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Parental Alcohol Dependence and the Transmission of Adolescent Behavioral Disinhibition: A Study of Adoptive and Non-Adoptive Families

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Abstract

Aim—To examine the genetic and environmental influences of parental alcoholism on offspring disinhibited behavior.

Design—We compared the effect of parental alcoholism history on offspring in adoptive and non-adoptive families. In families with a history of parental alcohol dependence, we examined the effect of exposure to parental alcoholism symptoms during the lifetime of the adolescent.

Setting—Assessments occurred at the University of Minnesota from 1998–2004.

Participants—Adolescents adopted in infancy were systematically ascertained from records of three private Minnesota adoption agencies; non-adopted adolescents were ascertained from Minnesota birth records. Adolescents and their rearing parents participated in in-person assessments.

Measurements—For adolescents, measures included self-reports of delinquency, deviant peers, substance use, antisocial attitudes, and personality. For parents, we conducted DSM-IV clinical assessments of alcohol abuse and dependence.

Findings—A history of parental alcohol dependence was associated with higher levels of disinhibition only when adolescents were biologically related to their rearing parents. Within families with a history of parental alcoholism, exposure to parental alcohol misuse during the lifetime of the adolescent was associated with increased odds of using alcohol in adopted adolescents only.

Conclusions—These findings suggest that the association between a history of parental alcohol dependence and adolescent offspring behavioral disinhibition is largely attributable to genetic rather than environmental transmission. We also obtained some evidence for parental alcohol misuse as a shared environmental risk factor in adoptive families.

Keywords

Adoption; Alcohol; Disinhibition; Genetic; Environmental

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Conflicts of Interest: None

Introduction

Family studies have consistently shown that the adolescent and young adult offspring of alcoholics are at elevated risk for alcohol use and misuse (1-5). This behavior, however, seldom occurs in isolation. Rather, these adolescents are also at excess risk for an array of difficulties including the misuse of other substances, conduct problems, and dispositional characteristics associated with impulsivity and behavioral undercontrol (1,4-8). In this respect, the offspring of alcoholics are similar to young people with non-alcoholic parents who experiment with substances(9). The robust associations typically found among these behavioral and dispositional tendencies have led researchers to hypothesize the existence of a general core vulnerability that links these indications etiologically as well as phenotypically (10,11). In this paper, we use the term behavioral disinhibition to refer to this general vulnerability to substance use, delinquency, antisocial attitudes, and impulsivity.

Why does parents' alcoholism predict behavioral disinhibition in their children? Some researchers hypothesize that the rearing environment shaped by an alcoholic parent is responsible for the surfeit of problem behaviors in their children. Research has indeed shown that this environment is likely to be marked by conflict and dysfunctional life experiences (12-14). Further, individuals with substance abusing parents may also grow up in homes where substance use is modeled and parental monitoring of children's behavior is inconsistent(14,15). Recent evidence from twin studies indicates that influences in the environment shared by family members may be salient for substance initiation and use in adolescence (16,17).

Researchers studying the offspring of monozygotic (MZ) twins discordant for alcoholism have attempted to measure the environmental effect of having an alcoholic parent while controlling for shared genetic influences. Children-of-twins studies have, however, produced disparate findings. Jacob et al.(18) reported that the offspring of alcoholics were at greater risk for alcohol disorders than were the offspring of their unaffected MZ co-twins, suggesting that a lower-risk environment can reduce the effect of genetic vulnerability. In contrast, a more recent study found no difference in alcohol dependence symptoms between the children of discordant MZ co-twins (19)

Adoption designs provide another powerful research tool because they directly estimate the environmental effect of rearing a child in the home of an alcoholic adoptive parent. However, existing adoption studies have also produced inconsistent results. A landmark adoption study from Sweden studied individuals adopted by alcohol-abusing adoptive parents. These researchers found no significant difference in the prevalence of alcohol abuse in adoptees with an alcohol-abusing rearing parent as compared to adoptees without an alcohol-abusing rearing parent(20,21). In these studies, however, the prevalence of alcoholism was estimated entirely from archival data. The findings contrast with those from adoption studies conducted in the United States in which adoptive parents and adoptees were directly interviewed. In two adoption studies, Cadoret and colleagues(22,23) reported that a history of alcohol problems in the rearing family (parents or siblings) increased the risk for alcohol disorders in adoptees. A third US adoption study found an effect of adoptive sibling, but not parent, drinking on drinking behavior in adolescent adopted offspring although this study did not focus directly on alcohol misuse in the adoptive parents(24).

In contrast, genetic contributions to parent-child similarity for alcohol use and misuse have been evidenced by both twin and adoption studies. Adoption studies have demonstrated that a history of alcoholism in the biological family heightens the risk for alcoholism in adopted-away sons and, less consistently, daughters (20-23,25-27). Twin studies have found substantial genetic influences on alcoholism in adult males with weaker evidence in female

samples(28). Studies of younger twins, however, suggest that genetic influences may be somewhat less for substance initiation and use during adolescence (29-31). Finally, recent research indicates that shared genetic influences are largely responsible for transmission of a general vulnerability to all the externalizing disorders from parent to child(32). In this view, the constellation of behaviors observed in the children of alcoholics may stem from a general inherited vulnerability for which alcohol misuse is but one manifestation.

In the present study, we contrast adopted and non-adopted adolescents reared in families where at least one parent received a lifetime diagnosis of alcohol dependence to those reared in families where neither parent received a lifetime diagnosis of either alcohol abuse or dependence. A significant strength of the present study is that parental diagnoses were obtained directly from in-person clinical interviews. In addition, adolescents were measured on a variety of outcomes indicative of behavioral disinhibition including, but not limited to, alcohol use. To the extent that adopted and non-adopted offspring of alcoholic rearing parents are similar on substance use and other behavioral outcomes, shared environmental influences will be implicated in the etiology of behavioral disinhibition. Genetic influences will be implicated to the extent heightened risk is found in the biologically related offspring of alcoholic parents when compared to adoptees reared by parents with a history of alcoholism. We will also be able to determine whether the genetic and environmental factors contribute differentially to the various indices of disinhibition.

Of course, parents with a history of alcohol dependence may not have exhibited symptomatic behavior within the lifetime of their children. Thus, as a more stringent test of the environmental impact of parental alcoholism, within families where at least one parent has been diagnosed with alcohol dependence, we will compare adolescents exposed to parental alcohol abuse or dependence *within the lifetime of the adolescent* with adolescents who have not been directly exposed to parental alcohol misuse.

We hypothesize that a history of alcoholism in non-adoptive parents represents a non-specific genetic risk for disinhibited behavior in their adolescent children including, but not limited to, alcohol use. Further, we hypothesize that parental alcohol misuse represents a specific environmental risk for adolescent alcohol use only. Thus we expect to observe increased alcohol use among both the non-adoptive and adoptive offspring of alcoholic parents, but increased rates of other disinhibited behavior in the non-adoptive offspring only. Even more specifically, we hypothesize that exposure to parental alcohol misuse will be associated with increased alcohol use in adolescent offspring, but will have no association with other indices of disinhibited behavior.

Method

Participants

The Sibling Interaction and Behavior Study (SIBS) is a population-based family study under the auspices by the Minnesota Center for Twin and Family Research (MCTFR). SIBS participants were 409 adoptive and 208 non-adoptive families, each with an adolescent sibling pair between the ages of 11 and 21 years (mean=14.92; SD=1.9) and no more than five years apart in age. In the adoptive families, the siblings were biologically unrelated, although one sibling could be the biological offspring of the adoptive parents. All adoptees were placed permanently in their adoptive home prior to age 2 years (average age of placement = 4.7 months; SD=3.4); no information is available on their birth parents. In the non-adoptive families, the siblings were full biological siblings, selected to have a distribution of sex and age comparable to those in adoptive families. Both types of families were required to live within driving distance of the University of Minnesota, and neither adolescent could have any physical or mental disability that might preclude their completing

the in-person assessment. Subsequent to their participation, two adolescents were judged ineligible. The final SIBS sample contained 1232 adolescents and one or both of their rearing parents. For a complete description of the SIBS sample, see McGue et al.(33).

Adolescents were included in the analyses reported here if at least one parent had a lifetime diagnosis of alcohol dependence or if neither parent received a lifetime diagnosis of alcohol dependence *or* abuse. Thus, 193 adolescents, with data available on both parents, were excluded because neither parent was diagnosed with dependence but at least one was diagnosed with abuse. For an additional 140 adolescents, data for one parent was missing, and the other parent did not meet the inclusion criteria. The final sample included 899 adolescents, from 453 families. Fifty-eight percent of the sample was adopted with 68% of these adoptions originating from South Korea; 96% of the non-adopted adolescents were Caucasian. A total of 132 adopted and 113 non-adopted adolescents had at least one parent with a lifetime diagnosis of alcohol dependence. Within these families, data on exposure to parental symptoms were available for 241 adolescents. Of these, 71 adopted and 78 non-adopted adolescents were exposed to a parent who had at least one symptom of alcohol abuse or dependence within the adolescents' lifetimes.

Parent Diagnostic Measures

Alcohol Dependence—Parental alcohol dependence diagnoses were obtained using a modified version of the expanded Substance Abuse Module of the Composite International Diagnostic Interview(34). Parents were independently interviewed in private rooms by extensively trained interviewers with a B.A. or M.A. degree in psychology. For each symptom, ages of onset and recency were obtained. Symptoms were reviewed in case conferences where individuals with advanced clinical training reached a consensus as to whether a symptom was present. Diagnoses were assigned via computer using DSM-IV algorithms. A lifetime diagnosis of alcohol abuse or dependence was made if the respondent met DSM-IV criteria at either a definite (all diagnostic criteria satisfied) or probable (all but one diagnostic criteria satisfied) level of certainty. The use of both definite and probable cases is supported by recent work from the National Comorbidity Study, which demonstrated that a difference in risk for negative outcomes at follow-up was greater between mild cases and non-cases than between cases that were mild and moderate in severity(35). Inter-rater reliability estimates of substance use diagnoses were high (kappas > .90).

Adolescent Measures of Behavioral Disinhibition and Substance Use

Multiple attitudinal, personality, and behavioral indices of disinhibition were selected. The broad age range of the sample constrained our choice to measures of disinhibition that were similarly defined for 11- to 21-year-olds. Thus, we focus on developmentally-relevant, quantitative indicators of behavioral disinhibition and measures of substance use, including lifetime use of tobacco, alcohol, and marijuana.

Delinquent Behavior Inventory—The 36-item Delinquent Behavior Inventory (DBI) (36) asked whether the adolescent has ever engaged in delinquent behaviors spanning a range of severity from skipping school to using a weapon in a fight. Items were summed to compute a total score ($\alpha = .89$).

Peer Deviance—This inventory was designed by the MCTFR to tap behaviors and attitudes endorsed by the adolescent's peer group. Four items addressed peer substance use (e.g., "My friends drink alcohol or beer"), and five covered peer delinquency (e.g., "My friends get into fights with other kids"). Items were summed to produce a general measure of peer deviance ($\alpha=.89$).

Antisocial Attitudes—The eight-item Antisocial Attitudes scale, also developed by the MCTFR, assessed rule-breaking attitudes rather than actual rule-breaking behavior (e.g., “If I knew I could get away with it, I might take something from a store without paying”). Items were summed to compute a total score ($\alpha = .87$).

Personality Traits of Impulsivity and Aggression—Adolescents completed the Multidimensional Personality Questionnaire (MPQ)(37), a widely used personality questionnaire. In the current investigation, we have included the Control and Aggression scales from the MPQ. The Control scale measures the degree to which an individual exerts restraint. This scale was reflected so that higher levels indicate less control; the reflected scale is labeled Impulsivity ($\alpha = .83$). The Aggression scale measures the tendency toward hurting others or being vindictive; higher levels indicated more aggressive personality characteristics ($\alpha = .84$).

Adolescent Substance Use—Adolescent lifetime use (yes/no) of 11 substances, including alcohol without parental permission, tobacco, marijuana, amphetamines, tranquilizers, barbiturates, inhalants, cocaine, psychedelics, opiates, and steroids was assessed via a private, computer-administered questionnaire. We examined use of tobacco, alcohol, and marijuana separately as well as a count of the total number of substances ever used.

Disinhibition Factor—We constructed a disinhibition factor composed of the six continuous measures described above. All six measures loaded on one large principal component accounting for 58.1% of the variance; factor loadings ranged from .56 to .87. A factor score was estimated for each adolescent using least squares regression.

Statistical Analyses

Descriptive statistics were calculated, by adoption status and history of parental alcohol dependence, for sex, ethnicity, age at assessment, and all outcome measures. To determine the effects of parental alcohol dependence and whether these effects were moderated by biological relatedness, we conducted two sets of analyses. Quantitative outcome variables were examined using multilevel ANCOVAs with adolescent's age and gender as covariates and two between-subjects factors: history of parental alcohol dependence (yes/no) and adoption status (adopted/non-adopted). These analyses were conducted in version 9.1 of SAS/STAT software for Windows, using PROC MIXED. Random intercepts were included in the models to account for similarity within sibling pairs on the dependent variables. Significant interactions between parental alcohol dependence and adoption status were followed with tests of simple main effects to determine the nature of the parental alcoholism effect on adopted and non-adopted adolescents separately. Effect sizes (ESs) were estimated by dividing the difference in the covariate-adjusted means by the square root of the residual variance from the multilevel ANOVA. They represent the difference in means as a function of the level-1 variance, or variance not due to sibling resemblance. Markedly skewed outcome measures, specifically delinquent behavior, peer deviance, and the substance use tally, were log-transformed prior to analyses.

Lifetime prevalences of alcohol, tobacco, and marijuana were analyzed using logistic regression, with history of parental alcohol dependence and adoption status as dichotomous predictors and the adolescent's age and gender as covariates. These were conducted using Generalized Estimating Equations (GEE) (38,39) in PROC GENMOD to control for the correlated nature of within-family observations. Covariate-adjusted odds ratios (ORs) and confidence intervals were estimated as the measure of effect.

Because failure to find effects of parental alcohol dependence history may simply reflect lack of exposure to parental alcohol symptoms, we conducted a second set of analyses to determine whether exposure to parental alcohol problems during the adolescent's lifetime was related to these same outcomes, and whether this exposure effect differed between adopted and non-adopted offspring. We considered exposure to have occurred if any parental symptoms of alcohol abuse or dependence occurred during the child's lifetime. The exposure effect was examined in the subsample of adolescents in families where at least one parent ever had an alcohol dependence diagnosis. Analyses were identical in form to our primary analyses, with exposure to parental alcohol problems substituting for a history of parental alcohol dependence.

Results

Focusing on the descriptive statistics provided in Table 1, adopted and non-adopted adolescents were not significantly different in age or gender composition. Although they did differ significantly in ethnic composition ($<.001$), ethnicity was unrelated to any of the outcomes included in the analyses reported here, and thus was not considered further. In addition, non-adopted adolescents were more likely to have at least one parent with a history of alcoholism; however, this difference between groups was not significant.

Effects of parental alcohol dependence

The results of the analyses are presented in Table 2 which gives test statistics and effect sizes for the effects of adolescent adoption status, parental alcoholism history, and their interaction, statistically adjusting for age and gender. There were no main effects of adoption status. There were two significant main effects for lifetime parental alcohol dependence. Adolescents with at least one rearing parent with a history of alcoholism scored significantly higher on delinquent behavior and aggressive personality measures than adolescents whose rearing parents had no lifetime alcohol diagnoses. The interaction between adoption status and parental alcohol dependence was significant for all outcome measures except peer deviance and any marijuana use, indicating that many of the effects of parental alcohol dependence were conditioned on adoption status. Tests of simple main effects in mixed-model ANCOVAs and the appropriate contrasts in GEE models confirmed that parental alcohol dependence was significantly associated, in non-adopted adolescents only, with increased risk for delinquent behavior, antisocial attitudes, aggressive personality, impulsive personality, tobacco use, alcohol use, total number of substances used, and the summary disinhibition factor (ESs for significant effects range from .37-.62). For adoptees, only the simple main effect for impulsive personality was significant (ES=-.29). In this case, however, parental alcohol dependence was associated with *decreased* levels of impulsivity. The interaction of parental alcohol dependence history and adoption status is illustrated in Figure 1 for the summary disinhibition factor.

Effects of exposure to parental alcohol problems

These analyses were limited to those families with a history of parental alcohol dependence. They were identical in form to the main analyses, with the presence of at least one symptom of parental alcohol abuse or dependence during the lifetime of the adolescent as an independent variable. Table 3 provides the descriptive statistics for the exposure analysis. Adopted and non-adopted adolescents in this subsample were not significantly different in age or gender composition. Although non-adopted adolescents were more likely to have been exposed to symptoms of parental alcoholism, the difference between groups was not significant.

Table 4 gives test statistics for exposure to parental alcohol problems, adoption status, and their interaction. There was a main effect of adoption status for six of the nine outcome variables and for the summary disinhibition factor, indicating that levels of disinhibitory behavior in adolescents with at least one alcohol dependent parent were consistently higher in non-adopted than adopted adolescents. Significant effects of exposure to parental alcohol problems were limited to alcohol use. Although the interaction between exposure and adoptive status was not statistically significant ($p=.08$), inspection of the individual odds ratios demonstrates that the odds of having experimented with alcohol were more than four times greater for exposed adolescents, but only if they were adopted. For non-adopted adolescents, there was no significant difference in alcohol use between those exposed to parental alcohol problems (43% used) and those not exposed (42% used).

Discussion

The current study compared adopted and non-adopted adolescents reared in families where at least one parent had a history of alcohol dependence to those reared in families with no history of alcoholism. This investigation extends previous adoption research on the effects of parental alcohol dependence in three important respects: studying a large and representative sample of US adolescents; including, in addition to alcohol use, a variety of outcomes indicative of behavioral disinhibition; and examining the effect of exposure to parental abuse and dependence symptoms in the lifetime of the adolescent.

Our results support several conclusions. Effects of parental alcohol dependence history were conditioned on adoption status, such that a history of parental alcohol dependence was associated with higher levels of disinhibition only when adolescents were biologically related to their rearing parents. This finding suggests that the association between a history of parental alcohol dependence and offspring behavioral disinhibition is largely attributable to genetic rather than environmental transmission. Moreover, we found that parental alcohol dependence was associated with elevated levels in biological offspring on a variety of indicators of behavioral disinhibition, and not just on alcohol or other substance use. These results imply that what is transmitted genetically is a general tendency toward behavioral disinhibition, one that extends across substance use, personality traits, attitudes, and behaviors. These results buttress previous research, which established a highly heritable, externalizing factor linking mental disorders that share behaviorally disinhibited traits in common(10). They also support the contention that disinhibited, antisocial behavior and substance use are variable expressions of a common, general vulnerability (11,32,40).

In addition, we obtained some evidence for parental alcohol misuse as a shared environmental risk factor. A novel contribution of the present investigation was our ability to parse family environmental influences from genetic effects. Our analyses of exposure to parental alcohol misuse among adoptees provided a direct test of the environmental effect of exposing a child to a rearing parent with alcohol problems. We show that exposure to parental alcohol misuse was associated with a substantially increased likelihood of having used alcohol in adopted adolescents. The odds of having ever used alcohol were approximately four times greater among adoptees exposed to parental alcohol misuse relative to adoptees that were not similarly exposed.

Exposure to parental alcohol symptoms did not, however, increase the odds of alcohol use in non-adopted adolescents. This suggests that the environmental effect of exposure to parental alcohol problems does not significantly increment the risk associated with shared genetic endowment. These findings are not without precedent. DeLucia, Belz, and Chassin(41) previously demonstrated that the offspring of recovered alcoholic fathers drank more heavily than the offspring of control fathers. Further, Biederman and colleagues(42) showed that

adolescent offspring with alcohol use disorders spent a greater proportion of their lives exposed to their parents' alcohol dependence when compared with offspring who did not have an alcohol use disorder. This difference, however, was not significant when controlling for substance disorder history in the parent.

Our findings are limited by the nature of the sample. For example, all non-adopted adolescents were ascertained through Minnesota birth records and thus do not reflect the ethnic diversity of the larger U.S. population. Similarly, reflective of Minnesota adoption practices in the years relevant to this investigation, a preponderance of the adopted adolescents were of Asian descent. It will be important to replicate these findings in more ethnically diverse adoptee and non-adoptee samples. Finally, because our sample was relatively young, it will also be important to follow these participants into early adulthood to determine whether the effects we observe are lasting.

In summary, exposure to parental alcohol misuse during one's lifetime represented an environmental risk factor for alcohol use in adopted adolescents. It was a specific risk factor, however; none of the other effects of parental exposure on offspring disinhibition was significant. This stands in contrast to a history of alcohol dependence in a biologically related parent, which conferred a generalized, largely genetic vulnerability to disinhibited behavior in adolescent offspring that was independent of direct exposure to parental problems with alcohol.

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Figure 1.
Effect of Parental Alcohol Dependence History on Standardized Disinhibition Factor Scores
in Adopted and Non-adopted Adolescent Offspring

Table 1

Descriptive Statistics on Demographic and Outcome Measures as a Function of Adoption Status and Parental Alcohol Dependence History

	Adopted Adolescents (N=525)		Non-Adopted Adolescents (N=374)	
	Parent Dependent (N=132)	Parent not Dependent (N=393)	Parent Dependent (N=113)	Parent not Dependent (N=261)
	Quantitative Variables — Mean (SD)			
Age	14.92 (1.9)	14.92 (2.0)	15.09 (2.0)	14.78 (1.9)
Delinquent Behavior	4.82 (5.4)	5.79 (6.9)	8.22 (10.3)	4.41 (6.5)
Deviant Peers	12.98 (3.4)	13.27(4.0)	14.08 (4.9)	12.59 (3.6)
Number of Drugs Used	0.83 (1.5)	1.06 (1.6)	1.37 (1.9)	0.80 (1.4)
Antisocial Attitudes	13.05 (4.5)	14.01 (5.4)	15.19 (5.8)	13.06 (4.9)
Aggressive Personality	39.43 (9.8)	38.95 (10.4)	40.87 (12.2)	37.79 (9.6)
Impulsive Personality	44.88 (8.1)	47.00 (9.1)	48.32 (9.6)	45.28 (7.5)
Disinhibition Factor Score	-0.10 (0.9)	0.03 (1.0)	0.29 (1.1)	-0.21 (0.9)
	Categorical Variables — Percent			
% Male	51.5	42.7	45.1	48.7
% Caucasian	18.2	19.3	98.2	94.6
% Ever Use Tobacco	19.8	23.8	33.9	20.8
% Ever Use Alcohol	21.4	27.9	42.0	21.5
% Ever Use Marijuana	13.7	14.4	17.9	12.4

Table 2

Effect of Adoption Status and Parental Alcohol Dependence History on Measures of Disinhibition and Substance Use

Quantitative Variables	Adoption Status		Parental Alcohol Dependence		Interaction Effect		Adopted		Non-Adopted	
	F	p	F	p	F	p	ES	ES	ES	ES
Delinquent Behavior	.039	.53	7.00	<.01	15.17	<.001	-.103		.621	***
Deviant Peers	0.17	.68	1.60	.21	2.64	.10	-.027		.262	
Number of Drugs Used	1.77	.18	0.89	.35	10.83	<.01	-.203		.377	**
Antisocial Attitudes	2.00	.16	1.05	.31	13.38	<.001	-.239		.440	**
Aggressive Personality	0.01	.91	3.93	<.05	5.02	<.05	-.017		.373	**
Impulsive Personality	1.55	.21	0.32	.57	18.15	<.001	-.292**		.381	**
Disinhibition Factor Score	0.45	.50	3.20	.07	16.43	<.001	-.209		.586	***
Categorical Variables	χ^2	p	χ^2	p	χ^2	p	OR	OR	OR	OR
% Ever Use Tobacco	2.80	.09	.090	.34	5.50	<.05	(0.4-1.3) 0.69	(1.1-3.9) 2.91***		
% Ever Use Alcohol	3.60	.06	2.49	.11	11.79	<.001	(0.4-1.2)	(1.6-5.4)		
% Ever Use Marijuana	0.05	.82	0.48	.49	0.39	.53	1.02	(0.6-1.9)	1.38	(0.6-2.8)

Test statistic is an F ratio for continuous measures and χ^2 (with 1 df) for the measures of lifetime substance use. Effect sizes (ES) for quantitative variables are calculated as the difference in means between adolescents with an alcoholic parent and those without an alcoholic parent divided by the square root of the residual variance from the multilevel ANOVA. They represent the difference in means as a function of the level-1 variance, or variance not due to sibling resemblance. Odds ratios (with 95% confidence intervals) are given as estimates of effect size for the categorical variables. Covariates included age at assessment and gender. Significant effects are bolded (

* p < .05

** p < .01

*** p < .001).

Table 3

Descriptive Statistics on Demographic and Outcome Measures as a Function of Adoption Status and Exposure to Parent Problems with Alcohol

	Adopted Adolescents (N=132)		Non-Adopted Adolescents (N=109)	
	Exposed (N=71)	Not Exposed (N=61)	Exposed (N=78)	Not Exposed (N=31)
	Quantitative Variables — Mean (SD)			
Age	15.26 (1.7)	14.53 (1.9)	15.07 (1.9)	15.18 (2.2)
Delinquent Behavior	5.26 (5.4)	4.32 (5.5)	8.56 (11.5)	7.45 (7.5)
Deviant Peers	13.28 (3.5)	12.63 (3.4)	14.35 (5.2)	13.39 (4.3)
Number of Drugs Used	1.03 (1.6)	0.60 (1.3)	1.35 (1.9)	1.45 (2.0)
Antisocial Attitudes	13.83 (4.8)	12.15 (4.0)	14.96 (6.0)	15.74 (5.4)
Aggressive Personality	39.04 (9.7)	39.88 (10.0)	40.71 (12.4)	41.00 (12.3)
Impulsive Personality	44.58 (7.5)	45.23 (8.8)	48.38 (9.5)	48.37 (10.3)
Disinhibition Factor Score	-0.01 (0.9)	-0.22 (0.9)	0.28 (1.1)	.32 (1.1)
	Categorical Variables — Percent			
% Male	52.1	50.8	43.6	48.4
% Caucasian	16.9	19.7	97.4	100.0
% Ever Use Tobacco	23.9	15.0	32.5	35.5
% Ever Use Alcohol	32.4	8.3	42.9	41.9
% Ever Use Marijuana	18.3	8.3	16.9	19.4

Table 4

Effect of Adoption Status and Exposure to Parental Problems with Alcohol on Measures of Disinhibition and Substance Use

Quantitative Variables	Adoption Status		Parental Alcohol Exposure		Interaction Effect		Adopted		Non-Adopted	
	F	p	F	p	F	p	F	ES	F	ES
Delinquent Behavior	5.49	<.05	0.57	.45	0.07	.79	.181			.090
Deviant Peers	0.07	.79	0.70	.40	0.45	.50	.032			.254
Number of Drugs Used	5.67	<.05	.034	.56	0.27	.60	.178			.013
Antisocial Attitudes	8.99	<.01	0.04	.84	1.12	.29	.216			-.144
Aggressive Personality	1.31	.25	0.00	.99	0.03	.87	-.030			.026
Impulsive Personality	9.51	<.01	0.01	.94	0.02	.88	-.033			.010
Disinhibition Factor Score	6.65	<.05	0.22	.64	0.07	.79	.132			.039
Categorical Variables	χ^2	p	χ^2	p	χ^2	p	OR	OR	OR	OR
% Ever Use Tobacco	5.10	<.05	0.11	.74	0.28	.60	(0.5-3.3)	(0.4-2.5)		1.16
% Ever Use Alcohol	9.02	<.05	4.50	<.05	3.16	.08	(1.6-11.5)	(0.4-3.3)		
% Ever Use Marijuana	0.42	.52	0.51	.48	0.55	.46	(0.6-5.3)	(0.3-3.4)		

Test statistic is an F ratio for continuous measures and χ^2 (with 1 df) for the measures of lifetime substance use. Effect sizes (ES) for quantitative variables are calculated as the difference in means between adolescents with an alcoholic parent and those without an alcoholic parent divided by the square root of the residual variance from the multilevel ANOVA. They represent the difference in means as a function of the level-1 variance, or variance not due to sibling resemblance. Odds ratios (with 95% confidence intervals) are given as estimates of effect size for the categorical variables. Covariates included age at assessment and gender. Significant effects are bolded (

p < .01).