Serena King, Kristina Jackson, John Kramer, Kathleen Bucholz, Grace Chan, William Iacono, Samuel Kuperman, Jenny M. Larkins, Richard Longabaugh, Matt McGue, Linnea Polgreen, Kenneth J. Sher, Robert Stout, David Strong, and Robert Woolard

**Key Words:** Alcohol, Female Offspring.

IN THE PAST decade, significant advances have been made in understanding how genetic and environmental factors contribute to alcoholism and other psychopathology among children of alcoholic individuals. Potential biopsychosocial markers of risk (e.g., low level of response to alcohol, behavioral undercontrol, and family functioning variables) have been identified and indicate that both individual and environmental variables are highly relevant. Despite these advances, studies have predominantly focused on examining outcomes among sons of alcoholic individuals, with the consequence that relatively little is known about the risk for alcoholism and other psychopathology among daughters. Effective prevention and treatment strategies are predicated upon further knowledge of these risks among daughters as well as sons. This symposium will present recent findings using family, prospective, and cross-sectional research to elucidate the biopsychosocial correlates and the moderators of risk for alcoholism and other psychopathology among daughters from developmental trajectories spanning the periods of childhood, adolescence, and adulthood.

This symposium begins with a presentation by John Kramer in which high-risk daughters' and sons' alcohol and drug involvement are compared with respect to their predictors, drawn from demographic, familial, and personal domains. Next, Serena King focuses on the correlates of disinhibited behavior in males and females from adoptive and biological families, with an emphasis on parental alcoholism, genetic versus environmental influences, and differences between genders. This talk is followed by a presentation by Kristina Jackson, who examines the predictors of alcohol use disorders among young adults from high-risk and control families, including such factors as family history, negative affect, behavioral undercontrol, and childhood stressors. Finally, Aruna Gogineni addresses the familial predictors of adult daughters' alcohol problems and depression, focusing on the effects of maternal versus paternal alcoholism as well as family density of pathology.

*From the Brown University Medical School, Providence, Rhode Island (AG, KJ, RL, RS, DS, RW); the Department of Psychology, Hamline University, St. Paul, Minnesota (SK); the Department of Psychiatry (JK, SK), the Department Statistics (GC), and the Department of Economics (LP), University of Iowa, Iowa City, Iowa; the Midwest Alcoholism Research Center and the Department of Psychiatry, Washington University, St. Louis, Missouri (KB); the Department of Psychology, University of Minnesota, Minneapolis, Minnesota (WI, MM); and the Midwest Alcoholism Research Center and the University of Missouri, Columbia, Missouri (JML, KJS).*

*Received for publication November 4, 2005; accepted November 7, 2005.*

*This work was supported by NIAAA Grants AA012250-01, AA11886, U10AA08403, AA03938, AA07231, and AA11998*

*Reprint requests: John Kramer, PhD, Department of Psychiatry Research, University of Iowa, MEB, Iowa City, IA 52242-1000; Fax: 319-3553-3003; E-mail: john-kramer@uiowa.edu*



**DOI: 10.1111/j.1530-0277.2006.00042.x**

## CORRELATES OF DRINKING AND SUBSTANCE USE AMONG OFFSPRING IN ALCOHOLIC FAMILIES: ADULT DAUGHTERS AND SONS

*John Kramer, Grace Chan, Linnea Polgreen, Samuel Kuperman, Kathleen. Bucholz, and Victor Hesselbrock*

### *Introduction*

In the United States, women exhibit fewer clinically significant alcohol problems than do men. For example, in 2001 to 2002, 12-month prevalence rates of Diagnostic and

Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV) Alcohol Dependence or Abuse (American Psychiatric Association, 2000) were considerably lower for females (4.87%) than for males (Grant et al., 2004). Females also have a later age of onset, a more compressed course of symptom development, and a higher rate of physical complications (Bucholz et al., 1994b; Schuckit et al., 1998).

These differences raise the possibility that the antecedents of alcoholism for women and men may differ to some degree as well. The 2 genders share biological risk markers, such as reduced amplitude visual and auditory P3 (Prabhu et al., 2001; Suresh et al., 2003) and a diminished response to alcohol (Schuckit et al., 2003). In addition, heritability of alcohol problems is essentially the same for men and women (Heath et al., 1997), and specific genetic risk factors identified thus far, such as GABRG3 (Dick et al., 2004), are common to both genders. However, future investigations may reveal additional genetic and physiological antecedents of alcohol dependence that are sex-specific.

The same possibility applies to precursors that involve demography, family environment, child psychopathology, and parenting. The purpose of the present study is to examine whether predictors of drinking problems drawn from these domains are in any way different for males and females. We present multiple regression analyses conducted on a national sample of men and women from high-risk families. Two established variables, externalizing symptoms (Kuperman et al., 2001) and religion (Wallace et al., 2004), are anticipated to predict, respectively, increased and decreased risk of drinking problems in both genders. Two other widely acknowledged antecedents, demographic background and parental drinking, are expected to predict alcohol problems in a less even-handed fashion; data from other investigations suggest that parental drinking might be a more salient predictor among females and that demographic factors might play a more significant role among males (Curran et al., 1999; Ohannessian et al., 2004). Finally, we incorporate variables that address parenting and parent–child relationships separately for mother and father (e.g., mother consistency, father consistency); these are anticipated to discriminate between male and female offspring in at least some instances, although the precise direction of differences cannot be specified in advance.

## *Materials and Methods*

*Sample*. Subjects in the current study were drawn from the Collaborative Study on the Genetics of Alcoholism (COGA). For the present analysis, only high-risk families were included, ascertained through an alcohol-dependent proband in treatment and containing other first-degree relatives with alcohol dependence. Methodological details of the COGA project can be found in Begleiter et al. (1995). Subjects missing data on any independent or dependent variable were excluded. The final sample consisted of 586 participants, of whom slightly more than half (54.9%) were female and a minority (10.6%) were probands. The 2 genders were very similar in age (women: mean = 38.7; range = 18–77; men: mean = 39.2; range = 18–78).

*Assessment*. All subjects were administered the Semi-Structured Assessment for the Genetics of Alcoholism, developed for COGA. This instrument possesses adequate reliability and validity (Bucholz et al., 1994a; Hesselbrock et al., 1999) and assesses major DSM-IV psychiatric diagnoses, including alcohol dependence and abuse. In addition, the interview also queries the economic, religious, and familial characteristics of participants' childhoods.

*Independent Variables*. Potential predictors were selected from 5 domains. All retrospective variables were based on circumstances that prevailed when subjects were between 6 and 13 years of age. Demographic characteristics included age of participant at interview, whether he or she looked older than friends as an adolescent (Dick et al., 2000), size of town where the subject grew up, father's and mother's education, family's relative economic standing in the neighborhood (average, better off, worse off), whether the mother worked outside the home, and whether the father frequently was away from home (including reasons such as job, divorce, and separation). Family Environment variables included the following: (a) 2 religion questions (whether participant was raised in a fundamentalist denomination and whether his/her religion had rules against alcohol use); (b) 4-point scaled ratings of subject's relationship with parents, parents' relationship with each other, and conflict and tension in the home; and (c) dichotomous ratings of whether parents frequently fought in front of the participant and whether the subject saw parents hit each other. Parenting predictors addressed, separately, mother and father strictness/laxity (3-point scales), consistency (Y/N), and punishment styles (primarily physical vs nonphysical). Harsh physical punishment (from either mother or father) was assessed by asking participants whether they still hurt the next day and/or had to see a physician. Parental Pathology consisted of the maximum number of core alcohol symptoms (19 items) ascribed to the mother and to the father by other family members, using a validated family history interview (Rice et al., 1995). In addition, subjects were asked whether their mother or their father drank too much while they were growing up. Finally, Subject Pathology variables incorporated retrospective symptom counts drawn from 2 externalizing syndromes, Conduct Disorder (CD; 15 items) and Oppositional Defiant Disorder (ODD; 5 items) (American Psychiatric Association, 2000).

*Dependent Variables*. Subjects' lifetime involvement with alcohol was measured in the 3 following ways: (a) whether subjects ever met criteria for DSM-IV Alcohol Dependence; (b) the total number of alcohol problems endorsed [this count variable combined dependence and abuse criteria (12 items)]; and (c) the maximum number of drinks

subjects had ever consumed in 24 hours. Two other substance outcomes were tested as outcome variables as follows: (a) the total number of drug categories ever tried (marijuana, cocaine, stimulants, sedatives, opiates, PCP, hallucinogens, and solvents) and b) the typical number of cigarettes subjects consumed daily if they ever smoked regularly.

*Analyses*. Multiple regression was used to control for correlations among the predictors. All analyses were conducted separately for males and females. Maximum drinks in 24 hours and average number of cigarettes per day were binned prior to analysis to make their distributions smoother. A stepwise variable selection procedure was used, with significant predictors entered and nonsignificant ones removed at a $p < 0.05$ level. SAS binary logit was used for alcohol dependence, and cumulative logit was used for all other outcome variables (Allison, 2001).

### *Results*

For each outcome variable, predictors are described in descending order of contribution to the final model. *Alcohol dependence in females* (16.8% of female sample) was positively predicted by their childhood CD symptom count. In addition, women who rated their mothers as too strict or too lax were significantly more likely to meet criteria for alcohol dependence than were women who rated their mothers as appropriately strict. With both predictors taken into account, 76% of women were correctly classified on alcohol dependence. *Alcohol dependence in males* (35.6% of male sample) was positively associated with their CD symptom count, ODD symptom count, father being present in the home, parents fighting in front of subject, mother using physical punishment, father having less education, and mother working at home. With all independent variables combined, 80% of males were correctly classified as being alcohol dependent or not.

*Alcohol symptom count in women* (mean = 2.1; range = 0–12) was positively predicted by retrospective CD and ODD symptom counts, looking older as a teenager, growing up in a smaller hometown, harsh physical punishment, being raised in a religion that did not forbid alcohol, a poor relationship with the father, father drinking too much, and parents not fighting. *Alcohol symptom count in women* (mean = 3.6; range = 0–12) was positively associated with both CD and ODD symptoms, less father education, a religion that allowed alcohol, father's alcohol symptom count (attributed by family members), and father being in the home. The maximum rescaled $R^2$, an approximation of variance accounted for, was higher for men (0.34) than for women (0.27).

*Maximum drinks in 24 hours among females* (mean = 11.6; range = 0–120; maximum rescaled $R^2 = 0.17$) was predicted by ODD symptoms (+), age (−), whether the subject experienced severe physical punishment (+), and a poor relationship with the father (+). *Maximum drinks in 24 hours among males* (mean = 22.1; range = 0–144; maximum rescaled $R^2 = 0.28$) was significantly associated with CD symptoms (+), religion that forbade alcohol (−), ODD symptoms (+), paternal education (−), and looking older as an adolescent (+).

The final model for typical *daily cigarette consumption in women* (mean = 8.4; range = 0–60; maximum rescaled $R^2 = 0.08$) contained 3 predictors: father drinking too much (+), father consistency in parenting (−), and severe physical punishment (+). For *daily cigarette consumption in men* (mean = 13.2; range = 0–90; maximum rescaled $R^2 = 0.28$), the following 5 variables emerged: age (+), poor relationship with the mother (+), CD symptoms (+), maternal consistency (+), and household conflict (+).

Finally, *number of drug categories tried among females* (mean = 1.6; range = 0–8; maximum rescaled $R^2 = 0.17$) was positively associated with CD symptoms, severe physical punishment, father inconsistency, and smaller town size. *Number of drug categories tried among males* (mean = 2.6; range = 0–8; maximum rescaled $R^2 = 0.26$) was positively predicted by CD and ODD symptom count as well as by more maternal education.

### *Discussion*

The current sample was drawn from densely affected alcohol-dependent families, with probands ascertained in treatment centers. For this reason, our high-risk subjects are not representative of all individuals from alcoholic families, and our results should be considered preliminary until they are replicated in independent investigations. In addition, significant associations between predictors and outcome variables do not imply causality. For example, severe physical punishment could be (a) a cause of daughter alcohol problems, (b) a reaction to these problems, (c) both cause and effect, or (d) causally unrelated to such problems.

Certain predictors were shared by men and women: in both sexes, a history of externalizing symptoms (CD and/or ODD) positively predicted alcohol dependence, alcohol symptoms, maximum drinks, and number of drug categories tried. Looking older as an adolescent predicted alcohol use in females (symptom count) and males (maximum drinks). Religious sanctions against alcohol may have served as a protective factor, as it was associated with fewer alcohol symptoms in both genders and a lower number of maximum drinks in males. In addition to these shared predictors, several findings pointed toward differences between the sexes as follows:

1. For each of the 5 outcome variables, predictors accounted for a smaller amount of the variation among women than among men.
2. When parenting and parent-child relationships emerged as predictors of offspring alcohol and substance involvement, they almost always (7 of 8 instances) involved the opposite-sex parent.
3. For women, but not men, severe physical punishment (seeing a doctor and/or hurting the next day) was a

predictor of several outcomes (alcohol problems, maximum drinks in 24 hours, cigarette use, and number of drug categories tried). This variable may have served in some cases as a proxy for ongoing physical or sexual abuse.

4. There was no evidence for the hypothesis that, in females, parental drinking is more associated than socioeconomic status (SES) with substance outcome (each type of predictor emerged 2 times). There was limited support for the prediction that, among males, SES (as measured by mother and father education) was more associated with substance outcome (alcohol dependence, alcohol problems, and maximum drinks) than was parental drinking (father's alcohol symptom count), which only emerged as a predictor of alcohol symptoms.

It should be noted that counterintuitive predictors of female alcohol problems (parents not fighting), male cigarette use (mother consistency), and male drug categories tried (more mother education) were only significant in the multiple regression models; simple (bivariate) correlations between these predictors and their respective outcomes were in all instances nonsignificant.

The Collaborative Study on the Genetics of Alcoholism is poised to begin a prospective investigation of high-risk adolescents and young adults in which subjects will be evaluated every 2 years. Besides information gathered from interviews and questionnaires, the investigation will also incorporate data from genetic, neurophysiological, neuropsychological, and community environment domains. We anticipate that this longitudinal approach will elucidate both common and gender-specific risk factors that signal the subsequent development of alcohol and drug problems.

## GENDER DIFFERENCES IN PSYCHOLOGICAL ADJUSTMENT AMONG THE OFFSPRING OF ALCOHOLIC PARENTS: AN ADOPTION STUDY

*Serena King, Matt McGue, and William Iacono*

### *Introduction*

Several studies have demonstrated that parental alcoholism elevates risk for offspring substance use and childhood psychopathology, including acting-out behaviors and internalizing psychopathology (Bierut et al., 1998; Chassin et al., 1991, 1999). Although the association between parental alcoholism and offspring psychopathology is robust, it is unclear to what extent genetic and environmental mechanisms account for this association, particularly during early adolescence, a period of risk for experimentation with substances and the emergence of psychopathology. Moreover, we know relatively little about whether there are gender differences in the etiological contributions of parental alcoholism to early adolescent substance use and psychopathology.

The majority of studies on offspring of alcoholic parents have been conducted on sons of alcoholic subjects, as male offspring have been shown to be at greatest risk for the development of alcoholism. Consequently, little research has focused on gender differences and similarities in the familial transmission of alcoholism and related psychopathology. Although males have higher rates of alcoholism and acting-out behaviors, it is unclear whether there are etiologically relevant gender differences (i.e., differences in the degree of genetic and environmental influences on the behavior). A limitation of traditional family designs is that such designs cannot disentangle environmental and genetic effects.

The work presented here aims to examine etiological contributions to a range of behaviors characterized by behavioral disinhibition, in the context of a genetically informative adoption design. Specifically, we examine the degree to which adoptive and biological parental alcoholism are related to offspring psychopathology and substance use. If associations between parent alcoholism and offspring psychopathology are stronger for biological than for adoptive parents, this would imply a robust genetic effect of parental alcoholism. One benefit of the present study is the ability to determine whether such relationships are different for female and male offspring.

### *Materials and Methods*

*Sample.* The analyses were conducted using data from the Sibling Interaction and Behavior Study (SIBS), a population-based longitudinal study examining environmental and sibling interaction influences on mental health in a sample of biological and adoptive families gathered from the state of Minnesota. The sample was composed of 407 adoptive and 191 biological families consisting of 2 adolescents and their biological parents. Included among the 407 adoptive families were 123 families containing 1 adoptive and 1 biological child, which provided a test of whether differences between adoptive and biological offspring were because of systematic differences between adoptive and nonadoptive families. Biological and adoptive offspring did not differ significantly on age or gender composition (biological offspring, $M = 15.1$; adoptive offspring, $M = 14.4$; 52 and 56% female, respectively). A substantially greater proportion of adoptive than biological offspring were of Asian (primarily Korean) descent (adoptive offspring, 61% Asian, 21% White, 12% other ethnicities; biological offspring, 97% White), but the outcomes we investigated did not vary significantly by ethnicity. Higher rates of college education were found for adoptive mothers and fathers compared with biological mothers and fathers (61 and 63% and 45 and 45%, respectively). Adoptive mothers had significantly lower rates of alcohol dependence than biological mothers, but fathers did not differ on rates of dependence.

*Variables and Analyses*. Measures of parental alcoholism (DSM III-R dependence) were gathered via structured in-person diagnostic interviews conducted independently with mothers and fathers. Parents affected by alcohol dependence at a definite (met all DSM criteria) or probable (1 symptom short) level of certainty were considered affected with alcoholism. A range of adolescent indicators of disinhibited behavior and psychopathology were assessed, including the following: self-report measures of the adolescent's antisocial belief system, aggressive personality traits, delinquent behaviors, bad peer influences, the personality domain of constraint (from the Multidimensional Personality Questionnaire, where low constraint indicates poor impulse control), the number of drugs ever used, and a score that combines all of the above measures (disinhibition factor). To facilitate comparisons across offspring groups, scores were transformed to a *T*-score metric (i.e., mean of 50 and standard deviation of 10) prior to analysis. Outcomes were analyzed using a 3-factor analysis of variance, with sex, rearing status (adoptive vs biological), and parental alcoholism (either vs neither parent alcoholic) as the independent variables.

### *Results*

For all except one outcome measure (bad peers), the effect of having a parent with alcoholism was stronger in biological than adoptive families, resulting in a significant rearing status by parental alcoholism interaction and implicating the existence of genetic influences. In terms of gender effects, as expected, boys scored significantly higher than girls on all measures of disinhibition except number of drugs used and antisocial attitudes, for which the 2 genders did not differ significantly. The 3-way interaction was statistically significant for number of drugs used ($p = 0.02$), and marginally significant for antisocial attitudes ($p = 0.07$) and the disinhibition factor ($p = 0.09$). In each case, the nature of the 3-way interaction was a greater effect of biological parent alcoholism as compared with adoptive parent alcoholism among male than among female offspring, suggesting that biological effects were greater for male than female offspring. A similar pattern of results was found when analyses were restricted to mixed adoptive-biological families, indicating that our finding of reduced effects of parental alcoholism in adoptive compared with nonadoptive families was not attributable to systematic differences between biological and adoptive families.

### *Discussion*

Our findings demonstrate the following: (1) Parental alcoholism is associated with increased risk of behavioral disinhibition in offspring, as deviations in the offspring of alcoholic individuals is not limited to alcohol-related outcomes but rather are manifest across a range of disinhibited behavior; (2) this increased risk is predominantly mediated by genetic rather than environmental mechanisms, as significant deviations were observed in the biological but not the adoptive offspring of alcoholic individuals; and (3) biological effects may be greater in male than female offspring; for at least one of the outcome measures (number of substances used) the effect of biological versus adoptive parent alcoholism was greater for boys than girls. The latter result, although potentially important, needs to be interpreted cautiously as we only found suggestive evidence of similar differential effects on the other outcomes that we studied.

Collectively, the study findings suggest that male and female adolescents exhibit different levels of behavioral disinhibition on a phenotypic (or behavioral) level and that the underlying genetic and environmental transmission of risk for behavioral disinhibition may be different for the 2 sexes, at least during this stage of development. Perhaps as adolescents escalate in their substance use and delinquent behaviors during the transition to later adolescence and young adulthood, gender differences in etiological influences on behavioral disinhibition may become more pronounced. As adolescents leave the confines of parental control and gain independence, different environmental and genetic influences may become salient for men and women. Perhaps qualitatively different social and environmental mechanisms for drug and alcohol use are operant in girls and boys during adolescence. These findings underscore the importance of examining sex-specific etiological contributions to the familial transmission of alcoholism risk, using a variety of behavioral phenotypes across the life span. The longitudinal nature of the SIBS study will provide an opportunity to test these speculations as adolescents are followed during the passage to late adolescence and adulthood.

## GENDER-SPECIFIC MEDIATORS OF THE FAMILY HISTORY OF ALCOHOLISM: EFFECT ON YOUNG ADULT ALCOHOL USE DISORDERS

*Kristina M. Jacksson, Kenneth J. Sher, and Jenny M. Larkins*

### *Introduction*

A family history of alcoholism (FH) has been shown to be associated with increased risk of alcohol use disorder (AUD), with some evidence that this effect is stronger for women (Cotton, 1979; Crum and Harris, 1996; Pollock et al., 1987; Russell et al., 1990), although this finding has not always been reported. Possible mechanisms that underlie this association include temperament or personality (e.g., negative affectivity, behavioral undercontrol), disrupted family environment (stressors), drinking motives and expectancies, and pharmacologic vulnerability. Limited prior work has explored the mediation of the FH effect on alcohol involvement, including mediators such as alcohol expectancies, neuroticism, emotionality/negative

affectivity, behavioral undercontrol, externalizing behavior, parental monitoring, stress, and level of response to alcohol (Chassin et al., 1996; Colder et al., 1997; Hussong et al., 1998; LoCastro et al., 2000; Schuckit and Smith, 1996; Sher et al., 1991). However, no research has focused systematically on multiple mediators and gender differences. Prior work has been inconclusive in this area, suggesting that mechanisms underlying familial transmission of alcoholism may differ by gender. For example, there is some evidence that women with familial history of alcoholism report greater internalizing symptoms (Preisig et al., 2001) and that women may be more sensitive to the family environment (Russell et al., 1990).

The purpose of the current study is to examine the influence of FH on risk for a subsequent AUD and to determine the extent to which this effect differs between the sexes. Second, we examine the mechanisms that underlie this association, with a focus on identifying factors that are unique to women.

## *Materials and Methods*

*Sample and Procedure*. Data were taken from a prospective high-risk study on correlates of alcoholism (Sher et al., 1991). The sample was composed of 489 young adult participants, half (51%) with a FH, 46% male (113 Male FH−, 124 Female FH−, 118 Male FH+, 132 Female FH+). Respondents were prospectively assessed 6 times over 11 years (Years 1, 2, 3, 4, 7, and 11), between the ages of 18 and 28, by both interview and questionnaire. In all, 84% were retained at Year 11, and 81% had complete data across all waves. Attrition analyses show that those lost by Year 11 were similar at baseline to those successfully followed on sex, FH, race, and age. However, noncompleters were more likely to have had an AUD at Year 1 (34%) than were completers (22%) (Cohen's $h = 0.27$; $p < 0.05$).

*Variables*. Sex and FH were assessed at screening. The FH variable was based on history of alcoholism in the biological father in a 2-stage process, beginning with offspring ratings on the Short Michigan Alcoholism screening test (SMAST) (Selzer et al., 1975), adapted to refer to drinking patterns of the biological mother and the father (M-SMAST and F-SMAST) (Crews and Sher, 1992). Those who scored a 4 or more on the M-SMAST or F-SMAST were tentatively classified as high risk. Those with a 0 or 1 were tentatively classified as low risk. Tentatively classified participants then completed the Family History-Research Diagnostic Criteria (FH-RDC) (Endicott et al., 1978). A positive FH was coded if the biological father met FH-RDC criteria for alcoholism. Negative FH was coded if no first-degree relative received a diagnosis of alcohol, drug abuse, or antisocial personality disorders and if there was no alcohol or drug use disorder in a second-degree relative.

Alcohol use disorder was assessed at each wave and was coded positive if the respondent was diagnosed with past 12-month alcohol abuse and/or dependence. To maintain continuity, diagnoses were made according to criteria from DSM-III (American Psychiatric Association, 1980).

The following general constructs were modeled as latent variables; all measures significantly loaded on their respective constructs. First, Negative Affectivity (NA) consisted of 3 components, Neuroticism from the Eysenck Personality Questionnaire (EPQ) (Eysenck and Eysenck, 1975), Harm Avoidance from the Tridimensional Personality Questionnaire (TPQ) (Cloninger, 1987), and the Social Anxiety Scale (Fenigstein et al., 1975). Second, Behavioral Undercontrol (BU) was composed of TPQ Novelty Seeking, EPQ Psychoticism, and a lifetime DSM-III conduct disorder symptom count. Third, Cognitive Ability (COG) was assessed with 3 WAIS-R (Wechsler, 1981) subtests, Vocabulary, Block design, and Similarities. Fourth, 44 items addressing potentially positive effects of alcohol were used to assess Alcohol Expectancies (AE) (Sher et al., 1996). Previous factor analytic work on AE with this sample identified 4 factors as follows: Tension Reduction, Social Lubrication, Activity Enhancement, and Performance Enhancement. Finally, Childhood Stressors (CS) were assessed with the Childhood Life Stressors Interview (Sher et al., 1997), a 38-item structured instrument that retrospectively measures a range of childhood stressors that occurred before age 18. Items targeted effects of living in an alcoholic home (e.g., embarrassment, disrupted family rituals), as well as adaptations of previously used items examining psychological, physical, and sexual abuse.

## *Results*

The prevalence of alcohol use disorder decreased over time, from late adolescence to young adulthood [22, 21, 19, 18, 14, and 10%; analysis of variance $\chi^2(5) = 37.71$]. This decline did not significantly differ by FH or sex.

There was a significant relationship between family history and AUD across all waves; odds ratios (95% confidence interval) ranged from 1.62 (1.02, 2.58) to 2.75 (1.34, 5.68). The association was stronger in women (and was generally significant), echoing prior research, and was inconsistently significant in men. Logistic regressions predicting AUD from FH and sex revealed that there was a stronger association between FH and AUD among women at Years 1, 4, and 11 ($p < 0.05$ at Year 1, $p = 0.07$ at Years 4 and 11).

We examined mediation of the relation between family history and alcohol use disorder in a latent growth curve model framework (Muthen, 1991; Muthen and Curran, 1997), with an intercept and linear slope representing AUD over the 6 waves. We first estimated a simple growth model with FH modeled as an exogenous variable. Although FH had a significant effect on the intercept for the full sample (standardized $\beta = 0.21$) and for women ($\beta = 0.35$), it did not significantly predict the negative growth (decrease) in AUD over the 11 years.

**Table 1.** Mediators

| | Negative affectivity (NA) | Behavioral undercontrol (BU) | Cognitive ability (COG) | Alcohol expectancies (AE) | Childhood stressors (CS) |
|---|---|---|---|---|---|
| FH → Mediators[a] | | | | | |
| Overall | 0.08 | 0.32** | −0.13* | 0.16** | 0.56** |
| Men | 0.11 | 1.04** | −0.20 | 0.87 | 4.45** |
| Women | 1.53* | 0.60** | −0.49* | 2.65** | 7.88** |
| Mediators → AUD[b] | Int/Slope | Int/Slope | Int/Slope | Int/Slope | Int/Slope |
| Overall | 0.31**/–0.25* | 0.76**/–0.70** | −0.23*/0.25* | 0.53**/–0.47** | 0.20**/–0.12 |
| Men | 0.05***/–0.04 | 0.48**/–0.37** | −0.24***/0.24*** | 0.08**/–0.06*** | 0.02/–0.003 |
| Women | 0.12**/–0.09* | 0.83**/–0.69** | −0.29**/0.24*** | 0.10**/–0.08** | 0.05**/–0.03 |

*Note.* Overall parameters are standardized regression coefficients. Parameters for men and women are unstandardized regression coefficients. The model testing Mediators → AUD test each mediator in a separate framework. FH, family history of alcoholism; AUD, past 12-month alcohol use disorder; Int, Intercept for AUD; Slope, Linear slope for AUD.

[a]Overall $N = 452$; Men $n = 217$; Women $n = 235$.

[b]Overall $N = 396$; Men $n = 181$; Women $n = 215$.

**$p < 0.01$; *$p < 0.05$; ***$p < 0.10$.

The full mediational model contained FH, the AUD intercept and slope, and all 5 mediators (negative affectivity, behavioral undercontrol, cognitive ability, alcohol expectancies, and childhood stressors). First, we considered the extent to which FH predicted the purported mediators. Family history of alcoholism significantly predicted all 5 mediators for women, but only BU and CS for men. Table 1 presents regression coefficients for each mediator for the overall sample, for men, and for women. Standardized parameters are presented for the full sample to show a measure of effect size; unstandardized parameters are presented for men and women to compare across sex. Only the FH → CS relation significantly differed across sex, $\Delta\chi^2(1) = 16.07$, $p < 0.001$.

Next, we considered prediction of AUD by each of the mediators. Because of high multicollinearity among predictors, we examined mediation univariately. As illustrated in Table 1, women showed significant prediction of the intercept by all mediators, and men showed significant prediction by BU and AE. For the linear slope, NA, BU, and AE were significant for women, and BU was significant for men. Significant ($p < 0.01$) sex differences in the mediator–AUD relation were observed with the exception of prediction of intercept and slope by BU in both genders. Focused mediational tests concluded that BU was a significant mediator of the FH-AUD intercept relation for men ($z = 2.35$). Because of a sparse cell at Wave 6 (no FH-negative women were diagnosed with AUD), mediational tests did not converge for women.

### *Discussion*

Family history had a modest effect on the risk of developing AUDs assessed over early adulthood, providing evidence of familial transmission of alcoholism. This effect was stronger for women, consistent with some prior research, and appeared to be stronger at later waves. This is notable because FH was assessed at baseline, and one might expect the effect to become weaker over time.

In general, the mechanisms underlying the familial transmission of alcoholism appeared to be consistent with the general literature, with behavioral undercontrol clearly the strongest mediator for both genders. This may be in part because of an association between parental alcoholism and parental antisociality. However, both negative affectivity and, in particular, childhood stressors may be stronger correlates of AUD in women.

Our study has a number of strengths, including an intensive assessment of family history, as well as a prospective design, with 6 waves over 11 years and a high retention rate. However, the study has some drawbacks as well: the sample size was small for forming subgroups, our college student sample censored general deviance, and there was some differential attrition across AUD status. Finally, rates of AUD were high, in part, because of the high-risk nature of the sample, half of whom had a paternal history of alcoholism.

## GENDER DIFFERENCES IN PARENTAL ALCOHOLISM AND ALCOHOL USE PROBLEMS AND DEPRESSION AMONG ADULT DAUGHTERS

*Aruna Gogineni, Robert Stout, Richard Longabaugh, Robert Woolard, and David Strong*

### *Introduction*

Previous studies have clearly demonstrated that offspring of alcoholic individuals are at elevated risk for alcoholism (Cotton, 1979; Lieb et al., 2002; Sher, 1991). In addition, investigators have found that the gender of the alcoholic parent differentially affects the risk for alcoholism among offspring (Bidaut-Russell et al., 1994; Sher, 1991), although the pattern of findings reported has been somewhat inconsistent. In line with the general offspring of alcoholic individuals literature, several studies suggest that rates of alcoholism and alcohol-related negative consequences are higher among daughters of alcoholic mothers than among daughters of nonalcoholic mothers (Bidaut-Russell et al.,

1994; Harford et al., 1987; Sher, 1991). In addition, family (Cotton, 1979) and adoption (Bohman et al., 1981) studies indicate that same-sex parent–offspring alcoholism rates (e.g., mother–daughter pairs) are higher than opposite-sex rates (e.g., father–daughter pairs). However, other investigators find that paternal alcoholism is associated with a greater risk for alcoholism and alcohol-related negative consequences among adult daughters than among sons (Sher, 1991). Still other studies suggest that all offspring of alcoholic mothers, regardless of gender, have higher rates of alcoholism than offspring of alcoholic fathers (Harford et al., 1987).

In addition to the specific gender of the alcoholic parent, the density of familial alcoholism has been found to be associated with offspring's risk for alcoholism. Thus, individuals with 2 alcoholic parents are more likely to be alcoholic than individuals with only 1 alcoholic parent (Hesselbrock et al., 1982; Lieb et al., 2002). In addition, offspring from families with a larger number of alcoholic relatives are at increased risk for alcoholism compared with offspring coming from families with fewer affected relatives (Lieb et al., 2002).

Most of the aforementioned findings regarding gender differences in parental and offspring alcoholism are derived from clinical samples of treated alcoholic parents (West and Prinz, 1987) or from college student samples (Sher, 1991). Furthermore, most previous research has examined the effects of parental alcoholism on male offspring only. Few investigators have systematically examined the extent to which gender differences in parental alcoholism are associated with alcohol problems and other outcomes in adult daughters.

This study focuses on a community sample of female offspring drawn from an emergency department setting. We systematically compare (a) parental alcoholism with no parental alcoholism, (b) maternal versus paternal alcoholism, and (c) dual versus single parental alcoholism, with respect to rates of alcoholism, alcohol-related negative consequences, illicit drug use, and depression symptoms among adult daughters. In addition, our study addresses the extent to which these outcomes vary among adult daughters having a high versus low density of familial alcoholism by focusing specifically on the number of siblings with alcoholism.

## Materials and Methods

*Sample and Recruitment Procedure*. The current study was conducted in an urban emergency department medical setting in Rhode Island from which 245 females $\geq 18$ years of age were recruited. Women who had lived at least for 9 years, prior to 18 years of age, with biological parent(s) were screened for parental history of alcoholism using the SMAST for Mothers and Fathers (M-/F-MAST) (Crews and Sher, 1992; Sher and Descutner, 1986). Women who were eligible after initial screening (M-/F-MAST $\leq 1$ or $\geq 5$) were interviewed to confirm or rule out parental alcoholism using the FH-RDC (Endicott et al., 1978), and subjects were considered eligible to participate in the study only if they also met the criteria on the FH-RDC. Four groups of women with a history of (a) paternal alcoholism ($n = 63$), (b) maternal alcoholism ($n = 61$), (c) dual parental alcoholism ($n = 61$), and (d) no parental alcoholism ($n = 60$) were recruited into the study. The mean age of the sample was 30 (SD = 10.54) years, and the mean number of years of education was 12 years (SD = 2.41). Sixty percent of the sample was white.

*Measures of Parental Alcoholism and Other Pathology*. The adapted SMAST (Selzer et al., 1975) for Fathers (F-MAST) and Mothers (M-MAST) was used to assess for a family history of parental alcoholism. Each measure consists of 13 items that address fathers' and mothers' drinking behavior and alcohol-related negative consequences. The FH-RDC Interview (Endicott et al., 1978) was used to measure the participants' family history of parental alcoholism and other psychiatric disorders such as drug abuse, antisocial personality, and depression.

*Measures of Offspring Alcohol Involvement, Substance Involvement, and Depression*. Lifetime drinking consequences were assessed with the DrInC (Miller et al., 1995), which contains 45 true/false items with a range of consequences. Items from the Alcohol Use Disorders Identification Test (Saunders et al., 1993) were incorporated to assess recent quantity/frequency of alcohol use and past year symptoms of dependence. The Diagnostic Interview Schedule-Version IV (DIS) (Robins, 1999) was used to measure DSM-IV alcohol abuse and dependence symptoms. Lifetime use of illicit drugs was assessed through the drug use section of the Addiction Severity Index (McLellan et al., 1992). Recent depression symptoms among the adult daughters were measured through the administration of the Beck Depression Inventory (Beck et al., 1988). Finally, the FH-RDC (Endicott et al., 1978) was used to assess alcoholism in the siblings of the adult daughters.

*Analyses*. Univariate analyses compared family history groups on background variables of age, minority status, familial income, years of education, and parental psychiatric history. There were statistically significant but small differences between the groups on age, income, years of education, and the prevalence of parental psychopathology. These group differences were controlled by using these factors as covariates in subsequent analyses focusing on study outcomes of interest. Multivariate analysis of covariance was used to test the effect of group on current heavy alcohol use, drinking-related negative consequences, and lifetime symptoms of alcohol dependence, with the following planned comparisons: (a) all family history–positive groups (FH+) versus family history–negative groups (FH−); (b) maternal history only (MA+) versus paternal history only (PA+); and (c) dual parental history (Dual+) versus single parental history (MA+ or PA+). The 2-way interactions of family history status with

covariates were tested as a block after all other terms. In secondary analyses, we examined group differences in lifetime use of illicit drugs and current depression symptoms. It was anticipated that women with 2 alcoholic parents, compared with women with one or with no alcoholic parent, would manifest more (a) heavy alcohol use; (b) alcohol-related problems; (c) alcohol dependence; (d) illicit drug use; and (e) depression symptoms. Among women with 1 alcoholic parent, it was hypothesized that women with a history of maternal alcoholism would evidence more of the aforementioned outcomes than women with a history of paternal alcoholism because of the empirical literature relating to same-sex parent–offspring alcoholism rates.

*Results*

When differences in past-month alcohol use were examined across family history groups, we found that adult daughters with a positive FH had more heavy drinking days in the past month compared with the controls ($p<0.05$), but there were no differential rates within the family history–positive groups. We also found that women with a positive FH had more lifetime drinking-related negative consequences ($p<0.01$) and more lifetime alcohol dependence symptoms compared with women without a FH ($p<0.01$). There were no significant parental gender differences within the family history positive groups or among women who had dual versus single parental alcoholism.

In examining the family density variable, it was found that having a sibling with alcoholism was related to more symptoms of alcohol dependence ($p<0.05$). However, having a sibling with alcoholism was related to significantly more alcohol-related negative consequences regardless of the presence of parental alcoholism ($p<0.01$).

In examining the secondary outcomes of the study, we found that women with a positive FH had more depression symptoms than women without a FH. In addition, women with 2 alcoholic parents had higher levels of depression symptoms than women with 1 alcoholic parent, but there were no differences in depression symptoms among women with a history of maternal versus paternal alcoholism. Female offspring of maternal alcoholic individuals were 4 times more likely to use illicit drugs over the lifetime compared with female offspring without a FH, while there was no increased risk for adult daughters with a history of paternal alcoholism. Female offspring with both alcoholic parents were 3 times more likely to use illicit drugs.

*Discussion*

Our findings indicate that female offspring with a positive history of parental alcoholism were at increased risk for lifetime symptoms of alcohol dependence and alcohol-related negative consequences compared with female offspring without a positive FH. Women with a positive FH also reported more heavy drinking days ($>3$ drinks) in the past month and had more quantity/frequency of drinking compared with women who did not have a positive family history. These findings are consistent with those reported in the empirical literature, which delineates the increased risk for alcohol use problems among women with a positive history of parental alcoholism. However, no differences in daughters' alcohol involvement were observed with respect to maternal versus paternal alcoholism. We found a similar pattern when other secondary outcomes such as depression symptoms and lifetime illicit drug use were examined.

There were no differences found in alcohol use problems between women with a history of maternal versus paternal alcoholism or dual versus single parental alcoholism, although the trends were in the hypothesized direction for the latter comparison. Our finding that having a sibling with alcoholism relates to significantly more alcohol-related negative consequences regardless of the presence of parental alcoholism is an important and interesting one in that it underscores the relevance of considering environmental pathways in understanding offsprings' alcohol use problems.

Our investigation has several strengths, which include the use of a non–treatment-seeking sample of adult offspring, the use of rigorous criteria to establish/rule out a FH, and the use of a control group without a positive FH. However, our study's findings need to be viewed in the context of its limitations, such as the use of a cross-sectional naturalistic design, the presence of small sample sizes in the subgroups of adult offspring, and the low prevalence rates of alcohol problems in the sample.

## REFERENCES

Allison PD (2001) Logistic Regression Using the SAS System: Theory and Application. SAS Institute, Cary, NC.

American Psychiatric Association (1980) Diagnostic and Statistical Manual of Mental Disorders, 3rd Edition (DSM-III). American Psychiatric Association, Washington, DC.

American Psychiatric Association (2000) Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision (DSM-IV-TR). 4th ed. American Psychiatric Association, Washington, DC.

Beck AT, Steer RA, Garbin MG (1988) Psychometric properties of the Beck depression inventory: twenty-five years of evaluation.

Begleiter H, Reich T, Hesselbrock V, Porjesz B, Li TK, Schuckit M, Edenberg H, Rice J, Schuckit M (1995) The collaborative study on the genetics of alcoholism. Alcohol Health Res World 19:228–236.

Bidaut-Russell M, Bradford SE, Smith EM (1994) Prevalence of mental illnesses in adult offspring of alcoholic mothers. Drug Alcohol Depend 35:81–90.

Bierut LJ, Dinwiddie SH, Begleiter H, Crowe RR, Hesselbrock V, Nurnberger JI Jr, Porjesz B, Schuckit MA, Reich T (1998) Familial transmission of substance dependence: alcohol, marijuana, cocaine, and habitual smoking: a report from the collaborative study on the genetics of alcoholism. Arch Gen Psychiatry 55:982–988.

Bohman M, Sigvardsson S, Cloninger CR (1981) Maternal inheritance of alcohol abuse. cross-fostering analysis of adopted women. Arch Gen Psychiatry 38:965–969.

Bucholz KK, Cadoret RJ, Cloninger CR, Dinwiddie SH, Hesselbrock VM, Nurnberger JIJ Jr., Reich T, Schmidt I, Schuckit MA (1994a) A new, semi-structured psychiatric interview for use in genetic linkage studies: a report on the reliability of the SSAGA. J Stud Alcohol 55: 149–158.

Bucholz KK, Helzer JE, Shayka JJ, Lewis CE (1994b) Comparison of alcohol dependence in subjects from clinical, community, and family studies. Alcohol Clin Exp Res 18:1091–1099.

Chassin L, Curran PJ, Hussong AM, Colder CR (1996) The relation of parent alcoholism to adolescent substance use: a longitudinal follow-up study. J Abnorm Psychol 105:70–80.

Chassin L, Pitts SC, DeLucia C, Todd M (1999) A longitudinal study of children of alcoholics: predicting young adult substance use disorders, anxiety, and depression. J Abnorm Psychol 108:106–119.

Chassin L, Rogosch F, Barrera M (1991) Substance use and symptomatology among adolescent children of alcoholics. J Abnorm Psychol 100:449–463.

Cloninger CR (1987) Tridimensional Personality Questionnaire, Version 4. Unpublished Manuscript, Washington University, St. Louis.

Colder CR, Chassin L, Stice EM, Curran PJ (1997) Alcohol expectancies as potential mediators of parent alcoholism effects on the development of adolescent heavy drinking. J Res Adol 7:349–374.

Cotton NS (1979) The familial incidence of alcoholism: a review. J Stud Alcohol 40:89–116.

Crews TM, Sher KJ (1992) Using adapted short MASTs for assessing parental alcoholism: reliability and validity. Alcohol Clin Exp Res 16: 576–584.

Crum RM, Harris EL (1996) Risk of alcoholism and parental history: gender differences and a possible reporting bias. Genet Epidemiol 13:329–341.

Curran GM, Stoltenberg SF, Hill EM, Mudd SA, Blow FC, Zucker RA (1999) Gender differences in the relationship among SES, family history of alcohol disorders and alcohol dependence. J Stud Alcohol 60: 825–832.

Dick DM, Edenberg HJ, Xuei X, Goate A, Kuperman S, Schuckit M, Crowe R, Smith TL, Porjesz B, Begleiter H, Foroud T (2004) Association of GABRG3 with alcohol dependence. Alcohol Clin Exp Res 28:4–9.

Dick DM, Rose RJ, Pulkkinen L, Kaprio J (2000) Measuring puberty and understanding its impact: a longitudinal study of adolescent twins. J Youth Adol 30:385–400.

Endicott J, Andreasen N, Spitzer RL (1978) Family History-Research Diagnostic Criteria (FH-RDC). New York State Psychiatric Institute, New York.

Eysenck HJ, Eysenck SBG (1975) Manual of the Eysenck Personality Questionnaire. Educational and Industrial Testing Services, San Diego.

Fenigstein A, Scheier MF, Buss AH (1975) Public and private self-consciousness: assessment and theory. J Consult Clin Psychol 43: 522–527.

Grant BF, Dawson DA, Stinson FS, Chou P, Dufour MC, Pickering R (2004) The 12-month prevalence and trends in DSM-IV alcohol abuse and dependence: United States, 1991–1992 and 2001–2002. Drug Alcohol Depend 74:223–234.

Harford TC, Haack MR, Spiegler DL (1987) Positive family history for alcoholism. Alcohol Health Res World 12:138–143.

Heath AC, Slutske WS, Madden PAF (1997) Gender differences in the genetic contribution to alcoholism risk and to alcohol consumption patterns. in Gender and Alcohol: Individual and Social Perspectives (Wilsnack RW, Wilsnack SC eds), pp 114–119. US: Rutgers Center of Alcohol Studies, Piscataway.

Hesselbrock MN, Easton C, Bucholz KK, Schuckit M, Hesselbrock V (1999) A validity study of the SSAGA—a comparison with the SCAN. Addiction 94:1361–1370.

Hesselbrock VM, Stabenau JR, Hesselbrock MN, Meyer RE, Babor TF (1982) The nature of alcoholism in patients with different family histories for alcoholism. Prog Neuropsychopharmacol Biol Psychiatry 6:607–614.

Hussong AM, Curran PJ, Chassin L (1998) Pathways of risk for accelerated heavy alcohol use among adolescent children of alcoholic parents. J Abnorm Child Psychol 26:453–466.

Kuperman S, Schlosser SS, Kramer JR, Bucholz K, Hesselbrock V, Reich T, Reich W (2001) Developmental sequence from disruptive behavior diagnosis to adolescent alcohol dependence. Am J Psychiatry 158:2022–2026.

Lieb R, Merikangas KR, Hofler M, Pfister H, Isensee B, Wittchen HU (2002) Parental alcohol use disorders and alcohol use and disorders in offspring: a community study. Psychol Med 32:63–78.

LoCastro J, Spiro A III, Monnelly E, Ciraulo D (2000) Personality, family history, and alcohol use among older men: the VA Normative Aging Study. Alcoholism: Clin Exp Res 24:501–511.

McLellan AT, Kushner H, Metzger D, Peters R, Smith I, Grissom G, Pettinati H, Argeriou M (1992) The Fifth Edition of the Addiction Severity Index. J Subst Abuse Treat 9:199–213.

Miller WR, Tonigan JS, Longabaugh R (1995) The Drinker Inventory of Consequences (DrInC). An instrument for assessing adverse consequences of alcohol abuse. in Project MATCH Monograph Series. Rockville.

Muthen B (1991) Analysis of longitudinal data using latent variable models with varying parameters. in Best Methods for the Analysis of Change. Recent Advances, Unanswered Questions, Future Directions (Collins L, Horn J eds), pp 1–17. American Psychological Association, Washington, DC.

Muthen BO, Curran PJ (1997) General longitudinal modeling of individual differences in experimental designs: a latent variable framework for analysis and power estimation. Psychol Methods 2: 371–402.

Ohannessian CM, Hesselbrock VM, Kramer J, Kuperman S, Bucholz KK, Schuckit MA, Nurnberger JI Jr (2004) The relationship between parental alcoholism and adolescent psychopathology: a systematic examination of parental comorbid psychopathology. J Abnorm Child Psychol 32:519–533.

Pollock VE, Schneider LS, Gabrielli WF Jr., Goodwin DW (1987) Sex of parent and offspring in the transmission of alcoholism. A meta-analysis. J Nerv Mental Dis 175:668–673.

Prabhu VR, Porjesz B, Chorlian DB, Wang K, Stimus A, Begleiter H (2001) Visual p3 in female alcoholics. Alcohol Clin Exp Res 25:531–539.

Preisig M, Fenton BT, Stevens DE, Merikangas KR (2001) Familial relationship between mood disorders and alcoholism. Compr Psychiatry 42:87–95.

Rice JP, Reich T, Bucholz KK, Neuman RJ, Fishman R, Rochberg N, Hesselbrock VM, Nurnberger JI Jr, Schuckit MA, Begleiter H (1995) Comparison of direct interview and family history diagnoses of alcohol dependence. Alcohol Clin Exp Res 19:1018–1023.

Robins LN (1999) Diagnostic Interview Schedule for DSM-IV (DIS-IV). Washington University School of Medicine, St. Louis.

Russell M, Cooper M, Frone MR (1990) The influence of sociodemographic characteristics on familial alcohol problems: data from a community sample. Alcohol Clin Exp Res 14:221–226.

Saunders JB, Aasland OG, Amundsen A, Grant M (1993) Alcohol consumption and related problems among primary health care patients: WHO collaborative project on early detection of persons with harmful alcohol consumption-I. Addiction 88:349–362.

Schuckit MA, Daeppen JB, Tipp JE, Hesselbrock M, Bucholz KK (1998) The clinical course of alcohol-related problems in alcohol dependent and nonalcohol dependent drinking women and men. J Stud Alcohol 59:581–590.

Schuckit MA, Smith TL (1996) An 8-year follow-up of 450 sons of alcoholic and control subjects. Arch Gen Psychiatry 53:202–210.

Schuckit MA, Smith TL, Danko GP, Isacescu V (2003) Level of response to alcohol measured on the Self-rating of the effects of alcohol questionnaire in a group of 40-year-old women. Am J Drug Alcohol Abuse 29:191–201.

Selzer ML, Vinokur A, van Rooijen L (1975) A self-administered short Michigan alcoholism screening test (SMAST). J Stud Alcohol 36: 117–126.

Sher KJ (1991) Children of Alcoholics: A Critical Appraisal of Theory and Research. University of Chicago Press, Chicago.

Sher KJ, Descutner C (1986) Reports of paternal alcoholism: reliability across siblings. Addict Behav 11:25–30.

Sher KJ, Gershuny BS, Peterson L, Raskin G (1997) The role of childhood stressors in the intergenerational transmission of alcohol use disorders. J Stud Alcohol 58:414–427.

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 100:427–428.

Sher KJ, Wood MD, Wood PK, Raskin G (1996) Alcohol outcome expectancies and alcohol use: a latent variable cross-lagged panel study. J Abnorm Psychol 105:561–574.

Suresh S, Porjesz B, Chorlian DB, Choi K, Jones KA, Wang K, Stimus A, Begleiter H (2003) Auditory P3 in female alcoholics. Alcohol Clin Exp Res 27:1064–1074.

Wallace JM, Brown TN, Bachman JG, Laveist TA (2004) The influence of race and religion on abstinence from alcohol, cigarettes and marijuana among adolescents. J Stud Alcohol 64:843–848.

Wechsler D (1981) Wechsler Adult Intelligence Scale-Revised. (WAIS-R) Manual. Psychological Corporation, New York.

West MO, Prinz RJ (1987) Parental alcoholism and childhood psychopathology. Psychol Bull 102:204–218.