

Family History of Alcoholism and Childhood Adversity: Joint Effects on Alcohol Consumption and Dependence

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Empirical studies provide substantial evidence that having a family history of alcoholism increases the risk of developing alcohol dependence; however, some of this effect may be caused by nonspecific childhood socioeconomic adversity common in families with an alcohol-dependent parent. In this study, we examine joint effects of family history and childhood adversity within a sample of 509 men and 217 women over age 40. The measures analyzed were included in routine screening assessments for participants in various studies at the University of Michigan Alcohol Research Center. About 60% of the men and 45% of the women were alcohol-dependent. About 30% reported an alcoholic parent. Degree of family history affected drinking behavior for both men and women. There were also environmental effects on the same measures for both men and women. Childhood socioeconomic adversity was reported more frequently by participants with an alcoholic parent, but adversity effects were also shown for those with a negative family history. The risk of alcohol dependence was additively increased by a positive family history and childhood socioeconomic adversity. The environmental effects identified in this study are promising evidence for nonspecific factors that moderate family history risk for development of alcohol problems.

Key Words: Family History, Childhood Adversity, Risk Factors, Alcohol Dependence, Gender Differences.

EMPIRICAL STUDIES provide substantial evidence that a family history of alcoholism increases the risk of developing alcohol dependence.¹ Such a family history predisposes individuals to greater alcohol consumption and more severe consequences from use of alcohol and other drugs.²⁻⁹ Among alcohol-dependent patients, family history is associated with more severe symptoms.¹⁰⁻¹⁸ The majority of offspring of alcohol-dependent parents do not develop problems with alcohol, however, and only about one-third develop alcohol dependence.^{19,20} It must be kept in mind that genetic studies indicate that environment contributes to alcohol misuse as strongly as does biology, a pattern that applies to most psychiatric disorders.²¹

Environmental factors that moderate family risk need to be delineated more fully. Many of the effects of having alcohol-dependent parents may be nonspecific effects of parental impairment, as has been shown for parental

depression.²² Numerous effects on the family of parental alcohol dependence have been demonstrated.²³⁻²⁸ Reich et al.²⁹ found that children of alcohol-dependent parents experienced a worse home environment than did a control group, as measured by marital conflict and parent-child conflict. These problems are nonspecific, also occurring in families without an alcohol-dependent parent. Sher³⁰ concluded that home environments in families of active alcoholics are markedly different from those in families of both nonalcoholics and recovering alcoholics. One nonspecific adversity that is likely to affect outcome of children in families of active alcoholics is poverty. Very low socioeconomic status has been associated with higher rates of drug and alcohol use among youth³¹ and with higher prevalence of adult alcoholism.³² Another nonspecific risk might be a later birth order, because last-born offspring were more likely to imitate the drinking level of their same-sex parent.³³ Nonspecific effects of childhood environment do not appear to be sufficient to cause the development of alcoholism without the presence of family history risk,³⁴ but these factors have not been examined sufficiently.

The role of gender differences in the etiology of alcoholism is particularly unclear. In Cloninger's type I and II classification system, consisting of *milieu-limited* and *male-limited* subtypes, environmental effects are reportedly shown only in the first type.³⁵ Conflicting results from adoption studies with women show substantial increased risk for female offspring of alcoholic parents,³⁶ little genetic risk,³⁷ or genetic risk only for daughters of type I fathers.³⁸ Recent reviews of adoption studies have criticized in particular their inadequate specification of possible environmental or intrauterine influences, diagnostic criteria, and statistical analyses.³⁹⁻⁴¹

Two recent twin studies^{42,43} have reported higher concordance for *alcohol dependence* in monozygotic than dizygotic twins for both men and women. In both, family risk was largely attributable to genetic factors for alcohol dependence. Nonshared environment was a significant component of family risk for women only. Concordance in *alcohol abuse* showed a genetic influence only in male twins, in the one study that modeled it separately.⁴³ Therefore, environmental and genetic influences may vary for different aspects of drinking behavior, and these relationships may differ for men and women.

The current authors conducted a preliminary study of

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the associations among parental alcohol dependence, family environment, and alcohol misuse measures with young adults in their twenties.⁴⁴ Family history affected only substance abuse patterns, not psychological symptomatology. Although a high level of alcohol problems occurred in both groups, participants with an alcohol-dependent parent were more likely to be heavy drinkers and showed more symptoms of alcohol dependence. The most important correlates of alcohol misuse were degree of exposure to consequences of parental alcoholism and a history of abusive punishment by parents. Within families with an alcohol-dependent parent, positive family relationships were protective, moderating the potential negative effects of a family history of alcoholism.

In this study, we examine the effect of a family history for alcoholism on adult alcohol use. We analyzed these effects on a heterogeneous sample of participants in studies at the University of Michigan Alcohol Research Center (UMARC), a center funded to focus on alcohol and aging by the National Institute of Alcoholism and Alcohol Abuse. We expected that both men and women with a family history of alcoholism (FH⁺) would be more likely to report adverse consequences from drinking, alcohol dependence, and more severe dependence; we also expected that these effects would be stronger with a stronger family history. In addition, we expected men and women with a positive family history to be less likely to exhibit alcohol dependence or other negative outcomes if they experienced less childhood socioeconomic adversity.

METHODS

Sample

The study sample consisted of 509 men and 217 women recruited for studies at the UMARC. Analyses included only those participants who were at least 40 years old, an age that represents ~85% of the distribution of age of onset of the first alcohol problem for alcohol-dependent participants in UMARC studies. Table 1 shows the composition of the sample in recruitment source, demographic characteristics, alcohol dependence, and reported parental drinking status. Less than half the sample were recruited from alcoholism treatment settings; other sources included community outreach (advertising, direct mailings, and word of

Table 1. Sample Characteristics

	Men (n = 509)	Women (n = 217)
Age	60.6 (10.0)	63.8 (9.8)
Education	14.0 (3.0)	13.8 (2.6)
Race (Caucasian)	88.6%	92.7%
Ever married*	92.5%	92.2%
Occupational prestige	48.0 (23.4)	43.8 (17.6)
Recruitment sources		
Treatment units	43.6%	39.2%
Community outreach	34.8%	27.7%
Other	21.6%	33.2%
Alcohol-dependent	61.9%	45.6%
Familial alcoholism		
Parent	32.6%	31.8%
Mother	8.5%	7.4%
Father	28.5%	29.5%

Percentages or means and standard deviations are shown.

* Legal marriages only.

mouth) and recruitment from other medical settings, such as general hospital wards. Approximately 62% of the men and 46% of the women recruited were alcohol-dependent ($\chi^2 = 16.62, p < 0.001$), and ~32% of both groups had an alcoholic parent. The samples of men and women differed on age, occupational status, and recruitment source ($p < 0.05$). The average participant was a 60-year-old Caucasian volunteer with some college education.

Measures analyzed herein were extracted from the initial screening interviews used to assess eligibility for ongoing studies at UMARC during 1989–1991. Screening and assessment interviews were conducted in two phases. After an initial brief interview, those who consented and were eligible for further studies were given the structured diagnostic interview analyzed herein. Many of those initially screened (>2000) were ineligible for further studies because of diagnosis-related selection criteria (e.g., inpatients with a primary drug of abuse other than alcohol, community recruits who screened positive for alcoholism) or medical exclusion criteria for biological research projects, such as use of aspirin (for a study of alcohol and the immune system). Although about three-fourths of the participants were willing to have the 2nd level of screening interviews, only ~40% were included, based on eligibility for ongoing studies and priorities for recruitment. This subgroup participated in the structured diagnostic interviews that are used for the present analyses. Participants resemble local county residents in education, race, and marital status. The atypical average age of the participants resulted from the Center's focus on alcohol and aging research and the exclusion of younger subjects (under age 40) for the present analysis.

Measures

Psychiatric Diagnoses. The Diagnostic Interview Schedule (DIS-III-R)⁴⁵, was administered to all participants. The DIS takes ~1–2 hr and can be administered by trained interviewers without clinical expertise. DIS measures used were lifetime alcohol dependence, severity of dependence, and age of onset. Severity of dependence is presented as none or abuse only (collapsed over DIS codes 1 and 2), mild or moderate (DIS codes 3 and 4), and severe dependence (DIS code 5). The screening interview also included questions regarding past treatment history (number of treatment episodes and age at first treatment).

Alcohol Consumption History. Lifetime alcohol use was assessed using the Lifetime Drinking Patterns History, an instrument adapted from Skinner and Sheu.⁴⁶ This instrument was developed to define patterns of alcohol consumption during different periods throughout the life span. Drinking quantity, frequency, withdrawal symptoms, and so forth are recorded for each period. A pattern change was defined as a shift of a specified degree in the quantity, frequency, or reported symptoms of consumption. Measures analyzed from this interview included age at 1st drink, age of regular drinking, an estimate of lifetime total drinks, and an estimate of lifetime average for days one was drinking.

Family History. Family history of alcoholism was assessed during interviews with the participant. A pedigree was constructed, showing children, parents, siblings, aunts, uncles, and grandparents. For each relative, level of alcohol use was assigned (abstinent, social drinker, and probable or definite alcoholism). Alcoholism was coded using the criteria from the Family Informant Schedule and Criteria (FISC).⁴⁷ Probable alcoholism was coded when the respondent could give examples of the relative being frequently drunk, drinking regularly, and heavily, or "always has a glass in his hand." Definite was coded if the respondent could also name a specific consequence from the FISC list (legal, marital, work, or health problems, fights when drunk, or alcoholism treatment).

In most of the analyses presented herein, "probable" and "definite" codes were collapsed. Family history was analyzed using several different classifications, with FH⁺ first defined as parental alcoholism. Next, degree of family history was classified as "generational" [i.e., 1st- versus 2nd-degree relatives affected (herein, 1st degree was defined as sibling or parent; 2nd degree was grandparent, aunt, or uncle)]. Third, family history was classified by "lineality" [i.e., alcoholic relatives on one versus both maternal (mother or her siblings or parents) and paternal (father or

his siblings or parents) sides of a pedigree].¹⁰ Only relatives reported to be biological were included.

Multiple Environmental Risks. Some measures relevant to childhood adversity were available. Although measures of parental conflict or parent-child relationships were not included in the screening interview, measures of socioeconomic resources were. Stability of socioeconomic resources was assessed several ways. Parental occupations were given socioeconomic status ratings using Featherman and Steven's⁴⁸ index. Using this scale, an electrician is coded 30 and a lawyer 87. For men over age 45 in UMARC studies, this occupation rating has a Spearman correlation of ~ 0.7 (0.67 and 0.69) with personal and household income categories. In addition, one question assessed perceived poverty by asking about resources when the respondent was growing up. Potential responses ranged from, "We were poor and had a hard time paying for food and housing" to "We were well off or wealthy." This item was adapted from an interview used in a study of heroin-addicted patients in treatment.⁴⁹ In addition, having more than one sibling (with whom to share inadequate resources) was considered a risk, as was having a birth rank greater than the 1st (later-born siblings would be at a disadvantage in competing with older, larger siblings when resources were scarce). In general, younger siblings might be expected to have experienced a more advanced stage of a parent's alcoholism while at a more vulnerable age. An index of the number of childhood adversities was created by summing (1) presence of parental occupational prestige score below the group median (within gender), (2) perceived poverty in childhood, (3) more than one other sibling, and (4) a lower birth order (not 1st born). Adversity count, therefore, ranged from 0 to 4 for both men and women.

Statistical Analysis

Outcome measures were analyzed separately for men and women in parallel analyses; as previously noted, the samples of men and women differed on recruitment source and other characteristics, which makes gender effects on outcome harder to interpret. We tested the univariate effects of a 1st-degree family history on alcohol use measures, using χ^2 or analysis of variance. The multiple measures of alcohol use were then intercorrelated to determine which measures were redundant. Associations were calculated using Spearman correlation coefficients or Cramer's V (for categorical variables). The most independent, representative measures were subjected to further analyses. Alcohol use measures were examined for evidence of a biological pattern of family history effects, by testing family history three separate ways. Analyses were conducted classifying subjects first by parental alcoholism. Second, family history was classified generationally, as 2nd-degree relatives only, 1st-degree only, and both 1st- and 2nd-degree. Next, family history was classified lineally, as biparental (present in relatives of both mother and father) or uniparental (present in only one side). We intended also to analyze subgroups with both parents affected versus only the father affected. This last analysis was omitted, however, because only 20 men and 8 women had an alcoholic mother. To demonstrate cumulative change as degree and lineality of family history increased, we tested for a significant linear contrast effect subsequent to analysis of variance (for age at 1st drink and lifetime average drinks/drinking day) or logistic regression (for alcohol dependence).

Moderation of family history effects by cumulation of nonspecific socioeconomic risks was tested by examining the univariate associations of multiple risks (0-4) with outcome measures, within gender. Alcohol use variables were correlated with the index for multiple risks. The joint effects of family history and environmental risks were modeled for three drinking measures, separately for men and women. These factors and their interactions were simultaneously entered into multiple regressions, linear (age of 1st drink, drinks/day) or logistic (alcohol dependence). A second set of regression analyses was conducted with a reduced model, including only main effects as predictors.

RESULTS

Family History Effects

1st-Degree Family History. Table 2 shows the effect of having an alcohol-dependent parent on drinking patterns and alcohol dependence. Significant effects indicated are those between family history groups, separately compared for men and women. Parental alcoholism affected age at 1st drink, the probability of becoming dependent, consumption (drinks/day), and the frequency of substance abuse/dependence. Age at regular use, lifetime drinks, years of heavy drinking, and age at 1st treatment varied significantly with family history for men.

Because these measures of alcohol use were highly intercorrelated, three were chosen for further analyses of density of family history and childhood adversity: age at 1st drink, dependence severity, and lifetime drinking/day average. Lifetime average drinks/day correlated highly with years of heavy drinking ($r_s = 0.80$) and lifetime total drinks ($r_s = 0.84$). Age at 1st drink was associated with age at regular drinking ($r_s = 0.53$) and age at onset of the 1st symptom of dependence ($r_s = 0.39$). Dependent and nondependent participants differed significantly on most other consumption measures: age at 1st drink, age at regular drinking, drinks/day, lifetime total drinks, and years of heavy drinking (all $p < 0.0001$).

Density of Family History. Two classifications of family history density were employed to test quasigenetic effects. Differences by the generational classification are shown in Table 3. Genetic effects are implied when there is a linear, stair-step increase in an outcome measure, with the cumulative levels of family history, that is significant using a linear contrast. For men, there was a clear effect of a multiple-generation family history on alcohol dependence (Table 3). Rates of alcohol dependence went from 42.6% for those with no family history to 52.8% for those with only a 2nd-degree family history, to 71.7% for those with 1st only, to 82.6% of those with a history in both generations [linear contrast $\chi^2(1) = 34.92, p < 0.001$]. Lifetime drinks/day [linear contrast $F(1,451) = 23.23, p < 0.001$] and age at 1st drink [linear contrast $F(1,472) = 13.32, p < 0.001$] showed the same pattern. Substance abuse varied, but did not show a clear linear pattern. Women showed a significant cumulative pattern only for lifetime drinks/day [linear contrast $F(1,168) = 9.84, p = 0.002$], although the other measures would show similar results if the 2nd-degree only group were averaged with the negative group. Alcohol dependence and substance abuse measures for women did not show such a clear linear pattern; the highest severity appeared to be in the group with only an alcoholic parent (Table 3).

We also analyzed density of family history using lineality, comparing those with reported alcoholism in one parent's family versus both parents' families (uni- versus biparental; Table 4). Genetic effects are implied when measures are higher for those with a uniparental family

Table 2. Effects of Parental Alcoholism on Alcohol Consumption Patterns and Dependence

	Men		Women	
	FH ⁻ (n = 343)	FH ⁺ (n = 166)	FH ⁻ (n = 148)	FH ⁺ (n = 69)
Age of 1st drink	15.3	13.4 (5.3)**	18.3 (5.4)	15.5 (6.0)**
Age of regular use	19.9 (7.3)	17.3 (5.1)**	25.6 (12.5)	23.3 (8.8)
Alcohol dependence				
None (%)	46.1	21.7**	64.9	31.9**
Mild/moderate (%)	27.4	29.5	16.2	34.8
Severe (%)	26.5	48.8	18.9	33.3
Age onset/dependence	24.2 (10.2)	24.2 (11.2)	40.4 (15.0)	36.0 (13.6)
Drinks/day	5.0 (4.5)	6.9 (5.3)**	2.8 (2.7)	3.9 (3.7)*
Lifetime consumption	44,723 (55,220)	67,490** (72,237)	20,716 (36,972)	23,189 (20,944)
Years heavy drinking	91.0 (12.8)	14.2 (14.0)**	4.5 (8.2)	6.0 (7.7)
Years abstinent	6.8 (7.5)	7.8 (8.8)	9.3 (8.4)	11.1 (11.3)
Alcoholism treatments†	2.2 (2.1)	3.0 (4.6)	1.8 (1.4)	2.0 (2.8)
Age of 1st treatment‡	54.1 (12.1)	49.8 (10.3)**	60.4 (10.1)	56.4 (11.8)
Substance abuse/dependence	2.9%	9.0%**	4.1%	14.5%**

Percentages or means and standard deviations are shown.

* $p < 0.05$.

** $p < 0.01$.

† FH⁺ refers to parental alcoholism.

‡ Sample sizes for these measures are 203 men and 62 women.

Table 3. Effects of Generational Degree of Family History on Alcohol Dependence and Consumption

	Degree of family history				p value*
	None (n = 169)	2nd only (n = 91)	1st only (n = 99)	Both (n = 149)	
Men					
Age of 1st drink	15.6 (6.0)	15.5 (4.2)	14.1 (5.2)	13.5 (5.7)	0.003
Lifetime drinks/day	3.7 (3.9)	6.2 (5.0)	6.0 (5.4)	6.9 (4.6)	0.001
Alcohol dependence	42.6%	52.8%	71.7%	82.6%	0.001
Substance abuse/dependence	0.0%	6.6%	2.0%	11.4%	0.001
Women					
Age of 1st drink	18.4 (5.6)	17.2 (4.2)	17.7 (4.8)	16.2 (6.9)	0.186
Lifetime drinks/day	2.3 (1.4)	2.5 (2.0)	2.8 (2.1)	4.5 (4.6)	0.002
Alcohol dependence	31.9%	22.2%	53.6%	64.5%	0.001
Substance abuse/dependence	4.2%	3.7%	8.9%	11.3%	0.346

Percentages or means and standard deviations are shown.

* p values < 0.001 are shown as 0.001.

history than for those with none and are higher still for those with a biparental family history (also tested using a linear contrast effect). Alcoholism on both sides of the pedigree increased rates of alcohol dependence in both men and women [linear contrasts, men: $\chi^2(1) = 27.72$, $p < 0.001$; women: $\chi^2(1) = 13.73$, $p < 0.001$]; lifetime drinks/day was similarly affected [linear contrasts, men: $F(1,452) = 24.27$, $p < 0.001$; women: $F(1,169) = 6.26$, $p = 0.002$]. Age at 1st drink and substance abuse/dependence varied with lineality for both, but was statistically significant for only the male group [linear contrast $F(1,473) = 6.87$, $p = 0.009$], probably because of a larger sample size.

Multiple Environmental Risks

Univariate effects of childhood socioeconomic adversity on adult outcome were examined. Over one-half the par-

Table 4. Effects of Lineality of Family History on Alcohol Dependence and Consumption

	Degree of family history			p value*
	None (n = 207)	Uniparental (n = 197)	Biparental (n = 104)	
Men				
Age of 1st drink	15.5 (5.8)	14.4 (5.4)	13.8 (5.1)	0.021
Lifetime drinks/day	4.0 (4.0)	6.6 (4.8)	6.9 (5.5)	0.001
Alcohol dependence	48.5%	67.7%	77.1%	0.001
Substance abuse/dependence	0.0%	7.1%	10.5%	0.001
Women				
Age of 1st drink	18.3 (5.1)	16.7 (5.7)	16.6 (6.6)	0.120
Lifetime drinks/day	2.4 (1.8)	3.1 (3.1)	4.6 (4.3)	0.002
Alcohol dependence	33.0%	46.0%	71.7%	0.001
Substance abuse/dependence	4.1%	8.1%	13.0%	0.164

Percentages or means and standard deviations are shown.

* p values < 0.001 are shown as 0.001.

ticipants had >1 sibling (men 64.8%, women 68.4%) or a lower birth order (men 57.6%, women 57.4%). One-third had perceived their childhood environment as poor (men 35.0%, women 35.9%). By definition of the measure, about one-half reported parents with low-prestige occupations (men 51.8%, women 49.8%). The incidence of multiple childhood adversities was similar for men and women, with $>20\%$ experiencing four of the risks [men: 0 or 1: 34.1%, 2: 29.9%, 3 or 4: 36.2%; women: 0 or 1: 35.5%, 2: 25.4%, 3 or 4: 39.2%; $\chi^2(2) = 1.61$, $p = 0.447$]. Most men and women with a 1st degree family history had experienced at least one of the analyzed adversities. Fewer childhood adversities occurred for the FH⁻ groups [FH⁺ men: 0 or 1: 25.0%, 2: 29.0%, 3 or 4: 46.0%; FH⁻ men: 0 or 1: 42.7%, 2: 30.8%, 3 or 4: 26.5%; $\chi^2(2) = 25.1$, $p < 0.0001$; FH⁺ women: 0 or 1: 29.7%, 2: 23.7%, 3 or 4:

46.6%; FH⁻ women: 0 or 1: 42.4%, 2: 27.3%, 3 or 4: 30.3%; $\chi^2(2) = 6.40, p = 0.041$].

Table 5 shows the univariate associations of multiple childhood socioeconomic risks with alcohol use measures. Effects of childhood adversity are apparent. Age at 1st drink tended to vary: men with more risks began drinking at a younger age, whereas the reverse was true for women. Alcohol consumption was also higher with more childhood adversity for both genders. Alcohol dependence was more likely with more childhood adversity for both genders, and was highly significant for men. Although the percentage dependent was higher for those women with 3–4 risks, there was no apparent difference between groups with two versus one risk. Substance abuse/dependence was not significantly related to risks in this sample.

Joint Effects of Family History and Environmental Risks

Because FH⁺ participants experienced more childhood adversity, it was necessary to examine joint effects. The effects of childhood environment, family history, and gender are shown in Fig. 1 and Table 6. Multiple regression analyses showed family history had a significant main effect on age at 1st drink (Fig. 1A), drinks/day (Fig. 1B), and dependence (Fig. 1C), for both men and women. Childhood socioeconomic adversity affected drinks/day and alcohol dependence for both men and women. The pattern of effects was additive; no interaction effect reached statistical significance. Because there were only 42 participants who were positive for substance abuse/dependence, it was not analyzed in this manner. Figure 1 shows results for all groups, even though interaction effects were not significant.

When regressions were conducted without interaction terms, parental alcoholism remained highly significant and adversity effects became stronger (Table 6B). Using the final main effects models, parental alcoholism predicted a younger age at 1st drink, whereas socioeconomic adversity had no independent effect (Table 6B), although it appeared to increase the age at 1st drink for women with an alcoholic parent (Fig. 1A). Parental alcoholism

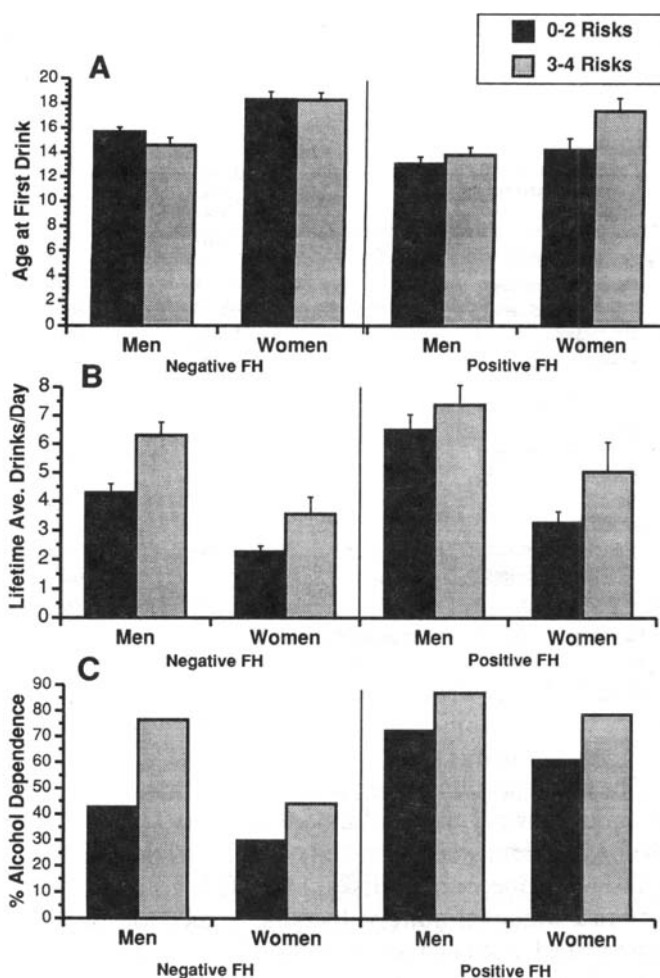


Fig. 1. Joint effects of parental alcoholism and childhood socioeconomic adversity on (A) age at 1st drink, (B) lifetime average drinks/drinking day, and (C) alcohol dependence.

and childhood socioeconomic adversity increased average drinks/day and the frequency of alcohol dependence for men and women (Table 6; Fig. 1, B and C).

DISCUSSION

The results corroborate other studies documenting the effect of a family history of alcoholism on alcohol and drug abuse or dependence. The increasing risk with greater density of family history is also congruent with other studies.¹⁰ Lineality and generational degree of family history affected drinking behavior for both men and women. Alcohol consumption and dependence measures appeared higher when women had at least a 1st-degree family history; a 2nd-degree family history had little effect, compared with no family history. There were environmental effects on measures of alcohol consumption and dependence for both men and women. Childhood socioeconomic adversity was more frequent for men and women with an alcoholic parent; nevertheless, adversity effects were shown within genders and family history groups. Although women appeared to be less likely to respond to socioeco-

Table 5. Association of Multiple Socioeconomic Risks with Various Outcome Measures

	Men (n = 509)	Women (n = 216)
Age of 1st drink	-0.09†	0.14*
Drinks/day	0.22**	0.17*
Alcohol dependence	V = 0.30**	V = 0.16†
0-1 risks	46.2%	40.3%
2 risks	57.2%	38.2%
3-4 risks	80.4%	55.3%
Substance abuse/dependence	V = 0.05	V = 0.06
0-1 risks	3.5%	6.5%
2 risks	5.3%	5.5%
3-4 risks	6.0%	9.4%

Spearman correlations or Cramer's V are shown.

*p < 0.05.

**p < 0.01.

†p < 0.10.

Table 6. Effects of Parental Alcoholism and Multiple Childhood Socioeconomic Risks on Alcohol Use by Men and Women

Effects	Age of 1st drink		Drinks/day		Alcohol dependence	
	F	p value*	F	p value	χ^2	p value
A. Full Model						
Men (n = 509)						
Parental alcoholism	13.69	0.001	15.03	0.001	9.72	0.001
Childhood adversity	1.93	0.147	5.80	0.003	35.81	0.001
Interaction	1.81	0.165	0.82	0.439	1.45	0.229
Model	4.01	0.001	6.44	0.001	74.45	0.001
Women (n = 217)						
Parental alcoholism	12.15	0.061	5.56	0.020	3.04	0.081
Childhood adversity	1.77	0.173	4.61	0.011	2.71	0.100
Interaction	1.80	0.168	0.20	0.818	0.00	0.977
Model	3.33	0.007	3.08	0.011	25.01	0.001
B. Main Effects Model						
Men (n = 509)						
Parental alcoholism	12.24	0.001	13.98	0.002	25.16	0.001
Childhood adversity	1.78	0.170	7.29	0.001	40.30	0.001
Model	5.46	0.001	10.19	0.001	73.02	0.001
Women (n = 217)						
Parental alcoholism	10.99	0.011	5.80	0.017	19.74	0.001
Childhood adversity	0.90	0.408	4.76	0.010	4.06	0.044
Model	4.32	0.006	5.05	0.002	25.01	0.001

*p values < 0.001 are shown as 0.001.

nomics or family history risks with alcohol dependence or heavy consumption, the pattern of effects was similar for men and women.

The risk of alcohol dependence was additively increased by family history and childhood adversity. Men with an alcoholic parent were likely to be alcohol-dependent, even with few socioeconomic risks. Men without a family history risk were still quite vulnerable to alcoholism if they experienced socioeconomic risks. This result contrasts with Valliant's longitudinal study, where poor childhood environment was associated with later alcoholism in only 5% of men with no family history.³⁴ In that study, 27% of men with alcoholic parents became alcoholic, even with no childhood environmental problems. The present results are similar, however, to our previous study with young adults with an alcoholic parent.⁴⁴ In that sample, virtually all the FH⁺ men were heavy drinkers, so little variation could be attributed to environmental effects.

Gender differences were not analyzed directly, because effects were tested separately for men and women. In the present sample, the female group had a lower frequency of alcohol dependence than males, but the predictors were similar; family history and adversity affected both groups. Family history effects are consistent with recent twin studies reporting genetic effects on alcohol dependence for men and women.^{42,43} Some results suggest possible gender differences for future research, however. For example, alcohol dependence and substance abuse measures for women did not show as clear a linear increase with degree of family history as for men; a 2nd-degree family history did not appear to be a risk factor for women. Women may require a higher susceptibility "load" before expressing alcohol dependence.⁵ Childhood socioeconomic adversity appeared to have different univariate effects on age at 1st drink; women with more risks began drinking at an older

age, whereas there was little effect for men. When joint effects of family history and adversity were modeled, however, adversity had no independent effect on age at 1st drink for men or women.

One limitation of this study is that the measures of childhood adversity and family history are retrospective self-report accounts; prospective family studies will be required for more precise measurement. Informant reports for psychiatric disorders show high specificity but generally low sensitivity. However, accuracy has been high for alcoholism.^{50,51} The family tree method has shown very high test-retest reliability for 1st-degree relatives, but lower reliability for 2nd-degree relatives.^{52,53} Mann et al.⁵² found better reliability for 2nd-degree relatives when using liberal criteria, such as the "probable" code used herein. Other studies report good correlations between adult children's reports of parental alcohol consumption and their parent's own reports.⁵⁴ As would be expected, agreement between siblings on parental drinking is better for objective than subjective items.⁵⁵ However, family history diagnoses are known to be biased in that probands with major depression or generalized anxiety overreport the same diagnosis in their parents, compared with the reports of a nonsymptomatic twin.⁵⁶ This bias is less evident for alcoholism, perhaps because the symptoms and consequences are more observable than are symptoms of anxiety and depression.⁵⁶ Family history data concerning alcoholism appears to be adequately reliable and less biased than informant reports about other psychiatric disorders.

Similarly, a potential bias exists if accuracy of retrospective recall by patients and nonpatients differs. In general, the effect of current psychiatric status on recall of childhood experiences depends on the type of experiences measured.^{57,58} A current diagnosis of depression consistently affected recall of subjective experiences, such as

relationships with parents, but not of more factual events, such as separation from parents.⁵⁸ Comparing sibling reports of childhood experience, Robins et al.⁵⁷ found good agreement on factual data (>70%), but worse agreement on value judgments or inferences about feelings (<50%). Patients did not systematically assess the family less favorably than did their well siblings. In a 30-year follow-up, factual data were also more often corroborated with clinic and juvenile court records than were subjective reports.⁵⁷ Of relevance to the present study, retrospective self-reports of low socioeconomic status or unemployment of one's parents were in agreement with records in 69% of cases.⁵⁷ In summary, retrospective self-reports of objective data are generally reliable and unbiased by diagnostic status of the informant.

More research on positive outcomes and protective factors is clearly needed. The protective factors that promote resilience are not well known, especially in families with an alcohol-dependent parent, but other studies have identified two primary ones: secure, stable, affectional relationships, and the experience of success and achievement.⁵⁹ Seifer and Samaroff⁶⁰ analyzed multiple risk factors on the development of children with schizophrenic mothers. Variables such as social status and family stress had joint effects that were complex. Another longitudinal study found that variation in risk for sons of alcoholic fathers depended on such variables as the mother's esteem for the father and her level of control over the son.¹⁵ For the Core City Sample of men in Felsman and Valliant's⁶¹ longitudinal study, the best predictor of adult positive outcome was the early measure of "boyhood competence," which is something like ego strength.

The simple adversity measure used herein (i.e., adversity due to low availability of socioeconomic resources) gives us only a hint of how nonspecific environment might affect the development of alcoholism. This nonspecific environmental factor was an independently important variable that increased risk in general, not just in families with alcoholic members. Thus, this factor is not interpreted as a mediator, but rather as a moderator of family history risk. In this study, the true biological risk status of the participants is unknown. If a good marker of biological/genetic risk existed, demonstrating the effects of childhood adversity would be clearer and more compelling.

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