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Externalizing Symptoms Among Children of Alcoholic Parents: Entry Points for an Antisocial Pathway to Alcoholism

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The authors examined heterogeneity in risk for externalizing symptoms in children of alcoholic parents, as it may inform the search for entry points into an antisocial pathway to alcoholism. That is, they tested whether the number of alcoholic parents in a family, the comorbid subtype of parental alcoholism, and the gender of the child predicted trajectories of externalizing symptoms over the early life course, as assessed in high-risk samples of children of alcoholic parents and matched controls. Through integrative analyses of 2 independent, longitudinal studies, they showed that children with either an antisocial alcoholic parent or 2 alcoholic parents were at greatest risk for externalizing symptoms. Moreover, children with a depressed alcoholic parent did not differ from those with an antisocial alcoholic parent in reported symptoms. These findings were generally consistent across mother, father, and adolescent reports of symptoms; child gender and child age (ages 2 through 17); and the 2 independent studies examined. Multialcoholic and comorbid-alcoholic families may thus convey a genetic susceptibility to dysregulation along with environments that both exacerbate this susceptibility and provide few supports to offset it.

Keywords: externalizing symptoms, parent alcoholism, integrative analysis, child psychopathology, high-risk development

In the study of alcohol disorders, the recent inclusion of a developmental perspective has encouraged the search for pathways of risk that define early antecedents and intervening mechanisms culminating in adult alcoholism. One of the most widely acknowledged pathways recognizes the central role of externalizing behavior in the development of early-onset alcoholism (Sher, 1993; Zucker, 2006). Variants of the antisocial pathway strive to explain the widely replicated finding that externalizing behaviors are a robust predictor of later alcohol involvement, abuse, and disorder (Zucker, 2006). Posited early precursors (or perhaps heterotypic indicators) of antisocial alcoholism in this pathway include temperamental difficulties and behavioral dysregulation as well as neurobiological deficits and maturational delays (Tarter et al., 1999). Rarely considered are factors present even before conception that identify a potential intergenerational transmission of risk.

Previous studies consistently have reported elevated externalizing symptoms among children of alcoholic parents (COAs), which makes this an important risk group in which to study the emergence and development of the antisocial pathway (Chassin, Rogosch, & Barrera, 1991; Puttler, Zucker, Fitzgerald, & Bingham, 1998). However, few studies have focused on the notable heterogeneity in risk among COAs or have considered how this risk unfolds over the first 2 decades of life, a key period of ontogeny just preceding the observed peak risk for alcoholism onset in young adulthood (Kessler et al., 2005). In the current study, we examine the relation between parental alcoholism and developmental trajectories of externalizing symptoms from ages 2 through 17, focusing on indicators of risk heterogeneity among COAs, as

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they may inform our understanding of intergenerational influences on an antisocial pathway for alcoholism.

Markers of Heterogeneity

Although the findings are not always consistent, previous studies have indicated that parental alcoholism may be a unique predictor of child externalizing symptoms after controlling for comorbid parental depression and antisocial personality disorder (ASPD; Loukas, Fitzgerald, Zucker, & von Eye, 2001; Loukas, Zucker, Fitzgerald, & Krull, 2003; although see Chassin et al., 1991). Moreover, children whose alcoholic parents also have ASPD show greater externalizing symptoms than children whose alcoholic parents do not (Zucker, Ellis, Bingham, & Fitzgerald, 1996). These findings support the relevance of parental alcoholism, and heterogeneity within COAs in particular, for understanding early patterns of child externalizing symptoms.

One potential marker of heterogeneity, comorbidity in alcoholic parents, distinguishes the two most consistently recognized subtypes of adult alcoholism, namely, antisocial alcoholism and depressive alcoholism (Zucker, 1994). In early to middle childhood, offspring of antisocial alcoholics show greater risk for externalizing symptoms as compared with children of nonantisocial alcoholic parents and children of nonalcoholic parents (Puttler et al., 1998; Wong, Zucker, Puttler, & Fitzgerald, 1999). Given that ASPD is rarely observed in the absence of alcoholism, antisocial alcoholism may be viewed as a component of antisociality rather than as a subtype of alcoholism (Zucker, 2006; Zucker, Ellis, Fitzgerald, Bingham, & Sanford, 1996). Thus, children of antisocial alcoholic parents may realize greater externalizing symptoms over time through those mechanisms implicated in the intergenerational transmission of antisocial behavior more broadly. These mechanisms include a heightened genetic liability for early conduct problems as well as cognitive deficits and high-risk environments characterized by such factors as greater family conflict, poor parent–child interactions, and maltreatment (Arseneault et al., 2003; Jaffee et al., 2005; Wong et al., 1999). These factors are suggested to underlie the emergence of a psychopathological form of antisocial behavior that may be difficult to distinguish cross-sectionally from the typical rise in antisocial behavior marking adolescence (Moffitt, 1993). Whether children of antisocial alcoholics continue to show greater externalizing symptoms than children of nonantisocial alcoholics during adolescence is unclear.

However, little evidence supports predictions about temperamental differences in the offspring of parents with differing subtypes of parental alcoholism (Sher, 1993). Thus, it is not clear that comorbid subtypes of alcoholism "breed true" (i.e., that children are most at risk for the subtype of alcoholism evident in their parents), which suggests that the greatest risk for externalizing symptoms may not be limited to children of antisocial alcoholic parents. It is notable that children of depressed alcoholic parents may share this risk. Studies of depressed mothers suggest that these children also experience greater externalizing symptoms, with difficult temperament, insecure attachment, maladaptive child-rearing practices, and exposure to distress serving to potentially mediate this risk (Cole & Zahn-Waxler, 1992; Zahn-Waxler, Iannotti, Cummings, & Denham, 1990). The extent to which children of parents with depressive alcoholism also show greater externalizing than children of parents with nondepressive alcoholism is currently unclear. A finding of equivalent risk for externalizing symptoms in children of antisocial and depressive alcoholic parents is important because it suggests that multiple entry points may lead children into an antisocial pathway for alcoholism.

A second marker of heterogeneity in COAs' risk for externalizing symptoms is the number of alcoholic parents in the family. Because of assortative mating (i.e., the tendency for individuals with alcoholism to marry one another; Maes et al., 1998; particularly in COAs; Boye-Beaman, Leonard, & Senchak, 1991) and lower base rates of alcoholism in women than in men (Grant et al., 2004), it is often difficult in practice to isolate the effect of having two alcoholic parents from that unique to maternal alcoholism. As such, children with two alcoholic parents rather than one may show greater externalizing symptoms because the primary caretaker is more likely to be affected; the familial stress load and dysfunction within the home are heightened (Chassin et al., 1991; Hussong & Chassin, 2004); and a potentially protective, nonaffected parent is absent (Werner, 1986; although this influence is not always supported; Curran & Chassin, 1996). In support of this hypothesis, children with two alcoholic parents show greater internalizing symptoms and neurobehavioral disinhibition and lower social competence than those with a single alcoholic parent as early as 3 years of age (Clark, Cornelius, Kirisci, & Tarter, 2005; Hussong, Flora, Curran, Chassin, & Zucker, in press; Hussong, Zucker, Wong, Fitzgerald, & Puttler, 2005).

Child gender may be a third marker of risk heterogeneity among COAs. Boys are more likely to display physical forms of aggression than are girls beginning in early childhood (Moffitt, Caspi, Rutter, & Silva, 2001; Silverthorn & Frick, 1999). Moreover, converging studies have suggested that boys may be more sensitive to the effects of family-related stress than are girls. That is, studies of divorce, family conflict, maternal depression, and nonresponsive caregiving have shown more negative effects of these family stressors on externalizing symptoms in boys than in girls (Dadds, Atkinson, Turner, Blums, & Lendich, 1999; Essex, Klein, Cho, & Kraemer, 2003; Malone et al., 2004; Martin, Maccoby, & Jacklin, 1981; Shaw, Keenan, & Vondra, 1994; Shaw et al., 1998). The extent to which this sensitivity to family-related stress also results in greater externalizing in male versus female COAs is unclear.

The Current Study

In the current study, we examined heterogeneity in COAs' risk for externalizing symptoms over the early life course as related to comorbid subtypes of parental alcoholism, the number of alcoholic parents in the family, and child gender. Moreover, we tested whether the number of alcoholic parents and child gender are unique markers of heterogeneity in COAs' risk for externalizing symptoms beyond parent comorbidity. Using an integrative analysis framework (Curran & Hussong, 2007), we conducted simultaneous analyses of two independent, longitudinal, high-risk studies that together assess a large sample of COAs and matched controls from ages 2 through 17. The studies contributing to our analysis have several methodological strengths that lend confidence to our pursuit of an integrative approach, including the use of a community-based sampling strategy, recruitment of matched

controls, multiple reporters of symptomatology, and direct ascertainment of parental alcoholism. Thus, a final contribution of this study is the demonstration of a multiphase approach to conducting integrative analyses.

Method

Samples and Procedures

The two studies contributing to the current analyses each used a longitudinal, high-risk design in which COAs and controls with nonalcoholic parents were assessed repeatedly. The Michigan Longitudinal Study (MLS) used a rolling, community-based recruitment process to assess three cohorts of children from families with alcoholic parents as well as children from matched, contrasting families without an alcoholic parent (Zucker et al., 2000). In Cohort 1, 338 boys (262 COAs and 72 controls), initially ages 2–5, and their parents completed a series of in-home interviews.[1] COA families were identified through court-arrest records for male drunk drivers (with a minimum blood alcohol concentration of 0.15% at first arrest or 0.12% if multiple arrests) as well as through community canvassing. Inclusion criteria for COA families were that fathers meet Feighner et al.'s (1972) diagnostic criteria for alcoholism during adulthood on the basis of self-reports, reside with their biological son ages 3–5, and be in an intact marriage with their son's biological mother at the time of first contact and that sons show no evidence of fetal alcohol syndrome. Contrast families were recruited through community canvassing in the neighborhoods in which COA families resided and were matched to COA families on the basis of age and sex of the target child and parallelism of community characteristics; both parents of controls had to be free of lifetime alcohol and drug disorders. Assessment waves involving both parents and the children were at 3-year intervals.

Cohort 2 consisted of girls from the Cohort 1 families who were recruited when Cohort 1 boys were at Wave 2. Because Cohort 1 inclusion criteria indicated that families have at least one male child and put no restrictions on other children, these families had fewer girls. To provide age parallelism with Cohort 1 when possible and to begin assessments at ages 3–5, we used a broader age range to recruit girls. One target girl per family was enrolled if she was ages 3–11, with those ages 3–5 receiving the Wave 1 battery, those ages 6–8 receiving the Wave 2 battery, those ages 9–11 receiving the Wave 3 battery, and (at follow-up) those ages 12–14 receiving the Wave 4 battery.

Similarly, Cohort 3 contained all additional siblings of the male target child in Cohort 1 who were ages 3–11 at the time of data collection, with assessment batteries structured by age as for Cohort 1. The siblings in Cohorts 2 and 3 were reassessed in all subsequent waves of data collection and received measures that paralleled those given to the male target children in Cohort 1 on the basis of age of assessment. Because children in Cohorts 2 and 3 were recruited later in time and could enter the study at older ages, fewer waves of data were collected from these participants by design. A total of 152 girls (from 152 families) comprised Cohort 2, and an additional 106 siblings (from 84 families) comprised Cohort 3.

Across all three cohorts, 596 children from 338 families provided four waves of data, separated by 3-year intervals. A total of 399, 339, 402, and 418 participants had reports on their functioning available at Waves 1–4, respectively, which yielded an overall participation rate of 73% for those with at least two waves of data in the sample (see Zucker et al., 2000). These data were augmented by annual assessments completed by participating children (but not parents) beginning at age 11 and ranging up through age 17 (for the current study).

Each family completed a primarily in-home assessment conducted by trained staff who were blind to family diagnostic status. Although protocol length varied by wave of assessment, parent assessments typically involved 9–10 hr of data collection, and child assessments were typically 7 hr (except for annual interviews, which took 1 hr), each spread over seven testing sessions. Families were compensated $300 for their involvement if the assessment was carried out on a one-child family and $375 if two children were involved. Seventy percent of eligible court families and 93% of community-canvassed families agreed to participate (overall participation rate was 84%).

In the Adolescent/Adult Family Development Project (AFDP; Chassin et al., 1991), 454 adolescents and their parents from 454 families completed repeated, computerized, in-home interviews. Of these, 246 included a biological and custodial alcoholic parent, whereas 208 were matched controls. COA families were recruited by means of court records ($n = 103$), wellness questionnaires from a health maintenance organization ($n = 22$), and community telephone surveys ($n = 120$). Inclusion criteria for COA families were that they be of Hispanic or non-Hispanic Caucasian ethnicity, reside in Arizona, have a 10.5–15.5-year-old adolescent, speak English, lack cognitive limitations that would preclude an interview, and include a biological and custodial parent who met *Diagnostic and Statistical Manual of Mental Disorders* (3rd ed.; *DSM–III;* American Psychiatric Association, 1980) lifetime criteria for alcohol abuse or dependence. Lifetime presence of parental alcoholism was determined through diagnostic interviews with parents via the Diagnostic Interview Schedule Version II (DIS; Robins, Helzer, Croughan, & Ratcliff, 1980) or through spousal report via the Family History Research Diagnostic Criteria (if the alcoholic parent was not interviewed; Feighner et al., 1972). Matched control families were recruited by phone screens of families identified through reverse directory searches based on identified COAs. Control families matched COA families on the basis of ethnicity, family composition, target child's sex and age, and socioeconomic status. Direct interview data confirmed that neither biological nor custodial parents met criteria for a lifetime alcoholism diagnosis. Recruitment biases have been found to be minimal (Chassin, Barrera, Bech, & Kossak-Fuller, 1992; Chassin et al., 1991). Although contact rates were low (38.3% from archival records and 44.2% from reverse directories), participation rates were high (72.8% of eligible COA families and 77.3% of eligible control families participated). No recruitment biases were found for alcoholism indicators (available in archival data), although lower participation rates were found among families with lower socioeconomic status and Hispanic families.

[1] Although 3-year-olds were targeted as the lower bound for study recruitment, because of assessment scheduling issues, 6 boys were assessed shortly before their 3-year birthday.

These families were initially interviewed when the adolescents were ages 11–15 (Wave 1) and reinterviewed on an annual basis when the adolescents were ages 12–16 (Wave 2) and 13–17 (Wave 3). Sample retention has been high, with 97% interviewed at all of the first three waves (for details, see Chassin et al., 1992). Adolescents and parents completed computer-based interviews separately on each occasion, and each received up to $65 for participation.

Because analyses used the accelerated longitudinal structure of these aggregate data (see Mehta & West, 2000), the mother-, father-, and adolescent-report samples are described with respect to the underlying age distribution rather than assessment waves (see Figure 1). Across MLS and AFDP, at least one assessment was available for 1,050 adolescents.[2] We created three samples to examine effects for each reporter of externalizing symptoms on the basis of the availability of complete parental psychopathology data and at least one report of symptoms between ages 2 and 17 (or between 10 and 17 for adolescent reports). These criteria resulted in a sample of 991 children from 748 families for mother-reported externalizing symptoms, 925 children from 700 families for the father-report sample, and 829 children from 608 families for the adolescent-report sample. These three samples were 63%–65% male, 12%–13% ethnic minority (primarily Hispanic), and 63%–67% COA; 7%–9% of families had parents with less than a high school education, and 27%–29% had parents with at least a college degree (Table 1). Analyses indicated some differences between retained and excluded cases on parental alcoholism, parental education, child ethnicity, child gender, and study membership. However, the use of missing data techniques that permitted the inclusion of cases with even a single observation reduced further potential bias.[3]

Measures

Demographic variables included child gender, age, and ethnicity, assessed by adolescent report when available and otherwise by parent report. Parents also reported on their educational attainment (maximum of either parent's educational status, assessed through parental report on a 6-point scale ranging from 0 = *less than 12 years or not a high school graduate* to 5 = *graduate or professional school training*).

Parental alcoholism was assessed by parent report in both studies.[4] In MLS, parental alcohol use disorder at Wave 1 was assessed by the DIS (Robins et al., 1980), the Short Michigan Alcohol Screening Test (Selzer, Vinokur, & van Rooijan, 1975), and the Drinking and Drug History Questionnaire (Zucker, Fitzgerald, & Noll, 1990). On the basis of information collected by all three instruments, a lifetime diagnosis was made by a trained clinician using *DSM–IV* (American Psychiatric Association, 1994) criteria. In each subsequent wave, past-3-year diagnoses were made. Interrater reliability for the diagnoses was excellent ($\kappa = .81$).

In AFDP, parents completed assessments for lifetime alcoholism at Wave 1 and for past-year drinking- and alcohol-related consequences at Waves 2 and 3. At Wave 1, biological parents were directly interviewed via a computerized version of the DIS to assess diagnostic status with *DSM–III* lifetime criteria. (Families in which parents were not directly interviewed were omitted from current analyses because parent comorbid diagnoses were not available.) For Waves 2 and 3, we created proxy diagnoses on the basis of parental reports of drinking frequency and their experience of alcohol-related consequences and dependence symptoms that reflected *DSM–IV* criteria for alcohol abuse and dependence (using items from Mayfield, McLeod, & Hall, 1974; Sher, 1993). Parents who endorsed at least weekly drinking and experiencing either one of four abuse symptoms or any three of seven dependence symptoms in the past year were diagnosed as having a current (within the past year) alcohol disorder (see Hussong et al., in press, for details). In the current analyses, families in MLS and AFDP were assigned to the impaired group if either biological parent met criteria for alcohol abuse or dependence at any wave of assessment.

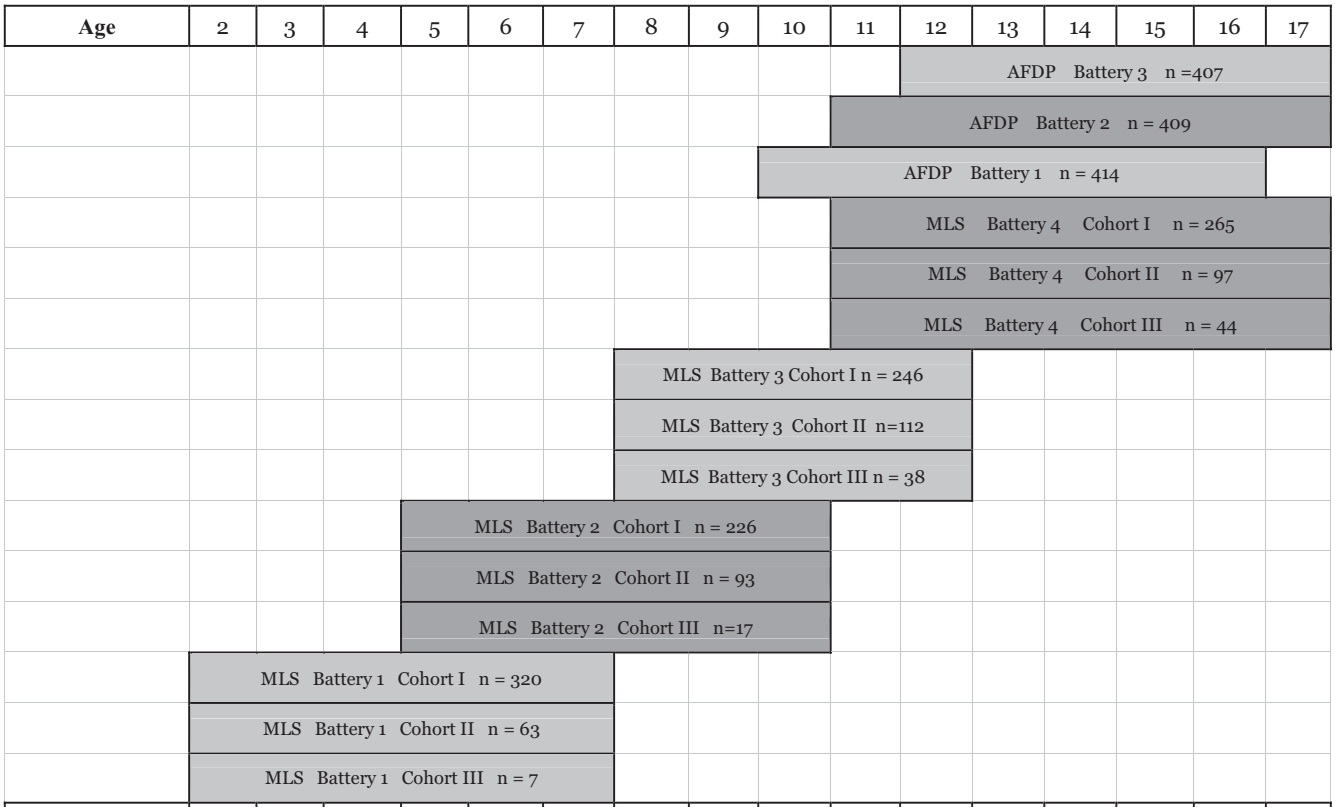
Parents' comorbid diagnoses were assessed via parent interview. Lifetime affective disorder (major depression or dysthymia) and ASPD were obtained by DIS interview in MLS and by the computerized DIS in AFDP. In AFDP, parents completed the DIS and received lifetime diagnoses of affective disorder or ASPD at Wave 1. In MLS, parents completed the DIS at each wave of assessment. The diagnosis of an affective disorder was based on meeting criteria at any assessment prior to the first wave of data collection for that child.[5] ASPD was based on Wave 1 only because this disorder, by definition, yields a lifetime diagnosis. The diagnosis was based on the DIS, supplemented by information provided by the 46-item self-report Antisocial Behavior Inventory (Zucker, Ellis, Fitzgerald, et al., 1996), which assesses the frequency of aggressive and antisocial activity in childhood and adulthood. For current analyses,

[2] We had a total of 154 of 2,713 observations on 991 cases with a single assessment in the mother-report analyses, 178 of 2,247 observations on 925 cases in the father-report analyses, and 30 of 2,822 observations on 829 cases in the child-report analyses. To evaluate the impact of including these cases in our analyses, we reestimated key models (Model 2 in Table 2 and the subtype analyses in Table 3) for each reporter with these cases eliminated. No substantive changes were noted in mother- or child-report analyses. In father-report analyses, the interaction between the first slope and parent depression dropped into the nonsignificant range (from $b = 0.05$ to 0.04), as did the estimate of the intercept ($b = -0.18$ to -0.14). Thus, changes were trivial and suggest that inclusion of cases with a single observation did not bias these findings.

[3] To test for effects of reporter differences resulting from sample membership, we reanalyzed key models (Models 2 and 3) using only participants who were in all of the three reporter subsamples ($n = 799$ participants from 608 families). No meaningful differences were noted, although the effects of parental depression on adolescent-reported externalizing symptoms became only marginally significant, and the comparison between having one alcoholic parent and having none became significant in predicting father-reported symptoms.

[4] In both studies, the parent of interest was the biological parent, regardless of residence. Given the inability of the current study designs to parse environmental and genetic risk, we consider this index the most appropriate for determining parental alcoholism, depression, and ASPD.

[5] Because parents could, for example, complete a lifetime assessment for their first child at Wave 1 and, subsequently, a past-3-year assessment for a second child entering the study at Wave 2, a diagnosis was given if the parent met criteria at any wave of assessment prior to that child's entry into the study. Thus, for each child, parental affective disorder was a child-level variable representing a lifetime diagnosis temporally precedent to the child's first wave of data collection.



Age	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
MLS n	16	137	118	95	124	114	103	154	135	113	153	119	102	11	1	0
AFDP n									28	98	169	242	265	226	137	53
Total n	16	137	118	95	124	114	103	156	163	211	322	361	367	237	138	53

Figure 1. Sample description by study, wave of assessment, and age. Sample frequencies refer to the mother-report analysis sample. AFDP = Adolescent/Adult Family Development Project; MLS = Michigan Longitudinal Study.

parental affective disorder and ASPD, respectively, were considered present if either biological parent received a diagnosis.

Child externalizing symptoms were assessed by mother, father, and adolescent reports. In each study, participants completed the Child Behavior Checklist (MLS parents) or Youth Self-Report (MLS adolescents) or an adapted form of these instruments (AFDP participants; Achenbach & Edelbrock, 1978). In the current study, we examined 30 items from the Child Behavior Checklist Aggressive and Delinquent Behavior subscales (defining a parallel set of items for boys and girls across the three reporters and the two studies). The response scale ranged from 0 to 2 for parental report and for self-report in MLS and from 0 to 4 for self-report in AFDP, with an assessment window of the past 6 months for MLS and the past 3 months for AFDP. (Differences in the assessment window for this instrument are part of the study effect, which was tested in all aspects of analyses.) For the current study, we chose to dichotomize items as absent (0) or present (> 0) because of sparse endorsement which introduced estimation problems and model instability.

Results

We used a multiphase approach to integrative analysis, simultaneously analyzing data from the two studies (Curran, Edwards, Wirth, Hussong & Chassin, in press; Hussong et al., in press). These phases concern measurement, trajectory estimation, and hypothesis testing through the pairing of item response theory (IRT; Thissen & Wainer, 2001) and mixed modeling (Raudenbush & Bryk, 2002) techniques. In particular, we used IRT to derive externalizing scale scores that optimized available data and were sensitive to item differences in severity, behavioral repertoire, and development. IRT has several advantages over traditional proportion scores (see Curran et al., in press) and provides a unique opportunity to consider two issues of particular importance to the study of externalizing behaviors. First, IRT permits differential weighting of individual behaviors as informed by overall patterns of item response. This is accomplished through the estimation of item-specific parameters that indicate the strength of the relation between the item and the construct being measured as well as the severity of the specific externalizing symptoms. This item-level

Table 1
Demographic Characteristics Within and Across Studies and Reporters

Variable	Mother report (n = 991)				Father report (n = 925)				Adolescent report (n = 829)			
	MLS	AFDP	Total	Exc.	MLS	AFDP	Total	Exc.	MLS	AFDP	Total	Exc.
% male	70.86	52.39	63.07	0.22	72.79	52.43	64.65	6.65*	70.40	53.50	63.69	0.35
% Hispanic or Black	1.75	27.03	12.41	21.58*	1.80	28.65	12.54	7.45*	1.80	27.05	11.82	11.24*
Parents' education:												
% with high school education or less	10.65	6.22	8.78	2.84a*	10.27	5.95	8.54	3.36a*	0.10	3.95	7.60	4.68a*
% college graduate	23.21	31.58	26.75		23.60	33.24	27.46		24.20	35.56	28.71	
% COAs	74.69	50.96	64.68	15.72*	74.05	53.78	65.95	0.08	75.00	54.10	66.71	0.66
% mother alcoholic	33.16	13.86	33.16	5.58*	32.79	9.73	23.57	2.42	32.60	9.73	23.52	2.11
% father alcoholic	72.43	44.74	72.43	13.63*	71.71	48.65	62.49	0.51	72.80	49.24	63.45	2.55
% two alcoholic parents	35.25	9.57	35.25		34.59	6.49	23.35		35.00	6.08	23.52	
% parental depression	31.76	15.07	31.76	0.02	30.99	15.68	24.86	0.19	32.20	16.72	26.06	3.59
% parental ASPD	20.24	8.85	20.24	2.19	20.18	8.92	15.68	2.38	18.40	9.12	14.72	0.39
% AFDP members			42.18	8.05*			40.00	33.20*			39.69	20.24*

Note. The columns with data on excluded cases (Exc.) report tests of statistic significance (chi-squares) comparing cases from the full sample of 1,050 that were excluded versus those retained in creation of each subsample. MLS = Michigan Longitudinal Study; AFDP = Adolescent/Adult Family Development Project; COA = child of an alcoholic parent; ASPD = antisocial personality disorder.
[a] This result is from a t test comparing those participants who were excluded versus retained on the continuous variable of parent education.
* $p < .05$.

information can be used to derive scale scores that take into account not only how many items were endorsed but which items were endorsed. Second, IRT permits tests of differential item functioning (DIF), which identify the extent to which items vary in their relation to externalizing symptomatology over subpopulations. When these scores are subjected to growth modeling analyses, we are able to consider developmental trajectories of externalizing symptoms that maximize meaningful variance in our data while also correcting for item variability along these dimensions.

Integrative Study Analysis Phase 1: Measurement

We first evaluated possible invariance resulting from study membership in our externalizing measure and used information about invariance to develop comparable scales that share a common metric across studies for each reporter. That is, we used IRT to evaluate DIF (Thissen, Steinberg, & Wainer, 1993) as a test of whether items functioned similarly in relation to the underlying construct of externalizing symptoms across important subgroups based on child age, gender, and study membership.[6] Next, we calibrated item parameters to determine the optimal approach to creating scale scores using a two-parameter logistic IRT model. Finally, we used the resulting parameters to estimate individual time-specific scores for each report of externalizing symptoms.

We used IRTLRDIF software (Thissen, 2001) to conduct sequential tests for DIF in each subgroup of interest. We initially examined whether items functioned differently across age (ages 2–11 vs. 12–17 for parent reports and ages 10–13 vs. 14–17 for adolescent reports), followed by gender and then study membership (i.e., MLS vs. AFDP). To do so, we relied on a calibration sample containing one randomly selected observation for each individual from among the repeated waves of assessment (ns = 1,026, 938, and 966 for mother, father, and adolescent reports, respectively).[7] DIF analyses then tested for group differences in either item severity or discrimination. Item severity is the level of the latent construct at which an individual has a 50% chance of endorsing a particular item; higher values denote items that require a child to engage in more externalizing behaviors before a participant is likely to endorse the item. Item discrimination (similar to a factor loading in factor analysis) describes the strength of the relation between an item and the latent construct. Because of the multiple tests involved in this procedure, we used the Benjamini–Hochberg adjusted chi-square tests to reduce potential for Type I error (Benjamini & Hochberg, 1995; see also Thissen, Steinberg, & Kuang, 2002). Significant parameter differences between groups were retained as subitems for subsequent DIF analyses (i.e., items with age DIF were split into two subitems, one for young participants and one for old participants, with the subitem not pertaining to a particular group coded as missing). This strategy allows for different IRT parameters to be used in scoring for items that operate differently across groups.

For mother-reported symptoms, 11 items showed age DIF, and 6 items each showed some form of gender or study membership DIF (with some items showing more than one form of DIF). For father-reported symptoms, 14 items showed age DIF, 12 showed gender DIF, and none showed study DIF. For adolescent-reported symptoms, 14 showed age DIF, 7 showed gender DIF, and none showed study DIF. On the whole, reporters varied considerably in the pattern of DIF. (Full results of DIF analyses are available from A. M. Hussong upon request.)

The resulting items and subitems (created to account for DIF) were then subjected to calibration and scoring procedures via

[6] We performed exploratory factor analyses to confirm that the scale was characterized by a dominant, unidimensional factor to meet assumptions of local independence for these models.

[7] Note that the calibration sample size was slightly larger than the analysis sample size because of the omission of cases in the analytic sample resulting from missing data on predictor variables.

MULTILOG (Thissen, 1991). Using the two-parameter logistic model, we estimated discrimination and severity parameters for all items and subitems and used these parameters to estimate maximum a posteriori (Thissen & Wainer, 2001) scores for each observation of externalizing symptoms for all waves and reporters. The resulting scores take into account differences in item parameters as a function of age, gender, and study as identified in DIF analyses and can be interpreted on a *z*-score metric. These scores served as the outcomes of interest in all subsequent analyses.

Integrative Study Analysis Phase 2: Constructing Trajectories

To model nesting of repeated observations within children sampled from the same family (in the MLS design), we used three-level mixed models for all trajectory analyses. We estimated all trajectory models separately for each reporter using restricted maximum likelihood as implemented in SAS's MIXED procedure (Littell, Milliken, Stroup, & Wolfinger, 1996), following strategies described in Singer and Willett (2003).

Our first step was to identify the optimal shape of externalizing trajectories within reporter through descriptive and iterative inferential tests. Mean IRT scores for externalizing within reporter and across ages suggested a decreasing pattern of externalizing behavior over time in parent reports and an increasing pattern in adolescent reports (see Figure 2). However, given the large developmental window and repeated assessments available in the current study, we also explored alternative functional forms of change as competing characterizations of the observed data. That is, we examined unconditional models in which time was modeled as a single linear decline (one-piece), two discontinuous linear trajectories (using multiple cutoffs; i.e., two-piece describing change between ages 2 and 7 and between ages 7 and 17 as distinct trajectories), and three discontinuous linear trajectories (for parent reports only) as well as a quadratic function. Potential models were compared visually via mean and individual trajectory plots, Bayesian information criterion and Akaike information criterion fit indexes, and chi-square difference tests (when available for nested models). On the basis of these criteria, the optimal functional form retained for mother- and adolescent-reported scores was a one-piece linear model, and for father-reported scores it was a two-piece linear model.[8] For fathers, the two-piece model delineated change from ages 2 to 7 and from ages 7 to 17 through two slope parameters, best reflecting the pattern of change evident in our data. Thus, patterns of change over time varied to some extent as a function of reporter, with the most striking difference in decreasing parent reports versus increasing adolescent reports.

Our final unconditional models examined change over time through each of these functional forms, with intercepts representing symptoms at age 13 across reporters, and estimated random variation in both the intercept and the slope parameters to account for individual variability in growth and levels of child externalizing.[9] (In the absence of interactions with age, main effects or predictions of the intercept represent a stable effect over time. When interactions with age were found, we probed alternative intercept coding to examine age differences in these effects.) Parameter estimates in the final model indicated significant, steady decreases in externalizing from ages 2 through 17 in mother-reported scores, steeper declines in externalizing for ages 2 through 7 than for ages 7 through 17 in father-reported scores, and steady increases over ages 10 through 17 in adolescent-reported scores. With the exception of nonsignificant random variation in the intercept for adolescent reports, significant random variation in both the intercept and the slope was found in models for all reporters, indicating individual variability in growth and levels of child externalizing symptoms.

Integrative Study Analysis Phase 3: Hypothesis Testing

Model 1: Externalizing trajectories conditioned on the number of alcoholic parents. To determine meaningful covariates for subsequent hypothesis testing, we regressed the random trajectories of externalizing symptoms on the three-way interaction among child age (as coded by reporter in unconditional models to reflect slopes), demographic variables (i.e., parents' education level, child's ethnicity, and child's gender), and study membership as well as on all contributing two-way interactions and main effects. Thus, these predictors tested for developmental changes in externalizing symptoms (i.e., slope effects), the main effects of demographic variables and changes in these effects over development (i.e., the interaction of age and demographic variables), study differences in change over time (i.e., the interaction of study membership and child's age), and study differences in both the effects of demographic variables (i.e., the interaction of study with demographic variables) and changes in these effects over time (i.e., the interaction of study, demographic variables, and child's age). As in all subsequent analyses, nonsignificant predictors ($p > .05$) were omitted for parsimony and model stability, although age and study participation (and their interaction) were retained regardless of significance because of their central role in integrative analysis. (Results did not differ substantively between trim and full models.) The resulting predictors were retained as covariates for subsequent model testing (and appear in Table 2 for each reporter).

For Model 1, we added two predictors to the covariate model to test the effects of having (a) one alcoholic parent versus none (36%, 34%, and 33% nonalcoholic families for mothers', fathers', and adolescents' samples) or (b) two alcoholic parents versus one (24%, 22%, and 25% two-parent families, respectively). Consistent with observed means (see Figure 2), mothers in the AFDP reported greater externalizing symptoms in their children than mothers in MLS did at age 13 ($b = 0.40$, $p = .005$), with

[8] Age was thus recoded as ranging from −11 to 4 for mother-report analyses and as ranging from −3 to 4 for adolescent-report analyses. For father-report analyses, two dummy variables representing age coded the two-piece functional form, with the first coded −5 to 0 to capture change from ages 2 through 7 (and 0 from ages 7 to 17) and the second coded −6 from ages 2 through 7 and −5 to 4 from ages 8 through 17 to capture change from ages 7 to 17.

[9] In addition to the random intercept and slope parameters, we also estimated the covariance between the random intercept and slope parameters and a time-specific residual. Given the relatively small number of families with multiple children with repeated assessments, only the family-level intercept was allowed to vary. The final baseline models for mother and child report each consisted of five total variance components, and that for father report consisted of eight components.

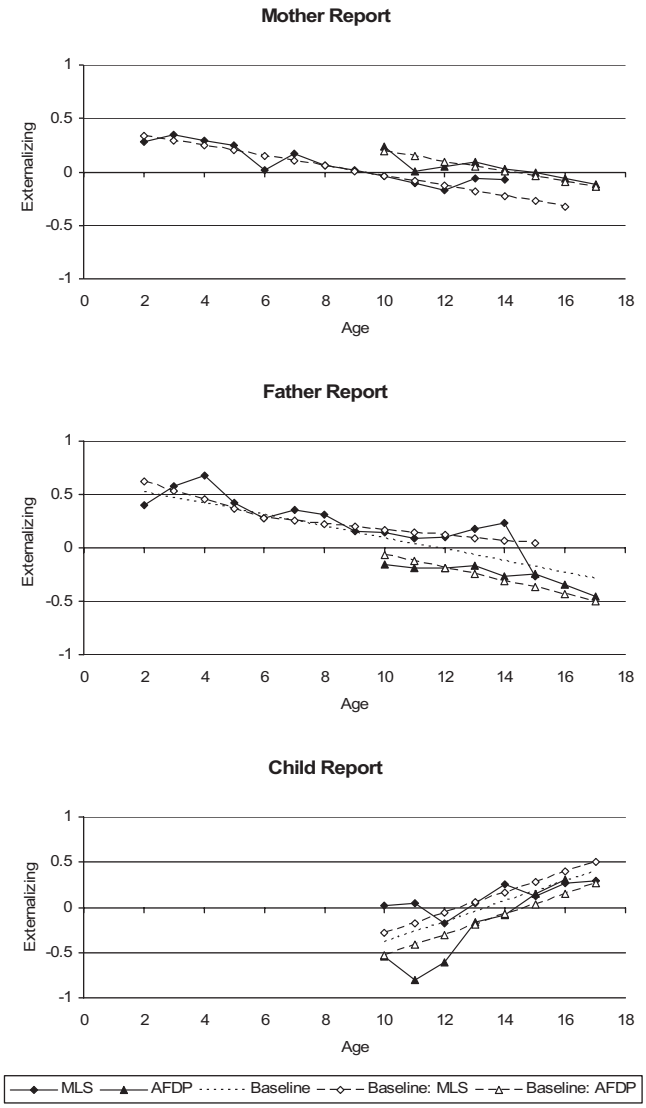


Figure 2. Unconditional fitted trajectories for externalizing symptoms. Solid trajectories indicate observed item response theory scores for externalizing symptoms over time, whereas dashed trajectories indicate estimated trajectories defined in the baseline model analyses. Ages with fewer than 12 observations are omitted from figures for simplicity. MLS = Michigan Longitudinal Study; AFDP = Adolescent/Adult Family Development Project.

symptoms in both studies decreasing significantly over time ($b = -0.05$, $<.001$; see Table 2).[10] Mothers reported greater externalizing symptoms in boys than in girls ($b = 0.27$, $p < .001$) and in children with lower parental education ($b = -0.07$, $p = .005$). Mother-reported symptoms were lower in children with no alcoholic parents versus those with one alcoholic parent ($b = -0.17$, $p = .007$) and significantly greater in children with two alcoholic parents versus those with one alcoholic parent ($b = 0.30$, $p < .001$).

Father-reported externalizing scores also decreased over time in both studies, with sharper decreases noted from ages 2 through 7 ($b = -0.08$, $p < .001$) than from ages 7 to 17 ($b = -0.03$, $p < .001$). Unlike mothers, fathers in the AFDP reported lower externalizing symptoms in their children ($b = -0.22$, $p < .001$) and nonsignificant change in symptoms from ages 7 to 17 ($b = 0.04$, $p = .03$) as compared with fathers in the MLS. In addition, fathers reported greater externalizing symptoms in boys than in girls ($b = 0.17$, $p < .001$). Consistent with mother reports, father-reported symptoms were lower for children with no alcoholic parents versus those with one alcoholic parent ($b = -0.17$, $p = .004$) and significantly greater for children with two alcoholic parents versus children with only one alcoholic parent ($b = 0.23$, $p < .001$).

In contrast, adolescent-reported externalizing symptoms increased significantly over time ($b = 0.06$, $p < .001$). This pattern differed over study and gender ($b = 0.13$, $p = .001$), such that girls showed increases in symptoms over ages 10 to 17, with stronger increases in the AFDP ($b = 0.28$, $p < .001$) than in the MLS ($b = 0.06$, $p < .001$). Similarly, AFDP boys showed significant increases over age in the AFDP ($b = 0.36$, $p < .001$), but MLS boys showed no significant change ($b = 0.01$, $p = .25$). We probed these gender differences by estimating a series of models in which the intercept was coded at each observed age within the sample. These analyses showed that, across studies, gender differences were apparent in younger adolescents (ages 10–13) and became increasingly nonsignificant with age. Moreover, adolescent-reported symptoms were lower in children with no alcoholic parents versus those with one alcoholic parent ($b = -0.26$, $p < .001$). However, an interaction between slope and the comparison of children in families with two alcoholic parents versus one ($b = 0.06$, $p = .003$) showed that children with two alcoholic parents reported greater externalizing symptoms than those with one alcoholic parent only at older ages (namely, 15.3 years and older).

Model 2: Controlling for comorbid parent disorder. To examine the unique effect of parental alcoholism on child externalizing symptoms, beyond the effects of parental comorbidity, we added indicators of parent depression and parent ASPD to Model 1 (Table 2). Across all three reporters, parental depression (bs = 0.13–0.20, $p < .05$) and parent ASPD (bs = 0.18–0.30, $p < .05$) predicted greater externalizing symptoms. For father reports, parental depression also predicted slower decreases in externalizing over ages 2 through 7 ($b = 0.05$, $p = .03$). For both mother and father reports, externalizing symptoms remained elevated in children with two alcoholic parents versus those with one after we controlled for comorbid parental disorder (bs = 0.27 and 0.21, $p < .002$, respectively), although no differences remained between those with only one alcoholic parent and those with no alcoholic parents. For adolescent reports, externalizing symptoms continued to differ between children with one alcoholic parent and those with none ($b = -0.22$, $p < .001$) and, at older ages, between those with two alcoholic parents and those with one ($b = -0.06$, $p = .003$).

[10] Because of differences across studies in age distributions, age and study effects appear partly confounded. However, we controlled for age and study differences simultaneously in these analyses, so that resulting study effects are unique from those for age. The overlap between ages 10 and 16 permits us to model these sources of influence separately in our analyses.

Table 2
Results of Mixed Models Testing Study Hypotheses

Predictor	Mother report (n = 2,713)			Father report (n = 2,425)			Adolescent report (n = 2,852)		
	Model 1	Model 2	Model 3	Model 1	Model 2	Model 3	Model 1	Model 2	Model 3
Intercept (age 13)	−.26*	−.42*	−.42*	−.06	−.18*	−.20*	.04	.04	−.05
First slope[a]	−.05*	−.05*	−.05*	−.08*	−.10*	−.10*	.06*	.06*	.06*
Second slope				−.03*	−.03*	−.03*			
Study membership (0 = MLS)	.40*	.44*	.44*	−.22*	−.18*	−.18*	−.33*	−.30*	−.29*
First Slope × Study	.03	.03	.03				.22*	.22*	.22*
Second Slope × Study				.04*	.03*	.03*			
Parents' education	−.07*	−.05*	−.05*						
Child's gender (0 = girl)	.27*	.27*	.26*	.17*	.16*	.18*	.16*	.16*	.18*
Child's Gender × Slope							−.05*	−.05*	−.05*
Child's Gender × Study							.01	.00	−.02
Child's Gender × Slope × Study							.13*	.13*	.13*
No versus one alcoholic parent[b]	−.17*	−.09	−.06	−.17*	−.09	−.07	−.26*	−.22*	−.22*
One versus two alcoholic parents[b]	.30*	.27*	.21*	.23*	.21*	.23*	.01	.00	.03
One Versus Two Alcoholic Parents × Slope[b]							.06*	.06*	.06*
Parental depression		.20*	.20*		.20*	.20*		.13*	.13*
First Slope × Parental Depression					.05*	.05*			
Parental antisocial personality disorder		.29*	.28*		.30*	.30*		.18*	.18*
No Versus One Alcoholic Parent × Child's Gender			−.05			−.03			.01
One Versus Two Alcoholic Parents × Child's Gender			.09			−.03			−.06

Note. Values are parameter estimates. MLS = Michigan Longitudinal Study.
[a] The first slope characterizes change over ages 2 through 17 for mothers' and adolescents' reports and 2 through 7 for fathers' reports; the second slope characterizes change over ages 7 to 17 in fathers' reports.
[b] Comparison group was those with one alcoholic parent for both the no versus one alcoholic parent and the one versus two alcoholic parents dummy codes.
* $p < .05$.

Model 3: Gender differences. To further explore the effects of the number of alcoholic parents on reports of child externalizing, we added interactions of child gender and dummy codes for the number of alcoholic parents to Model 2. These interactions were not significant across all reporters (Table 2).

Model 4: Comorbid subtypes of parental alcoholism. Finally, we considered subtype differences in the form of parent alcoholism by first classifying each parent into one of four categories: no alcohol diagnosis, an alcohol-only diagnosis (i.e., no comorbidity within the alcoholic parent for depression or ASPD), comorbid alcohol and depression diagnosis without ASPD (i.e., depressed subtype), or comorbid alcohol and ASPD diagnosis (i.e., antisocial subtype). Parents with all three diagnoses were classified into the antisocial subtype, because this group constituted a larger proportion of the ASPD than depressive subgroups. (When we classified these trimorbid cases under the depressed subtype, no differences in findings resulted.) Because these analyses aimed to disaggregate heterogeneous types of parental alcoholism, parents with depression (ns = 29 in mothers' reports, 28 in fathers' reports, and 23 in adolescents' reports) or ASPD (n = 2) but not alcoholism were considered controls, which thus provided a more conservative test of parental alcoholism risk.

On the basis of these categories for each parent's alcoholism, we then classified each child into one of four groups of families: control (i.e., no alcoholic parents; 37%, 34%, and 35% of families for mothers', fathers', and adolescents' samples, respectively), alcoholic only (i.e., one or both parents had an alcohol diagnosis but neither parent's alcohol diagnosis was comorbid; 41%, 43%, and 43% of families, respectively), depressed alcoholic subtype (at least one depressed subtype parent, but neither parent showed the antisocial subtype; 8% of families in all samples), and antisocial alcoholic subtype (at least one parent showed the antisocial subtype; 14%, 15%, and 14% of families, respectively). To probe differences among these four groups of participants in externalizing trajectories, we used both dummy-coded and effect-coded (linear contrast) variables in separate models (see Table 3).

Linear contrasts showed greater externalizing symptoms in COAs, regardless of parental comorbidity (i.e., across the three subgroups of COAs), as compared with controls across all reporters. Children in the comorbid-alcoholic families (either depression or ASPD) showed greater symptoms than children in the alcoholic-only families across reporters. However, children in antisocial alcoholic families did not differ from children of depressed alcoholic families on externalizing symptoms, regardless of reporter. Alternatively, dummy codes showed greater externalizing behaviors across reporters in children of antisocial alcoholic families compared with children of alcoholic-only families as well as in children with alcoholic-only families compared with controls. Children of depressed alcoholic families only showed greater externalizing symptoms than children of alcoholic-only families in parent reports (both mothers and fathers); adolescent reports did not differentiate these youths. In sum, these findings indicate the greatest risk for externalizing symptoms in children of alcoholic parents who show comorbidity, although the difference between depressive alcoholism and alcoholism alone was only significant in parent reports.

Table 3
Child Externalizing Symptoms Regressed on Subtypes of Parent Alcoholism

Predictor	Reporter		
	Mother (n = 2,713)	Father (n = 2,425)	Adolescent (n = 2,852)
Intercept (age 13)	−.28*	−.08	−.02
First slope[a]	−.05*	−.08*	.08*
Second slope		−.03*	
Study membership	.38*	−.23*	−.31*
First Slope × Study	.03		.21*
Second Slope × Study		.04*	
Parents' education	−.06*	.16*	
Child's gender	.26*	−.13*	.16*
Child's Gender × Slope			−.05
Child's Gender × Study			.00
Child's Gender × Slope × Study			.12*
Dummy code			
Controls vs. AO	−.14*	−.13*	−.22*
DA vs. AO	.29*	.23*	.12
AA vs. AO	.39*	.37*	.22*
Linear contrast[b]			
Controls vs. AO + DA + AA	35.22*	31.84*	33.34*
AO vs. AA + DA	23.85*	21.88*	7.11*
AA vs. DA	0.86	1.93	0.93

Note. Dummy and effect coding (linear contrasts) were included in separate analyses. Values are parameter estimates, except for results of linear contrasts, which are F values. AO = children of an alcoholic-only parent; DA = children of a depressive alcoholic parent; AA = children of an antisocial alcoholic parent.
[a] The first slope characterizes change over ages 2 through 17 for mothers' and adolescents' reports and 2 through 7 for fathers' reports; the second slope characterizes change over ages 7 to 17 in fathers' reports.
[b] Table values indicate parameter estimates, except for results of linear contrasts which are F values.
* $p < .05$.

Discussion

Through an integrative analysis of two longitudinal, community-based studies, we have identified meaningful sources of heterogeneity among COAs at risk for externalizing symptoms over the first 2 decades of life. That is, children in multialcoholic and comorbid-alcoholic families showed elevated levels of externalizing symptoms, although the pattern of risk varied somewhat by reporter and the child's age. The child's gender did not moderate COAs' risk for externalizing symptoms. These key findings were consistent across studies, thus providing an internal replication. Collectively, they define markers of heterogeneity among COAs, identifying a minority of youths who have either two alcoholic parents or a parent with comorbid alcoholism as showing the greatest risk for externalizing behavior. These findings also have implications for understanding early pathways of risk for alcoholism.

Multialcoholic Families

After we controlled for comorbid parental disorder, children with two alcoholic parents versus one showed greater externalizing symptoms over time in parent-reported symptoms. We found the same pattern only over ages 15 to 17 in adolescent-reported symptoms. Children with one alcoholic parent also showed greater adolescent-reported, although not parent-reported, symptoms over time compared with controls. Together, these findings suggest that COAs evidence greater externalizing symptoms across the early life span but that the added risk of having two alcoholic parents differs by reporter and age.

Particularly by adolescence, self-reports of antisocial behavior are considered more valid than reports of parents. Parents are clearly more cognitively sophisticated respondents than young children but are likely poorer reporters than adolescents, who often hide antisocial behavior from their parents. Our finding of decreasing trajectories of parent-reported externalizing symptoms but increasing trajectories of adolescent-reported symptoms is consistent with this observation. An additional source of potential bias is parental impairment, most notably parental depression (e.g., Youngstrom, Izard, & Ackerman, 1999). Consistent with the tendency for depressed parents to overestimate externalizing behavior in their children, we found that parental depression was a stronger predictor of parent-reported than of adolescent-reported symptoms. Such a reporter bias would result in exaggerated effects of parental depression and underestimated effects of model covariates on parent- versus adolescent-reported symptoms. This is consistent with our finding that children with one alcoholic parent versus none had greater adolescent-reported, but not parent-reported, externalizing symptoms after we controlled for parental depression. As such, reporter biases undermine confidence in parent reports of children's symptoms in those instances in which our findings vary by reporter. To be conservative, we focus on findings that replicate across reporter. With respect to our findings about multialcoholic families, we thus conclude that having at least one alcoholic parent increases risk for externalizing symptoms from an early age, but

the added risk posed by multialcoholic families does not consistently emerge until mid-adolescence.

The developmental timing of this increased risk may result from an interaction of familial and peer-based risk processes in adolescence. Peer-based risk processes, including the effects of social mimicry as well as peer encouragement and participation in deviant activities, have been linked to increased antisocial behavior in adolescence (Moffitt, 1993). Although they are sometimes viewed as causal factors that are more likely to impact normative rather than psychopathological deviance, these peer processes may also serve to increase involvement in antisocial behavior among those youths who are already engaging in a psychopathological form of deviance. This may be particularly true among youths who become increasingly peer oriented in adolescence as a way to escape family stress, conflict, violence, and abuse. These indicators of family-related stress may be particularly elevated in youths from multialcoholic families, who also lack the potential protective influence of an unimpaired parent. Thus, the confluence of familial and peer-based risks may escalate externalizing behavior in children of multialcoholic versus single-alcoholic families because of an increased deviant peer orientation fueled by a need to escape a more intensely chaotic and stressful home environment.

Comorbid-Alcoholic Families

Children in antisocial and depressed alcoholic families showed equivalent risk for externalizing symptoms, with children in antisocial alcoholic families showing greater risk than children in families of alcoholic parents without comorbid disorders. Children in depressed alcoholic families significantly differed from those in alcoholic-only families on parent-reported, not adolescent-reported, symptoms. Nonetheless, the pattern of findings across reporters indicates that children in depressed alcoholic families showed an intermediate risk in comparison with children in antisocial alcoholic and alcoholic-only families, with differences being weaker in adolescent reports. These group differences were consistent over time, suggesting that heightened risk for externalizing symptoms, particularly among children of antisocial alcoholic parents, is present early and persists through adolescence.

These findings are consistent with previous work showing greater externalizing symptoms in children of antisocial alcoholic versus alcoholic-only parents in childhood (Puttler et al., 1998; Wong et al., 1999) and provide evidence that this risk continues into adolescence. In addition, they are consistent with studies showing a strong familial pattern and genetic vulnerability for antisocial behavior and thus liken this form of alcoholism to one of many indicators of antisociality (Cadoret, Troughton, Bagford, & Woodworth, 1990; Zucker, 2006). Although genetic vulnerability has long been a recognized mechanism of risk for antisocial behavior, recent studies have identified important gene–environment interactions contributing to this risk (e.g., Arseneault et al., 2003; Jaffee et al., 2005). Two findings in the current study further support efforts to contextualize genetic vulnerabilities within environmental influences.

First, externalizing symptoms were equally elevated in children in antisocial and depressed alcoholic families. As such, parent antisociality did not stand alone as a marker of COAs' risk for externalizing symptoms, consistent with potential complexity in gene and Gene × Environmental mechanisms related to antisociality. Second, despite their greater risk for externalizing symptoms, not all children of antisocial alcoholic parents evidenced this risk. As depicted in Figure 3, 18% of children of antisocial alcoholic parents had externalizing trajectories that never surpassed the average levels of externalizing for the control sample of children. Thus, even in the absence of parent antisociality, greater risk for

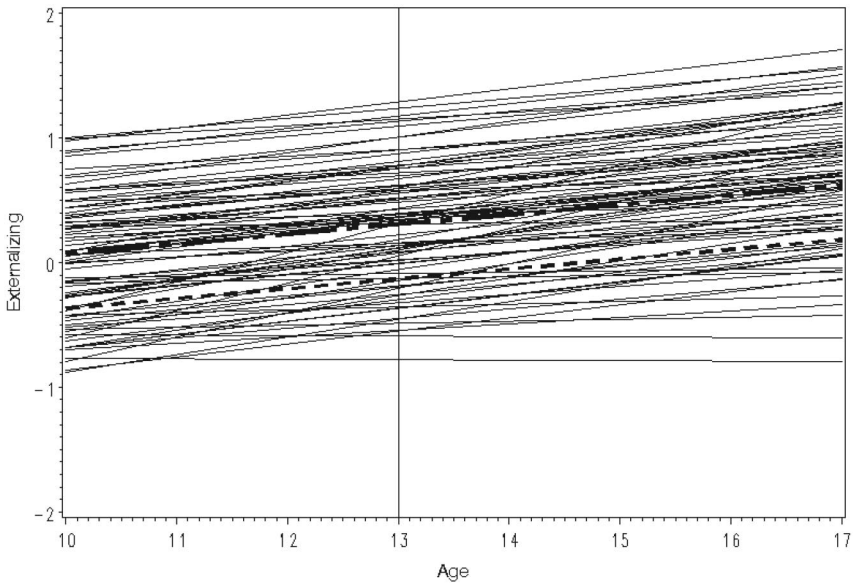


Figure 3. Comparison of individual model-implied trajectories (based on results for Model 4) for children of antisocial alcoholics (solid lines) versus group model-implied trajectories for children of antisocial alcoholics (dashed lines) and control participants (dotted lines).

externalizing symptoms was evident in families struggling with some form of parental disturbance, and even in the presence of antisociality, average or even low levels of externalizing symptoms were also observed. These findings suggest that some underlying diathesis–stress (or diatheses–stress) process, only imprecisely indexed by the antisocial alcoholism marker, is operating to produce these behavioral differences. Moreover, they also indicate that the study of parental alcoholism, and not simply antisocial alcoholism, may contribute uniquely to identifying children at risk for externalizing symptoms.

Implications for an Antisocial Pathway Toward Alcoholism

Although previous studies have shown a robust prediction of greater alcohol involvement from externalizing symptoms in children and adolescents (Zucker, 2006), these findings only partly inform our understanding of an antisocial pathway leading to alcohol abuse and dependence. The current study provides further support for the role of intergenerational transmission as marking an entry point to this pathway. It is notable that our finding of equivalent risk for externalizing symptoms in children of depressed alcoholic and antisocial alcoholic parents may tentatively suggest that subtypes of parent alcoholism are unlikely to breed true (although some specificity in risk resulting from parent alcoholism vs. other parental disorders has been shown; e.g., Chassin et al., 1991). Merikangas et al. (1998) showed that comorbidity, regardless of the co-occurring disorder (i.e., anxiety, mood, conduct, or antisocial behavior), predicted increased severity of substance use in adults with alcoholism or drug disorders. Similarly, severity of parent alcoholism may be more important than the form of comorbidity in determining COAs' risk for externalizing symptoms.

In this vein, comorbid-alcoholic and multialcoholic families may evidence risk via an inherited broad, underlying regulatory deficit that impacts not only externalizing symptoms but also social competence deficits, internalizing symptoms, and neurocognitive deficits previously found in these groups (Clark et al., 2005; Hussong et al., 2005, in press; McGue, Iacono, Legrand, & Elkins, 2001). This model has perhaps been best articulated for an antisocial pathway to alcoholism (Tarter et al., 1999; Zucker, 1994) and appears to be temperamentally mediated as well as affected by a nested high-stress environment, which is correlated with parent comorbidity. Multialcoholic and comorbid-alcoholic families may thus convey a genetic susceptibility to dysregulation along with environments that both exacerbate this susceptibility and provide few supports to offset it. However, these indicators of familial alcoholism are imperfect markers of risk. A primary task for future studies is to understand the mechanisms that account for this risk and thus explain the processes by which children enter and travel along an antisocial pathway to alcoholism.

Alternatively, children of antisocial alcoholic and depressed alcoholic parents may show similar levels of externalizing symptoms through different mechanisms related to their parents' comorbid disorders. This may, in part, be due to high associations between internalizing and externalizing symptoms that are evident by adolescence (Oland & Shaw, 2005). Similar to studies of parental antisociality (Cadoret et al., 1990), studies of depressed mothers have shown greater aggressive and oppositional behaviors in their children as compared with children of nonimpaired parents (Cole & Zahn-Waxler, 1992; Zahn-Waxler et al., 1990). It is not clear, however, whether children of antisocial alcoholic and depressive alcoholic parents follow similar or divergent pathways from this point of similar externalizing symptoms toward adult alcoholism.

Conclusions about the specificity between parent and child subtypes of alcoholism require a broad sampling of child outcomes. It is notable that other analyses of these data have shown that parents' depressive alcoholism was associated with an increased risk for children's internalizing symptoms as compared with parental antisocial alcoholism and alcoholism alone. Given the high correlation between internalizing and externalizing symptoms, particularly in adolescence, the question of specificity in the intergenerational transmission of alcohol subtypes requires the joint consideration of internalizing symptoms, externalizing symptoms, and, of course, alcohol disorders in offspring. Nonetheless, further research concerning an externalizing pathway to alcoholism should consider the relevance of depressed alcoholism as well as antisocial alcoholism as markers of initial risk in children for subsequent alcoholism via increased and persistent externalizing symptoms.

Conclusions

In sum, we found that children in multialcoholic families showed greater risk for externalizing symptoms that emerges at least by mid-adolescence and that children in comorbid alcoholic families showed a stable, early risk for greater externalizing symptoms compared with children in noncomorbid alcoholic families. These markers of risk heterogeneity among COAs were consistently supported in mother, father, and adolescent-reports, despite differences in the pattern of externalizing symptoms over time across reporters. We found no study differences concerning COA effects, which provides an internal replication of these findings.[11] These effects were also consistent across child gender. Although parents and adolescents (between 10 and 13 years old) reported greater externalizing symptoms in boys than in girls, the effects of parental alcoholism on child externalizing symptoms were the same across genders. Strengths of the current study lend additional confidence in these findings. These include careful attention to measurement in modeling trajectories of behavior over time, examination of two longitudinal samples with community-recruited risk and matched contrast participants, inclusion of both boys and girls and explicit testing of gender differences, incorporation of measurement invariance and consideration of multiple reporters in a large sample, high statistical power because of the combined analysis of two longitudinal studies, and use of a broad conceptualization of externalizing behaviors that (as our analyses evidence) is sensitive to differences in symptom expression across age and gender.

One limitation of the current study is potential bias in parent-reported symptoms. The ideal reporter of externalizing symptoms changes with development (see also Jester et al., 2005, for use of different reporters on the basis of variable content and arousal

[11] Although accounting for differences in our measurement structure by age and gender may seem to undermine predictions of change in externalizing over time based on these variables, our findings show little substantive change whether or not we account for DIF in constructing the externalizing scale.

context), although assessments of very young children preclude self-reports. The inclusion of additional sources (i.e., teacher reports, school records, arrest records) is thus an important consideration in understanding the development of externalizing symptoms over time. Our study is also limited by the inability to disaggregate genetic and environmental influences, a focus on patterns of risk rather than tests of specific risk processes or mechanisms, a limited number of items tailored to the youngest participants in our sample (which thus limits our ability to fully consider heterotypic continuity), and a small sample of antisocial alcoholic parents who did not manifest depression. (Given high rates of depression in antisocial alcoholics, as evidenced in this study, this final limitation is likely not specific to the current study.) Moreover, we are unable to account for the extent of the child's exposure to parental depression and alcoholism because of lack of specificity in our assessments.

These limitations all point to future directions for research, particularly in improving methods that address the role of heterogeneity in parental alcoholism as predicting children's externalizing symptoms. In addition, research is called for that examines mechanisms that account for children's risk associated with the imperfect markers of comorbid subtypes of parent alcoholism. Through examination of a broad array of child outcomes in concert with these three forms of alcoholism in parents, patterns of specificity and generality underlying individual variability in children's risk for various pathways leading to adult alcoholism may be illuminated.

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