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Parental Alcoholism and Co-Occurring Antisocial Behavior: Prospective Relationships to Externalizing Behavior Problems in their Young Sons[1]

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The hypothesis that parental alcoholism and co-occurring antisocial behavior would be indirectly linked to child externalizing behavior problems through child lack of control, current levels of parent depression, family conflict, and parent–child conflict was tested using manifest variable regression analysis. Participants were a community sample of 125 families with an alcoholic father and 83 ecologically matched but nonsubstance abusing families involved in the first 2 waves of an ongoing longitudinal study (with 3 years between each wave). All families had a biological son who was 3–5 years old at study onset. Results revealed that child lack of control mediated the relation between paternal alcoholism and the son's subsequent externalizing behavior problems. Family conflict was a significant mediator of maternal and paternal lifetime antisocial behavior effects and father–son conflict mediated paternal lifetime antisocial behavior effects. Study implications are discussed within the context of parental socialization of antisocial behavior.

KEY WORDS: Children of alcoholics; child externalizing behavior problems; child lack of control; family conflict; parental alcoholism.

Children of alcoholics (COAs) are at elevated risk for a variety of behavioral difficulties, foremost among which are aggressiveness, delinquency, and attention deficits (Sher, 1991). Collectively, these behaviors have been termed externalizing problems (Achenbach, 1991) and their emergence in COAs has been of central concern because they are hypothesized to be one of the first steps in a causal process leading to later alcoholism (Sher, 1991; Zucker & Gomberg, 1986). In fact, at least one longitudinal study has shown that externalizing behavior problems in adolescence, in part, mediate the relation between parental alcoholism and offspring alcoholism in young adulthood (Chassin, Pitts, DeLucia, & Todd, 1999).

Although adoption and twin studies show that parental alcoholism is related to child externalizing problems both through genetic and environmental factors (see Iacono, 1998), the mechanisms underlying and sustaining parental alcoholism effects are not yet well understood. Previous studies have shown that COAs differ from their counterparts on a number of individual- and family-level variables including impulsivity, family conflict, and parent–child conflict (Barrera, Chassin, & Rogosch, 1993; Fitzgerald et al., 1993; Moos & Billings, 1982). However, there has been a paucity of research to determine the role of such factors as mediators of the relation between parent alcoholism and child externalizing behavior problems. Moreover, whereas several studies have examined the development of problem outcomes for adolescent and young adult COAs (e.g., Chassin et al., 1999), substantially less research has been directed toward examining the problems

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encountered by this population during the preschool- and school-age years.

Examination of problem behaviors during the preschool years is particularly important given that behavior problems begin to emerge during this period of development (Campbell, 1995), may persist across time (Fischer, Rolf, Hasazi, & Cummings, 1984), and are predictive of later maladjustment (see Loeber & Hay, 1997 for review). For instance, previous research has demonstrated that the sustained presence of conduct problems in 3-year old boys is linked to the development of antisocial behaviors and alcohol dependence at age 18 (Moffitt, Caspi, Dickson, Silva, & Stanton, 1996). Furthermore, it is at the end of this period of development that gender differences in levels of problem behaviors become increasingly apparent. Beginning in early childhood and continuing into adulthood, more males than females show aggressive and antisocial behaviors (see Loeber & Hay, 1997). Given the implications for early behavioral difficulties in boys, the focus of the current research was the development of externalizing problems in a sample of male COAs and non-COAs who were 3–5 years old at the onset of the study and 6–8 years old at follow-up.

Children of alcoholics are a heterogeneous population and show substantial variability in their outcomes (Johnson & Jacob, 1995). Although a portion of COAs develop later problems, many do not show elevated levels of emotional or behavioral problems and most do not develop later alcoholism. Variability may result from individual differences in the etiology and developmental course of problems, but may also be due to the presence of factors other than parental alcoholism that exert an independent effect on child outcomes. For instance, the presence of other forms of parental psychopathology, such as antisocial personality disorder (ASPD) and depression, may increase risk for child externalizing problems (Cummings & Davies, 1994; Shaw, Owens, Vondra, Keenan, & Winslow, 1996).

The role of parent ASPD is of particular interest because it is the most common comorbidity of alcoholism (Regier et al., 1990) and has been found to discriminate between COAs with higher and lower levels of maladjustment (Puttler, Zucker, Fitzgerald, & Bingham, 1998; Zucker, Ellis, Bingham, & Fitzgerald, 1996). However, the co-occurrence between the two psychopathologies precludes firm conclusions regarding whether it is specifically parental alcoholism or ASPD that augments risk for externalizing problems in children. Research reporting on adolescent COA outcomes has demonstrated that the contribution of parent ASPD to offspring externalizing problems is unique and exists over and above the effects of parental alcoholism (Chassin, Rogosch, & Barrera, 1991). Little research, however, has been directed toward disentangling the effects of these two variables in the development of behavioral difficulties in very young COAs.

The goal of the current study was to assess how parental alcoholism and antisocial behavior operate over time to influence the development of sons' externalizing behavior problems. That is, we examined the independent contributions of parental alcoholism and antisocial behavior, assessed when boys were 3–5 years old, on boys' externalizing problems when they were 6–8 years old. The guiding framework of the study is a contextual one, in which maladaptive child outcomes are viewed as being maintained across time in circumstances where a set of individual risk factors is nested within high-risk environments (Zucker, Fitzgerald, & Moses, 1995). From this perspective, stability or maintenance of externalizing problems is most likely in cases where both individual and contextual risk factors have a sustained presence as the child develops. We assessed the role of (a) child lack of control as the individual risk factor and (b) current levels of parental depression, family conflict, and parent–child conflict as the contextual factors.

Based upon theory and previous research (briefly reviewed below), we hypothesized that each of the individual and contextual risk factors would mediate, or account for, the relations between parental alcoholism and antisocial behavior and follow-up ratings of the son's externalizing outcome. Thus, we expected that parental alcoholism and antisocial behavior would be indirectly related to boys' externalizing behavior problems through child lack of control, current levels of parent depression, family conflict, and parent–child conflict. Because family demographic variables have been linked to parental lifetime alcohol problems and antisocial behavior as well as to child maladjustment (see Fitzgerald, Zucker, & Yang, 1995), we also included examination of parental level of education in our model (see Fig. 1 for Hypothetical Model).

A number of individual difference variables have been implicated in the development of later externalizing problems. Among these are emotional liability, impulsivity, distractibility, inattention, and overactivity. For instance, Pierce, Ewing, and Campbell (1999) found that boys identified by their mothers or teachers as impulsive, noncompliant, and inattentive in preschool had more externalizing sympomatology in middle childhood and early adolescence. Caspi and his colleagues (Caspi, 2000; Caspi, Moffitt, Newman, & Silva, 1996; Caspi & Silva, 1995) have reported that individual differences identified in the preschool years are predictive of maladjustment and substance dependence well into early adulthood. In particular, children characterized by emotional liability, restlessness, short attention span, and negativism, collectively

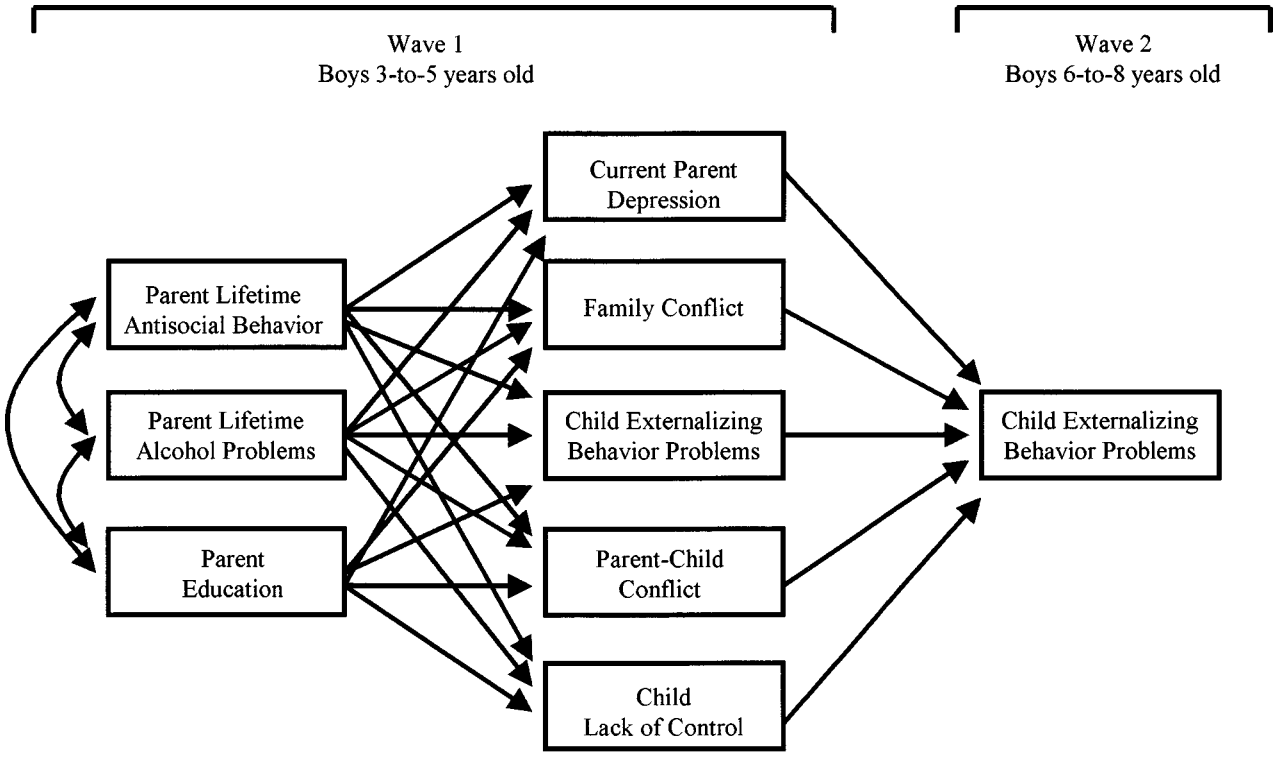

Fig. 1. Hypothetical mediational model of child lack of control and family contextual factors linking long-term parental psychopathology and years of education to subsequent child externalizing behavior problems.

referred to as lack of control, at ages 3 and 5 were more likely to show externalizing problems in later childhood and early adolescence. In comparison to their peers, undercontrolled children also scored higher on aggression and impulsivity at age 18 (Caspi & Silva, 1995), and were more likely to be diagnosed with alcohol dependence at age 21 (Caspi et al., 1996). Taken together, these studies indicate that attributes reflecting deficits in emotional and attentional control in childhood play a central role in the development of externalizing problems in adolescence and alcohol dependence in adulthood.

Children of substance abusing parents have been described as displaying poor emotional and attentional control, thus elements of lack of control may mediate paternal alcoholism effects on child externalizing problems (see Tarter & Vanyukov, 1994). For instance, in comparison to their counterparts, 3-year old sons of alcoholics are more impulsive on a delay on gratification task (Fitzgerald et al., 1993) and 10- to 12-year-old sons of substance abusing fathers score higher on measures of inattention and impulsivity (Martin et al., 1994). Children who are undercontrolled may be at increased risk for externalizing problems because they are high in emotional dysregulation and as such are more prone than their counterparts to become easily frustrated and act out (Cole, Michel, & Teti, 1994). However, such children are also likely to evoke or elicit negative responses from individuals with whom they interact, which further contributes to elevate risk for maladjustment (Lytton, 1990).

In addition to child individual characteristics, current level of parent psychopathology (e.g., depression) may also play an important role in the development of externalizing behavior problems. The role of contemporaneous levels of parent depression may be especially salient because depression is found at elevated rates in male and female alcoholics (Regier et al., 1990), is linked to a variety of child emotional and behavioral difficulties, and may be indicative of the prevailing quality of the family environment (see Cummings & Davies, 1994). Existing evidence indicates that parent depression heightens risk for both child internalizing and externalizing problems (Campbell, March, Pierce, Ewing, & Szumowski, 1991; Chassin et al., 1991), for experiencing less positive family interactions (Johnson & Jacob, 1995), and also more family conflict (Billings & Moos, 1983). Given that alcoholics and individuals who are antisocial often experience elevated levels of depression (Babor et al., 1992), we hypothesized that contemporaneous levels of parent depression

would mediate the parental alcoholism and antisocial behavior effects.

Family contextual factors—such as family conflict—are also likely to mediate the associations between parental alcoholism, antisocial behavior, and child externalizing behavior problems. Children of alcoholics experience elevated levels of family conflict (Clair & Genest, 1987) and family conflict has been found to be related to the development of child emotional and behavioral problems both in COAs (Moos & Billings, 1982) as well as the broader population of non-COAs (Lindahl, 1998). The effect of family conflict on child functioning can be viewed within the context of socialization. Social interaction theorists suggest that children learn directly from their parents how to behave in everyday situations (Patterson, 1982). Consequently, children who are repeatedly exposed to conflictual interactions among family members also learn to behave in this manner. Parents who experience high levels of distress, such as individuals who are alcoholic, antisocial, or depressed, are especially vulnerable to exposing their children to aggressive and conflictual interactions, thereby increasing the likelihood that their children will exhibit externalizing behavior problems (Patterson & Capaldi, 1991). Yet, despite the important socializing role that family conflict may play in the development of externalizing problems for COAs, very little research to date has examined the contribution of family-level characteristics to COA outcomes.

Family conflict is a global indicator of the interactions that occur within the family system and thus, does not provide any information regarding the quality of interactions between a specific child and each of his parents. To determine the effect of parent–child interactions on child outcome, we included examination of parent–child conflict as a putative mediator of parental alcoholism and antisocial behavior effects. Previous research has shown that COAs experience elevated levels of parent–child conflict (Reich, Earls, & Powell, 1988). Moreover, studies of children with elevated levels of externalizing behavior problems show that parents are negative toward their children (Campbell et al., 1991; Campbell, 1994) and children are fidgety, restless, and uncooperative in the presence of their parents (Campbell, Pierce, March, Ewing, & Szumowski, 1994), all of which may contribute to the quality of parent–child interactions. Like family conflict, parent–child conflict may act by socializing the child to behave in a conflictual and coercive manner (Patterson, 1982). However, actual level of externalizing problems may also elicit negative responses from the parent, further contributing to more conflictual interactions and thereby, increasing risk for externalizing problems (Lytton, 1990).

In sum, we examined the prospective contributions of parental history of alcoholism and antisocial behavior to the development of externalizing behavior problems in a sample of male children who were initially 3–5 years old. We expected that the parental lifetime variables would be indirectly related to sons' later externalizing problems through child lack of control, contemporaneous levels of parent depression, family conflict, and parent–child conflict.

METHOD

Participants

Participants were all 208 non-Hispanic White families (biological father, mother, and son) involved in Wave 1 and Wave 2 of an ongoing longitudinal study (Zucker et al., in press). Because non-Whites make up less than 4% of the population meeting inclusion criteria in the area sampled, they were not included in the original longitudinal study. The study is tracking the development of children being reared in high-risk environments characterized by parental alcohol abuse and related comorbidities. These 208 families are a subset of the larger study sample. Families not included in this subset were missing by design and were not accessed at Wave 2 because of funding problems at the time. However, there are no biases differentiating those missing from those included. At Wave 1, all 208 families were intact, as specified by inclusion criteria, and included a male target child who was 3–5 years old ($M = 4.21$, $SD = 0.90$). Wave 2 data collection occurred 3 years later when the male children were 6–8 years old ($M = 7.56$, $SD = 1.00$). By the end of Wave 2 data collection, 20 of the 208 families experienced a divorce or separation ($n = 17$ alcohol group families; $n = 3$ control group families). In all 20 cases noncustodial parents retained contact with their target sons and thus, Wave 2 data (i.e., information on child externalizing problems) were collected from all custodial and noncustodial biological parents.

Recruitment procedures for the larger study have been described elsewhere in detail (see Fitzgerald et al., 1995; Zucker et al., in press). The sample for this study included 125 alcoholic families and 83 nonalcoholic control families. Alcoholic families were recruited by way of father's drinking status in one of two ways. The first group of alcoholic fathers and their families ($n = 75$) was recruited by way of a net of administrative arrangements covering five local district courts and all drunk driving convictions in a four county area in mid-Michigan. The fact that the men in the alcoholic group were convicted drunk drivers indicated that their alcoholism was more

heavily combined with antisociality than was true of other alcoholics (Cloninger, 1987; Zucker, 1994). At initial contact, a positive alcoholism diagnosis was established using the Short Michigan Alcoholism Screening Test (SMAST; Selzer, 1975) and was subsequently verified by way of the NIMH Diagnostic Interview Schedule—Version III (DIS; Robins, Helzer, Croughn, & Ratcliffe, 1980). Using the Feighner Diagnostic criteria (Feighner et al., 1972), all alcoholic-group men met a "definite" or "probable" diagnosis for alcoholism during the life of the child with 92% making a "definite" diagnosis. Later, *DSM-III-R* diagnoses were also established although this was not a basis for study inclusion. Seventy-three percent of the alcoholic men met either moderate or severe alcohol dependence criteria.

Maternal drinking status was also assessed and revealed that 35 mothers in this group received a lifetime *DSM-III-R* diagnosis of alcohol abuse or dependence. However, maternal alcoholism was neither a requirement for inclusion nor a basis for exclusion from the study where risk families were concerned except that no family was included if their child manifested characteristics associated with fetal alcohol syndrome (FAS), including prenatal or postnatal growth retardation or both, central nervous system involvement, and characteristic facial dysmorphology (see Sokol & Clarren, 1989). On these grounds, two families contacted for study participation were excluded from later study involvement because of the presence of morphological indicators of FAS in their sons.

The second group of alcoholic fathers and their families ($n = 50$) was recruited from the neighborhoods in which the drunk driver alcoholic fathers resided. These families were accessed during neighborhood canvasses for nonalcoholic control families (shown later). Just as for the court-recruited alcoholic parents, alcohol diagnoses were initially established using the SMAST (Selzer, 1975) and all men met a "definite" or "probable" diagnosis for alcoholism, using the Feighner Diagnostic criteria (Feighner et al., 1972). Later, *DSM-III-R* diagnoses were also established but were not a basis for study inclusion. Maternal alcoholism was assessed but was not a basis for inclusion or exclusion from the study. Twenty-six mothers received a diagnosis of alcohol abuse or dependence according to lifetime *DSM-III-R* criteria and none of the children in this group manifested characteristics indicative of FAS. This group of families provided an ecologically comparable subset of high-risk families drawn out of the same social stratum as the drunk drivers, but where the alcoholism was identified by way of community survey.

The 83 nonalcoholic control families were recruited using door-to-door canvassing starting one block away from the alcoholic family and staying within the same census area where possible. The basis for choosing these families was geographic proximity and having a same-aged biological son (within 6 months) as the same-neighborhood alcoholic family. It was required that neither parent in this group receive an alcohol or drug diagnosis as per Feighner criteria (Feighner et al., 1972).

Data Collection

Data were collected by trained project staff who were blind to family risk status. Because of the large volume of data collected, a number of contacts with the family were necessary. Each wave of data collection took place across nine data collection sessions, seven of which took place in the family home and two of which took place on a university campus. At each wave the visits involved a contact time of approximately 9–10 hr for each parent and 7 hr for the target child. Contacts included questionnaire sessions, semistructured interviews, and interactive tasks.

Measures

In the current study, we used continuous measures of parent alcoholism, antisocial behavior, and depression rather than dichotomous measures of symptomatology (i.e., diagnoses). The decision to use this strategy was based on the premise that continuous measures provide more measurement information than do dichotomous measures and thus, are better able to represent the full history of psychopathology (Cummings & Davies, 1994). In particular, the assessment of parent alcoholism and antisocial behavior reflects the extensiveness of symptomatology that has been present over the life course, including early adolescence as well as adulthood (cf. Zucker, Davies, Kincaid, Fitzgerald, & Reider, 1997). However, for descriptive purposes, we provide information regarding clinical diagnoses of parental psychopathology, where available (e.g., for parental alcoholism and ASPD).

Developmental Scaling of Parent Alcoholism

The Lifetime Alcohol Problems Score (LAPS; Zucker et al., 1997) was the primary alcohol involvement variable used in the present analyses. The score was designed to assess differences in the magnitude and extent of drinking problems over the life course and was derived from information on the Drinking and Drug History Interview (Zucker, Fitzgerald, & Noll, 1990), the DIS (Robins et al., 1980), and the SMAST (Selzer, 1975). The LAPS provides a composite score derived from three component subscores: (a) the primacy component, involving

the squared inverse of the age at which the respondent reported first drinking enough to get drunk; (b) the variety component, involving the number of different drinking symptoms reported; and (c) the life percent component, involving a measure of the interval between the most recent and the earliest drinking problems, corrected for current age. The subscores are standardized and summed to form the final score. Given the generally lower level of alcohol related difficulty in women and to increase dispersion on the measure, all standardization was done within sex. This measure effectively distinguishes between alcoholics and nonalcoholics, among levels of severity of *DSM-III-R* alcohol dependence, and the index also is correlated with a wide range of external measures of alcohol-related difficulty such as blood alcohol level at arrest and treatment involvement (see Zucker et al., 1997). This lifetime measure of alcoholism was calculated only at Wave 1 for both mothers and fathers and higher scores on LAPS reflect more problems related to drinking.

Parental Lifetime Antisocial Behavior

To assess lifetime antisocial behavior, each parent completed the Antisocial Behavior Checklist (ASBCL) at Wave 1 (Zucker & Noll, 1980). The ASBCL is a 46-item revision of an earlier antisocial behavior inventory utilized in the Rutgers Community Study (Zucker & Barron, 1973) that has been modified so that items are also salient for adult antisocial activities. The ASBCL measures the frequency of the parent's participation in a variety of aggressive and antisocial activities in adolescence (e.g., lying to parents, being suspended from school) as well as in adulthood (e.g., being fired, resisting arrest, defaulting a debt). The scores for each item range from 0 (*Never*) to 3 (*Often*). Higher scores on the ASBCL reflect more antisocial behavior.

A series of reliability and validity studies with populations ranging from male and female college students to male and female jail inmates has shown that the ASBCL has adequate test-retest reliability (.91 over 4 weeks) and internal consistency (coefficient alpha = .93). The ASBCL differentiates between individuals with long histories of antisocial behavior (prisoners) versus individuals with minor offenses in district court versus university students (Ham, Zucker, & Fitzgerald, 1993), and between alcoholic and nonalcoholic adult males (Zucker, Noll, Ham, Fitzgerald, & Sullivan, 1994) and also strongly discriminates individuals with ASPD from those without the disorder (Ellis, Bingham, Zucker, & Fitzgerald, 1996). Although diagnostic information was not used in the current analyses, it is worth noting that 26 fathers (only one of whom was in the control group) and four mothers (all in the alcoholic group) participating in the current study received a *DSM-III-R* diagnosis of ASPD.

Parental Education

Highest level of parental education attained (in years) was ascertained at Wave 1 by a demographics questionnaire.

Parental Current Depression

Current levels of maternal and paternal depression were assessed at Wave 1 by the self-reported 13-item short form of the Beck Depression Inventory (Beck, Rial, & Rickels, 1974). This measure focuses on various areas of functioning known to be affected by depression, such as mood, appetite, and sleep. Considerable evidence supports its reliability and validity (Beck, Steer, & Garbin, 1988). Items were scored on a scale ranging from 0 (*higher functioning*) to 3 (*lower functioning*) and were summed so that higher scores reflect higher current levels of parental depression.

Child Lack of Control

Child lack of control was assessed at Wave 1 by five items from a modified version of the Conners Parent Rating Scale (CPRS; Conners, 1970). The CPRS is a 91-item questionnaire that has been used for screening and assessing behavior problems. Our 51-item version is similar to the 48-item Revised Conners Parent Rating Scales (Goyette, Conners, & Ulrich, 1978); however, 4 items were deleted and several items from the 91-item version were retained. The five items used in the present study (inattentive; demands must be met immediately, easily frustrated; gets overexcited easily; easily bored by a repetitive activity; fails to finish things he started) reflect poor child emotional and attentional control, do not directly overlap with items on the externalizing behavior problems subscale, and for brevity are referred to as lack of control. Mothers and fathers independently rated their sons on each item using a response scale ranging from 0 (*Not at all*) to 3 (*Very much*). The five items were summed and higher scores reflect higher levels of child undercontrol. Internal consistency of the five items was adequate for both maternal (coefficient alpha = .76) and paternal reports (coefficient alpha = .73).

Family Conflict

Conflict in the family environment was assessed at Wave 1 by the Conflict subscale of the Family Environment

Scale, Form R (FES; Moos, 1974). The FES is an empirically based taxonomy of family social environments as perceived by family members themselves. It requires fifth- or sixth-grade reading skills. Form R of the FES consists of a number of subscales that describe dimensions of the family climate with which each individual member must cope. The instrument has been subjected to extensive reliability and validity studies. The instrument provides scores in areas that have previously been significantly implicated in alcoholic and drug-abusing families; for example, conflict, cohesion, and expressiveness. Internal consistency reliability ranges from .64 to .78 and test-retest reliability ranges from .68 to .86 across the 10 subscales of the FES. For purposes of the current study, maternal and paternal ratings of the 9-item Conflict subscale were used. Items were scored either as being True (2) or False (1) and summed so that higher scores reflect more family conflict.

Parent–Child Conflict

The Child Rearing Practices Report (CRPR; Block, 1965) was used to measure maternal and paternal conflict with the child at Wave 1. The CRPR uses a forced-choice procedure to have parents rate 91 statements as most descriptive to least descriptive of their child rearing practices. The parents sort the cards into seven categories of 13 items each. The CRPR consists of 21 subscales but for purposes of the present study only the parent negative affect toward child subscale was used, which is referred to in the current study as parent–child conflict. The parent–child conflict subscale is comprised of three items: I often feel angry with my child; I feel my child is a bit of a disappointment to me; and There is a good deal of conflict between my child and me. Internal consistency (coefficient alpha) of the three items was .56 for maternal data and .65 for paternal data.

Child Externalizing Behavior Problems

Child externalizing behavior problems were assessed with the 118-item 4- to 18-year old version of the Child Behavior Checklist (CBCL; Achenbach, 1991). The CBCL is an objective assessment of the target child's social and emotional functioning. Mothers and fathers rated each of the items on a scale ranging from 0 (*Not true for my child*) to 2 (*Very true for my child*). All items are summed and higher scores reflect more behavior problems. Internal consistency of the total behavior problems score is good. Achenbach (1991) calculated the coefficient alpha to be .96. Wave 1 and Wave 2 maternal and paternal ratings of the broad-band subscale of externalizing behavior problems were used for the present study.

Missing Data Estimation

There were 36 missing data points (mothers were missing 15 data points and fathers were missing 21 data points) out of a possible 3744 (<1%). To maximize sample size and prior to substantive hypothesis testing, all missing scale scores were estimated (separately for maternal and paternal data) using a longitudinal estimation procedure developed by Petersen (1987; see also Bingham & Crockett, 1996; Bingham, Stemmler, Petersen, & Graber, 1999). This procedure utilizes two components: the nomothetic component (i.e., mean of each variable at each wave for each subgroup) and the ideographic component (the average distance, in standard deviation units, between the subjects' actual data points at the wave where data were not missing and the nomothetic component at the wave with missing data).

RESULTS

Prior to testing the hypothesized relations, we investigated change and stability in externalizing behavior problems by examining correlations and means of ratings across the two waves of data collection as well as by examining the proportion of boys for whom there was a change in the level of problems reaching clinical significance across time. Cross-temporal correlations revealed that externalizing behavior problems were highly stable across the 3-year interval for both paternal ($r = .70$, $p < .001$) and maternal ($r = .58$, $p < .001$) ratings. However, examination of mean differences in externalizing problems across Wave 1 (paternal $M = 11.62$, $SD = 6.34$; maternal $M = 12.18$, $SD = 6.40$) and Wave 2 (paternal $M = 10.39$, $SD = 6.36$; maternal $M = 10.91$, $SD = 6.53$) revealed an overall decrease in problem behaviors as boys increased in age [($t(207) = 3.58$, $p < .001$) and ($t(207) = 3.11$, $p < .01$) for paternal and maternal ratings, respectively].

Examination of the externalizing problems' T-scores revealed that at Wave 1, 23 boys ($n = 18$ COAs, $n = 5$ non-COAs) scored in the clinically significant range (i.e., a T-score at or above 64) of father-reported externalizing problems, whereas at Wave 2, 17 boys reached this criterion ($n = 13$ COAs, $n = 4$ non-COAs). Across-time comparisons revealed that 52% ($n = 9$ COAs, $n = 3$ non-COAs) of the boys who scored in the clinically significant range of father-reported externalizing problems at Wave 1 did not meet the criterion at Wave 2 and 3% ($n = 4$ COAs, $n = 2$ non-COAs) of the boys who did

Table I. Zero-Order Correlations Between Study Manifest Variables for Maternal ($n = 208$) and Paternal ($n = 208$) Data

Manifest variables	1	2	3	4	5	6	7	8	9
1. Lifetime alcohol problems	—	.57****	−.11	.05	.23****	.10	.19***	.27****	.19***
2. Lifetime antisocial behavior	.58****	—	−.36****	.21***	.29****	.11	.26****	.47****	.32****
3. Parent years of education	−.17**	−.29****	—	−.13*	−.11	−.08	−.03	−.07	−.13*
4. Wave 1 parent depression	.24****	.34****	−.07	—	.28****	.05	.11	.16**	.14**
5. Wave 1 family conflict	.26****	.31****	.02	.37****	—	.16**	.03	.22****	.26****
6. Wave 1 parent–child conflict	.03	.15**	−.11	.15**	.18***	—	.10	.22****	.31****
7. Wave 1 child lack of control	.26****	.10	−.08	.26****	.14**	.12*	—	.55****	.30****
8. Wave 1 child externalizing	.35****	.27****	−.13*	.31****	.39****	.17**	.57****	—	.58****
9. Wave 2 child externalizing	.27****	.25****	−.14**	.27****	.38****	.23****	.51***	.69****	—

Note. Maternal correlations are shown above the diagonal and paternal correlations are shown below the diagonal.
* $p < .10$. ** $p < .05$. *** $p < .01$. **** $p < .001$.

not meet the criterion at Wave 1 did meet the cut-off at Wave 2. Similarly, maternal reports revealed that 26 boys ($n = 19$ COAs, $n = 7$ non-COAs) scored in the clinical range at Wave 1 and 22 boys ($n = 14$ COAs, $n = 8$ non-COAs) met the cut-off at Wave 2. Of the boys who scored in the clinical range of externalizing problems at Wave 1, 50% (10 COAs, $n = 3$ non-COAs) did not score in the clinical range at Wave 2. Conversely, of the boys whose mother-rated externalizing behavior problems did not reach clinical significance at Wave 1, 5% ($n = 5$ COAs, $n = 4$ non-COAs) scored at or above the clinical cut-off at Wave 2.

The hypothesis that the relations between the parental lifetime variables of alcoholism and antisocial behavior and later childhood externalizing behavior problems would be mediated by contemporaneous levels of parent depression, child lack of control, family conflict, and parent–child conflict was tested by comparing three hierarchically related (nested) models. Manifest variable regression analysis (MVRA; Jöreskog & Sörbom, 1993) was used to contrast the direct effects model, the mediated model, and the mixed effects model. The direct effects model tested the hypothesis that the associations between the exogenous parental lifetime variables and each of the dependent variables was direct (i.e., no mediated effects). The mediated model tested the hypothesized indirect effects of the parental lifetime variables on the Wave 2 child outcome through the putative mediators (see Fig. 1), and the mixed effects model tested the hypothesis that the parental lifetime exogenous variables were related to the Wave 2 outcome both directly and indirectly through the mediators.

LISREL 8 (Jöreskog & Sörbom, 1993) was used to obtain the maximum likelihood estimates of the model coefficients and a covariance matrix was analyzed. In each of the models, the exogenous variables were freely correlated and Wave 1 (baseline) externalizing behavior problems were included as a predictor of the Wave 2 child outcome to partial out its effects. Moreover, the residuals for Wave 1 child externalizing behavior problems and each of the mediator variables were intercorrelated and based upon theory and previous research, the residuals for family conflict and current level of parent depression were freely estimated as were the residuals for child lack of control and parent depression.

Separate models were analyzed for maternal and paternal data (see Table I for zero-order correlations between study variables for maternal and paternal data) because previous research has indicated that each parent provides a unique assessment of their child's behavior (Phares & Compas, 1992). Thus, combining parental ratings may have obscured the results. However, to maximize sample size and decrease truncation of variance, data were collapsed across alcoholic and control group families (see Table II for means and standard deviations of study variables for maternal and parental data separate by risk group). The three competing paternal models were examined first. The paternal direct effects model resulted in a poor fit to the data. Although the GFI was adequate (.90), the CFI was smaller than .90 (.42) and the chi-square indicated significant model-data discrepancies [$\chi^2(17, N = 208) = 94.23$, $p < .001$]. Both the mediated model [$\chi^2(7, N = 208) = 12.05$, $p = .10$; GFI = .99; CFI = .99] and the mixed effects model [$\chi^2(4, N = 208) = 10.18$, $p = .04$; GFI = .99; CFI = .99] represented a good fit to the data. Although the resulting chi-square for the mixed effects model showed significant model-data discrepancies, the GFI and CFI were acceptably large.

To determine the best fitting model, chi-square comparisons were conducted between the mixed effects model and each of the other models. Analyses comparing the mixed effects model to the direct effects model demonstrated that the mixed effects model represented a significantly better fit to the data [$\Delta\chi^2(13) = 84.05$, $p < .001$].

Table II. Means and Standard Deviations for Study Variables for Maternal and Paternal Data in the Alcoholic and Control Families

	Alcoholic families ($n = 125$)				Control families ($n = 83$)			
	Paternal		Maternal		Paternal		Maternal	
Study variables	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
Lifetime alcohol problems	10.63	1.76	10.54	2.25	7.48	1.62	9.05	1.30
Lifetime antisocial behavior	19.81	10.49	11.80	7.56	10.67	6.29	7.92	4.83
Parental education level	13.48	2.20	13.28	2.09	14.55	2.06	13.65	1.77
Wave 1 parental depression	2.84	2.81	2.70	2.60	1.84	2.18	2.72	3.25
Wave 1 family conflict	14.34	2.10	14.22	2.12	15.11	2.26	14.71	2.02
Wave 1 parent–child conflict	5.51	2.44	5.90	2.48	5.16	2.92	5.63	2.42
Wave 1 child lack of control	4.57	2.57	4.08	2.83	3.86	2.06	3.82	2.25
Wave 1 child externalizing	12.90	6.37	13.03	6.74	9.68	5.81	10.90	5.66
Wave 2 child externalizing	11.39	5.81	11.46	6.58	8.88	6.87	10.07	6.34

Comparison of the mixed effects model to the mediated model showed that both models fit the data equally well [$\Delta\chi^2(3) = 1.87, ns$]. Examination of the structural relations in the mixed effects model, however, revealed that none of the direct paths from the exogenous variables to the Wave 2 child outcome were significant when the mediated paths were freely estimated. In support of the hypothesis, the results indicated that the mediated model represented the most parsimonious and best-fitting model, predicting 51% of the variance in the child outcome at ages 6–8 (Wave 2).

The final paternal mediated model showing the significant standardized coefficients is presented in Fig. 2. Results of the model demonstrated significant correlations in the expected direction among all three exogenous variables and structural paths showed that paternal lifetime

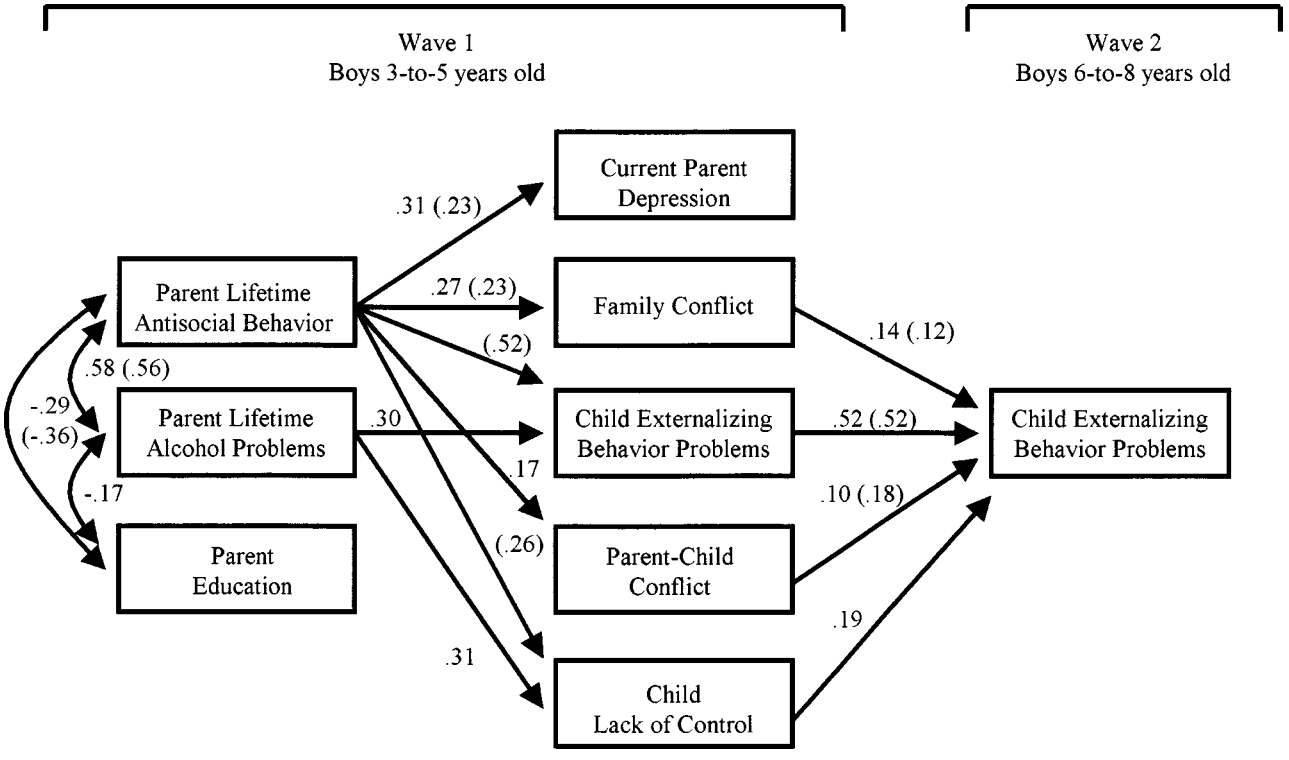

Fig. 2. Standardized solution for the final obtained paternal and maternal mediated models ($n = 208$). Maternal coefficients are shown in parentheses. To ease interpretation, only coefficients for significant ($p < .05$) paths are shown.

alcohol problems predicted baseline levels of externalizing behavior problems as well as child lack of control, both of which in turn predicted Wave 2 child externalizing problems. Higher levels of paternal antisocial behavior predicted elevated levels of Wave 1 parent depression, more family conflict, and more parent–child conflict. Increased levels of Wave 1 family conflict and parent–child conflict predicted increased rates of Wave 2 child externalizing problems. The resulting significant path from baseline externalizing behavior problems to the Wave 2 outcome demonstrated the stability of behavior problems across the 3-year interval corroborating the result from the zero-order cross-temporal correlation analysis. Moreover, after the influences of paternal lifetime alcohol problems, antisocial behavior and education were accounted for, paternal depression was still significantly and positively correlated with family conflict (standardized coefficient = .24) and child lack of control (standardized coefficient = .18). Baseline externalizing behavior problems were significantly and positively correlated with paternal depression (standardized coefficient = .19), child lack of control (standardized coefficient = .46), and family conflict (standardized coefficient = .24).

These results indicate that family conflict and parent–child conflict are potential mediators of paternal antisocial behavior effects, whereas child lack of control is a potential mediator of the relation between paternal lifetime alcohol problems and the child's outcome. To determine whether the effects of the exogenous variables on the Wave 2 outcome were indirect through the putative mediators, we used the methods described by Sobel (1986) and by MacKinnon and Lockwood (1999) to test the significance of the indirect effects.[6] Results revealed that both family conflict (estimate = .024, *SE* = .012) and father-child conflict (estimate = .012, *SE* = .007) were significant mediators of the relation between paternal lifetime antisocial behavior and later child externalizing behavior problems. Child lack of control mediated the relation between paternal lifetime alcohol problems and the Wave 2 child outcome (estimate = .157, *SE* = .070).

The same three models were then estimated for the maternal data. The direct effects model also resulted in a poor fit to the data. The GFI and CFI were smaller than .90 (.88 and .62, respectively) and the chi-square indicated significant model-data discrepancies, $[\chi^2(17, N = 208) = 157.61, p < .001]$. The hypothesized mediated model $[\chi^2(7, N = 208) = 6.60, p = .47;$ GFI = .99; CFI = 1.00] and the mixed effects model $[\chi^2(4, N = 208) = 5.23, p = .26;$ GFI = .99; CFI = 1.00] each reproduced the observed covariance matrix very well. Comparison of the chi-squares from the mixed effects and direct effects models revealed that the mixed effects model was a significant improvement in fit over the latter model $[\Delta\chi^2(13) = 152.38, p < .001]$; whereas comparison of the mixed effects model to the mediated model indicated that both fit the data equally well $[\Delta\chi^2(3) = 1.37, ns]$. However, results of the mixed effects model demonstrated that none of the exogenous variables directly predicted Wave 2 child externalizing behavior problems. Rather, the associations among the exogenous variables and the Wave 2 outcome were indirect, through the Wave 1 mediators. Results supported the hypothesis and indicated that the mediated model represented the most parsimonious and best fitting model, predicting 38% of the variance in Wave 2 child externalizing behavior problems.

The final maternal mediated model is also presented in Fig. 2 (standardized coefficients are shown in parentheses). Correlations between the exogenous variables showed that maternal lifetime antisocial behavior was significantly associated with more lifetime alcohol problems and lower levels of attained education. Examination of the structural relations revealed that maternal lifetime antisocial behavior was predictive of current level of maternal depression, child lack of control, family conflict, and baseline levels of child externalizing behavior problems. Lower levels of maternal education were marginally and positively associated with Wave 1 externalizing behavior problems. Family conflict and parent–child conflict each predicted Wave 2 child externalizing behavior problems. Similar to the paternal model and corroborating the resulting cross-temporal correlation analysis reported earlier, mother-rated child externalizing behavior problems remained relatively stable across time. Moreover, after accounting for the influences of the exogenous variables, the intercorrelations among the mediators were significant and positive for the relationships between maternal depression and family conflict (standardized coefficient = .23), baseline externalizing behavior problems and child lack of control (standardized coefficient = .42), baseline externalizing behavior problems and parent–child conflict (standardized coefficient = .13) and marginally significant for the relation between baseline externalizing problems and family conflict (standardized coefficient = .09). Contrary to expectations, maternal lifetime alcohol problems were not directly associated with any of the mediators,

[6] Mediation was tested by dividing the estimate of the mediated effect by its standard error (see Sobel, 1986). Significance of the overall effect was determined by comparing the resulting ratio with an empirical distribution derived through a series of simulation studies by MacKinnon and Lockwood (1999). These researchers have shown that because the mediated effect is not normally distributed, comparing it with the normal distribution to determine its significance results in decreased power. Thus, they recommend comparison of the effect with the empirical distribution (see MacKinnon & Lockwood, 1999 for details).

although they were significantly associated with higher levels of maternal antisocial behavior.

As described earlier, family conflict appeared to mediate the relation between maternal antisocial behavior and child externalizing behavior problems. To assess the significance of the mediational effect, a test of the indirect effects was conducted (see MacKinnon & Lockwood, 1999; Sobel, 1986). This test revealed that there was a significant indirect effect of maternal antisocial behavior on Wave 2 child externalizing behavior problems through Wave 1 family conflict (estimate = .031, SE = .018).[7]

DISCUSSION

Externalizing behavior problems in COAs are hypothesized to be one of the first steps in a causal pathway leading to later alcoholism (Zucker et al., 1995). Consequently, identifying factors that contribute to the early development of such problems may be an important precursor to understanding the etiology of alcoholism. In the current study, we used a prospective research design to examine the independent contributions of parental alcoholism and antisocial behavior to the development of externalizing behavior problems in a sample of male children who were initially 3–5 years old and who varied in level of risk for an eventual alcoholic outcome.

Consistent with previous research on non-COA preschoolers, we found that externalizing behavior problems remained relatively stable across a 3-year interval to the early school years (Fischer et al., 1984). However, results also revealed a decrease in the mean number of externalizing behaviors exhibited by boys across this 3-year period as well as a reduction in the proportion of boys whose problems reached clinically significant levels. These results corroborate previous research (see Campbell, 1995; Loeber & Hay, 1997; Mun, Fitzgerald, Puttler, Zucker, & von Eye, in press) and indicate that even though boys tend to display lower levels of externalizing problems as they grow older, they also tend to retain their relative ranking (in relation to their peers) in level of problems. Given that paternal alcoholism and maternal antisocial behavior were positively associated with baseline levels of externalizing problems, it is likely that preschool-age boys living in environments characterized by these parental psychopathologies also exhibit the highest levels of behavioral problems in the early school years.

The goal of the current study was to assess how parental alcoholism and antisocial behavior operate to elevate risk for externalizing behavior problems. We hypothesized that parental alcoholism and antisocial behavior would be indirectly related to later child externalizing behavior problems through current levels of parental depression, child lack of control, family conflict, and parent–child conflict. Our results supported the hypothesis and demonstrated that family conflict and father–son conflict were significant mediators of parental antisocial behavior effects on the boy's outcome and child lack of control mediated paternal alcoholism effects.

The mediational role of family conflict was consistent across both maternal and paternal models. Higher levels of parental lifetime antisocial behavior were related to more family conflict, which in turn contributed to the development of sons' externalizing behavior problems 3-years later. The effect of family conflict on externalizing behavior problems may represent a process of socialization, such that boys who live in environments that are conflictual and coercive may also learn to behave in this manner (Patterson, 1982). Alternatively, family conflict may act by disrupting parental ability to appropriately manage a family (Patterson, 1982). In particular, families with high levels of psychopathology experience elevated levels of distress that may interfere with parental abilities to provide appropriate child monitoring and discipline and thereby places the son at risk for problematic outcomes (Patterson, 1982; Patterson & Capaldi, 1991). The present study cannot disentangle the role of these two possibilities in the development of externalizing problems. However, given that each presents different implications for preventive intervention efforts, further research should be conducted to determine the processes by which family conflict exerts its effects.

Parent–child conflict was also identified as a significant mediator of paternal but not maternal antisocial

[7]The final paternal and maternal mediated models were reestimated with family alcohol diagnosis (0: *no*/1: *yes*) included as an exogenous variable. These analyses were conducted so that the effects of group membership could be accounted for and to verify that pooling the data across diagnostic category did not bias the resulting parameter estimates. In both models, alcohol diagnosis was allowed to freely correlate with the other exogenous variables and direct paths were estimated to each of the putative mediator variables, including Wave 1 child externalizing behavior problems. The resulting paternal [$\chi^2(8, N = 208) = 12.11,\ p = .15$; GFI = .99; CFI = .99] and maternal [$\chi^2(8, N = 208) = 6.62,\ p = .58$; GFI = .99; CFI = 1.00] models fit the data well. Examination of the structural relations revealed that family alcohol diagnosis was significantly associated with all three exogenous variables, namely parental lifetime alcohol problems, antisocial behavior, and education. However, once the relationships of lifetime alcohol problems and antisocial behavior were taken into account, residual effects of family diagnosis were not significantly associated with any of the putative mediators' variables or with the son's baseline levels of externalizing problems in either model. Moreover, inclusion of family alcohol diagnosis did not alter the pattern of associations between any of the variables in the original models. That is, all paths that were significant in the original models retained their significance.

behavior effects. Paternal antisocial behavior was associated with father–son conflict and father–son conflict predicted the son's later behavioral problems. Although mother–child conflict was also predictive of her son's externalizing problems, the current model did not support a relation between maternal antisocial behavior and mother–son conflict. Differences between maternal and paternal antisocial behavior effects on parent–son interaction quality could be related to the gender-specific socialization experiences provided to the child by each parent. That is, because fathers are likely to engage in gender-stereotyped activities with their sons (Siegal, 1987), they may be more willing than are mothers to promote and reinforce conflictual and coercive behaviors in specific interchanges with their children. Alternatively, although maternal antisocial behavior does not predict mother–son conflict in the preschool years, its effects may become more salient as the boy increases in age and enters adolescence. During adolescence, both male and female children experience the most conflict with their mothers, in comparison to their fathers (Montemayor, 1982). Thus, it is likely that as the number of conflictual interactions between mother and son increase, so too will the salience of maternal characteristics such as antisocial behavior.

Unlike family and father–child conflict, current levels of parent depression failed to meet criteria for mediation. Parents with higher levels of antisocial behavior experienced more contemporaneous depression (see Babor et al., 1992) but parental depression was not related to the boy's later externalizing problems. These findings are in contrast to previous studies showing a link between parental depression and externalizing problems both in very young boys (Campbell et al., 1991) and in adolescent boys and girls (Chassin et al., 1991). However, it is possible that parental depression is only indirectly related to child outcome through other more proximal variables, such as the quality of the family environment. For instance, analyses from the current study showed that parental depression was correlated with more family conflict, which in turn was directly predictive of the son's later outcome. This finding is consistent with theory and previous research suggesting that children of depressed parents may be exposed to parental negativity and unresponsiveness (Harnish, Dodge, & Valente, 1995), which may contribute to increasing family conflict and in turn lead to child maladjustment (Patterson, 1982). However, the mediational role of family conflict was not directly tested and thus, further research should be conducted to determine the factors that may mediate or account for the relation between parental depression and child outcome.

Other findings showed that parental level of education was positively associated with parental antisocial behavior and with paternal lifetime alcohol problems. Parents with lower levels of education were also likely to experience more problems related to drinking and to exhibit more antisocial behavior. Results did not, however, support the relation between parent education and any of the putative mediator variables, contradicting previous research showing the importance of familial demographic factors to child outcome (see Fitzgerald et al., 1995). Perhaps parental education alone cannot adequately assess the influence of familial sociodemographics on child outcome instead, it is likely that variables such as parental occupation and income must be included to obtain a clearer understanding of the role of familial demographic climate in the behavioral development of young male children. Alternatively, education may have an impact on child outcome only indirectly, through its influence on other more proximal factors such as parental alcoholism and antisocial behavior. Nonetheless, current findings parallel previous research showing that in comparison to other contextual factors, socioeconomic factors are relatively weak predictors of child externalizing problems (Shaw et al., 1996).

Results presented thus far indicate that parent alcoholism does not make a direct contribution to any of the contextual factors we examined. Rather, parental antisocial behavior predicted family conflict, father–child conflict, and current level of parent depression, over and above the effects of parental lifetime alcohol problems. Although unexpected, these findings are consistent with socialization theories suggesting that individuals with high levels of antisocial behavior are likely to be unskilled parents who experience elevated levels of distress and provide their children with environments that are conflictual and coercive (Patterson & Capaldi, 1991).

Paternal alcoholism did, however, play a central role in augmenting risk for child maladjustment. Paternal alcoholism was positively associated with deficits in the son's emotional and attentional control and in turn, this collection of variables predicted elevated rates of subsequent externalizing problems. Thus, child lack of control mediated paternal alcoholism effects on child externalizing problems. Children who display deficits in emotional and attentional control may be emotionally dysregulated and for this reason may be more prone than their counterparts to act out. Emotional dysregulation involves problems in modulating emotional experience and expression and has been previously implicated in the development of child psychopathology (see Cole et al., 1994). However, a substantial body of research indicates that child variables alone do not sustain emotional and behavioral difficulties but rather, maladjustment is most likely in circumstances where individual-level risk factors are nested

within risky environments (Zucker et al., 1995; Wong, Zucker, Puttler, & Fitzgerald, 1999). Importantly, elements of undercontrol have also been implicated in the development of later alcoholism (Tarter & Vanyukov, 1994). Consequently, boys who exhibit these individual differences and who live in environments characterized by family conflict, father–child conflict, and parental psychopathology may be at highest risk for subsequent maladjustment.

Paternal alcoholism was also predictive of the son's baseline levels of externalizing problems corroborating research showing that parental alcoholism raises risk for a diagnosis of conduct disorder in children and adolescents (Reich, Earls, Frankel, & Shayka, 1993; Kuperman, Schlosser, Lidral, & Reich, 1999). Although it could not be tested in the current study, it is likely that heritable individual differences, in part, underlie the relations between parental alcoholism and child lack of control as well as child externalizing problems. Findings from physiological studies as well as those from twin and adoption studies have demonstrated that genetic and biological mechanisms play an important role in these relations (see Iacono, 1998). Findings from our study as well as those reported by Reich and her colleagues (1993) and by Kuperman and his colleagues (1999) are in direct contrast to research showing that parental ASPD, and not alcoholism, is specific to the development of adolescent externalizing problems (Chassin et al., 1991). Inconsistencies across studies may be due to methodological differences including the measurement of different child outcomes (e.g., conduct disorder versus externalizing behavior problems) as well as differences in age and gender of target children/adolescents assessed.

Results from the maternal model support the specificity of parental psychopathology and revealed that the association between maternal lifetime antisocial behavior and her son's baseline levels of externalizing problems was unique, and existed over and above the effects of maternal alcoholism. The effect of maternal antisocial behavior on sons' lack of control further supports the specificity hypothesis and shows that sons of women with elevated levels of antisocial behavior are at risk for deficits in emotional and attentional control. Contrary to what was hypothesized, child lack of control did not mediate the associations between either of the maternal lifetime psychopathologies and the son's later externalizing problems. These results suggest that maternal reports of child lack of control are not associated with the development of externalizing behavior problems over the interval from preschool to middle childhood. These results are in contrast to those found in the paternal data and indicate that for mothers, contextual factors (family conflict and parent–child conflict) are better predictors of her son's later externalizing problems than is his level of undercontrol.

The current findings underscore the differential influence of maternal and paternal psychopathology on child emotional and behavioral problems. Whereas paternal alcoholism played a central role in child lack of control as well as in child externalizing behavior problems, it was maternal antisocial behavior that uniquely predicted these child variables. The discrepancy of parental psychopathology effects was not hypothesized. However, it must be noted that mothers in the current sample experienced fewer problems related to alcohol use than did fathers and were less likely than were their spouses to be diagnosed with alcoholism. Therefore, it is possible that maternal alcohol problems are not as salient as are the father's to the son's attributes, particularly in cases where only the father is diagnosed as alcoholic (the epidemiologically most common situation). Given this argument, why then is maternal antisocial behavior uniquely associated with the child variables, even though fathers in the current sample are more antisocial than are mothers? One potential explanation is that because mothers spend substantially more time than do fathers in direct interaction with their 3- to 5-year-old boys (Fitzgerald, Zucker, Maguin, & Reider, 1994), any expression of antisocial behavior is sufficient to directly influence the behaviors of their young sons, either through maternal modeling of such behaviors or through maternal reinforcement of inappropriate child behaviors (or both).

Given the conflicting findings across studies and across maternal and paternal models, further research is warranted to determine the relative roles of parental alcoholism and antisocial behavior in boys' externalizing behavior problems. Future research should examine the effects of living in a home where both parents are alcoholic or, alternatively, where one parent is alcoholic and the other meets criteria for a different diagnosis (e.g., depression). In the current study, almost half of all women married to alcoholic men were alcoholic themselves. Although this high rate of maternal alcoholism is not unexpected given the tendency for alcoholic women to marry alcoholic men (Hall, Hesselbrock, & Stabenau, 1983), not all alcoholic men are coupled with alcoholic women. The fact that a high proportion of the families in the present sample are composed of two alcoholic parents does have implications for the family environment and for child outcome. In particular, existing evidence indicates that families where both parents are alcoholic provide their children with rearing environments that are particularly risky (see Windle & Tubman, 1999; Wong et al., 1999). Such families appear especially likely to be characterized by familial violence and by poor parenting skills (Zucker et al., 1996). Given

the substantial variation in comorbidity found among both male and female alcoholics, it appears essential, for future studies to pay close attention to the comorbid occurrence of alcoholism and other parental psychopathologies, most notably ASPD and depression, and their moderating effects on child outcomes (e.g., Zucker et al., 1995).

Although the current research improved upon previous studies by assessing the unique effects of parental antisocial behavior on the externalizing behavior problems of young male COAs and non-COAs, by formally testing the mediational role of a number of variables in this relation and by using a prospective research design, it still has limitations. First, we examined the outcomes of young male COAs because they are at heightened risk for a variety of later problems (Sher, 1991). And although both genetic and environmental factors play a role in this risk (McGue, 1997; Iacono, 1998), the current study could not address the impact of genetic vulnerability upon the development of boys' externalizing problems. Second, study results are limited to young White male COAs and thus cannot be directly generalized to the population of female or non-White male children with alcoholic parents. However, previous research including the siblings of the target boys in the current sample has revealed few differences between young female and male children on a variety of behavioral indicators (see Wong et al., 1999). Finally, although information regarding the child and his environment was gathered from both mothers and fathers and these data were analyzed separately so that the unique assessment of each parent could be retained, the single-rater design of the models and the effects of shared method variance give rise to the possibility that the obtained relations are inflated. Thus, prior to drawing firm conclusions from the current study, findings should be replicated utilizing information from multiple sources including the children themselves, teachers, and peers.

In summary, the current study demonstrated that paternal alcoholism and maternal antisocial behavior were associated with boys' baseline levels of externalizing problems, which tended to remain stable from the preschool to the early school years. Paternal alcoholism and maternal antisocial behavior were also positively associated with child lack of control and according to paternal reports, this individual difference variable was predictive of increased rates of subsequent externalizing problems. Moreover, boys living in homes characterized by parental antisocial behavior had parents with elevated levels of depression, experienced more family conflict, and more parent–child conflict. In particular, family conflict was a significant mediator of the relation between parental antisocial behavior and sons' later externalizing problems and father–son conflict mediated paternal antisocial behavior effects on sons' later maladjustment. Thus, boys living in alcoholic or antisocial homes (or both of these) are at elevated risk for displaying risky individual-level characteristics and for living in risky enviornments characterized by conflictual familial interactions. Boys may learn through repeated observations to also use conflictual and coercive behaviors and thus, even as young as 3 years of age are at risk for elevated levels of externalizing behavior problems. Previous research suggests that the longer the child remains in such an environment, the more likely it is that the antisocial behaviors will be maintained and possibly develop into other deviant or even illicit behaviors (Patterson, 1982; Zucker et al., 1995). Specifically, through both modeling and genetic influences, these boys may be at elevated risk for the early initiation and problematic use of alcohol (McGue, 1997).

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