Journal of Studies on Alcohol, 66(1), 14-22

Parental alcohol use disorders and child delinquency: the mediating effects of executive functioning and chronic family stress

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Abstract

Objective: This study examines the relationship between parental alcohol use disorders (AUDs) and child violent and nonviolent delinquency. It also explores the mediating effects of executive functioning and chronic family stress on the parental AUD/child delinquency relationship, Method: Participants were 816 families with children (414 boys and 402 girls) born between 1981 and 1984 at Mater Misericordiae Mother's Hospital in Brisbane, Australia. Parents and children completed semistructured interviews, questionnaires and neuropsychological tests that assessed parental alcohol use, family psychiatric history, chronic family stress, child delinquency and child executive functioning. Re suits: Paternal (but not maternal) AUDs predicted child violent and nonviolent delinquency. Executive functioning mediated the relationship between paternal AUDs and violent delinquency, whereas family stress mediated the relationship between paternal AUDs and both violent and nonviolent delinquency. Conclusions: Results support a biosocial conceptualization of the paternal AUD/delinquency relationship. They suggest that paternal AUDs may be associated with child executive functioning and Family stress, which may in turn lead to child delinquency.

ALCOHOL USE DISORDERS (AUDs) are a widespread problem in the United States. Approximately 5-10% of the country's population suffers from DSM-IV alcohol abuse, and this figure appears to be growing (Anthony and Echeagaray-Wagner, 2000). Alcohol use problems affect spouses and children, unfortunately, in addition to the heavy substance users themselves. A recent study estimated that one in four American children have a parent who meets criteria for DSM-IV alcohol abuse (Grant, 2000).

Children of alcoholics (COAs) are at risk for a number of adverse behavioral outcomes. COA status has been associated with such delinquency-related behaviors as aggression, conduct disorder, oppositional defiant disorder and criminality (Barnow et al., 2002; Carbonneau et al., 1998; Puttler et al., 1998; Reich et al., 1993; Sher et al., 1991; Windle, 1997). COAs are also more likely than their peers to misuse alcohol and drugs (Sher et al., 1991).

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[*] This study was supported by funds from the National Health and Medical Research Council in Australia and by National Institutes of Health grant R01 MH52239.

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Although the COA/delinquency relationship appears robust, it should be noted that several studies have yielded negative results (Alterman et al., 1989; Berkowitz and Perkins, 1988; Fulton and Yates, 1990; Schuckit et al., 2000). COA studies also have been criticized on methodological grounds (Hatter, 2000; Sher, 1991). Many COA studies use small sample sizes and may be limited by inadequate power (Harter, 2000). Other COA studies utilize potentially nonrepresentative samples (e.g., college students, children of hospitalized alcoholics or children of alcoholics referred to correctional agencies; Heller et al., 1982; Sher, 1991). These samples are likely to over- or underestimate the degree of psychopathology among the general COA population. In addition, many COA studies fail to control for potential confounding variables (e.g., parental psychiatric history, parental education and socioeconomic status; Barnow et al., 2002; Sher, 1991; West and Prinz, 1987).

It is also unclear which factors mediate the relationship between COA status and delinquency. COA status is associated with many adverse environmental and biological conditions. These conditions may explain the process by which COA status affects delinquent outcomes. The current study will examine two potential mediators of the COA/delinquency relationship: chronic family stress and executive functioning deficits.

Family stress

Studies with a variety of methodologies have found a significant relationship between adverse family functioning and parental alcohol misuse. Barry and Fleming (1990) administered a family environment scale and the alcohol use scale of the Diagnostic Interview Schedule to alcoholics and nonalcoholics with and without a family history of alcoholism. Results showed that alcoholics with a family history of alcoholism tended to have conflictual noncohesive familial relationships. Shuntich et al. (1998) administered family environment and alcohol use questionnaires to a large sample of college students. Results showed that ratings of parental alcohol misuse were positively correlated with measures of parental aggression and negatively correlated with measures of parental affection. In an earlier study, Moos and Moos (1984) administered the Family Environment Scale to families of recovered and relapsed alcoholics as well as to community controls. Results indicated that families of relapsed alcoholics had lower cohesion, expressiveness and agreement about their family environment than families of recovered alcoholics or controls.

A large body of literature also suggests a relationship between family stress and child externalizing behavior (Shaw et al., 1998, 2001; Stormshak et al., 1997). Thus, it is possible that chronic family stress mediates the relationship between parental alcohol misuse and aggressive outcomes.

Executive functioning

A substantial body of literature suggests a relationship between executive functioning and COA status. COAs have been shown to perform more poorly on tests of planning, abstract conceptualization, conceptual shifting and psychomotor functioning than their peers (Drejer et al., 1985; Peterson et al., 1992). COAs have also been found to perform more poorly in school and on academic achievement tests than non-COAs (Hegedus et al., 1984; Knop et al., 1985).

To date, the causes of the executive functioning/COA relationship are unknown. Some research suggests that executive functioning deficits among COAs are heritable. Some recent studies have found more severe cognitive deficits among individuals with multigenerational, as opposed to unigenerational, family histories of alcoholism (Corral et al., 1999). Other studies, however, do not find an effect of genetic loading and instead implicate environmental causes (e.g., perinatal complications or parental antisocial personality disorder [ASPD]; Alterman et al., 1987; Reed et al., 1987).

It should also be noted that several studies have tailed to find relationships between familial alcoholism and executive functioning (Alterman et al., 1987; Reed et al., 1987; Schuckit et al., 1987). There are several possible explanations for these null results. Some executive functioning/ familial alcoholism studies, for example, have utilized high functioning samples (e.g., college students), in which cognitive deficits tend to be rare. In addition, some studies suggest that executive functioning deficits are more common among individuals with a family history of both alcoholism and ASPD (Cloninger, 1987; Poon et al., 2000). Studies yielding null results thus may focus on subgroups of COAs that are at low risk for cognitive impairment (for examples, see Alterman and Hall, 1989; Alterman et al., 1989: Schuckit et al., 1987).

Executive functioning has also been linked to externalizing and aggressive behaviors. Yeudall et al. (1982) found that violent juvenile delinquents performed more poorly than nondelinquents on an assessment of frontal lobe functioning, and Gorenstein (1982) found that criminal psychopaths performed more poorly than controls on a battery of frontal lobe tests. Other studies have found impaired executive functioning among domestic abusers (Westby and Ferraro, 1999), individuals with temper control problems (Krakowski et al., 1997) and violent psychiatric patients (Krakowski and Czobor, 1997).

Evidence for the executive functioning/aggression relationship also comes from direct measures of brain functioning. Recent functional brain imaging studies have detected reduced glucose metabolism and blood flow in the prefrontal cortexes of murderers (Raine et al., 1994), sex offeders (Garnett et al., 1988) and other violent criminals (Volkow and Tancredi, 1987). Event-related potential (ERP) studies suggest that attenuated amplitudes of the P300 waveform are related to both aggressive behavior (Barratt et al., 1997) and poor performance on tests of executive functioning (Hesselbrock et al., 1993). It is interesting to note that P300 amplitudes have not been related to tests of nonexecutive functioning; this suggests that they are markers of localized frontal lobe problems, rather than diffuse brain dysfunction (Giancola, 2000).

COA status, executive functioning and family stress: An integrative model

Moffitt (1997) has described a model in which neurologically compromised children become trapped in a cycle of negative social interactions that ultimately leads to behavior problems. She states that children with neurological problems tend to be hyperactive, irritable, inattentive and emotionally reactive. Offspring with these characteristics are often difficult to care for and may elicit negative affectivity and poor caretaking behavior. Child temperament also tends to be correlated with parent temperament (Plomin et al., 1990); impulsive, irritable and inattentive children often have impulsive, irritable and inattentive parents. These negative parent/child

interactions may increase stress and ultimately lead to conflictual family environments that are conducive to the development of offspring behavior problems. Because COAs are at heightened risk for neurological problems, they may be especially vulnerable to the effects of Moffitt's biosocial cycle of interactions. Familial stress also is likely to be exacerbated in families coping with alcohol use problems, even when the heavy drinking parent does not live in the home (e.g., due to lack of income, feelings of parental rejection, lack of parental role models, etc.). Thus, it is possible that parental alcohol misuse leads to family conflict and offspring neuropsychological problems that, in turn, increase the risk of deviant behavior among COAs.

Current study

One aim of the current study is to test the relationship between parental AUDs and child violent and nonviolent delinquency in a community sample of 816 Australian families. We also explore the degree to which executive functioning and chronic family stress mediate the parental AUD/child delinquency relationship. This study will add to the literature by examining the COA/delinquency relationship in a large community sample and will be the first to test the mediating roles of both family stress and neuropsychological functioning in the COA/delinquency relationship. It is hypothesized that (1) a parental history of AUDs will be related to higher levels of child self-reported violent and nonviolent delinquency, (2) a parental history of AUDs will be associated with child neuropsychological functioning and chronic family stress and (3) child neuropsychological functioning and family stress will mediate the relationship between parental AUDs and child delinquent outcomes.

Method

Participants

Participants in this sample were 816 families with children born between 1981 and 1984 at Mater Misericordiae Mother's Hospital in Brisbane, Australia. The sample for the present study was drawn from a larger birth cohort (N = 7,223) established in the context of the Mater University of Queensland Study of Pregnancy (MUSP: Keeping et al., 1989). The purpose of MUSP was to examine children's physical, cognitive and psychological health as a function of pregnancy and obstetric conditions and psychosocial history. The MUSP cohort was predominantly white (92%) and of lower-middle and working socioeconomic status.

Sample selection

In the context of MUSP, the mothers in the cohort completed self-report depression questionnaires during their pregnancy, at birth and when the child was 6 months and 5 years of age. Data from these questionnaires were used to select a sample that included a large number of women (about 70% of the selected sample) with a history of depressive symptoms (varying in chronicity and severity), along with a sample of comparison women who had no or few depressive symptoms. Further details concerning the selection of this high-risk sample are provided in Hammen and Brennan (2001).

High-risk and comparison families were contacted and asked to participate in a follow-up when their children were 15 years of age. They were informed that the purpose of the study was an examination of the relationship between maternal psychological and emotional functioning and youth behavioral and mental health outcomes. Families were included in the study if the mother and the child agreed to the interview; Fathers and stepfathers were included if available, including a small number of cases with divorced parents in which the father had substantial contact with the youth (parental divorce status was unrelated to youth diagnostic stares). All biological fathers who currently lived in the area were invited to participate in the interview.

From the sample still available for follow-up (N = 5,277; 73% of the original MUSP sample), 991 families were targeted for inclusion in the present study. Of the 991 families, 816 (82%) consented and were included; 68 families could not be located, 103 declined to participate in this phase, 3 included a child with a hearing or visual impairment that precluded participation, and 1 child had died. Children in the high-risk subsample were not significantly different from the original birth cohort in terms of gender ([chi square] 0.56, 1 df, p = .46), income (t = 0.10, 1/6,747 df, p = .92) or mother's education (t = 1.33, 1/7,164 df, p = .18). The subsample, however, had fewer ethnic minority members (8.5% vs 11.0%; [chi square] = 4.75, 1 df, p < .05) and older mothers (t = 2.03, 1/7,221 df, p < .05) compared with the unselected birth cohort from which it was drawn.

Sample characteristics

The mean (SD) age of the sample (N- 816, 414 boys) was 15 years, 2 months (3.48 months). The majority (92%) were white, and, at the time of the youth 15-year follow-up, median family income was AU$35,000-45,000. Median level of mother's education was Grade 10 (approximately equivalent to U.S. high school graduate), and the mothers' mean age was 41 years. Regarding mothers' marital status, 76.8% were currently married or cohabiting; 64.8% were currently married to the biological father of the youth.

Procedure

Interviews were conducted in the homes of the families. Interviewers were blind to the mother's depression status or history, and a team of two interviewers conducted the parent and child interviews separately and privately. Between interviews, the participants also completed a battery of questionnaires. The mother, father and child gave written informed consent/assent and were paid for their participation, which lasted approximately 3.5 hours.

Parental AUDs

Interviewed biological parents were classified as having an AUD if they qualified for past or present alcohol abuse or alcohol dependence on the Structured Clinical Interview for the DSM-IV (SCID; First et al., 1995). Of the 816 biological mothers interviewed, 26 (3%) met these criteria; 73 (16%) of the 454 biological fathers interviewed did. Reliability on diagnoses based on SCID interviews was computed separately for past and current disorders in four categories: anxiety, depressive,

substance use and "other" disorders (e.g., ASPD). Kappas ranged from 0.72 for past anxiety disorders to 0.99 for current "other," with overall mean kappa = 0.85 for current and 0.88 for past disorders. When the biological father of the child was not directly available for interview, mothers' reports of biological father alcohol disorders were obtained using the Family History Research Diagnostic Criteria (FHRDC; Andreasen et al., 1977). In 5 of these 362 cases, the mother reported that she did not have knowledge of the fathers' psychological status. In the remaining 357 cases, 79 biological fathers (22%) were reported to have a history of alcoholism. FHRDC-based diagnostic reliabilities were obtained from 55 randomly selected informants, yielding K = 1.0 for the presence or absence of lifetime disorders.

Youth self reported delinquency

Youth self-reports of both nonviolent and violent delinquent acts were obtained at the age 15 follow-up. These sell-report delinquency items were derived from the National Youth Survey and have established reliability and validity (Elliott et al., 1986). Acts of nonviolent delinquency on the survey included such behaviors as running away from home, skipping school and vandalizing property. Examples of acts of violent delinquency on the survey were such behaviors as attacking someone with a weapon and armed robbery. In this sample, the mean (SD) number of self-reported violent acts (ever committed) was 0.47 (0.86) and of self-reported nonviolent delinquent acts (ever committed) was 4.00 (4.03). Coefficient alphas for the violent and nonviolent delinquency scales within the sample were 0.56 and 0.86, respectively.

Youth neuropsychological functioning

Two separate neuropsychological measures of executive functioning were used in this study. Both of the tests that provided these measures were administered to the youths at the age 15 follow-up. The first measure was the difference score from the Dodrill format of the Stroop Color Word Test (Dodrill, 1978). In this task, youths were handed a sheet with a list of 176 color words printed in incongruous ink colors (e.g., the word "red" printed in green ink). They were asked to complete two separate trials with this word list, as quickly as possible. In the first trial, they were asked to simply read off each of the words on the sheet. In the second trial, they were asked to name the printed colors of each of the words on the sheet. The difference score was calculated as time in seconds to complete Trial 2 minus time in seconds to complete Trial 1. This difference score is the most widely used Stroop measure of selective attention (MacLeod, 1991) and is theorized to reflect a failure in response inhibition (Lezak, 1995). In the current sample, the mean (SD) Stroop difference score was 125.47 (41.55).

The second measure of executive functioning used in this study was the perseverative error score on the Wisconsin Card Sort Test (WCST; Berg, 1948). In this task, the youth was presented with a series of cards with different shapes of different colors displayed on them. They were asked to sort the cards by category (e.g., by shape or color) and given feedback about whether they sorted each card correctly. Rules for correct categorization change during the test (with no warning or explanation), and a failure to switch sets counts as a perseverative error. The Wisconsin perseverative error score has been found to be sensitive to frontal lobe damage in

neuropsychological studies (Lezak, 1995). The mean (SD) perseverative error score in this sample was 8.37 (6.88).

Chronic family stress

In order to evaluate adolescents' experience of stress in the family, a semistructured interview for adolescents was developed from earlier versions of chronic strain/functioning for children (Hammen, 1991) and adults (Hammen et al., 1987). The interview included a set of probes designed to assess stress in relationships with family members (parents and siblings). Chronic family stress was rated by the interviewer on a 5-point scale, with behaviorally specific anchor points indicating severity of ongoing stressful conditions (exceptionally good conditions = 1, extreme adversity = 5). Scoring was based on such factors as conflict resolution, adequate communication, cohesion, trust and acceptance. Reliabilities based on independent judges' ratings of audiotaped interviews yielded an intraclass correlation of 0.84 for this measure. The mean (SD) chronic family stress score for the youths in this sample was 2.34 (0.58).

Statistical controls

Family psychiatric history. Two types of family psychiatric history were controlled for in this study. First, due to the high-risk nature of the sample, we controlled for maternal depression status. Mothers were classified as depressed if they qualified for a lifetime diagnosis of major depression or dysthymia on the SCID (358 mothers were classified as such). Second, because we were assessing antisocial outcomes in the youths, and there is an overlap between AUDs and ASPD, we controlled for the presence of ASPD symptoms in the biological father (according to the SCID or FHRDC). There were 63 fathers fund to have a history of ASPD symptoms.

Intelligence scores. Youths were administered the vocabulary subtest of the Wechsler Intelligence Scale for Children-III (WISC-III; Wechsler, 1981) at the age 15 follow-up. The mean (SD) scaled score for vocabulary in this sample was 8.65 (2.61). This proxy measure of IQ was used as a statistical control m all analyses examining neuropsychological functioning.

Demographics. Maternal education was used as a proxy measure of socioeconomic status. Maternal education and whether or not the mother was currently married to the biological lather were included as demographic controls in all analyses.

Results

Exploratory analyses did not find any moderating effects of gender, so results were collapsed across male and female subjects, and gender was included as a covariate in all analyses. There were also no moderating effects of maternal depression, suggesting that the parental AUD/child delinquency relationship does not depend on maternal depression status. As noted above, all analyses controlled for biological fathers' ASPD symptoms, as well as mothers" education, depression status and whether or not she was presently married to the biological lather. In addition, all analyses examining neuropsychological functioning measures included a statistical control for IQ (WISC-III vocabulary subscale). There were several items on the youth

nonviolent delinquency scale that were related to both delinquency and alcohol problems. When analyses were run without these items, results were unchanged. Missing data resulted in varying sample sizes: sample sizes for each analysis are therefore reported throughout the results.

Bivariate correlations revealed small but significant correlations between paternal AUDs and child violent (r 0.13) and nonviolent (r = 0.10) delinquency. The correlations between maternal AUDs and child violent (r = 0.01) and nonviolent (r = 0.04) delinquency were nonsignificant (see Table 1).

It was predicted that chronic family stress and executive functioning would mediate the relationship between parental AUDs and child violent and nonviolent delinquency. To test this hypothesis, the data were examined for fit, with the conditions of mediation as outlined by Baron and Kenny (1986). These conditions are: (1) the predictor (parental AUD status) must be significantly related to the dependent variable (child delinquency), (2) the predictor must be significantly related to the potential mediators (chronic family stress and executive functioning measures), (3) the mediators must be significantly related to the dependent variable and (4) inclusion of the mediators in the regression model must substantially decrease the strength of the relationship between the predictor and the dependent variable.

Parental AUDs and child delinquency

Two-step hierarchical regressions were conducted to determine whether maternal AUDs predicted the dependent variables (i.e., child violent and nonviolent delinquency). All covariates (gender, marital status, maternal education, maternal depression and paternal ASPD) were entered into the first step of the model, and maternal AUD status was entered into the second step of the model. Results revealed that maternal AUDs were not significantly related to either child violent (F = 0.74, 1/796 df, p = .39; [R.sup.2] change 0.001) or child nonviolent (F = 0.37, 1/796 df, p = .54; [R.sup.2] change = 0.000) delinquency. Maternal AUDs were therefore dropped from further analyses.

Hierarchical regressions were also conducted to determine whether paternal AUDs predicted the dependent variables. Covariates were entered into the first step of the model, and paternal AUD status was entered into the second step of the model. Results revealed that paternal AUDs were related to both child violent and nonviolent delinquency (see Tables 2 and 3).

Paternal AUDs, chronic family stress and executive functioning

Two-step hierarchical regressions were conducted to determine whether paternal AUDs predicted the potential mediator variables (i.e., chronic family stress and executive functioning measures). Covariates were entered into the first step of the model, and paternal AUD status was entered into the second step of the model. Results revealed that paternal AUDs predicted chronic family stress (F = 12.26, 1/799 df, p < .01; [R.sup.2] change = 0.01) and Stroop difference scores (F = 4.83, 1/778 df, p = .03; [R.sup.2] change = 0.01) but not perseverative errors on the WCST (F = .004, 1/781 df, p = 0.95; [R.sup.2] change = 0.00; not shown in a table). Because

perseverative errors were not related to paternal AUD status, this variable was not considered for further testing as a potential mediator.

Chronic family stress, Stroop scores and child delinquency

Table 1: correlations among predictor, control and outcome variables

Variable	1	2	3	4	5	6	7
1. Violent delinquency	.58+	.13+	.01	.26+	.13+	.13+	.03
2. Nonviolent delinquency			.10+	.04	.32+	-.02	-.08*
3. Paternal AUD				.05	.17+	.09+	.02
4. Maternal AUD					.04	.03	-.03
5. Family stress						.02	.01
6. Stroop scores							.22+
7. WCST errors							

Notes. AUD = alcohol use disorder, WCST errors = Wiscousin Card Sort Test perserverative errors
*p<.05; +p<.01

Two-step hierarchical regressions were conducted to determine whether chronic family stress and Stroop difference scores (the potential mediators associated with paternal AUDs) were related to child delinquency. Covariates were entered into the first step of the model, and the relevant mediator variable was entered into the second step of the model. Results revealed that chronic family stress predicted both violent ($F = 54.99$, 1/796 df, $p < .01$; [R.sup.2] change = 0.06) and nonviolent ($F = 80.85$, 1/796 df, $p < .001$; [R.sup.2] change = 0.09) delinquency. Stroop difference scores predicted violent ($F = 3.94$, 1/783 df, $p < .05$; [R.sup.2] change = 0.01) but not nonviolent ($F = 0.64$, 1/783 df, $p = .42$; [R.sup.2] change = 0.001; not shown in a table) delinquency.

Mediating effects of chronic family stress and Stroop difference scores

A four-step hierarchical regression was conducted to determine whether Stroop scores and chronic family stress mediated the relationship between paternal AUD status and child violent delinquency. Covariates were entered into the first step of the model. Paternal AUD status was entered into the second step of the model. Stroop and WISC-III vocabulary scores were entered into the third step of the model, and chronic family stress was entered into the final step of the model. Results revealed that the beta associated with paternal AUD status decreased to nonsignificance after Stroop scores were entered into the model (Step 2 beta = 0.08, $t = 2.14$, $p = .03$; Step 3 beta = 0.07, $t = 1.87$, $p = .06$). The beta associated with paternal AUD status decreased even further after chronic family stress was entered into the model (Step 4 beta = 0.04, $t = 1.03$, $p = .30$; see Table 2).

A three-step hierarchical regression was conducted to determine whether chronic family stress mediated the relationship between paternal AUD status and child nonviolent delinquency. Control variables were entered into the first step of the model. Paternal AUD status was entered into the second step of the model, and chronic family stress was entered into the final step of the model. Results revealed that the beta associated with paternal AUD status decreased to nonsignificance after chronic family stress was entered into the model (Step 2 beta = 0.07, $t = 2.01$, $p = .045$; Step 3 beta = 0.04, $t = 1.01$, $p = .32$; see Table 3).

Discussion

The results of this study support the hypothesis that paternal AUDs are related to child violent and nonviolent delinquency. This finding is consistent with previous literature and supports the notion that COAs are at risk for externalizing behaviors (Barnow et al., 2002; Puttler et al., 1998; Reich et al., 1993).

It is important to note, however, that the effect sizes associated with paternal AUDs were fairly small. Thus, it is possible that paternal alcohol problems explain only a small portion of the variance in child delinquency. It is possible, alternatively, that paternal AUDs are strong predictors of child delinquency within certain subgroups of participants. Barnow et al. (2002), for example, found that the association between COA status and externalizing disorders was limited to children with three or more alcoholic relatives. Future studies are needed to identify other potential moderators of the COA/delinquency relationship.

Maternal AUDs did not predict child violent or nonviolent delinquency in our sample. There are several potential reasons for these nonsignificant results. First, only 26 of the 816 mothers in our sample met criteria for DSM-IV alcohol abuse or dependence. Thus, our analyses may have lacked the power needed to detect maternal AUD effects. Second, all of the alcoholic mothers in our sample were organized and responsive enough to participate in ongoing longitudinal research. Therefore, it is conceivable that these women comprise a particularly high-functioning group of alcoholic mothers. Last, several studies suggest that the AUD/delinquency relationship is passed from fathers to sons. Cloninger (1987), for example, proposes that there exists a subtype of male COAs at genetic risk for aggression, conduct disorder and early onset alcohol problems. To date, however, very few studies have examined the COA/ delinquency relationship among females. Future studies are needed to test this theory.

Table 2: Hierarchical regression analyses predicting violent delinquency

Predictor	*r*	Step 1 beta	Step 2 beta	Step 3 beta	Step 4 beta
Gender	-.21	-.21	**-.21**	**-.20**	**-.21**
Marital status	-.06	-.05	-.04	-.03	.01
Mother education	-.05	-.04	-.04	-.02	-.01
Mother depression	.07	.06	.06	.06	.03
Father ASPD	.02	.04	.02	.02	-.02
Father alcohol problem	.13		**.08**	.07	.04
WISC-III vocabulary	-.09			-.04	-.04
Stroop difference score	.13			.07	.07
Chronic family stress	.26				**.26**

Notes: Step 1: Adjusted R^2 = .06, F = 9.4, p<.001; Step 2: R^2 change = .01, F change = 4.6, significance (sig) of F change = .03; Step 3 = R^2 change = 0.1, F change = 3.3, sig of F change = .04; Step 4: R^2 change = .06, F change = 51.4, sig of F change <.001, Significant betas indicated in bold. ASPD = antisocial personality disorder; WISC-III = Wechsler Intelligence Scale for Children-III.

Results of our study also support the hypothesis that chronic family stress mediates the relationship between paternal AUD status and child violent and nonviolent delinquency. This finding suggests that fathers with severe alcohol problems tend to have high levels of family stress. Moreover, this finding seems to underscore the

protective effects of a positive home environment and to suggest the importance of system-level interventions for families dealing with alcohol-related problems.

As predicted, executive functioning also mediated the relationship between paternal AUD status and violent delinquency. Executive functioning did not, however, predict nonviolent delinquency. There are several potential explanations for this finding. First, it is possible that central nervous system damage directly increases aggressive responding. Researchers have found increased aggression in animals following brain lesions and neurotransmitter alterations (Johansson and Hansen, 2000; Johansson et al., 1999). Thus, it is conceivable that some COAs exhibit central nervous system abnormalities that directly predispose them to violence. These abnormalities may be the result of growing up in a chaotic, nonnurturing familial environment. Frontal lobe damage may also lead to violence by disrupting executive functions (e.g., impulse control and planning). It is possible that nonviolent delinquency involves forethought and organization (e.g., buying or selling stolen goods), whereas violent delinquency involves impulsive reactions to frustration or provocation (e.g., hitting or threatening others). As a result, executive functioning deficits may selectively increase violent (as opposed to nonviolent) delinquency. Last, it is possible that violent and nonviolent delinquency represent qualitatively different patterns of deviance. Moffitt (1993), in particular, has suggested that long-term persistent delinquency is more likely to have a biological basis than adolescent-limited delinquency. It is possible that violent delinquency represents a more persistent, neurologically based type of deviance than nonviolent delinquency.

Table 3: Hierarchical regression analyses predicting non-violent delinquency

Predictor	*r*	Step 1 beta	Step 2 beta	Step 3 beta
Gender	.01	-.01	-.01	-.02
Marital status	-.09	**-.08**	**-.08**	-.03
Mother education	-.01	-.01	.01	.01
Mother depression	.04	.03	.03	-.01
Father ASPD	.04	.07	.05	.01
Father alcohol problem	.10		**.07**	.04
Chronic family stress	.32			**.31**

Our data came from a large prospective sample, and we were able to control for maternal depression, paternal ASPD, intelligence scores, marital status, gender and maternal education. Nonetheless, our study had several limitations. First, nearly half of the mothers in our sample were at risk for depression. Although maternal depression status did not moderate the parental AUD/child delinquency relationship, future studies are needed to replicate these effects in nonselected community samples. In addition, we were unaware of the degree to which COAs had had contact with their alcoholic parents. It is unclear, therefore, whether COAs with minimal exposure to alcoholic relatives are at risk for delinquency.

Our results lend support to a biosocial conceptualization of the COA/delinquency relationship. They suggest that COAs are vulnerable not only to executive functioning deficits but also to stressful home environments that may exacerbate these deficits and lead to behavior problems. It is notable that post hoc analyses with our data indicated a significant interaction between Stroop scores and chronic family stress in predicting youth violent delinquency ($F = 6.23$, 1/780 df, $p = .01$). The pattern of this interaction

supports the contention that these risk factors may exacerbate one another in the prediction of youth violence. Future studies should investigate the process by which chronic family stress moderates the relationship between executive functioning and externalizing disorders, as increased understanding of this process may be relevant to early intervention and prevention. Future research also is needed to determine the effects of family-based interventions and cognitive remediation programs on delinquency among COAs.

Last, the current study has implications for the conceptualization of COA status. Recent investigators have argued against the idea of a COA "syndrome" (i.e., that COAs are a distinct, clinical population with "specialized treatment needs"; Harter, 2000). They state that the negative outcomes associated with a family history of alcoholism are not specific to COA status. The current study appears to support this hypothesis. The variability in delinquency within our sample was attributable to family stress and neuropsychological functioning rather than to COA status per se. These findings imply that not all COAs are at risk for delinquency and suggest the need for a more complex multifaceted approach to risk assessment.

Acknowledgments

The authors are grateful to the Brisbane project interviewers Cheri Dalton, Barbara Mann, Eileen Tone, Sandra Fergusson, Molly Robbins and Lisa Manning. They also thank the MUSP Team, MUSP participants, the Mater Misericordiae Mother's Hospital and the Schools of Social Science, Population Health and Medicine at the University of Queensland.

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Received: April 2, 2004. Revision: August 17, 2004.