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Parent Alcoholism Impacts the Severity and Timing of Children's Externalizing Symptoms

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Abstract

Although previous studies show that children of alcoholic parents have higher rates of externalizing symptoms compared to their peers, it remains unclear whether the timing of children's externalizing symptoms is linked to that of their parent's alcohol-related symptoms. Using a multilevel modeling approach, we tested whether children aged 2 through 17 showed elevated mother-, father- and child-reported externalizing symptoms (a) at the same time that parents showed alcohol-related consequences (time-varying effects), (b) if parents showed greater alcohol-related consequences during the study period (proximal effects), and (c) if parents had a lifetime diagnosis of alcoholism that predated the study period (distal effects). We used integrative data analysis to combine samples from two prospective studies to test these hypotheses. Distal effects of parent alcoholism on increased child externalizing symptoms were large and consistent. In addition, proximal and time-varying effects of parent alcohol symptoms were also found. Implications for preventing escalations in externalizing symptoms among this high-risk population are discussed.

Keywords

Parent alcoholism; Externalizing symptoms; Integrative data analysis; Intergenerational transmission; Time-varying effects

Previous studies show a well-established effect of parent alcoholism on children's externalizing symptoms (e.g., Eiden, Edwards, & Leonard, 2007; Hussong, Curran, & Chassin, 1998; Loukas, Fitzgerald, Zucker & von Eye, 2001; Loukas, Zucker, Fitzgerald & Krull, 2003). This effect is seen as early as age 2 (Hussong, Wirth et al., 2007) and extends into adulthood as disinhibited behavior (Sher, Walitzer, Wood & Brent, 1991). Multiple mechanisms may account for children of alcoholics' (COAs') increased risk for externalizing symptoms, with possible explanations ranging from the genetic to the neighborhood levels of analysis. Notably, behavioral genetic studies indicate that genetic variance associated with risk for alcohol use disorders is largely non-specific, reflecting a generalized risk for disinhibited behavior (Kendler, Prescott, Myers & Neale, 2003; King et al., 2009; Krueger, Hicks, Patrick, Carlson, Iacono, & McGue, 2002; McGue, Iacono, & Krueger, 2006). Risk processes implicated at the level of family interactions and environmental exposure (e.g., maternal sensitivity in early childhood, Eiden, Chavez, &

Leonard, 1999, or stress and poor monitoring, Chassin, Curran, Hussong & Colder, 1996) as well as at the level of neighborhood context (e.g., disorganization and availability of substances, see Buu et al., in press) similarly convey risk for externalizing symptoms more generally.

Work establishing the association between parent alcoholism and children's externalizing symptoms has almost exclusively focused on testing a severity hypothesis, addressing the question of whether children who have an alcoholic parent (or parents whose alcoholism varies in severity) have increased externalizing symptoms as compared to children without an alcoholic parent. However, some mechanisms explaining COAs' risk for externalizing symptoms, particularly those involving some form of environmental exposure, should predict not only who is at risk (i.e., COAs versus non-COAs) but also when this risk should occur (i.e., at the time of exposure to the parent's alcoholism). Currently, we do not know whether children are more likely to show externalizing symptoms in response to their parents' increases in alcohol-related symptoms (and thus at the same time as their parents increase their alcohol-related symptoms).

A previous study by our research group examined this question with respect to predicting children's internalizing symptoms. We found that children whose parents had a lifetime diagnosis of alcohol abuse or dependence showed greater internalizing symptoms than did their non-COA peers, but no additional risk for internalizing symptoms occurred during those periods when parents' were actively experiencing alcohol-related symptomatology (Hussong, Cai et al., 2007). In the current study, we extend this line of inquiry, testing whether three separate effects of parent alcoholism are differentially predictive of children's externalizing symptoms.

The first effect of parent alcoholism is a within-subjects or time-varying effect that indexes whether children show increased (or decreased) externalizing symptoms, over their usual baseline, at those times when their parents also show increased (or decreased) alcohol-related symptoms. The second effect of parent alcoholism is a between-subjects proximal effect that indexes whether children whose parents show greater alcohol-related consequences during the developmental period under study in turn show greater externalizing symptoms during that same period compared to children whose parents do not have alcohol-related consequences during this period. Thus, time-varying effects focus on issues of timing (whether children's externalizing symptoms get worse or better than usual at those times when their parents are more symptomatic) whereas proximal effects focus on individual differences (whether parents' average symptomatology over the developmental period helps us to identify those children showing elevated externalizing symptoms during this time).

Finally, the third effect of parent alcoholism is a baseline and (relatively) distal influence. This is also a between-subjects effect but the focus is on the impact of lifetime parent symptomatology that predates the developmental period under study and is not influenced by changes in parent symptomatology over the developmental period. This effect is operationalized by parents' lifetime diagnoses of alcohol disorder. These three effects are then conceptually distinct. For example, for a child followed from age 10 to 15, the time-varying effect is the within-person elevations and reductions in externalizing symptoms associated with parents' alcohol-related symptoms at a specific age; the proximal effect is the between-person differences in externalizing symptoms associated with averaged parents' alcohol-related symptoms between ages 10 and 15; and the distal effect is the between-person differences in externalizing symptoms associated with parents' baseline diagnosis (by age 10 of the child) of an alcohol use disorder.

Children of parents with a lifetime diagnosis of alcoholism show varying levels of exposure to their parent's alcohol-related symptoms, including a complete lack of direct exposure to symptoms in families where parents are in remission after the child's birth. Nonetheless, having a parent with a lifetime history of alcohol use disorders may increase risk for disinhibited behavior in children even without environmental exposure via genetic transmission (King et al., 2009). However, a greater risk for environmental disruptions that may or may not accompany genetic risk can occur for children whose parents show current alcohol-related symptoms. To the extent that these disruptions directly trigger children's externalizing behaviors (e.g., via maladaptive coping, reactions to impaired parenting), time-varying effects of parents' alcohol-related symptoms may be evident.

Parsing such distal, proximal and time-varying effects of parent alcoholism has the potential to inform etiology. The distal effect of having a parent with a lifetime diagnosis of alcoholism, which in our study occurred early in life or even before the child was born, supports the importance of mechanisms that operate early and provide a stable influence over the life course. A primary candidate for such influences is genetic liability (Zucker, 2006). However, early maladaptive environments characterized, for example, by maltreatment also have shown long-term impacts on children (Cicchetti & Toth, 2000; Egeland, Yates, Appleyard & van Dulmen, 2002), perhaps particularly when paired with genetic vulnerability for substance use (Caspi et al., 2002).

The proximal effects of parent alcoholism indicate that factors identifiable close in time to the child's symptoms identify who is at risk for externalizing symptoms whereas time-varying effects indicate the risk factors for when risk is increased. Supporting the possibility of time-varying effects, Loukas, Zucker, Fitzgerald, and Krull (2003) showed greater disruptive behaviors associated with the time-varying effects of parent alcoholism in a sample of young COAs and controls. In addition, DeLucia, Belz, and Chassin (2001) showed that children whose parents reported a high and decreasing pattern of alcohol dependence symptoms over time evidenced greater internalizing and externalizing symptoms than those whose alcoholic parents reported moderate and increasing or low and decreasing patterns, although adolescents' symptoms did not track changes in parent alcohol dependence over time. Together, these three effects of parent alcoholism thus inform our search for risk populations and periods, which inform developmental processes underlying externalizing symptoms.

We studied these effects of parents' alcohol-related symptoms using three reporters of children's externalizing symptoms. We used multiple reporters for two reasons. First, parents are clearly a more desirable reporter than children at the youngest ages (ages 10 and under) and adolescents are the more desirable reporter at the older ages (given the tendency of youth to hide their deviant behavior). For this reason, both parent and adolescent reports are useful in estimating children's externalizing symptoms across the wide age range of interest in this study (ages 2 through 17). In addition, a substantial literature indicates that parental psychopathology (particularly maternal depression, which occurs at elevated rates in families with an alcoholic parent) may result in biased reports of child behavior (Forehand & McCombs, 1988). Thus, by including both mother and father reports of the child's externalizing symptoms, we can see whether effects hold across reporter (e.g., whether effects of maternal alcoholism predict the outcome of father-reported child externalizing symptoms and vice-versa). This use of multiple reporters helps to reduce the risk that our findings are simply the result of shared method variance or reporter bias (as for example in the study of maternal depression, Offord et al., 1996) and provides a more thorough test of our hypothesis given differences in the predictors of psychopathology as a function of reporter (Collishaw, Goodman, Ford, Rabe-Hesketh, & Pickles, 2009).

In sum, we examined three effects of parent alcoholism on children's externalizing symptoms that we refer to as distal, proximal, and time-varying effects. These effects differentially focus on between-person (i.e., distal and proximal effects) and within-person (i.e., time-varying effects) differences and on influences that largely precede (i.e., distal effects) versus occur within the assessment period (i.e., proximal and time-varying effects). We also tested whether the proximal and time-varying effects of parents' alcohol-related symptoms differentially impact those children whose parents have a lifetime alcohol use disorder (i.e., COAs defined via our distal effect) versus those who do not. We examined these effects using an integrative data analysis approach (see Curran et al., 2008; Curran & Hussong, 2009; McArdle, Grimm, Hamagami, Bowles, & Merdith, 2009) in which we simultaneously analyzed two nationally prominent prospective studies of COAs and matched controls recruited from the community. Building on results of our previous analyses of these samples showing significant distal effects of parent alcoholism on the level of (but not changes in) children's externalizing symptoms (Hussong, Wirth et al., 2007), we focus here on the role of proximal versus time-varying effects and the unique contributions of all three effects of parent alcoholism when considered simultaneously.

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 non-alcoholic parents were assessed repeatedly.

The Michigan Longitudinal Study (MLS) used a rolling, community-based recruitment 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 one, 338 males (n=262 COAs and 72 controls), initially aged 2–5, and their parents completed a series of in-home interviews. 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 diagnostic criteria for alcoholism during adulthood based on self-reports (Feighner et al., 1972), reside with their biological sons aged 3–5, and be in intact marriages with their sons' biological mothers 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 child(ren) were at three-year intervals.

Cohort two were girls from the cohort one families who were recruited when cohort one boys were at Wave 2. Because cohort one inclusion criteria involved having families with at least one male child and no restrictions on other children, these families had fewer girls. To provide age parallelism with cohort one, where possible, and to begin assessments at ages 3–5, a broader age range was used to recruit girls. One target girl per family was enrolled if she was aged 3–11, with those aged 3–5 receiving the Wave 1 battery, those aged 6–8 receiving the Wave 2 battery, those aged 9–11 receiving the Wave 3 battery, and (at follow-up) those aged 12–14 receiving the Wave 4 battery. Similarly, the third cohort contained all additional male and female siblings of the male target child in cohort one who were within +/− 8 years of the cohort one target child at the time of data collection, with assessment batteries structured by age as for cohort one. The siblings in cohorts two and three were reassessed in all subsequent waves of data collection and received measures that paralleled the male target children in cohort one based on age of assessment. Because children in cohorts two and

three 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 two and an additional 106 siblings (from 84 families) comprised cohort three.

Across all three cohorts, 596 children from 338 families provided four waves of data (ages 2–17), separated by three-year intervals for mother- and father-reports of children's externalizing symptoms. A total of 399, 339, 402, and 418 participants had reports on their functioning available at waves 1–4, respectively, yielding an overall participation rate of 73% for those with at least two waves of data in the sample (see Zucker et al., 2000).

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 hours of data collection and child assessments were typically 7 hours (except for annual interviews which took one hour) 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. 70% 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 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 Hispanic or non-Hispanic Caucasian ethnicity, Arizona residency, having a 10.5–15.5 year old adolescent, English-speaking, lack of cognitive limitations precluding an interview, and a biological and custodial parent who met DSM-III lifetime criteria for alcohol abuse or dependence. Lifetime presence of parent alcoholism was determined through diagnostic interviews with parents using the Diagnostic Interview Schedule or through spousal report using the Family History Research Diagnostic Criteria (if the alcoholic parent was not interviewed). 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 among lower socio-economic status and Hispanic families were found.

These families were initially interviewed when the adolescents were aged 11–15 (wave 1) and re-interviewed on an annual basis when the adolescents were aged 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. Across MLS and AFDP, assessments of the target child's externalizing symptom by any of the reporters were available on 1050 adolescents. Three samples were created to examine effects for each reporter of externalizing symptoms, with each sample including cases for whom at least one report of symptoms for that reporter was available between ages 2 and 17 (or between 10 and 17 for adolescent-reports). These criteria resulted in a sample of 1026 children from 781 families for mother-reported externalizing symptoms and 938 children from 712 families for the father-report sample. For the adolescent-report sample, we only included reports from AFDP because parent-reported alcohol-related symptoms were assessed jointly with adolescent-reported symptoms at only one to two time points in MLS. Thus, the adolescent-report sample consisted of 454 children who were not nested in family (by design). Demographic characteristics for these three samples are reported in Table 1.

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).

Parent alcoholism was assessed by parent-report in both studies. Three indicators indexed the distal, proximal, and time-varying effects of parent alcoholism. The distal indicator was largely based on diagnostic interviews with parents conducted at baseline to assess lifetime diagnoses of alcohol abuse or dependence. Specifically, in the MLS, parental alcohol use disorder at Wave 1 was assessed by the Diagnostic Interview Schedule (version III; Robins et al. 1981, 1982), the Short Michigan Alcohol Screening Test (Selzer, Vinokur & Van Rooijan, 1975), and the Drinking and Drug History Questionnaire (Zucker, Noll, & Fitzgerald, 1988). On the basis of information collected by all three instruments, a lifetime diagnosis at the time of the baseline assessment was made by a trained clinician using DSM-IV criteria. Inter-rater reliability for the diagnosis was excellent (kappa=0.81). In AFDP, parents were directly interviewed at baseline using a computerized version of the Diagnostic Interview Schedule to assess diagnostic status. In cases where a biological parent was not directly interviewed (21% of fathers and 4% of mothers in the current subsample), the reporting parent was used as the informant using the Family History Research Diagnostic Criteria (Andreasen, Endicott, Spitzer & Winokur, 1977). Thus, a lifetime diagnosis at the time of the baseline assessment was made based on DIS self-reports or Family History-Research Diagnostic Criteria spousal-reports.

Both proximal and time-varying indicators of parent alcoholism were based on parents' self-reports at each wave of whether they had experienced any of 11 alcohol-related symptoms in the past year. The symptoms are based on indicators of DSM-IV criteria for alcohol abuse and dependence and include getting complaints from friends/family, losing friends, getting arrested for drunk drinking, getting arrested for other drinking-related offenses, missing school or work, losing a job or getting kicked out of school, drinking first thing in the morning, drinking more or longer than intended, feeling guilty about drinking, and suffering blackouts. All items were dichotomized (absent versus present) and summed within wave to form the repeated measures indicating the time-varying effects of parent alcoholism. These time-varying indicators were then averaged across wave (within-person) to create the proximal indicator of parent alcoholism.[1] By creating time-varying (or within-person) and proximal (or between-person) indicators in this manner, we were able to disaggregate within- and between- person effects within a multilevel modeling framework (as described in the results; also see Curran, Edwards, Wirth, Hussong & Chassin, 2007).

Child externalizing symptoms were assessed by mother-, father- and adolescent-reports. In each study, participants completed the Child Behavior Checklist (MLS) or Youth Self-Report (MLS adolescents) or an adapted form of these instruments (AFDP; Achenbach & Edelbrock, 1978). We used Item Response Theory (Thissen & Wainer, 2001) to create comparable measures of externalizing symptoms across study. Specifically, we examined 30 items from the Child Behavior Checklist aggression 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 0–2 for parent reports in both studies and from 0–4 for adolescent-reports in AFDP, with an assessment window of past 6 months for MLS and 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.

We then used Item Response Theory to derive externalizing scale scores that optimize available data and are sensitive to item differences in severity, behavioral repertoire, and development (for details of these analyses, see Hussong, Wirth et al., 2007). Item Response Theory has several advantages over traditional proportion scores (see Curran et al., 2007; Curran et al., 2008) including differential weighting of individual items (allowing for scale scores that account for the severity of the specific externalizing symptoms) and tests of differential item functioning that identify the extent to which items vary in their relation to externalizing symptomatology over sub-populations (such as that defined by study membership).

We first used Item Response Theory to evaluate differential item functioning (Thissen, Steinberg, & Wainer, 1993) using IRTLRDIF software (Thissen, 2001) as a test of whether items functioned similarly in relation to the underlying construct of externalizing symptoms across important subgroups based on child age (ages 2–11 vs. 12–17 for parent-reports and ages 10–13 vs. 14–17 for adolescent-reports), gender and 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 (N=1026, N=938 and N=966 for mother-, father- and adolescent- reports, respectively).[2] For mother-reported symptoms, 11 items showed differential item functioning across age groups and 6 items did so for gender or study membership (with some items showing differential item functioning for more than one group indictor). For father-reported symptoms, 14 items showed differential item functioning over age, 12 did so over gender and none did so across study membership. For adolescent-reported symptoms, 14 showed differential item functioning over age, seven did so across gender and none did so across study. On the whole, reporters varied considerably in the pattern of differential item functioning.

The resulting items and sub-items (created to account for differential item functioning) were then subjected to calibration and scoring procedures using MULTILOG (Thissen, 1991).

[1]This definition of the proximal effects of parents' alcohol-related consequences can be distinguished from distal effects both conceptually (as described earlier) and methodologically. It is of course true that variance in the proximal effects and overall levels of alcohol-related symptoms are higher in the COA families. Notably, descriptive analyses show a M=0.17, SD=0.39 for controls and M=1.05, SD=1.28 for COAs on father's alcohol-related symptoms (proximal effect) and M=0.09, SD=0.31 for controls and M=0.71, SD=1.14 for COAs on mother's alcohol-related symptoms. From another perspective, 76% of controls and 33% of COAs had reports of zero on father's alcohol-related symptoms over the study period, with 86% and 49% doing so, respectively, for mother's alcohol-related symptoms. Importantly, we tested these effects as unique from one another, so in our analyses we are testing whether these proximal variation in alcohol-related symptoms have predictive utility above and beyond distal effects.

[2]Note that the calibration sample size is larger than the analysis sample size due to the omission of cases in the analytic sample resulting from missing data on predictor variables. The adolescent calibration sample also included the MLS participants who provided symptomatology data but not concurrent reports of parent alcohol-related symptoms, and thus were not used in the current analysis samples.

Using the 2-parameter logistic (2PL) Item Response Theory model, we estimated discrimination and severity parameters for all items and sub-items 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 differential item functioning analyses and can be interpreted on a z-score metric. (The z-score metric is relative to the mean and standard deviation of the calibration sample as a whole rather than relative to each age period assessed.) These scores served as the outcomes of interest in all subsequent analyses.

Results

Our statistical approach permits simultaneous analysis of data drawn from the two longitudinal studies. These methods have recently been referred to as Integrative Data Analysis (Curran & Hussong, 2009). As noted in Table 1, the MLS and AFDP samples differ in a number of respects on key study variables. However, these differences do not undermine the value of integrative data analysis but rather augment it. By combining samples we increase heterogeneity among participants in developmental range, cohort representation, sampling and measurement strategies, and demographic features of the participants. We are careful to take into account these differences in harmonizing measurement (i.e., making our measures comparable across studies) and in conducting inferential tests of our hypotheses. Importantly, by combining the samples and including tests of differences in how our predictor variables relate to outcomes as a function of sample membership, we are able to directly test the generalizability of our findings across the two samples (akin to meta-analysis). These two features (increasing sample heterogeneity and testing generalizability of findings across the samples) are key advantages of integrative data analysis. The combined samples also have greater power to test interactions. And, importantly, the combined samples are able to test the hypotheses of interest over a longer developmental age range than is possible in any one of the two studies alone. (For a more complete discussion of the relative advantages and disadvantages of this approach, see Curran & Hussong, 2009.)

In the current application, our use of Integrative Data Analysis proceeded in three phases. First, we addressed the issue of missing data, both by design and attrition. Second, we estimated longitudinal trajectories of externalizing symptoms. And, third, we tested our specific study hypotheses. These methods parallel those previously reported in Hussong, Cai et al. (2008).

Integrative Data Analysis Phase 1: Imputing Missing Data

We addressed the issue of missing data in our time-invariant and time-varying covariates through multiple imputation for each of our three sub-samples (i.e., mother-, father-, and adolescent-report) independently (Schafer, 1997a). For these analyses, we used the externalizing Item Response Theory scores from 2,801 assessments of 1,026 participants in the mother-report sample, 2,448 assessments of 938 participants in the father-report sample and 1,349 assessments of 454 participants in the adolescent-report sample. We used SAS PROC MI (SAS, 1999) to impute missing data in the time-invariant covariates and the R package PAN (Schafer, 1997b) for imputation of the time-varying covariates. Specifically, we first created 10 data sets for which the missing data in the time-invariant covariates were imputed, and for each we proceeded to impute the missing time-varying covariate values using PAN. Following standard recommendations in the multiple imputation literature (Rubin 1996), we included all predictors in both imputation models and independent as well as dependent variables in the PAN model.

Integrative Data Analysis Phase 2: Constructing Trajectories

We then identified the shape of externalizing trajectories within each of the three sub-samples (for further details, see Hussong, Wirth et al., 2007). First, we plotted the means of Item Response Theory scores across age and examined iterative analyses to examine the optimal functional form of the resulting trajectories (i.e., linear, quadratic and piece-wise liner) following the strategies described by Bollen and Curran (2006). Intercepts in all models were centered at age 13. The nesting of repeated observations in multiple siblings within family resulted in 3-level models in the MLS. In the AFDP, we used a 2-level model due to the more simple nesting of repeated observations in target participants (who were independent of family). All trajectory models were estimated 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).

Competing models were compared visually using mean and individual trajectory plots, BIC and AIC fit indices, and chi-square difference tests (when available for nested models). Based on these criteria, the optimal functional form retained for mother- and adolescent-reported scores was a one-piece linear model and for father-reported scores was a two-piece linear model.[3] For fathers, the two-piece model delineated change from ages 2 to 7 and 7 to 17 through two linear slope parameters, best reflecting the pattern of change evident in our data. In unconditional trajectory models for each sub-sample, parameter estimates indicated significant, steady decreases in externalizing symptoms 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 (see Hussong, Wirth et al., 2007 for details).

Integrative Data Analysis Phase 3: Hypothesis Testing

To test our hypotheses, we estimated a series of conditional multilevel models. We fitted each model to all M=10 data sets with imputations of missing data and combined the parameter estimates and standard errors using SAS PROC MIANALYZE, which implements procedures developed by Rubin (1987). To test the effects of time-varying (i.e., within-person) versus proximal and distal (i.e., between-person) effects, we followed Raudenbush and Bryk (2002, p. 134– 141; see also Curran & Bauer, 2009). Specifically, we added person-mean centered time-varying covariates for mothers' and fathers' alcohol-related symptoms as repeated measures and the report of these symptoms averaged over repeated assessments as the proximal effect. We also added interactions between each of these predictors with study to test for differences in findings based on membership in MLS versus AFDP.

In baseline models, we first included control variables (i.e., child gender and parent education), study membership (i.e., a dummy variable with MLS=0 and AFDP=1) and interactions between control variables, study membership and the age-indicated time trends. This was a conservative strategy and we thus trimmed non-significant interactions for subsequent analyses. The interaction between one of the linear time trends (change between age 7 and 17) and study membership was significant for father-reported child externalizing symptoms and was thus retained. This interaction indicated that although externalizing scores decrease during childhood from age 2 to 7, this decrease slowed after age 7 to a

[3]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 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.

greater extent in MLS than in AFDP (likely because AFDP contributed 11–17 year olds only to this analysis). As reported in Tables 2 and 3 in Model 1, we then added the three types of effects (i.e., distal, proximal, and time-varying) for each parent's alcohol-related symptoms as well as interactions between these effects of parent alcoholism and study membership. Interactions including study membership were again trimmed when non-significant. Model 2 tested whether there were gender differences in the time-varying effects of parent-alcoholism as a post-hoc analysis. In final models (not tabled), we included interactions between the time-varying and distal effects (Model 3) and between the proximal and distal effects (Model 4) to test whether the effects of alcohol-related symptoms during the study period differentially effected risk for externalizing symptoms among COAs versus controls. Below, we report the combined results.

Results for model testing father- reported child externalizing symptoms—For the father-report model, distal effects of both mothers' and fathers' lifetime alcoholism diagnoses predicted greater father-reported child externalizing symptoms. In addition, proximal effects of father's (but not mother's) alcohol-related symptoms increased risk for father-reported child externalizing symptoms. A marginally significant time-varying effect for father's (but again not mother's) alcohol-related symptoms was also found to increase risk for father-reported child externalizing symptoms. No significant study effects were found, and thus these interactions were trimmed in the final model as reported in Table 2.

To examine whether the limited time-varying effects of parent alcoholism was due to moderating gender differences, we included cross-level interactions between the child's gender and the time-varying effects of mothers' and fathers' alcohol-related symptoms (see Model 2, Table 2). However, no gender differences were found. In addition, no interactions between parents' lifetime diagnosis of alcoholism and either the time-varying or proximal effects of parents' alcohol-related symptoms predicted father's report of the child's externalizing symptoms (Models 3 and 4, not shown).

Results for model testing mother- reported child externalizing symptoms— Results of the father-report model were largely replicated in analyses of mother-reported child externalizing symptoms. Specifically, for the mother-report model, distal effects of both mothers' and fathers' lifetime alcoholism diagnoses predicted greater mother-reported child externalizing symptoms. However, an interaction between the distal effect of father's alcoholism diagnosis and study indicated that this effect was only found in AFDP (β=0.20, t=2.29, p=0.02) and not in MLS (β= −0.06, t=−0.76, p=0.45). Proximal effects of father's alcohol-related symptoms increased risk for mother-reported child externalizing symptoms again, and a marginally significant proximal effect of mother's alcohol-related symptoms was also found. A time-varying effect for father's (both not mother's) alcohol-related symptoms was significantly associated with an increased risk for mother-reported child externalizing symptoms. Once again, tests of gender differences in the impact of time-varying effects of parent alcohol-related symptoms on mother-reported child externalizing symptoms were non-significant (see Model 2, Table 2).

However, mothers' lifetime diagnosis of alcoholism interacted with the time-varying effect of mother's alcohol-related symptoms in predicting mother's report of child's externalizing symptoms (β = −0.11, t=−2.39, p=.02). (No interaction was found for the proximal effects of parents' alcohol-related symptoms and lifetime diagnosis of alcoholism.) Probing of this interaction (following, Curran, Bauer, & Willoughby, 2006) revealed that children showed greater externalizing symptoms during those years when their mothers had higher alcohol-related symptoms (i.e., a higher time-varying effect) only if their mothers did not have a lifetime diagnosis of alcoholism (β =0.10, t=2.49, p=.01) rather than if they did (β = −0.01,

t=−0.43, p=0.67). Thus, the time-varying effect of maternal alcohol-related symptoms on mothers' report of children's externalizing symptoms was limited to non-COAs.

Results for model testing adolescent-reported externalizing symptoms— Because the child-report analyses included data only from AFDP, no study effect is included in this analysis. Effects differed slightly from those of the parent report analyses. A distal effect of only father's, and not mother's, lifetime alcoholism on the adolescent's externalizing symptoms was found. Moreover, a proximal effect of mother's, and not father's, alcohol-related symptoms on the adolescent's externalizing symptoms was found. No time-varying effects of either parent's alcohol-related symptoms were found. In addition, no gender differences in the time-varying effects of parent alcohol-related symptoms on adolescent-reported externalizing symptoms were found (see Model 2, Table 3).

However, mothers' lifetime diagnosis of alcoholism interacted with the time-varying effect of mother's alcohol-related symptoms in predicting adolescent's report of externalizing symptoms (β= −0.13, t=1.98, p=.05). (No interaction was found for the proximal effects of parents' alcohol-related symptoms and lifetime diagnosis of alcoholism.) Similar to mother-reports, probing of this interaction revealed that the children showed greater externalizing symptoms during those years when their mothers had higher alcohol-related symptoms (i.e., a higher time-varying effect) only if their mothers did not have a lifetime diagnosis of alcoholism (β =0.13, t=2.38, p=.02) rather than if they did (β =0.00, t=−0.05, p=0.96).

Discussion

In the current study, we tested whether the time-varying, proximal and distal effects of parents' alcohol-related symptoms predicted both who is at risk for externalizing symptoms (i.e., children of alcoholic parents and/or children with greater overall exposure to parents' alcohol-related symptoms) and when this risk occurs (i.e., at the same time as parents' alcohol-related symptoms). Strengths of our approach included the use of integrative data analysis to simultaneously test this question in two, prospective high risk studies of COAs and matched controls; direct ascertainment of parents' alcohol-related symptoms; and the inclusion of multiple reporters of children's externalizing symptoms. Overall, we found consistent and large distal effects of parent alcoholism confirming previous findings that COAs have greater risk for externalizing symptoms than do non-COAs, even though the initial diagnosis of parent alcoholism predates the timing of the children's symptoms. In addition, we found proximal effects indicating an additional risk for externalizing symptoms (above and beyond that attributed to distal effects) for children whose parents had greater alcohol-related symptoms during the study period when externalizing symptoms were assessed. Notably, we also found time-varying effects of parents' alcohol-related symptoms, showing that in some cases children increase their externalizing symptoms during those years when their parents report more alcohol-related symptoms. Importantly, these findings did not differ across gender; rather, these effects of parent alcoholism were equally relevant for boys and girls in predicting externalizing symptoms. These findings differ somewhat from our previous analyses that tested whether these three types of effects of parent alcoholism predicted children's internalizing symptoms. We discuss implications of the current findings in light of our previous analyses as well as effects of reporter and developmental timing on our conclusions.

Distal, proximal, and time-varying effects

The dominance of distal, over proximal and time-varying, effects of parent alcoholism on children's externalizing symptoms is consistent with our previous analyses predicting children's internalizing symptoms. Although these findings are not consistent with theories of child psychopathology that conceptualize parent-child influences as dynamic and driven

by real-time processes (e.g., Granic & Patterson, 2006), they are consistent with alternate views pointing to a long-term deleterious effects of high genetic vulnerability coupled with stressful, chaotic and sometimes abusive environments. Such posited gene by environment interactions may have long-term implications for subsequent adjustment due to increasing constraints on positive or even corrective environmental inputs (e.g., lower school readiness and parental involvement resulting in school failure and lack of exposure to the benefits of school success; Zucker, 2006). Thus, in some cases, distal influences may be so substantial as to reduce the odds that more proximal influences will significantly alter risk for symptomatology.

These distal effects were unique from (i.e., net the effect of) the proximal effects of parents' alcohol-related consequences on children's externalizing symptoms. Proximal effects may suggest that children acquire additional risk through concurrent exposure to their parents' drinking and related consequences that goes beyond the risk that is conveyed through more genetically or biologically mediated mechanisms. Proximal effects were present across reporters, and thus evident early in life and through adolescence. Moreover, proximal effects increased risk for externalizing symptoms regardless of whether parents met diagnostic criteria for an alcohol use disorder or not (i.e., regardless of distal effects), and thus even non-COAs showed greater risk for externalizing symptoms if their parents' had greater alcohol-related consequences.

Unlike distal and proximal effects, time-varying effects of parents' alcohol-related consequences are within-person comparisons and thus address questions of timing. Time-varying effects of parents' alcohol-related consequences were only a marginally significant predictor of father's report of children's externalizing symptoms but they were a significant predictor of both mothers' and adolescents' reports of externalizing symptoms, though only among non-COAs. Thus, our most consistent finding is that children are at greater risk for externalizing symptoms when their parents' are actively abusing alcohol, but only if their parents are below diagnostic levels. This effect was limited to maternal symptoms, perhaps reflecting a greater disruption to the caregiving environment or the effect of a later developing form of alcohol-related problems. For families in which parents meet diagnostic criteria for alcohol use disorders, this lifetime diagnosis and the proximal effects of parent's alcohol-related consequences have a much greater impact on the child's risk for externalizing symptoms; the timing of children's externalizing symptoms does not appear linked to that of their alcoholic parents' alcohol-related symptoms.

One reason for the dominance of distal effects over proximal and time-varying effects may be that the proximal and time-varying effects of parent alcoholism on children's externalizing symptoms are heavily impacted by meaningful protective factors. Previous studies show that diminished risk of negative outcomes for COAs whose families are able to maintain rituals and a regularity of routine (e.g., observing holidays, eating meals together) despite having an actively alcoholic parent (Wolin, Bennett, Noonan, & Teitelbaum, 1980). Moreover, some work has suggested that the functioning of the non-alcoholic parent may play a protective role, though findings are mixed (Curran & Chassin, 1996; Werner, 1986). Finally, adolescent COAs with greater cognitive coping styles, typically considered more adaptive when used in response to uncontrollable stressors, have shown reduced risk for alcohol involvement (Hussong & Chassin, 1997). Each of these protective factors may differentially impact the distal, proximal and time-varying effects of parent alcoholism. However, no research has examined whether these protective influences are differentially operative for adolescents as a function of when their parents are actively alcoholic.

Previously, we also posited that the distal effects of parents' alcohol-related symptoms were stronger and more consistent than proximal and time-varying effects because these latter

effects may be relatively weaker for internalizing symptoms as compared with other forms of psychopathology, such as externalizing symptoms, for which COAs show a greater risk (e.g., Chassin et al., 1991). Indeed, effects for proximal and time-varying effects were more evident in analyses predicting children's externalizing than internalizing symptoms. Thus, this argument may be at least partially supported. Further analyses should expand upon this specificity of risk question, particularly given that previous studies show the strongest specific effects of parent alcoholism on child functioning indices are, not surprisingly, for alcohol involvement itself (Chassin et al., 1991). As such, the effect of parent alcoholism on some outcomes may be limited to distal influences, as appears to be the case for internalizing symptoms, but others may be a combination of distal, proximal, and time-varying effects, as appears to be the case for externalizing symptoms.

Although the question of whether distal, proximal, or time-varying effects have a greater impact on child functioning may help us differentiate the relative salience of potential mechanisms of risk, we anticipate that in practice these effects operate collectively and likely in interaction. The presence of time-varying and proximal effects of parents' alcohol-related symptoms in the presence of such distal effects underscores the multiple ways in which parent alcohol-related symptoms may convey intergenerational transmission of risk for adjustment problems in COAs. Distal factors may remain dominant because they set children on an early risk trajectory but the continuance of that behavior may then function autonomously from the original cause as new causes take over. For example, children may engage in more disinhibited behavior as a result of parent drinking problems, but once they establish a certain level of externalizing symptoms they may gain entry into antisocial peer context (Dishion, Duncan, Eddy, & Fagot, 1994). These peer contexts in turn fuel the maintenance and perhaps escalation of externalizing symptoms. As such, the original externalizing trajectory starts because of parental drinking but then it becomes attached to a broader set of predictors, which may in part include parental drinking, over time.

Reporter differences and developmental timing

Analyses of father- and mother- reported children's externalizing symptoms provided results that were highly consistent with one another, however those based on adolescent's self-reports showed some notable differences. It is difficult to clearly attribute these differences to reporter effects in the current design because parent-report models tested the effects of parents' alcohol-related symptoms on children's externalizing symptoms from ages 2 through 17 whereas adolescent's self-report models included symptoms from ages 11 through 17. In addition, parent-report analyses were based on participants from the MLS and AFDP studies (and thus a larger sample) whereas child report analyses were based only on participants from AFDP.

Nonetheless, we found that distal effects for fathers' alcoholism were consistent across reporters (with the exception of the MLS sample in mother-report analyses) whereas we found distal effects for mother's alcoholism only in parent-report analyses. Moreover, we found proximal effects of father's alcohol-related symptoms in both father- and mother-report analyses and those for mother's alcohol-related symptoms were found in both mother- and adolescent-report analyses. Finally, we found the time-varying effects of father's alcohol-related symptoms in mother-, adolescent- and, to some extent, father-report analyses. Given potential parental bias in reporting children's symptomatology related to the parents' own psychopathology, the pattern of findings bolsters our confidence that reporter effects alone are not responsible for these effects. (For example, effects of maternal alcoholism were found in father-report analyses and not just mother-report analyses as well as vice-versa.)

However, the potential effect of developmental timing in comparing the findings across the three reporters of children's symptomatology deserves greater research attention. The time-varying effect of father's alcohol-related symptoms among children of alcoholic parents was limited to parent report. This may reflect a relatively greater impact of these timing of risk indicators in childhood versus adolescence. This finding would be consistent with the view that there is greater potential for malleability in children's functioning earlier in development before these trajectories of externalizing behavior become entrenched and reinforced through cascading, negative consequences (e.g., antisocial peers, negative labeling in schools).

Conclusion

The current findings indicate that parents' lifetime diagnosis of alcoholism has a large and consistent effect on identifying which children show increased externalizing symptoms that is unique from the average, proximal effects of parents' alcohol-related symptoms. In addition, parents' alcohol-related symptoms increase risk for children's externalizing symptoms during those periods when their parents' are actively experiencing alcohol-related symptoms. The size of this effect was small, though perhaps not surprisingly so given that the analysis was conservative in testing these three unique effects of parent alcoholism jointly. However, these effects appear largely limited to externalizing, as opposed to internalizing, symptoms and were not as consistent as the distal effects of parents' alcoholism on children's symptomatology. One reason why distal effects may have been more consistent in this study was the greater measurement precision for the lifetime diagnosis assessment than for the proximal and time-varying effects of symptomatology. Future studies that address this problem of measurement equivalence would be informative. Moreover, studies that further consider the time-scale on which parent alcoholism impacts children's functioning are important in that they inform the search for underlying etiological processes. To the extent that distal processes dominate the effect of parent alcoholism on children's externalizing symptoms, mechanisms that operate early and provide a stable influence over the life course should be primary targets for exploration. Moreover, such distal effects indicate that prevention efforts targeting COAs' externalizing and internalizing symptoms per se should occur in early childhood. However, parents' alcohol-related symptoms continue to show proximal and time-varying effects on children's externalizing symptoms even in adolescence. Thus, the findings may suggest that family-based programs that consider the impact of parents' alcohol-related symptoms in addressing child functioning are likely to continue to be effective over development.

These implications should be tempered by limitations of the current study. We were unable to measure children's actual exposure to parents' alcohol-related consequences and the timing of this exposure directly. Thus, further study is needed to understand the role that exposure-based mechanisms play in accounting for the current pattern of findings. Limited ethnic diversity in our samples also constrains the generalizability of these findings. Previous analyses of these data showed no differences between non-Caucasian Hispanic and Caucasian youth in the effect of parent alcoholism on externalizing symptoms, but no other ethnic/racial groups were represented in these samples. Given differences in environmental stress due to factors associated with ethnicity/race, the additional impact of parent alcoholism on fluctuations in child functioning over time may vary across groups. Moreover, our assessments of externalizing symptoms rely on symptom checklists, which may not relate directly to risk for disorder, and targets the externalizing spectrum broadly. Finally, participants were initially recruited from intact families, perhaps limiting the generalizability of these findings to more disturbed families who experience early dissolution.

In conclusion, the current study suggests that parents' alcohol-related symptoms may impact children in multiple ways. The distal effects of having a parent with a lifetime diagnosis of alcoholism does not fully account for children's risk for externalizing symptoms associated with parent alcohol involvement. Rather, more proximal risk mechanisms need to be considered such as the disruption of parental drinking to family environment and organization, increased stress and potentially violence in the home, and impairments in parenting. These proximal effects appear largely limited to predicting externalizing rather than internalizing symptoms. Further understanding the relations among the risk mechanisms underlying these highly co-occurring forms of symptomatology is needed to better understand the multiple ways in which parent alcoholism may impact children's functioning over development.

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References

Achenbach TM, Edelbrock CS. The classification of child psychopathology: A review and analysis of empirical efforts. Psychological Bulletin. 1978; 85:1275–1301. [PubMed: 366649]

Andreasen NC, Endicott J, Spitzer RL, Winokur G. The family history method using diagnostic criteria. Reliability and validity. Archives of General Psychiatry. 1977; 34:1229–1235. [PubMed: 911222]

Bollen, KA.; Curran, PJ. Wiley Series on Probability and Mathematical Statistics. New York: Wiley; 2006. Latent curve models: A structural equation approach.

Buu A, DiPiazza C, Wang J, Puttler LI, Fitzgerald HE, Zucker RA. Familial and neighborhood influences over the course of childhood and the development of drug involvement and other psychopathology in late adolescence. Journal of Studies on Alcohol and Drugs. 2009 in press.

Caspi A, McClay J, Moffitt T, Mill J, Martin J, Craig I, Taylor A, Poulton R. Role of genotype in the cycle of violence in maltreated children. Science. 2002; 297:851–854. [PubMed: 12161658]

Chassin L, Barrera M Jr, Bech K, Kossak-Fuller J. Recruiting a community sample of adolescent children of alcoholics: A comparison of three subject sources. Journal of Studies on Alcohol. 1992; 53:316–319. [PubMed: 1619925]

Chassin L, Curran PJ, Hussong AM, Colder CR. The relation of parent alcoholism to adolescent substance use: A longitudinal follow-up study. Journal of Abnormal Psychology. 1996; 105:70–80. [PubMed: 8666713]

Chassin L, Rogosch F, Barrera M. Substance use and symptomatology among adolescent children of alcoholics. Journal of Abnormal Psychology. 1991; 100:449–463. [PubMed: 1757658]

Cicchetti, D.; Toth, SL. Development processes in maltreated children. In: Hansen, D., editor. Nebraska Symposium on Motivation, Vol. 46: Child maltreatment. Lincoln: University of Nebraska Press; 2000.

Collishaw S, Goodman R, Ford T, Rabe-Hesketh S, Pickles A. How far are associations between child, family, and community factors and child psychopathology informant-specific and informant-general? The Journal of Child Psychology and Psychiatry. 2009; 50:571–580.

Curran PJ, Bauer DJ. The disaggregation of within-person and between-person effects in longitudinal studies of change. 2009 To appear in Annual Review of Psychology.

Curran, PJ.; Bauer, DJ.; Willoughby, MT. Testing the probing interactions in hierarchical linear growth models. In: Bergeman, CS.; Boker, SM., editors. Methodological issues in aging research. Mahway, NJ: Erlbaum; 2006. p. 99-129.

Curran PJ, Chassin L. A longitudinal study of parenting as a protective factor for children of alcoholics. Journal of Studies on Alcohol. 1996; 57(3):305–313. [PubMed: 8709589]

Curran, PJ.; Edwards, MC.; Wirth, RJ.; Hussong, AM.; Chassin, L. Alternative Categorical Measurement Models for the Analysis of Individual Growth. In: Little, T.; Bovaird, J.; Card, N., editors. Modeling Ecological and Contextual Effects in Longitudinal Studies of Human Development. Mahwah, NJ: LEA; 2007. p. 89-120.

Curran PJ, Hussong AM. Integrative data analysis: The simultaneous analysis of multiple data sets. Psychological Methods. 2009; 14:81–100. [PubMed: 19485623]

Curran PJ, Hussong AM, Cai L, Huang W, Chassin L, Sher KJ, Zucker RA. Pooling data from multiple prospective studies: The role of item response theory in integrative analysis. Developmental Psychology. 2008; 44:365–380. [PubMed: 18331129]

DeLucia C, Belz A, Chassin L. Do adolescent symptomatology and family environment vary over time with fluctuations in paternal alcohol impairment? Developmental Psychology. 2001; 37:207–216. [PubMed: 11269389]

Dishion T, Duncan T, Eddy J, Fagot B, Feltrow B. The world of parents and peers coercive exchanges and children.s social adaptation. Social Development. 1994; 3:255–265.

Egeland B, Yates T, Appleyard K, van Dulmen M. The long-term consequences of maltreatment in the early years: A developmental pathway model to antisocial behavior. Children's services: Social policy, research, and practice. 2002; 5:249–260.

Eiden RD, Chavez F, Leonard KE. Parent infant interactions in alcoholic and control families. Development and Psychopathology. 1999; 11:745–762. [PubMed: 10624724]

Eiden RD, Edwards EP, Leonard KE. A conceptual model for the development of externalizing behavior problems among kindergarten children of alcoholic families: Role of parenting and children's self-regulation. Developmental Psychology. 2007; 43:1187–1201. [PubMed: 17723044]

Feighner JP, Robins E, Guze SB, Woodruff RA, Winokur G, Munoz R. Diagnostic criteria for use in psychiatric research. Archives of General Psychiatry. 1972; 26:57–63. [PubMed: 5009428]

Forehand R, McCombs A. Unraveling the antecedent-consequence conditions in maternal depression and adolescent functioning. Behaviour Research and Therapy. 1988; 26(5):399–405. [PubMed: 3190649]

Granic I, Patterson GR. Toward a comprehensive model of antisocial development: A dynamic systems approach. Psychological Review. 2006; 113:101–131. [PubMed: 16478303]

Hussong AM, Cai L, Curran PJ, Flora DB, Chassin LA, Zucker RA. Disaggregating the Distal, Proximal, and Time-Varying Effects of Parent Alcoholism on Children's Internalizing Symptoms. Journal of Abnormal Child Psychology. 2007; 36:335–346. [PubMed: 17891557]

Hussong AM, Chassin L. Substance use initiation among adolescent children of alcoholics: Testing protective factors. Journal of Studies on Alcohol. 1997; 58:272–279. [PubMed: 9130219]

Hussong AM, Curran PJ, Chassin L. Pathways of risk for accelerated heavy alcohol use among adolescent children of alcoholic parents. Journal of Abnormal Child Psychology. 1998; 26:453–466. [PubMed: 9915652]

Hussong AM, Wirth RJ, Edwards MC, Curran PJ, Chassin LA, Zucker RA. Externalizing symptoms among children of alcoholic parents: Entry points for an antisocial pathway to alcoholism. Journal of Abnormal Psychology. 2007; 116:529–542. [PubMed: 17696709]

Kendler KS, Prescott CA, Myers J, Neale MC. The structure of genetic and environmental risk factors for common psychiatric and substance use disorders in men and women. Archives of General Psychiatry. 2003; 60:929–937. [PubMed: 12963675]

Krueger RF, Hicks BM, Patrick CJ, Carlson SR, Iacono WG, McGue M. Etiologic connections among substance dependence, antisocial behavior, and personality: Modeling the externalizing spectrum. Journal of Abnormal Psychology. 2002; 111:411–424. [PubMed: 12150417]

Littell, RC.; Milliken, GA.; Stroup, WW.; Wolfinger, RD. SAS System for Mixed Models. SAS Institute; Cary, NC: 1996.

Loukas A, Fitzgerald HE, Zucker RA, von Eye A. Parental alcoholism and co-occurring antisocial behavior: Prospective relationships to externalizing behavior problems in their young sons. Journal of Abnormal Child Psychology. 2001; 29:91–106. [PubMed: 11321632]

Loukas A, Zucker RA, Fitzgerald HE, Krull JL. Developmental trajectories of disruptive behavior problems among sons of alcoholics: Effects of parent psychopathology, family conflict, and child undercontrol. Journal of Abnormal Psychology. 2003; 112:119–131. [PubMed: 12653420]

McArdle JJ, Grimm KJ, Hamagami F, Bowles RP, Meredith W. Modeling lifespan growth curves of cognition using longitudinal data with multiple samples and changing scales of measurement. Psychological Methods. 2009; 14:126–149. [PubMed: 19485625]

McGue M, Iacono WG, Krueger R. The association of early adolescent problem behavior and adult psychopathology: A multivariate behavioral genetic perspective. Behavior Genetics. 2006; 36:591–602. [PubMed: 16557361]

Mehta PD, West SG. Putting the individual back into individual growth curves. Psychological Methods. 2000; 5:23–43. [PubMed: 10937321]

Offord DR, Boyle MH, Racine Y, Szatmari P, Fleming JE, Sanford M, Lipman EL. Integrating assessment data from multiple informants. Journal of the American Academy of Child and Adolescent Psychiatry. 1996; 35:1078–1085. [PubMed: 8755805]

Raudenbush, SW.; Bryk, AS. Hierarchical Linear Models: Applications and Data Analysis Methods. 2. Thousand Oaks, CA: Sage Publications, Inc; 2002.

Robins LN, Helzer JE, Croughan J, Ratcliff KS. National institute of mental health diagnostic interview schedule: Its history, characteristics, and validity. Archives of General Psychiatry. 1981; 38:381–389. [PubMed: 6260053]

Robins LN, Helzer JE, Ratcliff KS, Seyfried W. Validity of the diagnostic interview schedule, version II: DSM-III diagnoses. Psychological Medicine. 1982; 12:855–870. [PubMed: 7156256]

Rubin, DB. Multiple imputation for nonresponse in surveys. New York: Wiley; 1987.

Rubin DB. Multiple imputation after 18+ years. Journal of the American Statistical Association. 1996; 91:473–489.

SAS Institute. SAS/STAT user's guide: PROC MI, version 9.1.3. Cary: SAS Institute; 1999.

Schafer, Joseph L. Analysis of Incomplete Multivariate Data. London: Chapman & Hall; 1997a.

Schafer, JL. Unpublished Technical Report. 1997b. Imputation of missing covariates under a multivariate linear mixed model.

Selzer ML, Vinokur A, van Rooijan L. A self-administered short Michigan Alcoholism Screening Test (SMAST). Journal of Studies on Alcohol. 1975; 36:117–126. [PubMed: 238068]

Sher KJ, Walitzer KS, Wood PK, Brent EE. Characteristics of children of alcoholics: Putative risk factors, substance use and abuse, and psychopathology. Journal of Abnormal Psychology. 1991; 100:427–448. [PubMed: 1757657]

Singer, JD.; Willett, JB. Applied longitudinal data analysis: Modeling change and event occurrence. New York: Oxford University Press; 2003.

Thissen, D. MULTILOG user's guide: Multiple, categorical item analysis and test scoring using item response theory. Chicago, IL: 1991.

Thissen, D. IRTLRDIF v. 2.01b: Software for the computation of the statistics involved in item response theory likelihood-ratio tests for differential item functioning [Computer software]. Chapel Hill, NC: L.L. Thurstone Psychometric Laboratory; 2001.

Thissen, D.; Steinberg, L.; Wainer, H. Detection of differential item functioning using the parameters of item response models. In: Wainer, H.; Holland, P., editors. Differential item functioning. Hillsdale, NJ: Lawrence Erlbaum Associates; 1993. p. 67-113.

Thissen, D.; Wainer, H. Test scoring. Lawrence Erlbaum Associates, Publishers; 2001.

Werner EE. Resilient offspring of alcoholics: A longitudinal study from birth to age 18. Journal of Studies on Alcohol. 1986; 47(1):34–40. [PubMed: 3959559]

Wolin SJ, Bennett LA, Noonan DL, Teitelbaum MA. Disrupted family rituals: A factor in the intergenerational transmission of alcoholism. Journal of Studies on Alcohol. 1980; 41:199–214. [PubMed: 7374140]

Zucker, RA. Alcohol use and alcohol use disorders: A developmental-biopsychosocial systems formulation covering the life course. In: Cicchetti, D.; Cohen, DJ., editors. Developmental Psychopathology, 2nd Edition, Volume Three: Risk, Disorder and Adaptation. Hoboken, NJ: Wiley & Sons; 2006. p. 620-656.

Zucker, RA.; Fitzgerald, HE.; Refior, SK.; Puttler, LI.; Pallas, DM.; Ellis, DA. The clinical and social ecology of childhood for children of alcoholics: Description of a study and implications for a

differentiated social policy. In: Fitzgerald, HE.; Lester, BM.; Zuckerman, BS., editors. Children of addiction: Research, health and policy issues. New York: Garland Press; 2000. p. 174-222.

Zucker, RA.; Noll, RB.; Fitzgerald, HE. Unpublished questionnaire. East Lansing, MI: Michigan State University; 1988. Drinking and Drug History Questionnaire-Revised Edition (Version 3).

Table 1

Descriptive Statistics for Samples Defined by Father-, Mother-, and Adolescent-Reported Symptomatology

	Father-Report Sample				Mother-Report Sample				Adolescent- Report Sample
Variable	**MLS**	**AFDP**	**Total**	**Study Comparison**	**MLS**	**AFDP**	**Total**	**Study Comparison**	**AFDP**
SAMPLE SIZE	564	374	938	--	583	443	1026	--	454
Child Age (in years)	8.34 (3.54)	13.75 (1.68)	10.67 (3.94)	-31.36^{***}	8.48 (3.52)	13.73 (1.67)	10.90 (3.84)	31.63^{***}	13.73 (1.68)
Child Gender (% male)	76.69	52.89	66.42	7.58^{***}	74.90	53.26	64.94	7.29^{***}	52.82
Parent Education (% with high school education or less)	9.64	5.50	7.85	2.42^{*}	10.07	6.37	8.36	2.18^{*}	7.65
Proximal Effect of Mom's ARS	0.23 (0.54)	0.14 (0.52)	0.19 (0.53)	2.56^{**}	0.26 (0.58)	0.23 (0.77)	0.24 (0.67)	0.68	0.25 (0.77)
Proximal Effect of Dad's ARS	0.72 (1.07)	0.63 (1.22)	0.68 (1.14)	1.16	0.74 (1.08)	0.69 (1.18)	0.72 (1.12)	0.70	0.81 (1.25)
Distal Effects of Mom's Alcohol Diagnosis (%)	32.30	9.57	22.49	9.13^{**}	32.65	13.12	23.66	7.75^{***}	12.70
Distal Effects of Dad's Alcohol Diagnosis (%)	69.71	48.82	60.70	6.47^{**}	70.86	46.66	59.72	7.99^{***}	47.62

NOTE: Reported numbers indicated % for dichotomous variables and means (with standard deviations) for continuous variables. ARS = alcohol-related symptoms. Means and standard deviation for the time-varying covariate were calculated pooling over time. Note that the average proximal and (over time, within person) time-varying effect of parental alcohol-related symptoms are the same. Study comparisons are z-tests for independent proportions and two-sample unpooled t-tests, as appropriate.

*, ** *and* *** indicates that MLS and AFDP comparison is statistical significant at p<.05, p<.01, and p<.001 respectively.

Table 2

Results of Multilevel Analyses Testing Study Hypotheses for Parent-Reports

	Father-Report Models						Mother-Report Models					
	Model 1			Model 2			Model 1			Model 2		
Predictors	**est**	**t-val**	**OR**	**est**	**t-val**	**OR**	**est**	**t-val**	**OR**	**est**	**t-val**	**OR**
Control Variables												
Intercept	**−0.40**	**−4.46**	**0.67**	**−0.40**	**−4.45**	**0.67**	−0.09	−0.99	0.91	−0.09	−0.99	0.91
Linear Age	**−0.07**	**−5.36**	**0.93**	**−0.07**	**−5.27**	**0.93**	**−0.04**	**−9.28**	**0.96**	**−0.04**	**−9.29**	**0.96**
Linear Age Part 2 (L2)	0.01	0.78	1.01	0.01	0.75	1.01						
Study	**0.23**	**3.79**	**1.26**	**0.23**	**3.79**	**1.26**	**−0.22**	**−2.48**	**0.80**	**−0.22**	**−2.49**	**0.80**
L2 x Study	**−0.04**	**−2.35**	**0.96**	**−0.04**	**−2.33**	0.96						
Gender (GEN)	**0.17**	**3.76**	**1.19**	**0.17**	**3.76**	**1.19**	**0.27**	**5.75**	**1.31**	**0.27**	**5.75**	**1.31**
Parent Education	−0.02	−0.83	0.98	−0.02	−0.84	**0.98**	**−0.06**	**−2.75**	**0.94**	**−0.06**	**−2.75**	**0.94**
Time-Varying Effects												
Mom ARS	−0.02	−0.65	0.98	0.00	0.01	1.0	0.02	0.87	1.02	0.02	0.60	1.02
Dad ARS	*0.02*	*1.70*	1.02	0.01	0.35	1.01	**0.03**	**2.34**	**1.03**	0.04	1.54	1.04
Mom ARS x GEN				−0.02	−0.40	0.98				−0.01	−0.15	0.99
Dad ARS x GEN				0.02	0.70	1.02				−0.01	−0.31	0.99
Proximal Effects												
Mom mean ARS	0.02	0.34	1.02	0.02	0.34	1.02	*0.07*	*1.82*	*1.07*	*0.07*	*1.82*	*1.07*
Dad mean ARS	**0.06**	**2.82**	**1.06**	**0.06**	**2.82**	**1.06**	**0.08**	**2.91**	**1.08**	**0.08**	**2.91**	**1.08**
Distal Effects												
Mom alc diagnosis	**0.19**	**2.98**	**1.21**	**0.19**	**2.98**	**1.21**	**0.30**	**4.48**	**1.35**	**0.30**	**4.48**	**1.35**
Dad alc diagnosis	**0.12**	**2.12**	**1.13**	**0.12**	**2.12**	**1.13**	**0.18**	**2.20**	**1.20**	**0.18**	**2.20**	**1.20**
Dad alc diagnosis x Study							**−0.23**	**−2.08**	**0.80**	**−0.23**	**−2.08**	**0.79**

Note: Time-varying predictors are the repeated annual assessments of parents' alcohol-related symptoms (ARS) within the past year, proximal predictors are the average of these repeated annual assessments within each person, and the distal predictors are the wave one lifetime reports of parents' lifetime alcoholism diagnosis (alc diagnosis). OR=Odds Ratio. Bold estimates are significant at p<.05, italicized are significant at p<.10.

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Table 3

Results of Multilevel Analyses Testing Study Hypotheses for Adolescent-Reports

	Adolescent-Report Models					
	Model 1			Model 2		
Predictors	**est**	**t-val**	**OR**	**est**	**t-val**	**OR**
Control Variables						
Intercept	−0.17	−1.53	0.84	−0.17	−1.53	0.84
Linear Age	**0.06**	**4.16**	**1.06**	**0.06**	**4.18**	**1.06**
Gender (GEN)	**0.13**	**2.04**	**1.14**	**0.13**	**2.04**	**1.14**
Parent Education	−0.04	−1.46	0.96	−0.04	−1.46	0.96
Time-Varying Effects						
Mom ARS	0.04	1.34	1.04	0.03	0.57	1.03
Dad ARS	−0.02	−0.85	0.98	−0.02	−0.66	0.98
Mom ARS x GEN				0.03	0.46	1.03
Dad ARS x GEN				0.00	0.00	1.0
Proximal Effects						
Mom mean ARS	**0.13**	**2.67**	**1.14**	**0.13**	**2.67**	**1.14**
Dad mean ARS	−0.03	−1.10	0.97	−0.03	−1.10	0.97
Distal Effects						
Mom alc diagnosis	0.15	1.30	1.16	0.15	1.30	1.16
Dad alc diagnosis	**0.37**	**5.15**	**1.45**	**0.37**	**5.15**	**1.45**

Note: Time-varying predictors are the repeated annual assessments of parents' alcohol-related symptoms (ARS) within the past year, proximal predictors are the average of these repeated annual assessments within each person, and the distal predictors are the wave one lifetime reports of parents' lifetime alcoholism diagnosis (alc diagnosis). OR=Odds Ratio. Bold estimates are significant at p<.05.